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The Lower Limb Tendinopathies

Etiology, Biology and Treatment

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Chapter 1
Tendonitis, Tendinosis, or Tendinopathy?

Gian Nicola Bisciotti and Piero Volpi

Abstract The term tendinopathy does not seem more suited to describe the processes that the tendon undergoes during its rearrangement in the case of its biological and structural distress. In effect, the inflammatory process would seem to be absent, or in any case very limited, from a temporal point of view, while it would seem to prevail the biological degeneration process. For this reason, it would seem preferable to use the term "tendinopathy." In effect, this term would describe much better the profound processes of biological and structural rearrangement that the tendon suffers. However, one cannot ignore the fact that often inflammatory and degenerative processes can coexist.

1.1 Introduction

Historically the term "tendonitis" was used to describe chronic painful symptoms on a tendon with overt algic symptoms, a concept which implied the existence of an inflamed state as a primary disease process. However, in spite of this definition, the fact that normal anti-inflammatory therapies became more evident showed very limited effect on the aforementioned tendonitis [1, 2]; simultaneously to this statement, the results of the first studies of histology appeared in literature, which showed the presence, in such clinical pictures, of a degenerative process which was coexistent with that of inflammation. All of this put seriously into doubt the concept of

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centrality of the same inflammation process of disease on the tendon tissue [3, 4]. Ever since then the term tendonitis was progressively abandoned, to be substituted by a more generic term, that of "tendinopathy."

1.2 The Histological Aspect of the Tendon Affected by Tendinopathy

From a point of view of structural framework, in the healthy tendon, the fibers are laid in a parallel way and are strongly linked to one another. On the other hand, in the injured tendon, the fibers show a clear increase of their wavy aspect and a marked structural separation, thus showing a clear loss of their normal structure. In the tendinopathic tendon – or one which has maintained its continual structure, in spite of the development of disease – we can observe an increase of the wavy aspect of the fiber which is less accentuated in regard to that of the injured one. The nuclei of the tenocytes of the affected tendon by tendinopathy generally appear flat and tapered and, sometimes, distributed in line. In the case of severe tendinopathy, the tenocytes assume an aspect similar to chondrocytes. In the injured tendon, the tenocytes appear smaller and the nuclei are rounded. In some cases, the injured tendons show anarchic vascularization, often linked with the degenerative process; this neovascularization runs parallel to the collagen fibers. We may also observe an increase in glycosaminoglycans (GAG) which could influence the structure of the fibro and their organization, inducing a reparative response which can contemplate even a neovascularization process [5]. Histologically the changes of degenerative character are classified as:

(i) Hypoxic
(ii) Hyaline
(iii) Mucoid and myxoid

Furthermore, to this situation is often associated – above all in some specific tendon areas, for example, the rotator cuff – a lipid degeneration.

1.3 The Coexistence of Degenerative and Inflammatory Changes

Many authors agree on the fact that phenomena such as inflammation and degeneration may rarely be shown in an isolated way and which instead, more often, coexist in adjacent areas of the observed anatomic sample [2, 6–8]. Generally, in fact, macroscopic changes, at an intra-tendon level, in the case of tendinopathy, may be described as the formation of a scarcely marked area inside which we can identify a focal loss of the tendon structure. The tendon portion affected by tendinopathy loses its translucid aspect, and it appears grayish and amorphous. The tendon appears

much thicker, in a fusiform and nodular way, and inside we may sometimes observe calcification, fiber calcification, and bone metaplasia. The different portions of the degenerated tendon area show an ample variety regarding cellular density; in fact in some areas, we may observe an increase of the contextual cellular density and a high rate of metabolic activity. On the other hand, we may observe only a few pyknotic[1] cells in certain areas, or we may compare their total absence. Some changes in disease are often observed even in the tendon matrix, where we can frequently observe contextual mucoid material by a separation of the collagen fibers. The collagen fibers usually show irregularity, a difference and an increase in their crimping, as well as a loss of visibility in the transversal band. The degenerated fibers may be replaced by calcification areas or by infiltrated lipids, which give origin to the tendolipomatosis phenomena. A clear increase may be noted in the type III collagen which is poor, with respect to type I collagen, concerning the number of cross-links between and inside the tropocollagen units [9]. In spite of the evidence of such degenerative alterations, in the tendon tissue affected by tendinopathy, their clinical relevance is still not clear. Degenerative hypoxia, mucoid degeneration, calcification, and tendolipomatosis all represent phenomena which, either singularly or together, are visible in a high percentage of tendons in healthy and asymptomatic individuals at the age of 35 years and over [5, 10].

In the case of tendinopathy, we may observe frequent changes in the peritendinous structure, which appear more often in tendons showing a synovial sheath – i.e., posterior tibia, peroneal, and flexors and extensors of the wrist and of the fingers [11, 12]. In the acute phase of tendinopathy, in the histological examination, we may frequently see the presence of fibrinous exudates, followed by a second phase characterized by a diffused proliferation of fibroblasts. Following a macroscopic observation, the peritendinous tissue appears thick, and phenomena of adhesion are often visible between the tendon and paratenon [2]. During a chronic phase, the main cells of the paratenon are fibroblasts and myofibroblasts. Regarding the myofibroblasts, it is interesting to note that during the remodeling process, the fibroblasts assume their own characteristics, both from a morphological and biochemical point of view of the contractile cells. For this reason they are defined as myofibroblasts. The myofibroblasts possess a modest amount of actin inside their cytoplasm and thus have a certain contractile capacity. Due to these characteristics, the myofibroblasts may induce and maintain, in time, a lengthened state of contraction in a frame of peritendon adhesion, causing, at the same time, a state of vascular constriction perturbing intra-tendon circulation, which then probably starts up a reactive process of vascular proliferation. For some authors, peritendinitis is a process of inflammatory nature [2].

In an animal model – specifically the rabbit – damage provoked to the tendon tissue causes an infiltration of inflammatory cells which becomes evident at a distance of 6 h; on the other hand, when damage to the tendon tissue is provoked by

[1] *Pyknosis: in cytology, the contraction of the cell nucleus (pyknotic core) or of all the protoplasm, which looks like a mass intensely colored without regular pattern. It is generally a degeneration sign.*

overuse, we may merely observe histological changes of degenerative type [13, 14]. Still in an animal model, this time equine, the superficial flexor tendons of the digits undergoing overload show a precocious phase of inflammatory type, followed by a phase of degenerative type [15, 16]. These experiments, even though out of date, induce us to consider the hypothesis that the inflammatory process may represent the precocious phase of the tendinopathic process, to which follows a second phase of degenerative type, even if we have to admit that the relationship between the two phenomena is to date still not clear. Further studies, still on an animal model, show the existence of damage of oxidative type and an increase of the apoptotic phenomena when tendon structure is put under chronic stress [17]. Studies executed on rabbits have proven that mastocytes adjacent to the neural structure release neuropeptides – in particular neuropeptide substances P (SP) and calcitonin gene-related peptide (CGRP) – as well as specific mediators (mast cell mediator) such as histamine, prostaglandin, and leukotrienes able to influence both the activity of the fibroblasts and the vascular permeability [18].

Studies carried out on man are obviously more pertinent than those on animals; however, we must consider the fact that almost all of the studies carried out on man are performed on symptomatic tendons representing undoubted limitation. In such a way, it is rarely possible to observe the first phase, asymptomatic, of tendinopathy. Therefore, the main studies carried out on man may be interpreted by considering such limitation of the experimental model. In studies done by Alfredson [4], the authors refer how the level of prostaglandins E2 (PGE2) is similar in the case of chronic tendinopathy as in the case of absolute biological and histological normality, excluding the presence of an inflammatory process in later phases of the tendinopathic process. Yang and colleagues are not of the same opinion [19], who, on the other hand, observe how the mechanical stress on the patellar tendon induces and increases the production of PGE2 on behalf of the fibroblasts. The PGE2, as well as representing a strong inhibitor of the synthesis of collagen type I [20–22], shows a marked catabolic effect to the damage of the tendon structure where inhibition of the collagen production is provoked [23]. Other experiments show us how the levels of lactate grow significantly in the diseased tendon, witnessing the fact that the majority of anaerobic mechanisms of the tendon affected by tendinopathy represents the answer to the insufficiency of oxygen to the tissue [24, 25]. This hypothesis is confirmed by the fact that hypoxia observed in the suffering tendons of degenerative phenomena induces the production of hypoxia-inducible factor which, in turn, provokes the expression of vascular endothelial growth factor (VEGF) [26, 27]. Apart from its angiogenic properties, the VEGF is able to provoke an upregulation of the expression of matrix metalloproteinase (MMP) which, in turn, increases the degradation of extracellular matrix (ECM) altering the mechanical properties of the tendon [28, 29, 30]. The neoangiogenesis is followed by a proliferation of the nervous terminals and by the production of algogenic substances which, together with a high level of glutamate, typically found in tendinopathy, are the responsible factors of the outbreak of algic symptoms [2]. All these mechanisms may lead to a situation of repeated microtraumas which may show in a tendon breakage.

1.4 Study Models of Tendinopathy

Studies of tendinopathy disease carried out on man, even though being, without doubt, of the most interest, meet difficult objectives. Human tissue obtained surgically, or by biopsy, generally come from subjects who have developed advanced disease, without taking into account difficult objective in finding healthy tendon samples to use as a control model. For this reason, to the end of deepening the knowledge of mechanisms which determine the outbreak of tendinopathy, model tendons are used which may be in vitro, ex vivo, or in vivo.

1.4.1 In Vitro Models

In literature we find some interesting studies, carried out in vitro, based on chronic reminders in the lengthening of the tendon. The chronic stretching of the tendon structures is, in fact, considered as one of the main risks of the development of tendinopathy. In studies in vitro of this type, in general, they undergo a stretching cycle of the tenocytes or of the fibroblasts, and the mechanic strain has the aim of observing the effects of cellular deformation of potential cellular mediators and molecules [31]. Some of the studies [32–34] have highlighted how the deformation of tenocytes and fibroblasts have determined an increase in the production of PGE2, cyclooxygenase-1 (COX-1), cyclooxygenase-2 (COX-2), cytosolic phospholipase A1 (cPLA1), secretory phospholipase A2,[2] and leukotrienes B4. Some authors [19] have underlined the importance of the role of interleukin-1β (IL-1β) in the inflammatory process in a human model in vitro made up of fibroblasts of the patellar tendon. The authors noticed that, when IL-1β was present, a lengthening cycle of the tendon fibers equal to 4 % was sufficient to cause a decrease in the expression of COX-2, of the MMP-1, and of the production of PGE2; on the contrary, a lengthening cycle equal to 8 % of the length of the fibers showed an increase of the expression MMP-1. So, the conclusions of the study were that moderate stretching showed an anti-inflammatory effect, whereas more accentuated stretching ended in a pro-inflammatory action. The advantages of the experiments in vitro in the studies of tendinopathy present the allowance of observation in a simple and quick way of a good number of cellular processes, for example, the synthesis of DNA, mitosis, the genic expression, and the cellular differentiation [32, 34–42]. As well as this undoubted advantage, during the experiments in vitro, cells may undergo lengthening in a similar way to that of what happens in an in vivo situation. This latter aspect carries particular importance, as a change in the mechanism of lengthening may involve a different type of cellular response [43]. However, we must note that during such type of experiment, we may see that the inconvenience that the lengthening

[2] *The phospholipase A2 are enzymes that have the task to separate the fatty acids from the oxidized phospholipids.*

imposed on the substratus is not completely transferred to the cell, becoming imprecise both in length of time and frequency [44]. In any case, the main limitation of in vitro studies is represented by the fact that the study of isolated cells in an environment avoided of cellular matrix (ECM) and neurovascular connections, limits the interpretation of the obtained results when we want to refer these latter in a situation of natural activation.

1.4.2 Ex Vivo Models

Ex vivo models provide, in tendinopathy, the possibility to observe and study the changes and answers to different stimuli by the entire tendon tissue and not only from a few isolated cells, as happens in in vitro models. These models allow interaction between the tendon cells and the ECM, which, remaining intact, is able to provide a biological environment so as to allow efficient control of the experimental conditions. The main methods of study applied in ex vivo models are cyclical loading, creep loading, and stress deprivation.

(i) *The cyclical loading*
Exactly as what happens in in vivo models, the cyclical loading (or cyclical stretching) in ex vivo models is carried out in order to observe the effects of a repeated chronical load on the tendon tissue which shows intact ECM. After about 24 h of cyclical loading, the tissue shows a decrease in value of the mechanic failure (ultimate failure strength), becoming significantly weaker from a structural point of view [45]. In such conditions, we also assist in an increase of cellular turnover and of the quantity of collagen and unmistakable signs of tissue degradation [46]. Devkota and colleagues [46] also noted how, when the tendon sample was put through cyclical loading, it was linked to the production of PGE2. Also the levels of MMP-1 would seem linked to the length and the frequency of the imposed load; low loading and low frequency would cause an inhibition of the expression MMP-1, whereas the application of high loads at high frequency would entail the complete inhibition of the expression [47]. Some authors recommend using, during cyclical loading, experiments done ex vivo on the tendon, tension superior to 5 % of the maximum supporting tension; it seems that for tension inferior to this level, we are not able to produce appreciable structural deformation [48, 49]. The ex vivo method doesn't entail the same complexity as the in vivo method. In fact, as in the in vitro method, during ex vivo experiments, the damage provoked by cyclical loading is not completely repaired; complete repair cannot come about both because of the absence of vascularization and the deficiency of molecules of the signaling systemic.

(ii) *Creep loading*
As well as a repetitive chronic load, a situation also reproduced during cyclical loading experiments, the tendon tissue may undergo structural damage also through the application of sustained and prolonged loading. This type of situation, during experiments in ex vivo, is reproduced by using the creep loading

method. In this environment, Wren and colleagues [49] studied the different intercurrents between the effects caused by cyclical loading and creep loading on the human Achilles tendon, observing in both cases a significant structural damage. So, both cyclical loading and creep loading represent two valid ways of investigation in the study of tendinopathy.

(iii) *Stress deprivation*
Studies on stress deprivation resolve the aim of observing changes induced by immobilization in the sample in question. In studies of this type – ex vivo, in vivo, and in vitro – the immobilization causes an increase in the expression MMP-1 mRNA, a factor which involves an expiry of the tensile characteristics of the tendon [47, 50, 51]. For such a reason, the experiments of stress deprivation represent a valid study model of tendinopathy. However, in this type of experiment, the tensile properties of the tendon are altered without which a simultaneous change in the diameter of the collagen fibers occurs. For this reason, experiments of stress deprivation are not adapt to the study of the role of collagen in the context of tendinopathy. A complete understanding of the processes which regulate the outbreak and the evolution of tendinopathy – even when referring to experiments in vitro and ex vivo, which represent an important model of understanding regarding a specific cellular answer – it cannot be exempt from the use of experiments in vivo. To this aim, numerous experimental models have been conceived and developed so as to induce specific answers, at a tendon level, before the application of interfering experiments of chemical and mechanic nature. The necessity of having more than one model available is dictated by the fact that each experimental model is able to represent only one aspect of disease in tendinopathy. A further important aspect to remember is represented by the fact that we are obliged to choose models coming from different animal species as well as knowing the gene sequence, because no animal model exists, which possesses tendons, with the same characteristics as that of humans [52]. There are advantages and disadvantages connected with the different methods of induction of tendinopathy based on chemical and mechanical induction. In the first case, for example, we receive an answer from the part of the tendon tissue of acute type, which does not represent changes induced from a chronic tendinopathy in the human tendon; on the other hand, however, chemical induction shows the advantage of being less laborious in comparison with a mechanical one and is able to induce consistent tendon damage.

1.4.3 In Vivo Chemical Induction Models

The in vivo chemical induction models (in vivo CIM) can be divided into four main categories. Depending on the different techniques used, we can in fact have:

(i) In vivo CIM injecting collagenase

(ii) In vivo CIM injecting cytokines
(iii) In vivo CIM injecting prostaglandins
(iv) In vivo CIM injecting fluoroquinolones

(i) In vivo CIM injecting collagenase
One of the chemical induction models of tendinopathy consists in injecting collagenase into the tendon undergoing experimentation. One of the first studies that used the injecting of collagenase in the tendon in order to induce a tendinopathic process was done by Foland and colleagues [53]. The animal model chosen was the horse, and the injecting of collagenase was performed in three different areas of the flexor digitorum superficialis tendon. The tendon reaction was the development of an injury in the injected area, with a consequent repair process implying the proliferation of collagen type III. Other similar experiments used the injecting of collagenase in the supraspinatus tendon in a mouse model [54], which induces a strong cellular increase, the destruction of the collagen organization, and the increase of vascular tissue. The effects of the injecting of collagenase were completely resolved and repaired only after a period of about 12 weeks [54] even though some authors have recently observed the persistence of the phenomena for a longer time, around 32 weeks [55]. In all these experiments, we may observe a substantial loss of an initial inflammatory process. However, other studies show that inflammation became evident after 15 days from the injecting of collagenase, and at a distance of 16 weeks, it was able to see a chondral metaplasia with ossification [56]. Even though through this type of experiment we may obtain many similar characteristics to those of a human tendon affected by tendinopathy – such as hypercellularity, loss of organization of the matrix, an increase in vascularization, and absence of an inflammatory process – we cannot ignore the fact that the injecting of bacterial collagenase in an animal model may present substantial differences from that of human collagenase and hence inducing likewise substantially different reactions [57, 58].

(ii) In vivo CIM injecting cytokines
Since tendinopathy may present, even in its precocious phase, a process of inflammatory type, followed by a second phase of degenerative type, the injecting of cytokines represents an experimental model whose aim is that of inducing an inflammatory reaction. Following this species-specific injecting of cytokines on a patellar tendon in a rabbit, Stone and colleagues [58] observed after 4 weeks an increase in cellularity which drifted toward normality only after about 16 weeks. However, the injecting of cytokines was able to provoke only slight and reversible structural damage, without damage to the matrix or the degradation of collagen. So, the injecting of cytokines would seem not to represent a trustworthy model in order to reproduce a tendinopathic process. In any case, since the injecting of cytokines is species specific, this method has the advantage of normalizing intraspecies differences.

(iii) In vivo CIM injecting prostaglandins
Since studies in vitro [31, 33] and in vivo [59] have shown that repetitive mechanical stress induces the production of prostaglandin, on behalf of fibroblasts in the

human tendon, the injecting of these may represent a valid study model of tendinopathies. In the first week of weekly administration of PGE1 on the Achilles tendon in mice, Sullo and colleagues [60] met a growing content of contextual water to an inflammatory process. In the third week, half of the treated tendons showed signs of fibrosis on the paratenon associated with adhesions and degeneration, whereas the other half showed evident signs of inflammation. In the fifth week, all the treated samples showed signs of fibrosis on the paratenon with adhesions and degeneration. The authors concluded that the repetitive injecting of PGE1 caused, at the beginning, an inflammatory process which then tended toward a degenerative state. Also, the repetitive injecting of PGE1 in the patellar tendon of the rabbit [61] caused an increase in cellularity, disorganization and degeneration of the collagen matrix, and a decrease in the diameter of the collagen fibrils. Furthermore, the processes of tendon degradation are directly linked to the dose of injected prostaglandin [61]. The injecting of prostaglandin on an animal model causes a sequence of phenomena similar to those in a human tendon, affected by tendinopathy which are hypercellularity, deformation and degeneration of the tendon structure, and dilution of the collagen fibrils. So, chemical induction models based on the injecting of prostaglandin show a valid model for study on tendinopathy in an animal model, even if further studies are necessary in order to obtain full comprehension of action mechanisms of the same prostaglandins.

(iv) In vivo CIM injecting fluoroquinolones

As is well known, the use of fluoroquinolones induces the onset of severe tendinopathy which may involve the complete breakage of the tendon [62–65]. Single administration of 300 or of 900 mg/kg of pefloxacin [7] (PFLX), or 900 mg/kg of ofloxacin (OFLX) in mice, causes an infiltration of pro-inflammatory cells of peritenon and of the Achilles tendon, contextual to a disorganization of collagen strips [66]. Following the administration of PFLX or OFLX, the fibroblasts presented a fragmentation of nuclei and showed signs of dead cells; such a picture tended to become less severe in the case of OFLX but not in the case of PFLX [64–66]. Some authors carried out analyses regarding the possible effects of different quinolones, observing a certain variability on the different tested products [67]. In each case, at least in mice, the more toxic fluoroquinolones, and thus causing more serious injuries on the Achilles tendon, would seem to be fleroxacin (FLX) and PFLX. Other fluoroquinolones show minor toxicity and cause tendon injuries only for doses between 300 and 900 mg/kg, whereas norfloxacin, ciprofloxacin, and tosufloxacin do not show adverse effects on tendon structure up until doses equal to 900 mg/kg [44]. Further studies show that the injuring mechanism on a tendon activated by the assumption of PFLX and FLX is represented by the inhibition of the synthesis of proteoglycans and by the induction of oxidative damage of collagen [67]. We may, therefore, conclude that, in mice, the administration of fluoroquinolones, in particular PFLX and FLX, induces the onset of tendinopathy with characteristics (injuries and edema) similar to those in the human tendinopathic tendon [68]. However, the injecting of fluoroquinolones in mice presents

several important differences in comparison to that of the human. The major difference is made up of the fact that, in mice, the injecting of fluoroquinolones does not involve breakage of the tendon, as it does in man [65]. Furthermore, in man, even a low dose seems to involve much more serious effects on the structure of the tendon in comparison to that on mice. In the light of this data, we may affirm that the assumption of fluoroquinolones stimulates several specific reactions which induce the onset of severe tendinopathy in man (not showing in mice); this renders the mouse model less interesting in reproducing the effects of tendinopathy induced by the use of such pharmaceuticals.

1.4.4 In Vivo Mechanical Induction Models of Tendinopathy

The etiology of the overuse of tendinopathy is, in literature, largely shared [69–72]; for this reason an ample use of mechanical models of induction of tendinopathy has become widespread. Since the mechanical models are able to induce chronic tendinopathy, mainly taking advantage of an overuse mechanism, and not of acute tendinopathy as in chemical induction models, it appears clear that the application of a mechanical model is longer and more laborious. In literature, we find five categories of in vivo mechanical induction models of tendinopathy, of which we will briefly illustrate the main characteristics.

1.4.5 Mechanical Induction Models of Tendinopathy Based on Electrical Stimulus

Electrical exogenous stimulus of a muscle provokes a contraction which produces a flexo-extension movement which mechanically loads the relative tendon. Backmand and colleagues [13], by using electrostimulation (ES), managed to reproduce, on an animal model, tendinopathic processes with similar characteristics observed in man. The ES of the triceps muscle of a rabbit resulted in the tendon degeneration with neo-capillarization and the presence of pro-inflammatory cells. Also, Nakama and colleagues [73] and Asundi and colleagues [74] obtained similar results by electrostimulating the deep flexor of the digits of a rabbit. The ES thus represents a mechanical induction model of tendinopathy with satisfying reproducibility and relatively contained experimental timing. However, it is important to remember that some authors noted that ES may have different effects in different areas of the tendon undergoing experimentation [73, 74]. Furthermore, in some cases [75], an outbreak of tendinopathy was also seen in the contralateral tendon not undergoing ES, which could doubt the control conditions of the experimental protocol based on ES.

1.4.6 Mechanical Induction Model of Tendinopathy Based on Downhill Treadmill Running

Several experiments carried out on mice have proven to be like a downhill run – carried out with a 10 % incline, at the speed of 17 km/h, with the frequency of 1 h a day, 5 days a week, and for a variable number of weeks – done on a treadmill (downhill treadmill running, DTR) and induce severe structural changes on the tendon of the supraspinatus [76, 77]. The changes observed on the tendon included an increase in cellularity, a cell deformation, a nonalignment of the collagen fibers, and an increase of the cross-sectional area of the tendon: such structural alterations were evident for a period between 4 and 16 weeks. As well as these structural modifications, an increase of the genic expression of COX-2 was clear after 8 weeks, followed by its normality after about 16 weeks, whereas the VEGF values increase for the whole 16 weeks. This data proves the evidence both of the onset of inflammatory phenomena and of neoangiogenesis. Other experiments done with the DTR method evidenced an increase of the expression interleukin-18 (IL-18), interleukin-15 (IL-15), and interleukin-6 (IL-6) [78] and an increase in the expression of mediators in the process of apoptosis and of the levels of heat shock proteins (HSP).[3] In general, the DTR method may be considered a valid means of mechanical induction of tendinopathy. From a practical point of view, the method presents disadvantages connected to a long experimental period, necessary to induce the disease changes on the tendon, and undoubted difficulty in the training of guinea pigs. Furthermore, the DTR on mice would seem to affect only the supraspinatus tendon and not the Achilles one [79].

1.4.7 The Mechanical Induction Model of Tendinopathy Based on Uphill Treadmill Running

Since experiments carried out using the DTR method have not been able to show, in mice, tendinopathic induction on the Achilles tendon, some authors [80] introduced an experimental method based on uphill treadmill running (UTR). By using the UTR method, Glazebrook [80] obtained, on the Achilles tendon of mice, a disorganization of collagen fibers, contextual to a phenomena of neovascularity, and an increase in fibroblasts. So, the UTR method seems more adapt, in comparison with the DTR method, in inducing tendinopathy of the Achilles tendon in mice. The reason of this further efficiency in inducing tendinopathy of the Achilles tendon of the UTR method may be explained by the fact that uphill treadmill running requires a further eccentric activity of the complex muscle tendon involved [81], showing further tendon damage [82–84].

[3] *Heat shock proteins (HSP) are a class of functionally linked proteins, involved in folding and unfolding of other proteins.*

1.4.8 The Mechanical Induction Model of Tendinopathy Based on Fatigue

One of the most common etiological causes of tendinopathy is represented by a chronic overloading on the tendon able to alter the genic answer associated with the outbreak of the same tendinopathy. In other words, the fatigue phenomena may be in such conditions the *primum movens* of the outbreak in the tendinopathic process. Sun and colleagues [85], by inducing the fatigue phenomena through the application of cyclical loading on the patellar tendon of mice, of a magnitude between 1 and 35 N, with 1Hz frequency, managed to induce a series of microstructural damage together with an upregulation of MMP-13 and IL-1β. Using the same method, Fung and colleagues [86] showed the linking existence between the level of fatigue induced and the structural damage caused on the matrix and collagen fibers, as well as highlighting an increase in the expression of collagen of types III and V. The mechanical model based on the induction of fatigue, thus, was able to induce reproducible and controllable structural damage on the tendon of mice, showing, however, the limit of being able to induce such damage in an acute way or through an application of a single load, even if the application of the latter is reiterated in time, and not through chronic and intermittent mechanical stress, as is typical in man.

1.4.9 The Mechanical Induction Model of Tendinopathy Based on Disuse

Even though its effects are still not fully understood, several studies indicate how inactivity may induce tendinopathy [87, 88]; for this reason we may find, in literature, mechanical induction models of tendinopathy based on disuse. Nagawa and colleagues [89], by applying the disuse model, through suspension, on mice, observed a decrease in this surface of collagen fibers. However, the results of the experiment were nullified by the fact that the period of suspension induced a slackening of growing processes in animals which could have induced a successive slackening in the growth of collagen fibers. Since, in man, not all tendinopathies are caused by an overuse mechanism, the full comprehension of the effects of disuse of the onset of the tendinopathic phenomena could reveal great importance; for this reason, further and deeper studies are necessary in this environment.

1.5 The Iceberg Theory

In order to perfect our actual incomplete understanding of the intercurrent relationship between inflammatory phenomena and those which are more degenerative, the "Iceberg Theory" (IT) proposed by Abate and colleagues [2] seems to us of

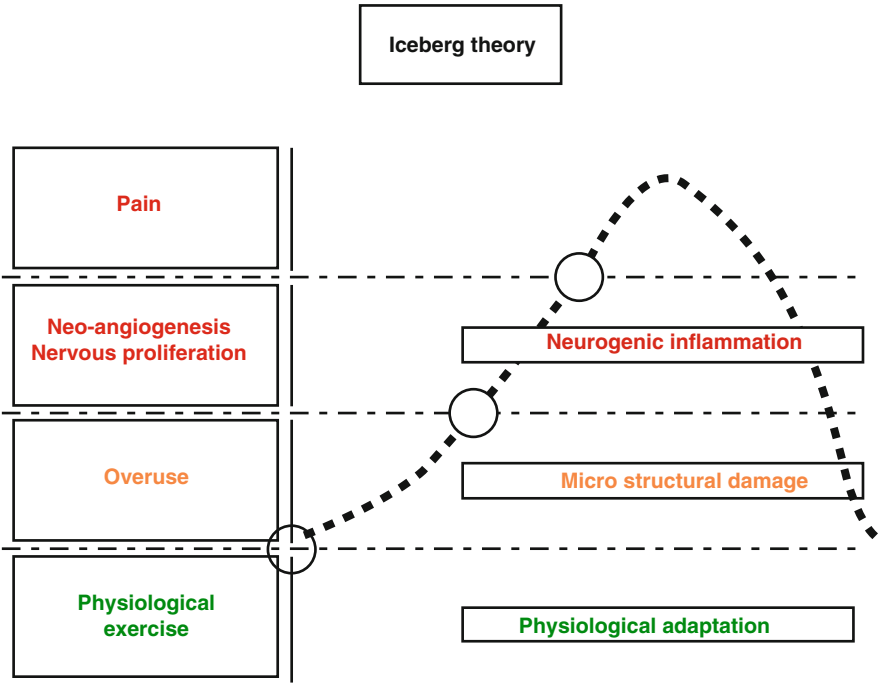

Fig. 1.1 The iceberg theory proposed by [2] allegorically illustrates the pathogenic cascade that marks the onset of tendinopathy through the levels of a hypothetical iceberg, whose base is formed by the condition of physiological normality and the tip represents the onset of the painful symptoms (From Abate et al. [2] modified)

particular interest. According to IT (Fig. 1.1), the sequence of events that follow, and which in many cases overlap, in tendinopathy may be compared to an iceberg where it is possible to see numerous levels. The base of the iceberg represents all which happens on a tendon in physiological conditions. In the initial stage of tendinopathy, we may recognize two phases: the asymptomatic phase and the symptomatic phase. This division implies that pain is the alarm symptom which marks the pathway from the first to the second phase. The first consideration is represented by the fact that it is extremely improbable that during the first phase – or shall we say non-algic – the subject undergoes an imaging examination, so the extreme base of the iceberg remains unexplored. Physical exercise, if well done and up to a certain limit, contributes in a substantial way to the increase of the mechanical resistance of the tendon, but after a certain point, we may consider likewise a true "breakpoint," and we may activate a microtraumatic mechanism which, once woken up, may lead to two different possible pictures:

1. An adequate regenerative pause may be given to the microtraumatized tendon – not necessarily by drastically diminishing physical activity but also, much more simply, by using correct workloads – such as to allow regenerative processes to prevail over the traumatic ones. In such a way, the tendon tissue maintains a functional balance.

2. An adequate regenerative pause may not be given to the microtraumatized tendon, a situation which involves a clear prevalence of tissue destruction phenomena on the regenerative ones; the tendon structure is launched toward the development of an overt tendinopathy.
   In the light of the first level iceberg exploration, we are able to draw two considerations:

   (i) The definition of tendinopathy may identify itself in a breakage of balance – existing when the tendon undergoes a physiological workload – between the auto-reparative processes and the microinjury ones.

   (ii) The borderline which marks the trespassing between physiological workload, which allows the homeostasis of the condition of the tendon, and a nonphysiological workload, responsible for the breakage of this delicate balance, is extremely unstable.

Once this existing balance has been broken between regenerative and destructive processes, and thus deviated in tendinopathy, we assist in the production of pathogenic cascade in pro-inflammatory cytokines, pro-angiogenic factors, and free radicals, which cause progressive degeneration of the tendon structure, with a possibly associated development of neural proliferation, which is responsible for the outbreak of algic symptoms.

1.6 Conclusions

The final consideration we have to make – both thanks to the amount of data of the various studies in literature and a correct and objective interpretation of the IT – is that the inflammatory and degenerative processes are not necessarily excluded but, on the contrary, may find a mutual collocation in the pathogenic cascade which distinguishes tendinopathy. So, the term tendinopathy appears more appropriate, in comparison to the definitions tendonitis and tendinosis, and able to describe the complex biological and structural rearranging, which the tendon undergoes in the case of functional sufferance.

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Chapter 2
Healing Processes of the Tendon

Gian Nicola Bisciotti and Piero Volpi

Abstract The biological principles on which the healing process of the tendon is based are quite different from the biological principles that regulate the muscle healing process, although some aspects may be considered as similar. Especially the last stage, namely, the remodeling and maturation phases, is very different especially regarding the temporal length that in the tendon, in respect to the muscle, is much greater. However the healing process between the tendon and the muscle will not only differ in the length time. In effect, the extrinsic and intrinsic healing mechanisms are a peculiar feature of the tendon healing that have no similarity with what occurs in the muscle during its healing process. Therefore it is of fundamental importance, especially after tendon surgical treatment, to know the biological principles that guide the healing process of the tendon.

2.1 Introduction

For a better understanding of the biological principles on which the healing process of the tendon is based, we are going to make a brief yet detailed review, in this chapter, on the various healing phases of the tendon tissue after undergoing injury or surgery. The full comprehension in a biological sense of these different phases makes up the absolutely necessary introduction to be able to fully understand etiology and the development of tissue disorder on the tendon tissue and the pathways

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which may compromise the complete anatomical and functional recovery. Such theoretical base is, and thus the prerequisite, to be able to put into practice efficient strategies whose aim is the streamlining of the natural healing processes of healing the tendon. The healing process of the tendon tissue, on the other hand, as all other soft tissues, may essentially base itself on three biological principles or the regeneration, the repair, or a combination of both. Regeneration represents a form of biological healing which comes about through the production of new tissue whose structural and fundamental characteristics are identical to those of the primitive tissue [1]. Tissue regeneration would therefore represent, in theory, the ideal healing process for the injured soft tissues, exactly as in the case of the skeletal muscle [3]; the healing of the tendon tissue comes about thanks to a repair process which shows itself in the formation of a more or less conspicuous scarring area, which once again, exactly as in the case of the skeletal muscle, presents itself of connective nature and with structural and functional properties inferior to those of original tissue [4]. The tendon tissue, in comparison with the muscular one, presents reduced self-repairing capacity given by the scarce vascularity which, in its turn, involves a reduced amount of oxygen and nutriment of tissue, even though some authors sustain that the self-repairing processes of the tendon are, however, underestimated [5–7].

From a general point of view, the process of tendon repair is divided into the same phases in which the repair process of the skeletal muscle is structured, or into three consecutive phases but, at the same time, extremely interconnected, which are:

- The inflammatory phase, which begins in immediate injury, which is prolonged until about the fourth to the seventh day
- The proliferative phase, which starts from the end of the first post-injury week until the fourth–sixth week
- The maturing or remodeling phase, which from the end of the proliferative phase may prolong itself for up to 1 year from the outbreak of injury

As in the case of muscle tissue repair, the pathway from one phase to another is extremely soft, creating pictures of biological coexistence between the two considered phases. Let us consider now the different biological stages before briefly describing them in their principle details.

2.2 The Inflammatory Phase

The inflammatory phase, also called the exudative phase, begins in the immediate post-trauma period as a physiological answer of structural damage. Following the damage in the vascular network, blood, plasma, and tissue fluids are spilt inside the injured area. The platelets present in the injured area link with collagen exposed by trauma and release phospholipids which stimulate the mechanisms of coagulation [8]. About an hour after trauma in the injured area, we may observe fibrin and fibronectin which form cross-links with the injured collagen fibers [9, 10]. Already, these

first stages bring about the formation of a tenuous glue-like structure which acts as a real "cork," even though still structurally fragile, which in any case banks the local hemorrhage and mechanically supports the damaged tendon fibers when supporting the tensile strengths, which they undergo during this first post-injury phase. In a few hours, a large migration of leukocytes of the injured area comes about, polymorphonucleates and monocytes. Such cellular infiltration happens before 24 h from the trauma and carries on for the next 2–3 days. The inflammatory phase represents a relatively short time, about 1 week [8, 11, 12] The cellular elements, the polymorphonucleates leukocytes, the monocytes, and the macrophages migrate inside the injured area, attracted by particular substances produced inside the injured area, named chemotactic agents. Among these substances, histamine stands out, a substance which is released by mastocytes, granular leukocytes, and platelets. Histamine performs a vasodilator action, increasing the vascular permeability. Still among the chemotactic substances, fibronectin also stands out, which performs a chemotactic action on the leukocytes and the macrophages, and bradykinin, which, as well as increasing vascular permeability, stimulates the releasing of prostaglandin during the inflammatory stage. This latter one is influenced by prostaglandins, in particular prostaglandin E (PGE) which increases vascular permeability and the prostaglandin E2 (PGE2) which has the capacity to attract leukocytes. Immediately after injury we may also observe a rapid increase of DNA inside tendon cells, which then stabilizes itself in the following phases of proliferation, of remodeling, and of maturation [13–15]. In the later period of the inflammatory phase, the PGE and the PGE2 may start up a precocious process of repairing, continuing, and, at the same time, the inflammatory reaction, providing a first example of how the various phases are often overlapping. One of the main tasks of the pro-inflammatory cells is to remove necrotic and refuse produce from the injured area; only after these have been removed, or after 5–7 days from the injury, the proliferative phase may fully begin. As we have already mentioned, a clear subdivision between the inflammatory phase, the proliferative phase, and the last phase of maturation does not exist; however, we may observe rather a continuum of biological activity, which presents overlapping aspects [8]. Several studies have suggested how the different characteristics, which the inflammatory phase may assume, are crucial for success, or, on the contrary, for the failing of the healing processes of the tendon [16]. For example, on an animal model, neutropenia accelerates the healing process from cut injuries [16] but does not influence the healing processes of a tendon which has been surgically repaired [17]. The depletion of the macrophages compromises the healing processes of the skin, causing both diminution of the processes of collagen deposition and angiogenesis [18, 19]. However, this discussion in literature data is contrasting; in fact, on the one hand, several authors indicate that the activation of macrophages may represent new and interesting therapeutic approaches on tissue repair, for example, regarding damage on cardiac tissue in the case of ischemia [20]; on the other hand, other studies indicate that the depletion of the macrophages implies a substantial improvement both of the morphology and of the mechanical properties of the interfacial tendon bone after surgical reconstruction of LCA [21]. This incomplete comprehension of the role of neutrophils and macrophages in the process of tissue reparation

may be, at least in part, justified by the fact that during the inflammatory phase, a molecule may adhere to various function and, at the same time, different molecules may perform substantially overlapping roles [22]. Taking this into consideration, it is opportune to remember that the different grades of the severity of the injury may determine different states of activation of the macrophages [23, 24] or:

(i) A first type of activation named "innate," triggered by lipopolysaccharides or by interferon-γ (IFN-γ)-inducing factor (IGIF) associated with the pro-inflammatory state and the production of interleukin 6 (IL-6), interleukin 1-β (IL-1β), and tumor necrosis factor-α (TNF-α)
(ii) A second mode of inflammation called "classic" activated by the action of IL-4 and IL-23 associated with the action of TGF-β, TGF-α, basic fibroblastic growth factor (b-FGF), PDGF, and VEGF.

In fact, several studies, even though preliminary, indicate a high level of complexity in the activation of macrophages, a level which depends on the nature of interaction and from the combination of biological stimulus to which the same macrophages are exposed [25]. The resolution of the inflammatory processes is, in the end, regulated by the fibroblast activity, which contributes in a substantial way, allowing the infiltrated leukocytes both to move toward apoptosis processes and to leave the tissue through the lymphatic circle [26]. Furthermore, several studies which report the observation of an increase in mastocytes during the inflammatory phase in patients affected by chronic tendinopathy with evident vascular hyperplasia and who complained about excel algic symptoms must be noticed [27, 28]. Since mastocytes contain numerous granules rich in heparin, histamine, and tryptase,[1] the release of these last two substances on behalf of the mastocytes during the degranulation phase participate in the release of substance B, which is responsible for the algic symptoms [27–29].

2.3 The Proliferative Phase

The proliferative phase begins with an accumulation of fibroblasts, myofibroblasts,[2] and endothelial cells inside the injury area [30–33]. The processes of migration and proliferation of these cells are promoted by the presence of growth factors produced both by the platelets and by macrophages [8]. Proliferation of new capillaries commences in this stage, which begin to functionally communicate with the

[1] *Tryptase is a proteolytic enzyme present in mast cell granules.*

[2] *The myofibroblasts are connective tissue cells with contractile capabilities similar to the smooth muscle. Discovered in 1970, at these cells, an important role is recognized in the process of wound healing, tissue fibrosis, and pathological fascia contractures. Their evolution generally occurs from normal fibroblasts to proto-myofibroblasts, until the complete differentiation into myofibroblasts and to end to a terminal apoptosis that is influenced by mechanical tension, cytokines, and specific proteins from the extracellular matrix.*

pre-existing capillary network. During the proliferative phase, the fibroblasts and the myofibroblasts, which may come from the same tendon, from the epitenon, from the tendon sheath, and from paratenon [34], show a strong proliferative activity and a synthesis of the extracellular matrix components (ECM). An important role is played by the b-FGF above all regarding the cellular proliferation and the vascularity inside the injured area [35]. The interaction between neoformed capillary fibroblasts and myofibroblasts and ECM give origin to the granulation tissue, and the original "cork" of glue-like substance, formed in the first stages of the inflammatory phase, is substituted by a more stable structure. At the same time, fibronectin makes the migration and the adhesion of fibroblasts better. In the initial stage of the proliferative phase, more precisely starting from the seventh day of the injury, The fibroblasts produce glycosaminoglycans of the ECM (mainly hyaluronic acid) and collagen type III, even if a clear increase of the synthesis of collagen is observable only from the post-injury third week. The new collagen fibers which form possess, however, neither a consistent organizational structure nor a clear anatomical orientation. The last period of the proliferative phase registers the production of collagen type I, which continues until the end of the maturation and remodeling phases [2]. Collagen type I starting at about the 12th to 14th day begins to substitute collagen type III, and in the meantime, the granulation tissue is further maturing, and the scar formation assumes its solidity structure. During this phase, we may observe a decrease in the activity of the oxidative enzymes and a clear increase of the anaerobic enzymes [4]. It is interesting to note that also in the injured skeletal muscle in a few hours from injury, the consumption of oxygen at rest, inside the injured muscle, heightens dramatically, generating an imbalance between the refueling and the request of O_2, which determines a rapid descent of the tension of O_2 inside the insulted area; contextually we assist in an increase of the lactate concentrate inside the injury. The proliferative phase in the tendon lasts approximately from 3 to 6 weeks, a period which is progressively substituted by the maturation and remodeling phases.

2.4 The Remodeling and Maturation Phases

This latter one is temporally the longer one; it may go on for 1 year from the injury [8]. During the remodeling phase, the number of macrophages, fibroblasts, myoblasts, and capillaries diminishes in a slow and progressive way and contextually also the activity of synthesis drops. The scar area becomes less dense, its capillarity decreases, and also its matrix loses a certain fluidity; in the phase we assist, at first, in a progressive substitution of the reparation granular tissue on behalf of the fibrous tissue and, from the tenth week onward, a further substitution of the fibrous tissue on behalf of the tendon tissue [36]. Also the quantity of the glycosaminoglycans slowly decreases, changing its own distribution. The tendon collagen becomes less dense in its structural compactness and is mainly composed of collagen type I. In all, during this last third phase, we assist in a remodeling of the neoformed

collagen fibers, until the latter ones do not form a strong permanent structure [1, 2]. The full maturation of the collagen and a total realignment of the fibers usually need a period of 5 or 6 months from the outbreak of the injury. Toward the end of the remodeling phase, the fibroblasts, ceasing their biosynthetic activity, transform themselves into fibrocytes. In spite of this large remodeling process, the biomechanical and biochemical losses following the trauma may indefinitely maintain themselves [1, 2, 37]. The tensile strength of the tendon may reduce itself to over 30 % [1, 2, 37], and the structure of the latter may present defects in the distribution of collagen with an increase of collagen type III and V at the expense of that of type I, of the positioning of the fibers, and of the content in water, DNA, and proteoglycans [33]. It is interesting to note that the mechanism just described is represented by the proteolysis process. The proteolithic activity, in fact, results as a biological component essential for both tissue growth and its maintenance, not to mention of its adaptation and reparation processes. After an injury the proteolysis becomes necessary both for the removal of the damaged matrix and for the remodeling of the scar area [38].

2.5 The Role of the Nervous Response in the Healing Processes of the Tendon

Following trauma, of any nature, the initial stress of the organism is codified in a neural sign [26]. Despite the fact that the tendon is essentially lacking in its own nervous component [39], the unmyelinated axons which innervate the peritenon and the endotenon receive molecular products coming from the injury and, thus, transmit a recorded signal in order to modulate the efferent neural response with the immune response [40] So, the nervous system plays a fundamental role in the regulation of the processes of tendon reparation; this shows the fact that the application of calcitonin gene-related peptide (CGRP), of substance P [40–42], or of nerve growth factor (NGF) [43] improves in an animal model, the process of repairing the tendon, whereas denervation, both in mice and rabbits [44], respectively, worsens and delays the processes of tissue repair of the medial collateral ligament and Achilles tendon. In fact, obviously, a denerved anatomical system does not possess a physiological potential such as to allow it to face the integration request of the various and multiple biological requests coming from tissue in a reparation phase. On the other hand, also the scarce repairing capability of cartilage is substantially due to its un-neural and un-vascular nature [45]. The nerves and the blood vessels therefore adopt a synergic strategy of reciprocated support in the repairing of the tendon. In mice, for example, we may observe how nerves and vessels proliferate together from the peritenon during the proliferative phase, whereas during the remodeling phase, we may observe a strong neo-innervation in the surrounding areas in comparison with the area of repair tissue. This neo-innervation aims at reducing angiogenesis during the same remodeling phase [39].

2.6 The Role of the Apoptosis Process in the Last Stage of the Healing of the Tendon

The return of a normal situation of homeostasis tissue after injury is conditioned by a clearance of neoformed fibroblasts [47]. The density of the fibroblasts normally grows up until the fourth post-injury day to then decrease in a constant way. In each case, during the healing processes of the tendon, the density of the fibroblasts remains high by a percentage equal to 6–7 times the base value. This notable increase of the fibroblast activity is justified by the fact that the fibroblasts cover a fundamental role in the depositing and remodeling processes of ECM; however, this also indicates the biological necessity that such events return to normality at the end of the healing process of the tendon. Some authors hypothesize that this downregulation comes about, thanks to apoptosis phenomenon, a process characterized by the condensation of chromatin, fragmentation, formation of mass around the cellular casing, and destruction of the cytoskeleton [47–49]. All these phenomena show in a contraction of nuclear casing and of the cellular membrane, driving the cell to programmed self-elimination. The apoptotic cells are then removed by a silent physiological mechanism where the role of the central regulator is carried out by caspasis (a group of protease which contain cysteine in the active site). A certain number of studies witness the existence of the apoptotic phenomenon in tendon fibroblasts both on man and animals, in vivo and in vitro [49–51]. Both electromagnetic fields [52] and the oxidative stress [53] and the fluoroquinolones [54] are able to provoke the apoptosis phenomenon on tendon fibroblasts in culture. In the animal model, the rate of apoptosis appears very low in a structurally and biologically healthy tendon (range 0.56–1.3 %) [51], whereas it appears much higher in the case of tendinopathy [55]. On the contrary, in samples of human tendons devoid of disease, we may observe a particularly high amount of apoptotic cells, with an index of about 35 % in active remodeling sites and on average 26 % of tenocytes [56]. Since the ratio of apoptosis in tendon tissue, affected by tendinopathy, does not differ to that observed in a healthy tendon, or, to say 34 % versus 35 % [56], we may reasonably suppose that, in man, the apoptotic phenomenon is naturally linked to the normal turnover of tendon cells inside which we find the most complex remodeling process of ECM and that such a process may be seen in both normal and pathological conditions. In a pathologic picture represented by the healing process of the tendon, apoptosis is involved in the clearance mechanism of the excessive proliferation of fibroblasts which may be seen in the site of injury repair. The apoptosis phenomenon during the process of tendon repair, as witnessed in the caspasis activity, shows starting from the 14th day to then, it reaches its peak around the 28th post-injury day, even if some authors form the hypothesis that the apoptosis may be mediated also by other proteins in comparison with those of caspasis [46, 57]. The apoptosis interests not only the fibroblasts but also the myofibroblasts which disappear, because of the latter, in the final phase of healing [58, 59]. Apoptosis is a fairly rapid process, which requires from just a few minutes up to 1 h, and so reveals difficulty in sample testing, seeing as some cells may respond quicker than others. It is probably for this reason we may

explain the relative lack of studies and the consequent necessity for further analysis in this area. However, given that the cellular density of the tendon at the end of its repairing processes, it may be established by the ratio between the growth and the death of cells which may be seen in the injured area, the ability to deepen the knowledge of the processes which regulates such a phenomenon, and first of all apoptosis, it allows us to fully comprehend the mechanisms which permit the tendon tissue to reach a new situation of homeostasis once the repairing processes have been completed.

2.7 The Role of Growth Factors in the Healing Process of the Tendon

The growth factors (GF) assume a very important role inside the various phases of the healing process of the tendon, a role based on their specific operating target and their heterochronism action. The full understanding of how and when the various GF and their receptors in the process of tendon repair may be expressed by representing a future and most important stage in research by optimizing the processes of tendon tissue repair. We may thus resume the role and the timing of the GF in the course of the three stages of tendon tissue repair:

The platelet-derived growth factor (PDGF) is only produced for a short time immediately after the injury and stimulates the other GF [60, 61].

The transforming growth factor-beta (TGF-ß) is active during the inflammatory and proliferative phases but assumes an even more important role during the second of the said phases. By separately analyzing its three isoforms, we may observe how the TGF-ß contributes to the sedimentation of ECM and how its overexpression appears in the formation of fibrotic tissue; furthermore it is possible to note how TGF- ß2 acts in a similar way to that of TGF-ß1 and that in the end TGF-ß3 shows the capacity to improve the scar tissue. The peak of the activity of the receptors of the expression of TGF-ß is registered around the 14th post-injury day and begins to decrease by around the 56th day [62–64].

The vascular endothelial growth factor (VEGF) stimulates the proliferation of endothelial cells, improves the angiogenesis, and increases the capillary permeability. Inside the injured area, the expression of VEGF RNA may be observed from the seventh post-injury day, whereas its peak is registered around the tenth day [65, 66].

The isoforms of nitric oxide synthase (NOS)[3] are expressed, through different expression patterns, during all three phases of tendon repair [67] (Table 2.1).

[3] *NO synthase is an enzyme distributed almost ubiquitously in tissues and in living organism in general that provides to produce NO starting from arginine that is converted to citrulline (intermediate metabolite of the urea cycle).*

Table 2.1 Diagram of the main cellular alterations and of the matrix regarding the reparation period

Period	Main cellular and matrix alterations
Immediately after injury	The presence of erythrocytes often grouped together in the form of small clots. Presence of fibronectin (within 1 h) and macrophages
24 h later	The presence of polymorphonuclear leukocytes, monocytes, and macrophages (also before in case of mechanical breakage and later on case of spontaneous breakage) Beginning of the synthesis of hyaluronic acid, followed by the synthesis of glycosaminoglycans (in any case later on)
Fourth to fifth day	The presence of fibroblasts
From the seventh day onward	Slow and progressive diminution of leukocytes, macrophages, and fibroblast activity Increase in the presence of fibronectin No presence of procollagen before the seventh day From the seventh day, collagen synthesis begins in the epitenon but not still in the endotenocytes The presence of myofibroblasts in the granulation tissue
Second week	The granulation tissue becomes more compact The fibroblasts (tenoblasts) show orientation on the main axis of the tendon The collagen synthesis is evident also in areas detached from the repair zone The neoformed collagen type III (formed in the injured zone) is progressively substituted by collagen type I (formed outside the injured area) A progressive increase in tensile strength of the tendon begins with the substitution from collagen type I to type III
Fourth week	The number of fibroblasts, myofibroblasts, and capillaries starts to decrease The number of macrophages clearly diminishes Collagen forms dense packages of fibers
From the fourth week onward	The remodeling and maturation phases continue, which may carry on for a period between 4 and 11 months

2.8 The Role of Angiogenesis in Cellular Proliferation

The high concentration of GF and cytokines secreted at first by the platelets and the leukocytes followed by the macrophages produces a rapid increase in several specific cellular populations, like endothelial cells, migrant fibroblasts, and resident tendon cells. Above all, it is important to note that the number of tenocytes contextually increases the phenomenon of angiogenesis [26]. In an animal model, the VEGF-A is precociously present in the post-traumatic phase of tendon damage [68], whereas others just as important GF for proliferation and vascular stability (like TGF-β, PDGF-BB, and angiopoietin-1) are observable inside the injury only in the following phases [69].

2.9 The Intrinsic and Extrinsic Healing Processes of the Tendon

Several authors sustain the hypothesis that the healing processes of the tendon have origin from the two injured tendon stumps. This theory is called "the theory of intrinsic healing." Other studies, instead, attribute tendon healing to the sole cellular activity in peritenon tissue. This second theory is known as "theory of extrinsic healing." In the end, a third thought is to recognize the two aforementioned processes as an equally complemented role.

2.9.1 Extrinsic Healing Mechanisms

Since 1962 [70] showed that in a cut tendon, repaired by suturing and thus immobilized, the reparation came about thanks to the formation of granular tissue coming from the peritendon structures, where an intense proliferative activity was observable. During this repairing process on the peritendon, the author observed that the tendon tissue remained inert and following this observation concluded that the tendon tissue was missing in any type of repair capacity whatsoever. So, the total loss of self-reparative properties of the tendon tissue should show its healing processes which entirely entrust the formation of scars. Other authors hypothesize that during the reparative process of the tendon, the phenomenon of neovascularity has origin mainly from the paratenon and other peritendon tissues and that its own vascularity has a minor role [71]. Also other authors [72] support this hypothesis, confirming that tendon repair processes come about through the surrounding tenosynovia in the area where the fibroblast cells cover the injury on the tendon body. However, an obvious observation regarding this affirmation is in the fact that one of the main causes of failure in the recuperation of full tendon functionality following a breakage is represented by the formation of scars between the suture area and the peritendinous structures [73]. So, the fact that extrinsic repair of the tendon bases itself on the formation of adherence, and, at the same time, these latter ones strongly limit the full recovery of the tendon flow represents an oxymoron [74]. Indeed, from the moment that full functionality of a tendon depends largely on its flow capacity, the peritendinous adherences, very often, have to be surgically removed. In the specific case of the Achilles tendon, we must remember that, since it presents reduced movement, the eventual formation of adherences does not limit its functionality in a dramatic way, as however happens in the case of finger tendons, which, on the other hand, present a much more ample movement [75, 76].

2.9.2 Intrinsic Healing Mechanisms

Even before the studies of Potenza [70], Wheeldon [77] referred to how the use of a cellophane membrane to rebuild the flow sheath after suturing the long extensor tendon of the thumb obtained full anatomical healing of the tendon and a total

recovery of its functionality without the formation of peritendinous adherences. Further studies confirmed the capacity of intrinsic repair in the tendon tissue, both in breakages of the flexor tendons of man [73, 74] and animals in vivo and in vitro [14, 15, 78–80]. All these studies showed the intrinsic healing capacity on behalf of the tendon, both in vivo and in vitro, based on experiments which foresaw, during the repair process of the tendon, the exclusion of all possible external cellular contributions such as circulation and the influence of synovial liquid. In such a situation, phagocytosis comes about through the transformation of epitenon fibroblasts, whereas the synthesis of collagen is mainly performed by the endotenon cells, whose migration on the injured tendon has been observed also in an in vivo model [81, 82]. In all studied models, the nutritive contribution necessary for tendon healing processes is provided by the synovial fluid, and repair comes about without the formation of adherences. In normal clinical practice, on the contrary, the lysis of the tendon adherences is necessary in 20–30 % of cases [76]. The diatribe between the sustainers of the mechanisms of extrinsic and intrinsic repair may substantially settle by keeping the hypothesis that the intra-tendon micro-circle, and the production of synovial fluid is preserved thanks to the type of surgery used, and if, at the same time, the injured tendon is immobilized in time (compatible with its repair processes), the tenocytes are able to genetically express a self-repairing program and thus give life to intrinsic repair. If instead, the nutritive contribution of the tendon, following surgical repair is jeopardized the mechanisms of extrinsic repair may prevail over those of intrinsic repair above all if we add an excessive immobilization period [83, 84]. In any case , we must remember that the precise effects of mechanical stimulation of a tendon in repair in man are still not clear [85].

2.10 The Molecular Bases of Neoformation of the Tendon

Even though no markers of the tendon morphogenesis have been indicated as a potential target of the neoformation processes of the tendon, evidence exists that such a process may be influenced by the activation of specific factors. The factors which have the most documentation in this area are the growth and differentiation factors (GDFs) and scleraxis (Scx).[4] The GDFs represent a group of the superfamily of transforming growth factor-β – bone morphogenetic protein (TGF-β/BMP) and are secreted in the form of mature peptides which form homo and heterodimers[5] [86]. Initially some studies have shown how the GDFs, the GDF6 and the GDF7 were, in

[4] *The protein scleraxis (Locus: Chr. 8 q24.3) is a member of the superfamily of transcription factors basic helix-loop-helix (bHLH). It is expressed in mature tendons and ligaments of the limbs and trunk but also in their progenitors. The gene coding for Scx is expressed in all the connective tissues that mediate the connection of the muscle to the bone structure, as well as in their progenitors that are found in primitive mesenchyme.*

[5] *A dimer is a molecule formed by the union of two subunits (called monomers) of an identical chemical nature (homodimer) or of a chemical nature different (heterodimer).*

mice, implicated in the processes of osteogenesis through endochondral ossification or the bone formation that begins with the condensation of mesenchymal cells [67, 87]. The first studies which identified a marker of articular development in mice in the GDF5 go back to 1996 [88]. In these experiments the authors showed how GDF5 were necessary and sufficient for the cartilage development process on animals. In mice the role of GDF5 in tendon formation on subjects which had tendon abnormalities has recently shown, for example, an insufficient development of the patellar tendon, due to structural alterations of collagen [89]. Even more recently [90] it has been observed, in mice and in subjects which present a deficiency of GDF5, an incomplete development of femoral condyles and of intra-articular ligaments of the knee. Regarding this, it is interesting to observe that, in studied subjects, a large and excessive apoptosis of mesenchymal cells in the area of development of the knee articulation has been seen. However, if both these studies show, with sufficient evidence, the role taken on by GDFs in the development of articulation, otherwise may not be said regarding the morphogenesis of the tendons. We must, however, remember that a study by Wolfman and coll. [91] had already shown that the expression of human GDF5, GDF6, and GDF7 in ectopic sites in adult animals induced the formation of connective tissue rich in collagen of type I similar to the neoformation of tendon and ligament tissue. Furthermore, Wolfman and coll. [91] observed that the co-implant, intramuscular or subcutaneous of GDF5, GDF6, and GDF7 with BPM-2, induces the formation, in a tissue containing contextually bone and tendon tissue, suggesting in such a way that the GDFs perform a tenogenic effect also in the presence of BMP-2 and in osteogenic conditions. More recent studies [92] also use the hypothesis that the GDFs have, on an adult animal, a stimulating effect on the regeneration and the neoformation of the tendon, as well as in the tendon morphogenesis on animals in development. The administration of human recombining GDFs (rhGDF5) in the injured area of a sutured tendon in mice induces a significant improvement of the healing processes, which results in a higher tensile strength and in stiffness of the tendon compared with the counter-lateral, equally cut and sutured, but which has not received the administration on rhGDF5 [92]. To obtain an effective improvement of soft injured tissue through growth factors (e.g., GDF5 in the case of tendon tissue), a crucial point is represented by the full comprehension of all the temporary sequence of events which happen during the natural healing processes of the various types of tissue considered. In the specific case of the tendon, when it undergoes a structural injury, we assist in the formation of a hematoma in the injured area which works as a matrix for the following invasion on behalf of the mesenchymal cells which, as we know, carry out a determining role in the processes of tissue repair [85]. The injecting of GDFs inside the hematoma during the formation phase has been considered by some authors as a promising therapeutic approach able to improve tendon healing processes [93]. The administration of transgenic GDF5 through an adenoviral vector in the area of the Achilles tendon in mice shows an improvement in terms of caliber and strength of the repaired tendon, if compared to the counter-lateral which has not received GDF5 [94]. It is, however, important to underline the fact that the authors, during said experimentation, observed an abnormal proliferation of cartilage tissue inside the formed repaired tendon tissue, a fact

which indicates possible disturbance of the repair processes of GDF5. We may, anyway, assume from the various available studies on the argument that GDF5 may be considered as a reasonable candidate regarding the tendon neoformation and the possible improvement of tissue repair processes. In spite of this, the fact that GDF5 in vivo may induce bone and cartilage neoformation could prevent the use of a factor of tendon regeneration [95, 96, 97]. However, since the effects of GDFs are, in mice, of dose-dependent type (300 μg of rhGDF5 induces bone and cartilage formation, whereas 500 μg only provokes bone formation), maybe it is possible that fine regulation of the dose may be the key to the solution of the problem, allowing an improvement of tendon tissue in healing, excluding the formation of other undesired neo-tissues. As well as GDFs, much research also indicates Scx as a possible molecule marker of the processes of tendon neoformation. The protein Scx (scleraxis-locus: Chr. 8 q24.3) is a member of the superfamily of transcription factors basic helix-loop-helix (bHLH) and is expressed in mature tendons and in ligaments of the limbs and the trunk but also in their pro-parents. The gene which codifies for Scx is expressed in all connective tissues which mediate the connection of the muscle to bone structure, as well as in their pro-parents which are found in the primitive mesenchymal. Scx is the best marker of tendon morphogenesis, and there is growing evidence on the fact that it can cover the same role also regarding the processes of tendon neoformation. As already mentioned, Scx is a bHLH transcription factor [98], and it may link to DNA sequences containing the "E-box[6]" consensus sequence[7] though it is bHLH [98]. During embryogenesis in mice, the transcription of Scx is observable both in areas of formation of pro-parent tendons and in the somite[8] of the same pro-parent tendons called sindetoma [99]. The analysis of sequence of Scx shows the presence of all the amino acids which characterize the bHLH[9] family [100]; however, other residues of the base regions are different in comparison with other transcription factors of bHLH, suggesting, in such a way, that Scx ties a specific group of E-box [100]. So, despite the fact that in pro-parent tendons, or in other bone and cartilage structures, an important formation of collagen type I and II is required, we may observe high levels of Scx transcription, whose role would seem limited to the function of progenitor tendons [99]. Scx is expressed in anatomical sites similar

[6] *An E-box is a DNA sequence that is typically located upstream of a gene in a "promoter region."*

[7] *In molecular biology and bioinformatics, a "consensus sequence" refers to the most common amino acid or nucleotide in a particular position after more aligned sequences.*

[8] *Somite [from the Greek "soma," body-ite], in embryology, is each of the segments in which it divides the dorsal mesoderm (or epimer), left and right of the spinal column. The somites give rise to elements that will form the dermis of the skin of the trunk (dermatomes), the muscles (myotomes), and the axial skeleton (sclerotomi).*

[9] *The myogenic regulatory factors are transcription factors belonging to the family "basic helix-loop-helix" (bHLH), because they contain a basic domain involved in binding to the DNA and a domain HLH needed to form homodimers or heterodimers with other proteins containing HLH domains. The bHLH motif is found in many transcription factors that are ubiquitously expressed in a tissue-specific manner.*

to those in which we observe the expression MyoD[10] which determines muscular morphogenesis. This would suggest that Scx acts in the area of tendon development in close association with the phenomenon of muscular development but without overlapping the action of MyoD [99]. This represents an important aspect of research in the area of factors which can improve the tendon healing processes, because it is obvious that the choice does not necessarily fall on the molecular target which does not imply, at the same time, muscular neoformation.

Even though many studies demonstrate an active role of Scx in tendon morphogenesis, it is still not evident that this may induce the phenomenon of tendon neoformation. Scx ties to the E-box consensus sequence as a heterodimer with E12 (a member of the family of E proteins which forms heterodimers with the bHLH protein and ties to DNA to regulate the genic expression). Furthermore, Scx is a powerful trans-activator of the genic expression [100]. A study by Lèjard and coll. [101] shows how Scx regulates the expression of the codifying gene for collagen type I in the fibroblasts of the tendon, or the COL1A1. In a recent experiment, done on mutant homozygous mice for an invalid allele Scx (Scx mice), we observed a strong disturbance of the processes of differentiation and of tendon formation [102]. The severity of the disturbance in these processes was variable, in some cases reaching a true destructive phenomenon, whereas in others, the tendon unity remained substantially intact. This study would thus use the observation previously executed by Lèjard and coll. [101] and would confirm the fact that Scx would activate the expression of the genes involved in tendon development even though the exact functions of such mechanisms remains, for now, unknown. So, we may conclude that the transcription factor bHLH Scx may, in all effect, be considered as an important marker of tendon neoformation; thus its involvement in neoformation processes also uses the hypothesis that Scx, once activated, would be able to induce the regeneration of tendon tissue, even though such an affirmation is today missing in sufficient evidence.

2.11 Conclusions

The processes of tendon repair, even though they largely trace the stages of skeletal muscle repair, maintain their specificity, differing themselves from a muscular model under numerous and non-under-valuating aspects. For example, the mechanisms of intrinsic and extrinsic healing represent a peculiarity of the mechanisms of tissue repair of the tendon, which do not find analogy in the healing processes of the skeletal muscle. For this reason, the rehabilitation process of the injured tendon is completely different from that applicable in the case of muscle injury. Also, the process of tendon neoformation in the adult covers fundamental importance, above all considering the fact that their optimization could resolve the long-standing

[10] *The MyoD gene encoding a transcription factor involved in the differentiation of the muscle, in particular, induces fibroblasts to differentiate into myoblasts.*

problem of the healing of tendon tissue, a problem which today has still not been resolved. The perfect healing of tendon tissue requires a sequential and coordinated expression of numerous molecules and GF, each responsible for a specific and distinct process. In the final part of this work, we have taken into consideration the molecules which present themselves as potentially more valid candidates for the activation of the processes of tendon tissue neoformation. Regarding this, it would seem possible that the use of recombining GDFs could be approved for clinical use in the treatment of tendon breakages [103]. Even the Scx would show an applicative interest in this sense, even if it should be used through a gene therapy approach (the most probable of these would seem to be the use of nonviral vectors) since an extracellular application of the protein would not generate any on site effect [103]. However, in this area, further and deeper studies are still necessary which evidence that characterization of the optimal factors adapts to induce the neoformation of tendon tissue in various models of tendon breakage and tendinopathy.

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Chapter 3
Adductor Tendinopathy

Jean-Marcel Ferret, Yannick Barthélémy, and Matthieu Lechauve

Abstract Adductor pain is very common in sports, but it is essential to distinguish among true tendinopathy, which is an enthesopathy (adductor longus insertion pain on the pubis), a tear of the myotendinous junction, which is rarer, and projected pain, where the adductors are affected the victims rather than being the cause: in abdominal groin pain (pubalgia) and all hip problems, especially femoral acetabular impingement (FAI), which affects young athletic population. Adductor tendinopathy can be isolated, but is also often associated with pubalgia. Once a positive diagnosis has been established, treatment can be tailored to the cause: medical for isolated tendinopathy, and often surgical in the form associated with pubalgia. Abdominal parietal pain is often the evolution of neglected adductor tendinopathy, which is why we must encourage those in the sporting environment to be more rigorous in the management of this pathological condition.

3.1 Introduction

Adductor pain is very common in sport, especially in activities with acceleration, deceleration, sudden changes in direction, blocking, trunk rotation, sliding tackles, and kicking: football, rugby, handball, and ice hockey [1, 2]. According to different authors, the epidemiology of adductor pain varies from 5 [2] to 16 % of all injuries in soccer players [3, 4].

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Adductor tendinopathy corresponds to one of the four forms of sport groin pain:

- Abdominal parietal failure
- Pubis arthropathy
- Enthesopathy adductors
- Enthesopathy rectus abdominis

These pathological conditions do indeed have a common location, all around the symphysis pubis, which plays the role of a true anatomical and functional crossroads. They occur within a common framework, consisting of mechanical stress to the pubic region, due to the practice of sports. Moreover, these four pathological conditions are often intertwined in athletes, although they can occur in isolation. The tendinopathy physiopathology is a multifactorial process, involving intrinsic and extrinsic factors that evolve independently or in combination [5]. In particular, repetitive mechanical loads and/or the application of loads exceeding the strength of the tendon may gradually cause microscopic and macroscopic lesions. Collagen fibers start to denature, gradually causing tendinitis [6].

3.2 Anatomy

3.2.1 Adductor Muscle

The adductor muscles (longus, brevis, magnus, pectineus, and gracilis) have a proximal insertion into the subpubic area with the internal obturator. The pectineus lies above and beyond the pubic tubercle, along the pubic crest. The gracilis originates inside the adductor on the front of the pubis and internal third of the lower edge of ischiopubic branch (Fig. 3.1).

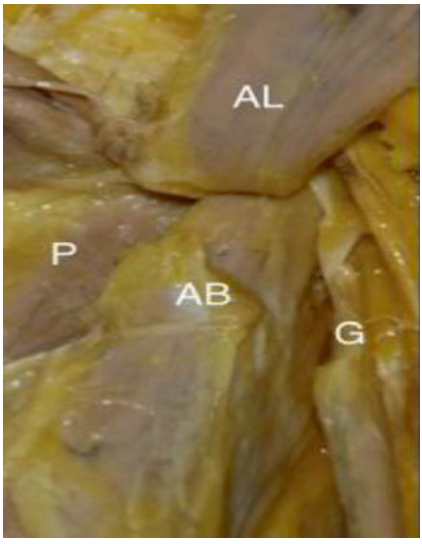


Fig. 3.1 Cadaveric adductor anatomy (Pesquer collection)

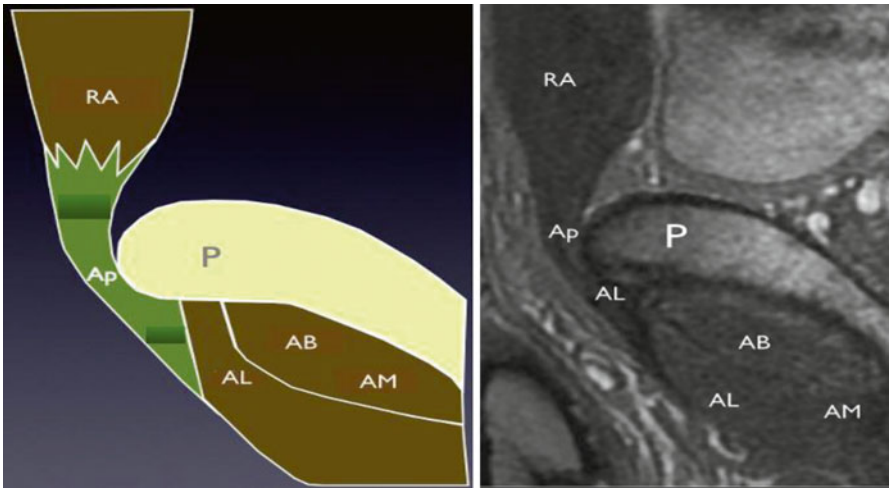


Fig. 3.2 Adductor anatomy on MRI (Pesquer Collection)

The proximal insertion of the adductor longus is close to the pubic tubercle and pubic symphysis through the tendon fibers (40 %) in area and the muscle fibers (60 %) in depth. Brevis and magnus adductor insertion is only muscular [7, 8]. The adductor longus tendon is small, measuring 11.6 mm long and 3.7 mm wide, according to a cadaver study [9]. It is the only adductor, to have an individualized tendon. There is an anatomical continuum through a common fascia between the surface fibers of the adductor longus tendon and the distal tendon of the rectus abdominis. This fascia is in contact with the fibrocartilaginous disk of the pubic symphysis and the capsular structures. These anatomical data explain why painful irradiation of one or more of the affected structures can extend to the thigh and the abdomen (Fig. 3.2) [10].

3.2.2 Inguinal Region

The inguinal region is located in the lower part of the abdomen, delimited by the outer edge of the rectus abdominis inside, by the oblique and transverse abdominal muscles outside, by the inguinal ligament at the bottom, and by the oblique internal and transverse muscles at the top. The inguinal canal lies between the muscle layers. The anterior inguinal wall consists of the external oblique fascia; the fibers diverge to create the superficial inguinal ring. The space between the inguinal ligament at the bottom and the conjoint tendon at the top is closed by the transverse fascia. It is a thin and solid membrane that is in contact with the peritoneum. The outer part of this area is the deep inguinal orifice, through which spermatic cord components pass to the testis. The peritoneum is located behind the transverse fascia plane. The iliohypogastric, ilioinguinal, and genitofemoral nerves pass behind the external oblique fascia, in front of the muscles.

3.3 Definition and Differential Diagnosis

The type of complaint is not unequivocal: we immediately eliminate acute pathological conditions, such as bone avulsion of the pubis and a myotendinous tear of the adductor. Moreover, distinction should be made between true adductor tendinopathy, i.e., insertional tendinopathy or enthesopathy [7, 11], and pain of locoregional origin such as groin pain due to abdominal injury or hip arthropathy. Indeed, in this case, different pathological conditions are associated, and the adductors are the victims rather than the cause. Our aim is to treat only the isolated adductor enthesopathy. Adductor enthesopathy essentially concerns the adductor longus with a prevalence of 45–60 % [12]. First, we must eliminate all pathological conditions with groin pain that is not caused by adductor longus enthesopathy and especially abdominal groin pain and hip arthropathy [13].

3.3.1 Abdominal Groin Pain

In the case of abdominal groin pain, the pathological condition may start with a mild pain of the adductor, which is often neglected, but can quickly become complicated abdominal wall pain with an unpredictable evolution. Clinical examination of the abdominal wall is aimed at helping to refine the diagnosis: rectus abdominis tendinitis, pathological condition of the inguinal ring and abdominal wall (athletic pubalgia), inguinal hernia, tunnel syndromes, and obturator, iliohypogastric, ilioinguinal, and symphysis arthropathy [14].

Questioning together with clinical and complementary examination (imaging, EMG) easily eliminate rectus abdominis tendinitis, symphysis arthropathy, inguinal hernia, and tunnel syndromes. Examination of the inguinal canal is imperative in the context of long adductor tendinopathy. The deep inguinal ring, including failure of the transverse fascia, would then be involved. In the chronic form, the painful symptoms are often exacerbated by the increased abdominal pressure.

For athletic pubalgia, the primary functional sign is inguinal pain with coughing, especially after effort, and abdominal pain when athletes sit up in bed after waking up. External orifice and deep orifice palpation must be bilateral and comparative information on a patient standing, the scrotum is pumped and forefinger palpates the channel up, out, and back. A Valsalva maneuver helps to inform the test. Clinical examination finds an enlarged inguinal ring, and an impulsive and painful cough. This form, if it does not show improvement after 2 months of well-conducted rehabilitation work, requires a surgical opinion [15]. The disorder differs from a real inguinal hernia in that only one protrusion or bulging fascia was found during Valsalva maneuvers [16]. Adductor tendinopathy, in this context, is an associated pathological condition.

The treatment is adapted to the pathological condition, which requires a very precise diagnosis because there are numerous intricate forms. Early diagnosis is essential for rehabilitation treatment to be efficient. Surgery has its place in forms

resistant to medical treatment, particularly in cases of inguinal canal posterior wall deficiency [14, 17].

In professional clubs, individual screening for athletes at risk should be systematic early in the season and preventive care should be performed throughout the season.

3.3.2 Hip

Hip pain is the other essential diagnosis. Indeed, any abnormality of the hip can cause adductor pain. This phenomenon is well known for hip osteoarthritis, but less so in femoral acetabular impingement (FAI) [18, 19]. When diagnosing any adductor pain that is resistant to treatment, a hip problem must be eliminated [20, 21]. This will be systematically reviewed and any limitation of the range of motion (ROM) taken into consideration. In FAI, a pathognomonic sign is pain on hip flexion, adduction, and internal rotation (FADIR: Fig. 3.3). Another important sign is pain, after effort, on hip flexion (for example, in our experience, pain after a soccer game when a player uses his hip in flexion >90° braking or acceleration), a sign never found in abdominal groin pain. FAI concerns physical activities with ballistic hip movements of large amplitude, such as soccer, martial arts, and artistic dance [18].

Characteristics of FAI are:

- Cam lesion: additional bone seen, more frequently at the antero-superior position of the femoral head neck junction [22, 23]; it is the most frequent lesion in soccer [24].
- Pincer impingement of the hip due to a change in the acetabulum: deep or retroverted acetabulum [29].
- A combination of the two.

The premature contact of the head–neck junction of the hip with the acetabulum caused by a cam or pincer bony abnormality causes pain in the inguinal area for activities with flexion, adduction, and medial rotation [25].

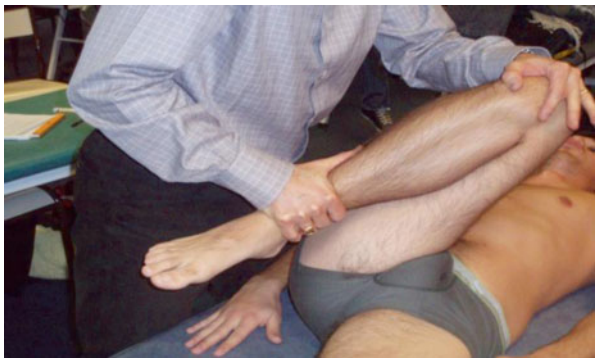


Fig. 3.3 Pain in flexion, adduction, internal rotation (FADIR) of the hip

High-level activities in childhood give rise to the risk of cam-type deformity [26], possibly in relation to a minimally slipped capital femoral epiphysis [27].

Imaging is important for evaluating FAI: to define the type of lesions and to adapt therapy. Plain X-rays and contrast-enhanced MRI are sufficient.

Rest or decreased activity: avoidance of hip hyperflexion, associated with hip rehabilitation therapy (on a bicycle with no resistance, 100 turns per minute for 20 min daily), lumbar and gluteal muscle strengthening, and postural work are sufficient, in most of cases, to enable a return to sport. However, in some cases, surgery may be needed (arthroscopy, mini-arthrotomy [28, 29].

Other pathological conditions of the hip to eliminate by rigorous clinical examination and imaging are:

- Coxarthropathy
- Acetabular labral tear
- Slipped capital femoral epiphysis (childhood pathological condition)
- Femoral head osteonecrosis
- Femoral neck and ischiopubic branch stress fracture
- Osteochondrosis or avulsion of the antero-inferior iliac spine (adolescent pathological condition)

3.3.3 Other Differential Diagnoses

- Hip muscle injury: iliopsoas, lateral and medial rotator, and iliopsoas bursitis
- Radicular syndrome: obturator neuralgia
- Inflammatory rheumatism: rheumatoid polyarthritis and ankylosing spondylarthritis
- Infectious lesions: symphysis osteitis
- Metabolic arthropathy: hyperuricemia
- Benign tumors: osteoid osteoma; malignant tumors, osteosarcoma

3.4 Etiopathogenesis

This intimate relationship among the adductor longus, the rectus abdominis, and the pubic symphysis is the anatomical substrate of biomechanical assembly to ensure the stability of the anterior pelvis and transmission of forces from both sides of the pubis [7]. This “confluence” of anatomical elements explains the frequency of associated damage among the different clinical forms [30]. Pesquer et al., in a retrospective series of 180 patients operated on to treat adductor tendinopathy, found that 80 % of patients had associated parietal failure requiring surgical treatment [31].

There are two types of risk factors for adductor tendinopathy.

3.4.1 Intrinsic Factors

The pathogenesis of tendinopathy adductor insertion is related to functional overload and repetitive strain injuries caused by twisting and pulling tendon insertions [32, 33]. In particular, some sports predispose to groin pain, such as soccer, Australian and American football, and ice hockey.

Soccer player morphotype characterized by association of a lack of extensibility of the anterior and posterior chains, hyperlordosis with pelvic anteversion, and genu varum and flexum of the knee, causes an overload of the adductor. The analysis of the morphological type of an athlete is important. The examination of the spine and lumbopelvic hinge assesses the overall static equilibrium of the patient's pelvis and the joint mobility of the different segments. A pelvic tilt due to legs of unequal length can be corrected if necessary. Anteversion of the pelvis is also reported to be a risk factor for groin pain [19, 37]. The use of a biplanar imaging system with a low X-ray dose (EOS) seems promising in the overall systematic analysis of the static lumbar–pelvic complex.

Many authors believe that the origin of the pain is due to a muscle imbalance in stabilizing the pelvis between the strong and retracted adductors and a weak abdominal wall [34, 35]; other authors describe an imbalance of the hip adductor and abductors as a cause of groin pain [36].

Any limitation in the ROM of the hip causes an adductor malfunction. Soccer is the best example because at 20 years old, professional players have a significant limitation of flexion (8 %), abduction (30 %), and adduction (35 %) in comparison with the general population of the same age [38].

The ballistic movements of large amplitude inflict significant pressures and microtraumas upon the femoroacetabular joint, notably during shooting with to arm it: hip extension, abduction, lateral rotation, and at the end of movement flexion, adduction, and medial rotation [18]. This could explain the limitations in amplitude in the young professional player (Figs. 3.4 and 3.5).

Fig. 3.4 Beggining of shooting movement: extension, abduction and lateral rotation of the hip, knee flexion

Fig. 3.5 End of shoot movement

Flexion, adduction, and medial rotation of the hip, and knee extension:

- The succession of accelerations, blockages, direction changes, rotations, jump receptions, and striking involve so much pressure being exercised all over the lumbar–pelvic–femoral complex, explaining the hyperuse of adductors and the abdominal wall. The abdominal cavity consists mainly of water and therefore is slightly compressible. Any work done without abdominal sheathing and pelvic retroversion increases abdominal pressure with a push on the anterior abdominal wall. Repeated abdominal strengthening exercises in a bad position, e.g, pelvis anteversion or hyperlordosis, contribute to abdominal wall distension, in addition to the practice of sports involving rotation and pivoting that exert considerable force on the lumbar–pelvic–femoral complex with increased abdominal pressure [39].

Other Etiologies

- Static troubles of the lower extremity: inequality, static disorders of the feet.
- Hygienic or dietary problems:
  - Lack of hydration
  - Acidifying diet: food too rich in sugars and animal proteins
  - Poor dental status promoting chronic inflammation

3.4.2 Extrinsic Factors

Extrinsic factors that are involved in this overuse at groin, particularly:

- Ground quality: hard ground, for example, road training, frozen ground, soggy pitch
- Atmospheric conditions: wind and rain
- Change of playing surface: grass, synthetic or canvas shoes, crampons
- Excessive physical activity, a sudden increase in training load, loss of fitness after injury
- Bad programming of training, inappropriate content, disorganized competition schedule, overpressure of abdominal work

3.5 Clinic

Precise questioning must precede the clinical examination: pain severity (diagnostic consultation is often late), pain location at the root of the thigh, sudden appearance or gradual, irradiation of the medial face of thigh, pain on effort or after effort, no pain when coughing, no pain when the subject sits up in bed upon waking or in hip flexion (sitting or driving) [18]. The clinical examination should be performed at rest and after effort, which increases symptoms and enables a more accurate diagnosis and prediction of evolution. In the case of adductor tendinopathy, examination is characterized by adductor insertion pain; it radiates into the medial face of the thigh with adductors more or less painfully and contracting.

Its intensity is variable, it may:

- Prevent sports activity
- Cause only gene decreasing sports performance
- Cause annoyance in daily life
- Rest has a favorable influence; sleep is usually not disturbed, but possibility of adductor mornig stiffness.

The main symptom is pain on palpation of the adductor longus insertion on the pubis, this being the only adductor with an individualized tendon; indeed, other adductors fit directly to the bone owing to their muscle fibers [8, 31].

Two other signs confirm the diagnosis:

Pain on adductor stretching due to hip abduction, knee in flexion (FABER; Fig. 3.6)
Pain on resisted adduction from the same position, but also:

- Absence or mild soreness when resisting contraction of hip flexors; indeed, adductors are hip flexor accessories
- Absence of pain on abdominal wall palpation: rectus abdominis pubis insertion, symphysis, inguinal ring
- Absence of pain on trunk flexor when resisting contraction
- No pain when coughing
  It should be noted that in many cases, the clinical form is associated, and it is not rare to find positive tests for several anatomical entities. The tests used must be as sensitive, specific, and reproducible as possible [40].

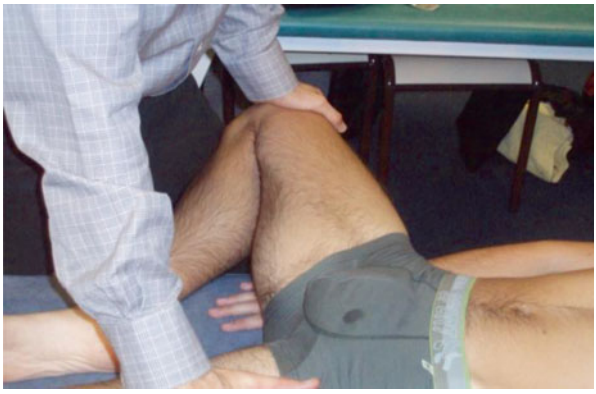


Fig. 3.6 Flexion abduction external rotation (FABER) movement

3.5.1 Biology

Any abnormality must move towards another diagnosis.

3.5.2 Evolution

The evolution is unpredictable from the start: rapid healing or chronicity. Recurrence is very common: 26 % in a population of athletes followed for up to 22 months for a pathological condition of the adductor longus [41].

3.6 Imaging

3.6.1 X-Ray

Adductor longus tendinopathy diagnosis is based on ultrasound and MRI, but standard X-rays are essential for studying the bone matrix, the pubic symphysis, the femoral hip, and pelvic imbalance. Standard radiography with the patient in a balancing on one leg also demonstrates the instability of the pelvis. Instability is discussed when there is a mismatch of ischiopubic branches of 2- or 7-mm diastasis [42].

3.6.2 MRI

Adductor longus tendinopathy manifested as peripheral and central T2 signal enhancement without fluid character and enhancement after gadolinium injection is associated with morphological abnormalities (thickening of the tendon, fissure or avulsion) [31, 44]. These discrepancies, notably T2 signal enhancement, are still

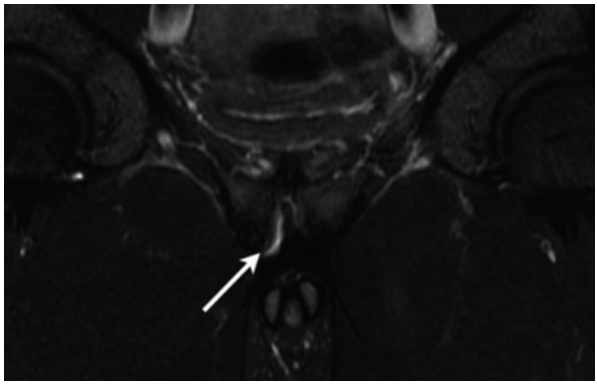


Fig. 3.7 Secondary cleft (Pesquer collection)

inconsistent in the absence of a fissure, recent bleeding or avulsion. This could be explained by the frequency of fibrotic lesions, calcifications and microcalcifications found intraoperatively and pathologically. One study showed that intratendinous abnormalities are sometimes minimal or absent on MRI and edema of the trabecular bone of anterior inferior topography is the only evidence of impairment of the aponeurosis (proven by surgery). The monitoring over time of these anomalies also shows that there is a progression of lesions to a typical aspect of a "secondary cleft" (Fig. 3.7) [10]. The secondary cleft corresponds to an extension of the lesion of the common fascia deep to the capsule down to the groin and in continuity with the primary physiological cleft [10, 31].

There is a strong correlation among the side of the pain, the presence of the sign, and the abnormalities found on MRI and symphysis pubic arthrography [43].

3.6.3 Echography

Ultrasound is widely used because of its ready availability and the spatial resolution allowed by the latest generation of hardware. It is performed with the patient lying down, the hip in flexion, abduction, and external rotation, using a linear high frequency transducer [31, 46]. The adductor analysis is guided clinically by identifying the protrusion of the adductor longus to the anteromedial side of the thigh. Longitudinal and axial slices are usually made and mandatorily compared with the contralateral side.

In sports, asymptomatic microcalcifications are often associated with cortical irregularities, which reflect an old enthesopathy. It is important to analyze the superficial side of the adductor longus tendon: it appears, in healthy subjects, regular and irregular, associated with ulcers of tendinopathy [31, 44]. If enthesopathy is recent or not, it is difficult to distinguish with certainty, and hyperemia can be recognized on color Doppler, but remains an inconstant sign. At a more advanced stage, the tendon is hypertrophic and there are often fissures, which may be longitudinal or transverse. These are more common and correspond to the secondary cleft described on MRI [45].

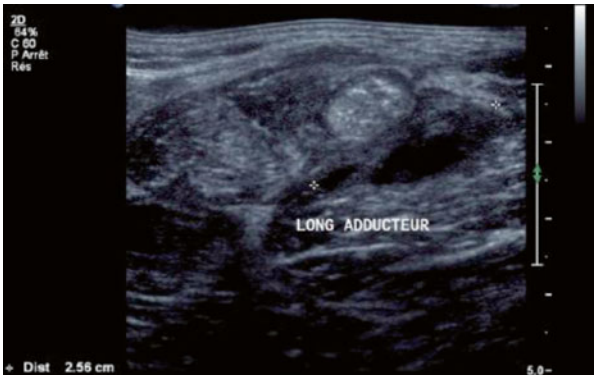


Fig. 3.8 Adductor longus disinsertion (Ph. Tanji collection)

In cases of acute pathological conditions, disinsertion may be asserted if the muscle belly of the adductor longus is significantly retracted, i.e., more than 2 cm from the bone insertion and if there is hematic fluid collection (Fig. 3.8).

Finally, ultrasound study, thanks to its dynamic specificity, is useful for looking for signs of parietal weakness in the superficial inguinal ring. In athletes, we do not expect to see a hernia sac to surface through the inguinal canal, but rather a bulging medial side (direct hernia) or lateral side (indirect hernia) of the caudal epigastric artery during the Valsalva maneuver [19]. This bulge is often symmetrical. There are a significant number of false-positives with impressive bulges but without groin pain. Ultrasound is thwarted in this diagnosis of sports hernia, which remains mainly a clinical diagnosis and may be the cause of long adductor tendinopathy [46].

3.7 Treatment

3.7.1 Conservative Treatment

3.7.1.1 Medical

Early diagnosis and management are crucial. Any adductor pain, even minor, must be supported with a rigorous full clinical examination to clarify the diagnosis and management, but in any case a few days' (5–7) rest is essential, combined with physiotherapy and mechanical treatments: stretching, slow speed eccentric load, and, optionally, NSAIDs. In our experience in professional soccer, this course of action is often sufficient to avoid complications and chronicity. The mistake not to make is to let the player continue with training and matches while on NSAIDs, because under these conditions the transition to chronicity is almost inevitable.

In chronic forms, treatment will be the same, but for a much longer period, emphasizing the eccentric loads at slow speed not only on the adductors, but also on abdominal wall and hip flexors owing to the entanglement of the anatomical structures. We believe that an isokinetic dynamometer is an indispensable tool for this type of treatment.

Oral administration of NSAIDs can be a useful adjuvant treatment. However, the corticosteroid injections should be avoided because of the risk of collagen disorganization and tendon rupture. They should be progressively replaced by PRP (platelet-rich plasma, plasma rich in growth factors [PRGF]) [74] techniques, whose results are notable as they are associated with the eccentric working protocol at a low speed on an isokinetic dynamometer [71].

3.7.2 Rehabilitation Protocol

The results of nonsurgical management are based on commitment to the proposed treatment by the athlete and long-term compliance. Rehabilitation is carried out in stages, with close medical supervision. The duration of a level depends on the ability of the athlete to perform the exercises without pain.

It has been proven several times that the use of active programs (unlike the passive physiotherapy) decreases recovery time and return to the game, produces the best long-term results and decreases the risk of recurrence. In particular, mention is made of interesting results (level 1 evidence) through programs combining flexibility exercises, in particular, adductor strengthening and/or abdominal muscles [19, 47].

The first randomized study showed a return to sport 7 months after the beginning of the treatment, to the previous level without pain, in 79 % of the athletes of the group that undertook "active exercises", compared with only 14 % in the group that had "passive physiotherapy" [48].

The rehabilitation program is codified into four stages, which vary in length from 8 to 12 weeks.

3.7.2.1 The First Phase

The goal of the first phase is pain reduction and the elimination of triggers. At least a relative rest from sports of varying duration is essential, in addition to strict refrain from movements that trigger pain.

The practitioner begins his work by normalizing muscle tension (osteopathy, contracting–released stretching, myotension and myofascial work) in painful areas, but also working remotely on pelvic balance, particularly the psoas. It continues with work on mobility of the hip and spine, Cyriax deep transverse massage (DTM) or administration of extracorporeal shock waves to the affected entheses.

From the 3rd day, the practice of manual eccentric loads to the adductor gradually improves tendon resistance by resisted adduction and hip flexion (adductors are accessory hip flexors). Owing to the entanglement of the anatomical structures, the same technique is used on the anterior and lateral abdominal sides to strengthen the fascias concerned. The return to the starting position is passive to avoid concentric action. The protocol implements the rules to determine its effectiveness: very slowly and with low intensity in the initial stages, these two parameters being gradually

intensified strictly according to absence of pain during and between sessions: the ROM very gradually integrating into the sector of maximum elongation [50, 71].

3.7.2.2 The Second Phase

The second phase involves strengthening the abdominal muscles (cladding) and the spinal and hip extensors the deficits of which are frequently associated.

Eccentric adductor work continues with intensification. Indeed, the eccentric muscle loads have been the basis of our treatment of tendon injuries since the work of Stanish et al. [51]. The tendon structure must also receive appropriate training to improve its mechanical and protect against microlesions resulting from external constraints.

The exercise incorporates the resilient elongation parameters of the musculotendinous unit (according to a defined ROM), speed of movement, and intensity of contraction. The selected joint displacement integrates with the maximum elongation sector (external course) as soon as possible.

To achieve this eccentric work, use of an isokinetic dynamometer has many advantages, as it enables quantified control of the resistance and speed, optimal contraction due to the auto-adaptive resistance, instant feedback, individualization of the workload, and control of the ROM.

The effectiveness of eccentric work in the treatment of tendon injuries has been regularly demonstrated over the last few decades [49, 50, 52] (Barthélémy et al. 2014).

If isokinetic equipment is the modality of choice for technique building, the eccentric rehabilitative exercises can be delivered through the use of different devices: manual application by the physiotherapist, use of direct charges or mobilized by ropes or chains, elastic resistance. Each category presents advantages and limitations regarding the availability and the cost of equipment, the efficiency and specificity of training, and patient safety. However, the management of the adductor longus tendinopathy need the implementation of this eccentric work protocol.

3.7.2.3 The Third Phase

The third phase includes the neuromuscular reprogramming form of muscle activation with pelvic stabilization exercises for the abdominal and lower limbs, first on balancing trays.

The objective of this phase is to develop a good lower abdominal muscle synergy between the pelvis and limbs and to improve muscle control and balance. During this phase we can use a work on the Klein ball, and the Pau-Toronto protocol [53]. Sports with many alternate unipedal movements benefit greatly from this phase. This stage continues on the ground, where the work will be more specific to the sport practiced: the movement is first to be made in the center and if no pain or when the 80 % threshold in a painless maximum eccentric contraction is reached they will become a real

preparation for the sporting gesture. The development of aerobic capacity and the maximal aerobic power and finally the support work (changes in direction, front-back race, shuffle) are part of the last part of this phase.

3.7.2.4 The Fourth Phase

The fourth and final phase is the continuation of specific movements for recovery, still without pain. This is a true program of progressive reintegration into the team. Monthly medical monitoring is necessary to address adductor longus fissural rebel tendinopathy. Platelet-rich plasma infiltration (PRP) could represent a therapeutic hope following the results of various in vitro and animal experiments [74]. The addition of eccentric loads in the aftermath of the injection of PRP improves the efficiency of this type of treatment [54].

3.7.3 Surgical Treatment

The surgical treatment consists of a tenotomy under general anesthesia. An incision of about 3 cm is made. After dissection of the superficial plans, the adductor longus tendon component is resected and pushed down (sometimes using pliers if an old calcific tendinopathy) until there is a soft area measuring 3–4 cm. The femoral fascia and the subcutaneous tissue are closed. The postoperative course is simple, with few bruising complications (2 %) and little infection (0.05 %) [72].

Adhesions occur in 15–25 % of cases, usually within a month or 3 months of surgery. After an adductor longus tenotomy, mobilization of the hip from the first attempt to lift by the patient is required, to prevent adhesions between the tendon and femoral fascia, which burden this surgery [55]. The patient needs to practice hip movements such as active abduction, flexion, extension, and rotation, avoiding active contraction of the adductors, which is painful at this stage.

The practice of administering manual eccentric loads to the adductor gradually facilitates the healing of tendons by collagen stimulation and prevents scar adhesions.

Rehabilitation takes place in several stages. Walking is allowed from the day after surgery if no or light pain. Static exercises can be considered as soon as possible within the first week. During the second and third weeks, a straight course is recommended: running very slow stopping when there is pain, with 1 or 2 days of rest between sessions. In parallel to this work, abdominal strengthening is gradually increased. During the fourth week, rehabilitation of the abdominal wall and groin is intensified with the implementation of a program of re-athletization.

The specific training for a return to sport is progressive and controlled, postponing ball-striking until around the 45th day.

A combination of ductal forms and chronic lesions of the adductor insertion is common [56]: 55 out of 80 soccer players according to Rifi et al. in 2009 [57]. In the experience of Reboul, in a retrospective series of 180 patients operated on for tendinopathy adductor, 80 % of patients had associated parietal failure requiring surgical treatment [31].

Surgical treatment is very effective for abdominal pathologies, which are often resistant to medical treatment. In the case of an abdominal pure form or a proven mixed form, surgery becomes necessary if rest and rehabilitation care for the duration of 2 months do not allow sports recovery [58].

The two most frequently used surgical techniques in athletes are [14]:

- Nesovic, the principle of which is a bilateral tensioning of the broad muscles of the abdomen by lowering of the inguinal ligament.
- Shouldice, administered to fight against the enlargement of the inguinal canal using a suture in three planes: the transverse fascia is retightened using an overcoat suture between the inguinal ligament and the conjoint tendon, closing the aponeurosis of the abdominal external oblique muscle.

The results obtained were good or very good in 80–90 % of cases.

A minimal repair technique exists: using endoscopy only, opening the transverse fascia and doubling the reverse fascia. The indication is only for a light deficiency of the abdominal posterior wall, with no pain of longus adductor insertion.

In our opinion, endoscopic single-shutter techniques with prosthetic reinforcements, which are practiced by visceral surgeons, do not have a place in athletes, because they do not meet the aim of rebalancing the constraints of the pubic symphysis.

3.8 Prevention

Adductor tendinopathy is very common in an athlete's life. It is the result of intrinsic factors such as postural dysfunction, hyperlordosis, a weakness of the abdominals. Extrinsic factors can also set off the tendinopathy. Indeed, overtraining is a strong element leading to stress. Furthermore, soft fields may be a factor.

Primary prevention is identifying subjects at risk: deficiency in the abdominal wall with dehiscence or Malgaigne's sign: oblique protrusion, ROM limitation of the hip, anteversion of the pelvis [59]. An improvement program with regard to these weak points will have to be made throughout the individual's career to avoid possible decompensation. Prevention of this consists of four forms of groin pain, not to mention the prevention of extrinsic factors including: the effective management of workloads, suitable muscle building, and avoiding abdominal work that involves significant increases in intra-abdominal pressure.

The frequency of groin pain has declined sharply among top athletes over the past 15 years, whereas it has increased in other disciplines and among amateur athletes [60]. This is undoubtedly because of prevention programs that are now in

place and systematically applied at the highest level. However, they must be reviewed regularly, as with any prevention program, by medical staff, technicians, and by athletes themselves, so that they do not reoffend. This is especially true as training and competition increase in frequency and intensity.

According to some studies, a significant recurrence rate has been reported in professional team sports: 38 and 44 % [11, 12], and 25–32 % of professional players suffer a recurrence during the season [61]. The prevention programs should take into account the training load, the morphotype, potential imbalances between the stabilizing muscles of the pelvis and hip, loss of ROM or abnormalities of the hip joint.

It is important to understand the function of the adductor complex; they mainly aid hip adduction and the thigh rotations (lateral or medial, according to the muscles and parts). Thus, it is essential to consider the rotator aspect of this muscular complex. We find this injury more frequently in collective sports such as soccer, basketball, handball, all requiring explosivity, acceleration, deceleration, and rotation. In soccer, the adductors participate in passing and shooting too. Here is a way of preventing adductor tendinopathy.

First of all, during the pre-season, some evaluations can be made to determine a player's profile. Postural tests and screening are fundamental [62]. The static posture is observed, then the dynamic posture through assessment using the National Academy of Sports Medicine's overhead squat test and the single-leg squat test. These tests enable us to determine any imbalances and compensations. Each dysfunction is linked to underactive muscles and overactive muscles. The Functional Movement Screen [63] can be used too. These kinds of tests provide information that can explain recurrent injuries or risks of injuries (i.e., the knee valgus and the risk of an ACL tear). Here are few examples of dysfunctions and imbalances that increase the risks of developing an adductor tendinopathy: hyperlordosis with weak core muscles (especially the external oblique muscle), a weak gluteus maximus, and a weak gluteus medius.

Second, there is the lower-crossed syndrome, showing the link with the weak gluteus, weak abdominals and tight hip flexors, and a tight erector spinae (causing the hyperlordosis). The relationship between the weakness of abdominals and the adductor tendinopathy is well known. During activity, the core plays important role in balancing the body, maintaining the pelvis (and forcing transmission, resisting contact…). If the core is weak, the body compensates by over-activating the peripheral muscles as adductors or hamstrings, and could lead to tendinopathy.

Third, mobility is a key in the prevention of any injury (especially in the groin area). We know that soccer players have a limited ROM of the hips owing to the particular role of the adductors [64]. Many mobility exercises and stretching postures exist to fight stiffness and to reduce the risk of developing inflammation.

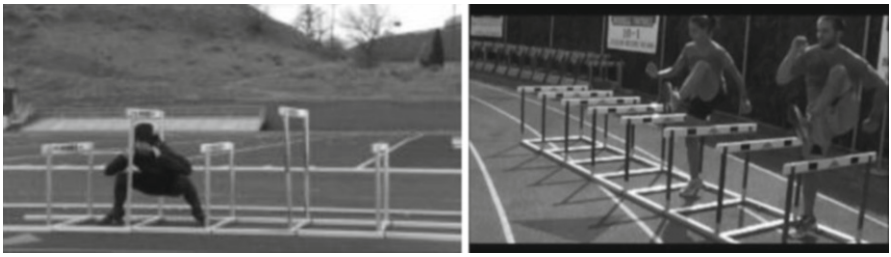
Fourth, proprioception should be included in the protocol of prevention for adductors. Indeed, the adductors are tonic, i.e., they contribute to maintaining the posture. They should be worked in any situation (including rotations) to reduce the risk of inflammation.

Strengthening is essential for prevention. There are two ways to prevent the adductor tendinopathy. The first one is by strengthening the adductor complex (brevis, magnus, longus, pectineus, and gracilis) and the hips flexor complex, both in eccentric mode [51, 65–68]. The manual work can be undertaken by physiotherapists. Bodyweight and machines also help to strengthen these muscles. We repeat

that the eccentric mode, which is used mainly for lengthening and stretching, should be reserved for these muscles. It should be added that training with an elastic band is not very interesting for the adductor complex because the resistance with bands exists mainly in the internal course (when the insertions get closer), whereas we look for the lengthening of the muscles. The second goal is global strengthening. The main idea here is to increase the strength of the gluteus and of the core to stabilize the pelvis and the trunk to release the over-tension of the adductor complex.

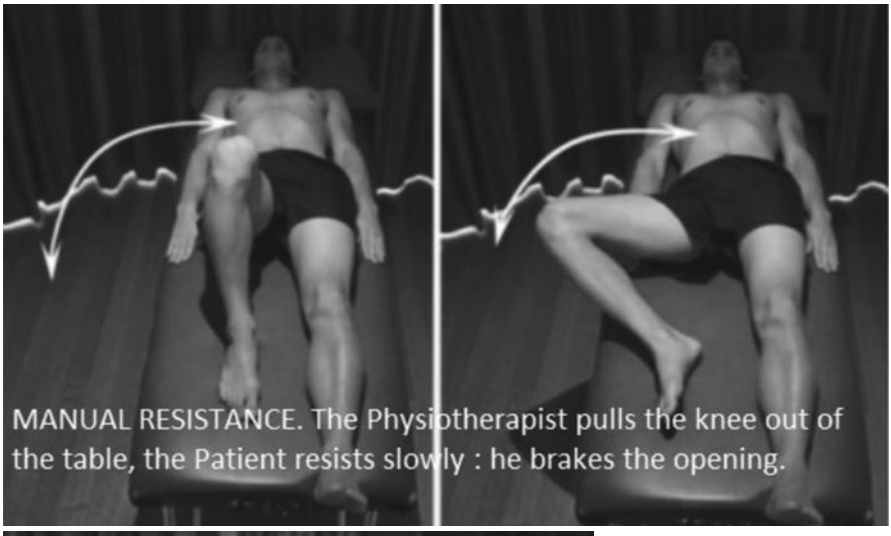
Last, the management of the training load is very useful for preventing all kinds of stress injuries, such as tendinopathies. Medical and technical staff must work together to define the limits and risks for each player. Many methods exist to control the training load through subjective tests (questions, rating of perceived exertion [RPE], feelings, sleep, fatigue, etc.), and/or objective tests such as heart rate variability [69, 70].

Here are some examples for preventing adductor tendinopathy:



Hip mobility

- Mobility: static deep squats, hurdle crossing:
  - Stretching, and myofascial release with a foam roller. For adductors, hip flexors, erector spinae (if hyperlordosis)
  - Eccentric strengthening of the adductors: lying with bent knees, lying with straight legs, seated; with manual resistance, with bodyweight through lateral lunges (using a slider pad), with machines
  - Eccentric strengthening of the hip flexors: with manual resistance, or bodyweight, or an additional load
  - Global strengthening of the adductors: sumo squat, sumo deadlift, split squat, lunges, step-ups
  - Strengthening of the gluteus maximus: with hip extension, hip thrusts, or through global movements such as deep squats or as a deadlift. Strengthening of the gluteus medius: lying on the side and hip abduction, lateral movements with an elastic band over the knees as lateral steps, or as lateral lunges, or even global movements described above, with an elastic band
- Core training: front and lateral planks and all variations (static or dynamic; with med-ball or physio-ball). Specific work on the oblique muscle can be added.
- Proprioception: single-leg stance with many situations, surfaces, static or dynamic, closed or open-eyed, manipulation of tools, rotations, etc.

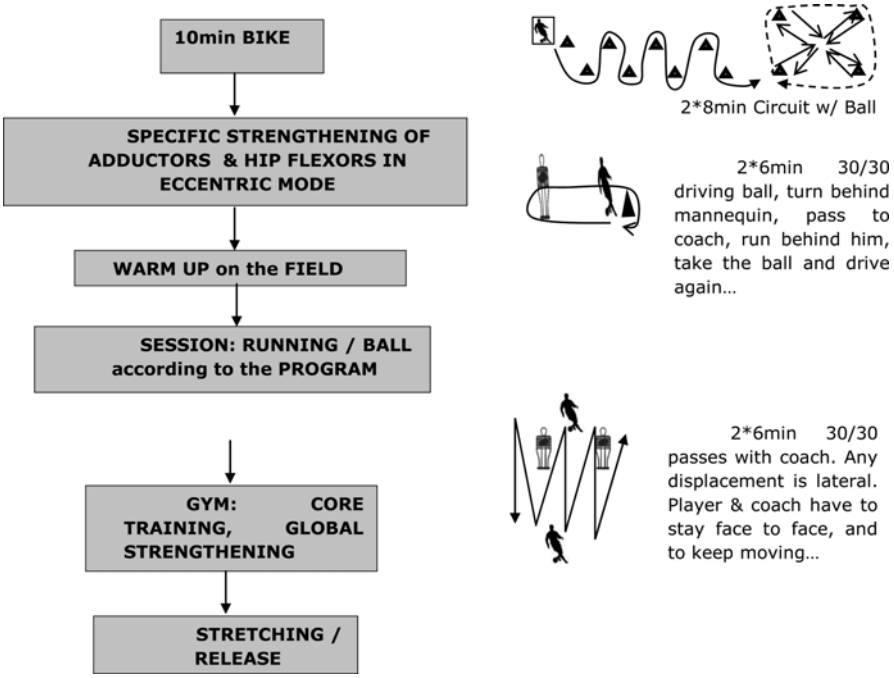


Let us have a look now at an example of rehabilitation for this kind of injury. We talk about conditioning and the ball parts. We add treatment and strengthening before and/or after these sessions with exercises as explained previously. Here is a proposed a session: players warm up on a bicycle, then perform a specific protocol for the adductor

complex (manual exercises with physiotherapists, or on a isokinetic dynamometer, or a machine). After that, they warm up on a field with skills, before starting the field program that follows. The last part of the session is in the gym with global strengthening, a core work-out, stretching or self-myofascial release. After a number of sessions of eccentric work, the players are able to start running. Players first run for progressively longer periods: 10 min, 2 × 10 min, 2 × 15 min. Then, we introduce accelerations; progressively, day after day, the players have to accelerate within the width of the box, then within the width of the field, and then within the length of the field. We introduce the ball during that period, with easy technical workouts first. Day after day, players perform circuits with more or less intensity. The next step is the intensity: players carry out interval training (2 × 8 min, 10 s at maximal aerobic velocity/20 s passive recovery). In the same period, the ball workouts gain in importance, and are more intensive for conditioning and muscles. In the field program, we mean running and ball workouts, and the players have to realize in all situations that they live in a game: running at different speeds, with changes in direction, rotations, accelerations/decelerations, driving the ball, passes, contacts, jumps, etc. At any time during the protocol, we control the training load and adjust the sessions if necessary.

3.9 Synthesis and Therapeutic Indications

Inguinal pain is very common in sport, but among many diagnoses, adductor insertion tendinopathy is one of the four most common entities either in the associated or isolated form. Our purpose was to detail clinical, etiological, and therapeutic plans, while providing the criteria by which to eliminate other pathological conditions. Under



these conditions it is essential to establish as soon as possible a specific positive diagnosis. Early consultation is crucial; it is easy in professional clubs with a medical staff present, but it is more difficult for amateur athletes who may be extremely hampered in their physical activities or in everyday life to consult a physician.

3.9.1 Subject Seen Early: Acute Mode

- Confirm the diagnosis of adductor longus enthesopathy by pain at palpation, adductor stretching, and painful adduction resistance
- No pain at the hip flexors and trunk to eliminate rectus abdominis tendinopathy or athletic pubalgia with in this case a pain when coughing after effort and a painful inguinal ring that expands on impulsive coughing
- No limitation of hip amplitudes for eliminating arthropathy
- No pain on FADIR to eliminate FAI

Treatment consists of rest, bicycle riding, manual eccentric movements in the internal course without pain during and after increasing the intensity, ROM, and speed gradually one single criterion at a time. When the external position is reached, painless recovery on the ground is possible.

3.9.2 Subject Seen Late: Chronic Mode

Confirmation of the diagnosis is much more difficult because of the spread of pain due to the chronicity and anatomical intricacy of the neighboring structures. At first, the pathological hip condition should be eliminated.

Clinically, pain includes:

- Palpation of the adductor longus insertion
- Lodge adductor roughly contracted
- Limited and painful lodge stretching (FABER)
- Resisting adduction
- Resisting hip flexion
- Resisted trunk flexion
- Palpation of the rectus abdominis insertion on the pubis
- Testing of the contralateral adductor

but the absence of impulsivity to affirm the isolated form. Although this is rare at this stage, the frequency of the association between chronic enthesopathy of the adductor and the weakness of the posterior wall is very high [31].

Treatment is associated with NSAIDs, stretching, and eccentric loads of slow speed limiting the ROM in a nonpainful area, low intensity, gradually increasing the criteria and respecting the rule of indolence, using the same protocol on hip flexors and trunk and rotators, manually then with an isokinetic dynamometer to follow a

numerical progression of evolution and allow the resumption of running with objective criteria. Ultrasound and MRI clarify the degree of tendon damage and eliminate or not the associated forms. Two injections of PRP at 3-week intervals associated with eccentric submaximal work may allow complete pain relief in less than 2 months. After resuming racing, the increase in physical activity is highly codified. The collaboration among physician, physiotherapist, and medical trainer is essential for the full recovery of the player.

In the case of failure: persistent pain after 2 months despite well performed treatment and in the case of a deficit of wall association, surgery is programmed with only a superficial tenotomy [14, 58, 73] or associated with abdominal wall strengthening using the Shouldice technique.

3.10 Conclusion

Groin tendinopathy is a difficult disease requiring good clinical knowledge. It is part of one of the four groin syndromes of sportsmen: adductor enthesopathy, rectus abdominis enthesopathy, posterior abdominal wall weakness, and pubic osteoarthropathy. It may be isolated, but is often associated with other clinical forms. The diagnosis should specify the various forms of damage so that treatment is appropriate: conservative and medical in isolated forms, often surgical in associated forms after the failure of medical treatment within 2–3 months, especially when treatment was delayed. The identification of subjects at risk is crucial in professional sports, but impractical in amateur sports. Information in sporting circles and in the general public on the importance of early medical consultation in this type of pathological condition would be a very good thing.

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Chapter 4
Rectus Femoris Tendinopathy

Stefano Dragoni and Andrea Bernetti

Abstract Rectus femoris tendinopathies are not particularly frequent occurrences in sports traumatology.

It is possible to observe their presence in sports activities requiring strong, repeated, and eccentric loading with maximal lengthening in lower limbs such as during sprinting and kicking.

The symptoms show a gradual and progressive onset of pain and discomfort in the anterior aspect of the hip and are typically aggravated by exercise especially during jumping and running.

Patient history and clinical evaluation, including direct palpation of the tendon and resisted muscle activation, are essential to make the appropriate diagnosis.

Imaging is helpful in confirming diagnosis. Ultrasound and magnetic resonance provide valuable diagnostic information, useful for planning therapeutic strategies.

Conservative treatment includes oral and topical anti-inflammatory drugs and physical therapy with a wide variety of modalities available.

Surgical treatment is unnecessary in most cases; surgery may be a valuable option in selected patients who have failed 3–6 months of conservative treatment or in painful calcific tendinopathy.

4.1 Introduction

Overuse disorders of tendons, bones, and joints represent a widespread problem in sport and a challenge to sports physicians. Achilles, patellar, and supraspinatus tendons are often site of overuse injuries, while rectus femoris (RF) tendinopathies are not so often observed in the clinical practice of sport medicine [1].

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Tendinopathy is an umbrella term that indicates that there is a non-rupture injury in the tendon or paratendon, which is exacerbated by mechanical loading and may lead to long-lasting disability [2].

In athletes, rectus femoris muscle/tendon acute injuries have a high incidence; indeed they are second only to the hamstrings tear among lower extremity acute injuries.

Rectus femoris acute injuries are common in soccer players because the athletic kicking motion involves forceful eccentric contraction while passively stretched at the onset of the forward swing phase.

The strong eccentric loading with maximal lengthening during sprinting and kicking predisposes the rectus femoris to injury. Although proximal injuries are mainly myotendinous or myoaponeurotic, proximal tendon avulsions or ruptures may also be seen, involving direct, indirect, or both tendons. Indirect tendon complete or partial tears have been reported to be more frequent than direct and conjoined tendon injuries. It has been proposed that proximal rectus femoris insertional injuries may progress from the indirect to the direct head and then to the conjoined tendon [3, 4].

The same mechanism of acute injury in a skeletally immature athlete could result in an avulsion fracture of the anterior inferior iliac spine (AIIS) at the origin of the direct head, owing to the vulnerability of the open apophysis, while the same gesture in adults, performed with high intensity for repeated time, may lead to tendinopathy [5].

4.2 Anatomy and Functional Anatomy

The rectus femoris is the most superficial and the only biarticular muscle of the quadriceps group. Its proximal insertion is complex, with two main proximal origins.

The direct or straight tendon is located at the anterior hip, arising from the anterior inferior iliac spine (AIIS) and forming the anterior aponeurosis.

The indirect or reflected tendon is located at the anterolateral hip, arising from the superior acetabular ridge and posterior coxofemoral capsule, and forms the deep central aponeurosis, extending up to the lower third of the muscle. The two heads unite at an acute angle and form the conjoined tendon around 2 cm distal to their origin (Fig. 4.1). A third and inconsistent head may arise from the inferior edge of the indirect tendon, attached superficially to the gluteus minimus muscle and deeply to the lateral iliofemoral ligament [6–8].

Sometimes, between the straight tendon and AIIS, there can be, although not constantly, the interposition of a bursa.

The tendon, devoid of synovial sheath and covered by peritendon, presents morphological and structural characteristics comparable to those of similar anatomical structures as the Achilles or quadriceps tendon.

Usually the integration of tendon into the bone occurs at a specialized interface known as *enthesis*, through a multitissue interface [9].

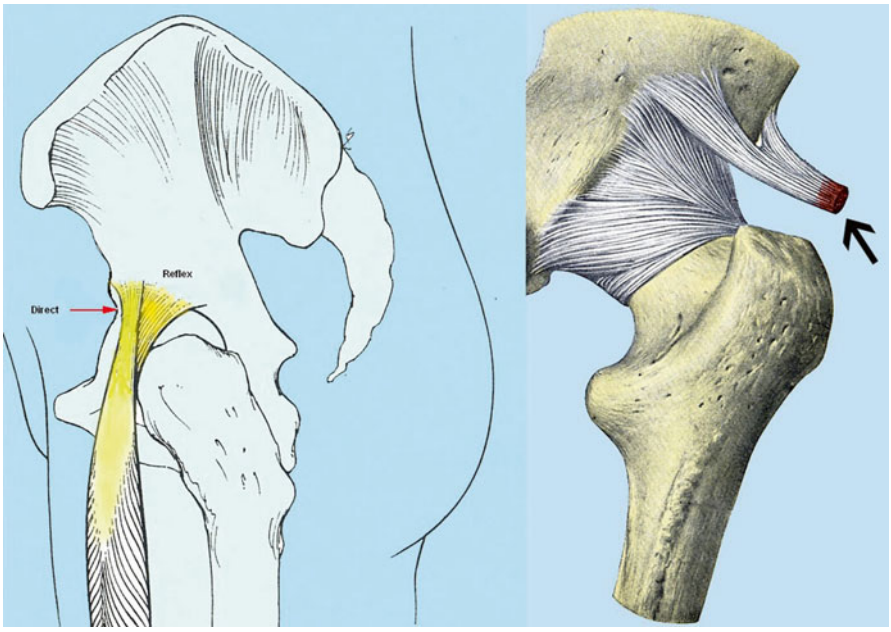

Fig. 4.1 On the left, direct and reflex tendon of rectus femoris; on the right, conjoined tendon (*black arrow*)

Tendons and ligaments may attach to the bone via either fibrous or fibrocartilaginous insertions which are more common than fibrous.

The straight tendon of rectus femoris muscle belongs in its junctional site to the fibrocartilaginous entheses with four transitional layers from the bone to tendon; the first zone, distal to bone-tendon junction, consists of tendon and is populated by fibroblasts; the second zone consists of uncalcified fibrocartilage which is populated by fibrochondrocytes; the third is composed of mineralized fibrocartilage; and the fourth consists of bone [10].

Concerning functional aspects, electrophysiological studies have shown that the rectus femoris muscle functions independently from the vastus muscles during walking. It extends the knee, flexes strongly the hip, and stabilizes the pelvis on the femur during weightbearing. These anatomical specificities, together with the strong eccentric loading with maximal lengthening during sprinting and kicking, may predispose the rectus femoris to acute and overuse injuries as mentioned before [3, 11, 12].

4.3 Pathophysiology

The classic description of the tensile load-bearing region of tendon includes three main components: (1) type I collagen fibers which are predominantly longitudinally oriented, (2) a well-hydrated, non-collagenous extracellular matrix (rich in glycosaminoglycans), (3) and cells. The predominant cell population in healthy tendon is traditionally categorized as collagen-producing fibroblasts, responsible for the synthesis of the collagen fibers and extracellular matrix. In addition to the primary load-bearing part of the tendon, there is an extensive network of septae (endotenon) where the nerves and few small vessels are mainly located [13].

The main cause of tendinopathies is overuse, as the result of excessive, repetitive, or improper overloads to the tendons, bones, and joints.

The causes that could lead to a tendinopathy are many, involving biological, anatomical, and mechanical factors; frequent actions involving quick accelerations and decelerations or eccentric activities are related to these pathologies [14].

Furthermore, imbalances between strength and flexibility around certain joints, training errors, faulty technique, and incorrect equipment may predispose to injury.

Structural damage to the tendon occurs from repetitive strain and loading from either endurance or skill activities that require technique and power. In the rectus femoris tendon, overuse acts usually through kicking and explosive movements as in sprint starts.

Each patient can present with a unique cluster of risk factors that may be relevant in the development of a tendinopathy and for the clinician to decide which to emphasize in assessment and treatment.

Extrinsic factors include excessive volume, magnitude, or speed of loading; training errors such as poor equipment and abrupt or acute changes in amount or type of load (e.g., sudden change to a different shoe type); environmental conditions such as temperature (e.g., cold weather, which makes the tendon stiffer); and ground conditions.

Intrinsic factors include individual biomechanics (malalignments, muscle weakness or imbalance, decreased flexibility), age, and adiposity.

In early-stage disease, inflammation has been demonstrated in peritendinous tissues but is unclear if the inflammation is associated with the etiology of tendinopathy; in end-stage disease, classic inflammatory changes are not frequently seen in tendon pathology in humans [15].

Histologic descriptions of tendinopathies demonstrate degenerative changes and disorganized arrangement of collagen fibers and an increase in vascularity [16].

In the insertional tendinopathies, there are often areas of focal fatty, mucoid, cystic, and hyaline degeneration, calcification, and a change in collagen fiber structure with the loss of the normal parallel bundles [17].

Patients with tendinopathy display a tendon thickening but a reduced energy-storing capacity, meaning that for the same load, their tendons exhibit higher strains than those of healthy individuals.

Furthermore, once symptoms and pain develop, ensuing movement dysfunction may contribute to the chronicity of symptoms. In particular, tendon pain causes

widespread motor inhibition in the affected region, evidenced by decreased muscular activity as assessed with electromyography. Individuals with tendinopathy also tend to use movement patterns that place excessive or abnormal load on their tendons; the faulty movement may represent either a root cause or a reason for chronicity or slow resolution [18].

In some cases of chronic tendinopathy, a calcific tendinitis may occur near the insertion of the tendon.

The exact etiology of the origin and of resorption of the calcium deposition is uncertain. Acute calcific tendinitis is largely idiopathic, but some reports suggest that repeated or local trauma is a cause of the calcium deposition, while Uhthoff et al. suggested local hypoxia due to mechanical or vascular change may be the cause. Hypoxia leads to degeneration of tendon substance into fibrocartilage and subsequent calcification, mediated by chondrocyte. The angiogenetic response would allow eventual resorption of the deposit by macrophages, which restores normal perfusion and oxygen tension to the tissue. After resorption of the calcium, the tendon probably regains its original architecture by synthesis of new matrix [19].

Although calcific tendinopathy can occur at the insertion of any tendon, it most commonly affects the shoulder around rotator cuff, Achilles tendon, or patellar tendon. Generally, in the hip area, the calcifications are commonly found in the region of the gluteal tuberosity involving the tendons of the hip abductors. Calcific deposit in the rectus femoris origin is a rare disease first recognized in 1967 by King and Vanderpool, and in recent years, only few cases were reported [20].

Most of the published reports regarding calcific tendinitis of the RF describe involvement of the reflected head; compared to the direct head, calcific tendinitis of the reflected head is more likely to produce an acute episode because of the proximity to the hip capsule. An acute pain exacerbation caused by rupture of calcific material into the joint could be a further eventuality [21].

4.4 Clinical Aspects

RF tendinopathy symptoms include a gradual and progressive onset of pain and tenderness at the anterior aspect of the hip during sport activity which may be reproduced when attempting a straight leg raise or trying to lift the knee up against resistance. Pain and stiffness may be worst in the morning or after periods of rest.

A thorough clinical history and specific physical examination are essential to make the appropriate diagnosis and facilitate a specific treatment plan.

At history, there is often a change in training routine as an increase in mileage, a different training surface, an inadequate warm-up, or stretching program.

Subjectively, the athlete complains of pain when he starts exercising, and typically, the pain is aggravated by exercise, especially during warming up, jumping, and running.

In the cases of acute calcific tendinitis, clinical manifestations include severe localized pain, swelling, tenderness, and loss of function.

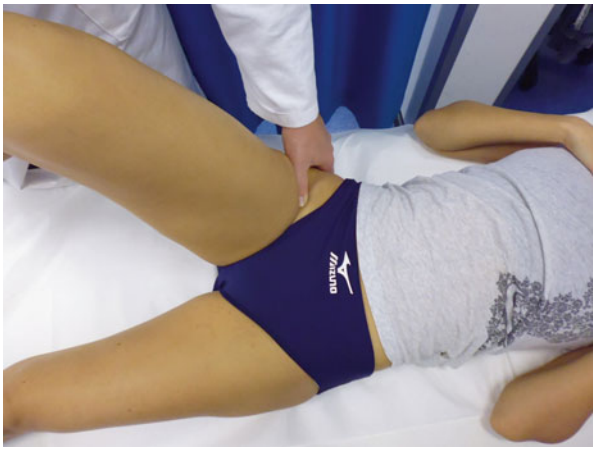

Fig. 4.2 Palpation of the tendon with the hip flexed

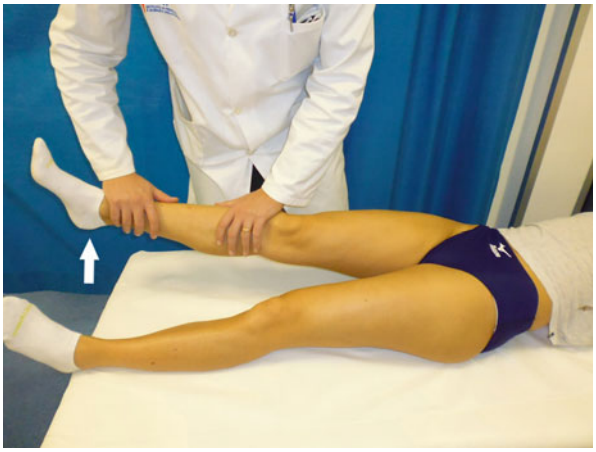

Fig. 4.3 Strength test of the rectus femoris with extended knee

After collecting accurately the clinical history, during physical examination, pain is usually felt by the patient during resisted muscle activation, passive stretching, and direct palpation over the tendon.

Typically, pain could be elicited by palpation and pressure on the tendon: the athlete lies on his back, the examiner is positioned by the affected side bringing the athlete's hip flexed to 90° passively with the knee flexed, and then he pushes on the tendon using as landmark the AIIS (Fig. 4.2).

Strength testing of the rectus femoris should include resistance of hip flexion both with extended and flexed knee (Figs. 4.3 and 4.4). Furthermore, adequate strength testing of the rectus femoris must include resisted knee extension with the hip flexed and extended (Fig. 4.5).

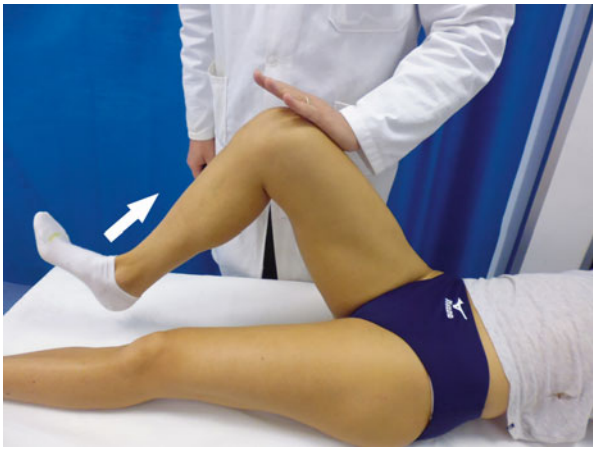

Fig. 4.4 Strength test of the rectus femoris with flexed knee

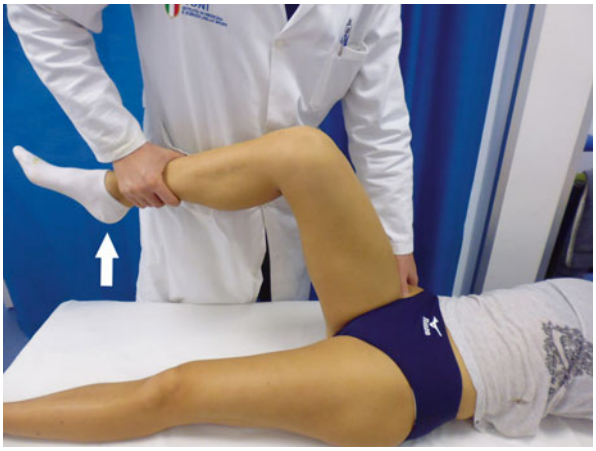

Fig. 4.5 Resisted knee extension with the hip flexed

4.5 Differential Diagnosis

The differential diagnoses are many and often difficult to reach; they must be placed with hip injuries as acute hip joint arthritis, periarticular infections, labral tears, degenerative changes and chondral lesions of acetabulum, stress fractures of femoral neck, and femoroacetabular impingement. During the diagnostic process, another cause of pain which must be kept in mind is the snapping hip. It is a syndrome characterized by pain and recurrent clicks or snaps during active motion of the hip. Three kinds of snapping hips are known: an intraarticular variety (attributable to loose bodies, labral lesions, chondromatosis, joint instability, or ruptured

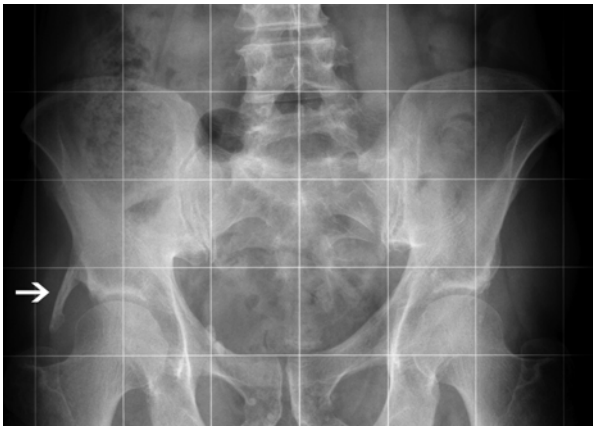

Fig. 4.6 X-ray examination of the pelvis; calcific tendinitis of rectus femoris (*white arrow*)

ligamentum teres), an external extraarticular variety (attributable to subluxation of the posterior aspect of the iliotibial band or the anterior border of the gluteus maximus over the greater trochanter), and an internal extraarticular variety (attributable to subluxation of the iliopsoas or direct RF tendon).

Even if calcific tendinitis of RF direct head is extremely rare, it can impinge against the overlying iliac muscle, resulting in a painful coxa saltans; other pathologic changes involving this tendon could result in similar thickening, potentially predisposing the development of an internal snapping hip [22].

An adequate history and physical examination are the first and the most important steps for a specific diagnosis. In cases of diagnostic uncertainty, advanced imaging techniques such as X-ray examination, ultrasound, or magnetic resonance imaging (MRI) are commonly utilized to assist in diagnosing these diseases.

4.6 Imaging

X-ray examination has only a limited role to play in the diagnosis of rectus femoris abnormalities; AP and slightly oblique view of the pelvis are able to show calcific tendinitis and the presence of major osseous or articular abnormalities (Fig. 4.6).

Tendinopathies can be easily studied with ultrasound and magnetic resonance (MR) imaging, both providing good anatomical details of the tendon and its alterations.

Ultrasonography has been shown to be a noninvasive imaging technique accurate and sensitive in diagnosing tendon injuries in the groin region. It has the advantage of being fast, inexpensive, and widely available, and it can be performed during dynamic tests. Normal findings are readily distinguished from pathological ones providing valuable diagnostic information in association with clinical evaluation, such as location and extent of the injury.

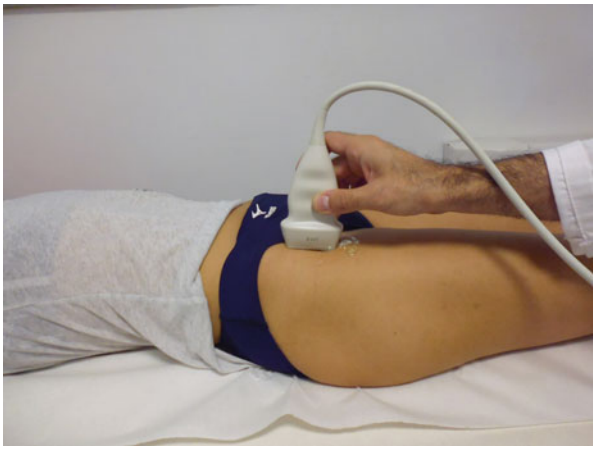

Fig. 4.7 The probe is placed longitudinally on the anterior aspect of the hip

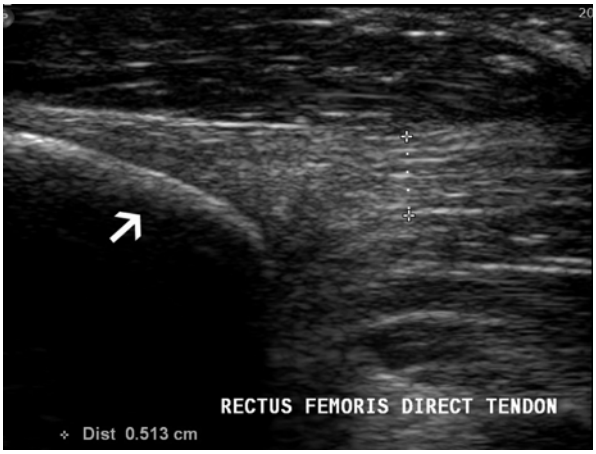

Fig. 4.8 Normal ultrasound longitudinal scan of rectus femoris tendon with a thickness of 0.51 cm at about 10 mm below the anterior inferior iliac spine (*white arrow*)

Rectus femoris direct tendon can be easily appreciated with high frequency transducers using sagittal scans performed taking as anatomical landmark (the AIIS) which can be readily identified by palpation (Fig. 4.7). The transducer is then placed over the anterior aspect of the hip on the axial plane and moved distally for 4–5 cm to visualize the muscle-tendon junction. The direct tendon is then examined with longitudinal scans moving the probe from medially to laterally providing an excellent morphological and structural visualization. In sagittal view the direct tendon is cylindrical with smooth borders, a thickness of about 0.5 cm, and an internal structure homogeneously hyperechoic (Fig. 4.8). On axial scans, the tendon appears as a more or less oval-shaped morphology, located over the AIIS (Fig. 4.9). Sagittal images

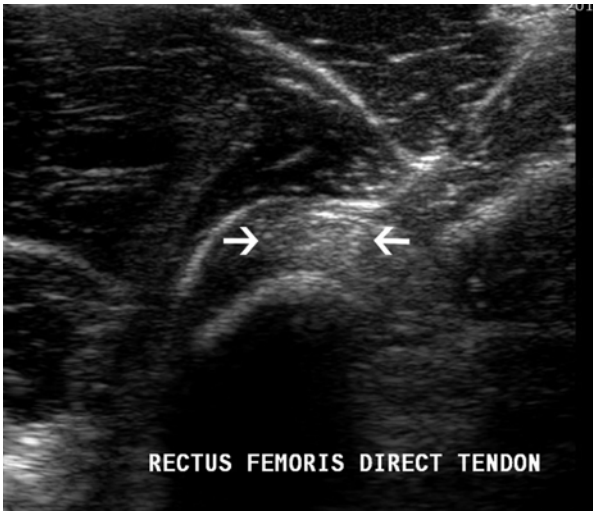

Fig. 4.9 Normal ultrasound axial scan of the tendon (*white arrows*) over the AIIS

allow complete exploration of the direct tendon from its origin at the AIIS to the myoaponeurotic junction. Moving the transducer laterally, the indirect tendon that follows an oblique course appears as a hypoechoic band descending toward the upper rim of the acetabulum, which is best visualized, on oblique axial images [23, 24].

It is known that the pathological modifications caused by overuse on the tendons are represented by changes in the morphology and structure. Pathologic changes may include an irregular, diffuse, or circumscribed fusiform thickening, with or without marked alterations of its structure as the loss of normal fibrillary texture and the presence of variable shape heterogeneously decreased echogenicity areas, corresponding to focal fatty, mucoid, cystic, and hyaline degeneration (Fig. 4.10).

Calcification of the tendon appears as hyperechoic formation of different sizes and shapes with posterior attenuation of the ultrasound beam.

MRI can obtain a tridimensional evaluation imaging, to visualize pathologic conditions of the tendon, clearly depicting all pelvic, hip, and groin changes. MRI is able to provide high intrinsic tissue contrast, which permits the distinction between normal and abnormal tendons, and its high spatial resolution permits detailed anatomic structures to be identified. MRI has superb resolution of the soft tissue structures, does not utilize ionizing radiations, and can aid to diagnose a variety of tendon disorders, representing the investigation of choice in all tendinopathies.

Normal tendons, ligaments, and fibrocartilage have low signal intensity in all sequences.

On the other hand, tendinopathy is characterized by thickening and an increased intrasubstance signal on T2-weighted imaging sequences, which is often the first sign of tendon abnormality. The high–intermediate signal intensity within the

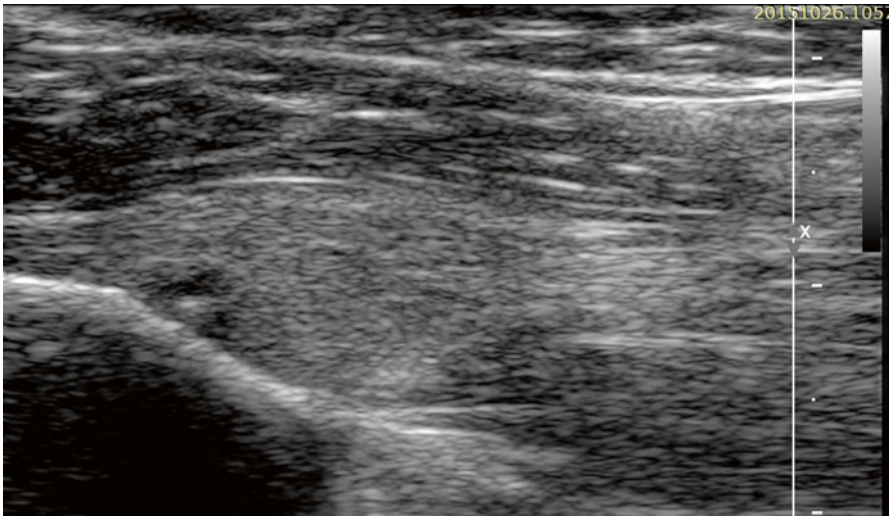

Fig. 4.10 Longitudinal ultrasound scan in rectus femoris tendinopathy; the tendon is thicker with changes in its structure

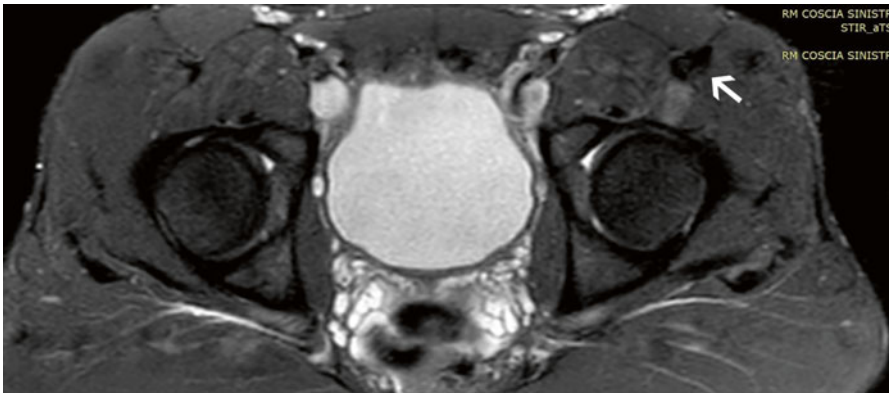

Fig. 4.11 Axial STIR MRI view of the pelvis; the tendon is thicker with minimal internal changes in its structure

substance of the pathologic tendon can be differentiated from fluid, as it appears brighter on T2 sequences.

MRI findings of tendinosis consist of foci of mildly increased signals within the tendon, often longitudinally oriented (Fig. 4.11).

In calcific tendinopathies, MRI shows on conventional spin echo (SE) T1- and T2-weighted images, lower signal intensity in the tendon; however, the calcifications are not always easy to detect with MR imaging.

The most valuable imaging planes are sagittal and axial along the course of the tendon. The coronal images are used when additional information is needed, for

example, to see changes at the myotendinous junction and/or at the bone-tendon junction.

It is recommended to acquire images in three planes with the appropriate field of view to include the tendon, the myotendinous junction, and the AIIS proximally.

The use of intravenous contrast is usually not part of the routine examination protocol of rectus femoris tendon as its use adds little in the identification of the lesion.

4.7 Conservative Treatment

Rectus femoris tendinopathy treatment is focused, in early stages, on pain and inflammation reduction, functional restoration, and prevention of reinjuries. Reported treatment strategies for tendinitis vary and mainly comprise conservative management.

Oral nonsteroidal anti-inflammatory drugs (NSAIDs) have been used extensively for decades to treat pain associated with tendon overuse injuries. More recently, the local administration of NSAIDs through gels or patches has been advocated. The evidences suggest both oral and local NSAIDS are effective in relieving the pain associated with tendinopathy in the short term of treatment (7–14 days) [25].

Not surprisingly, the patients who presented with a longer duration and greater severity of symptoms are more likely to have a poor response to both topical and oral NSAIDs.

In addition, long-term NSAID use increases the risk of gastrointestinal, cardiovascular, and renal complications associated with these medications. Overall, a short course of NSAIDs appears a reasonable option for the treatment of acute pain associated with tendon overuse. There is no clear evidence that NSAIDs are effective in the treatment of chronic tendinopathy in the long term.

The therapeutic protocol of rectus femoris tendinopathies should include physical therapy with a wide variety of modalities available in order to draw up a rehabilitation protocol.

First of all, cryotherapy provides acute relief of pain and its use is widely accepted. Repeated applications of melting ice water through a wet towel for 10-min periods are generally effective [26].

Concerning eccentric strengthening programs, they are widely used in rehabilitation; however, this kind of contraction could have a negative effect on the muscle as it is well known that eccentric exercise could cause greater muscle damages to patients than concentric exercise [27]. From these points of views, eccentric exercise should be introduced carefully in the rehabilitation protocols especially in the acute phases and could be performed under supervision at least in the first stages.

Otherwise, the use of manual therapy resulted in an improvement in pain scores and strength. Transverse friction massage has also been used to treat tendinopathy even if a Cochrane review evaluating deep friction massage found no benefit with deep friction massage versus other treatments [28].

Fig. 4.12 Kinesiotaping in flexed hip (*left*) and in extended hip and knee (*right*)

In some cases a quite new form of tape, called kinesiotaping, could be used. Kinesiotaping has been proved to be effective in various musculoskeletal conditions. Although its precise working mechanism has yet to be fully understood, it is believed to interact with neuromuscular function through mechanoreceptor activation. Kinesiotaping may represent an effective adjunct therapy in the physical rehabilitation program [29] (Fig. 4.12).

Furthermore, other treatment modalities can be applied with good results.

Low-level laser treatment (LLLT) has been studied extensively with mixed results. Some studies showed improvement, treating tendinopathy with LLLT compared to placebo LLLT [30].

Iontophoresis and phonophoresis are noninvasive therapeutic modalities using, respectively, ionizing current and ultrasound, to enhance the delivery of topically applied drugs through the skin; corticosteroids or NSAIDs are commonly used with these modalities to treat tendinopathy, but there is a lack of evidence on their efficacy.

Hyperthermia too has been used in the treatment of tendinopathy but not in the acute phases. This modality involves deep heating machines that combine a superficial cooling system with a microwave-powered heating system. This can increase the temperature of target tissues of approximately 4 °C without damaging the skin. Presumably, this increased temperature results in increased blood flow and subsequent healing to the damaged area. A randomized clinical trial has been published evaluating hyperthermia compared to therapeutic ultrasound in the treatment of tendinopathy demonstrating improvements in pain and patient satisfaction in the hyperthermia group compared to the ultrasound group [31].

Therapeutic ultrasound can be used in the treatment of tendinopathies. A systematic review of physical therapy modalities used for the treatment of shoulder pain suggested ultrasound appeared effective for the treatment of calcific tendonitis [32].

Even though the calcific tendinitis of the rectus femoris is rare and often it may be a self-limited disease, some of the patients do need treatment.

Some authors have reported the use of extracorporeal shock wave therapy (ESWT) with satisfying results [33].

Extracorporeal shock wave therapy has been advocated for treating several soft tissue conditions, including plantar fasciitis, lateral epicondylitis, and calcific and noncalcific tendonitis of the supraspinatus and Achilles tendon.

There is also evidence that tenocytes release growth factors in response to ESWT that may promote tendon healing. Chen et al. reported administering shock waves to a rat Achilles tendinopathy model resulted in increased tenocyte proliferation and increased expression of transforming growth factor-beta 1 and insulin growth factor 1 [34].

Improved efficacy has been demonstrated when using computer-guided navigation in applying shock waves to calcific tendonitis [35].

Eventually, in refracted cases, local steroid injections under computed tomography or fluoroscopic guidance are promising treatment. Local steroid and anesthetic injections are rapid and provide long-standing pain relief and a shortened clinical course, even if the mechanism involved is uncertain.

Eighty milligrams of methylprednisolone in combination with 2 ml of 0.5 % bupivacaine [36] or 40 mg of methylprednisolone with 1.5 cc of lidocaine has been used [37].

Other substances that can be administered by injection in the management of tendinopathies are represented by hyaluronic acid, platelet-rich plasma (PRP), and mesenchymal stem cells (MSC). Regarding the hyaluronic acid, a recent study shows how it could stimulate the synthesis of collagen type I [38]; furthermore Kumai et al. demonstrated how a single injection of high-molecular-weight hyaluronic acid in patients with enthesopathies (lateral epicondylitis, patellar tendinopathy, insertional Achilles tendinopathy, and plantar fasciitis) could be clinically effective [39].

A recent research shows how peritendinous injections of hyaluronic acid could also be used after a single steroid injection [40].

For what concern PRP, several studies underlined its use in tendinopathies. Devenport et al. demonstrate how ultrasound-guided intratendinous injections with PRP could be useful for treatment of proximal hamstring tendinopathy [41].

Furthermore, the majority of preclinical studies in literature show that PRP stimulates the tendon's healing process [42].

Mesenchymal stem cell therapy is a new regenerative approach for treating tendinopathy. For example, treatment of tennis elbow patients with single injection of mesenchymal stem cell showed a significant improvement in short- to medium-term follow-up [43].

In the future, such growth factor- and/or stem cell-based injection therapy can be developed as an alternative conservative treatment for patients with other kinds of tendinopathy such the rectus femoris one, especially in patients that have failed nonoperative treatment before surgical intervention is taken.

Currently, the challenge lies in conducting randomized, controlled clinical trials to determine the essential qualities of these technologies.

4.8 Surgical Treatment

Nowadays in the scientific literature, there are no significant indications on the surgical treatment of RF straight tendon, contrary to what happens to other forms of tendinopathy such as the tendinopathies of the supraspinatus or of the Achilles tendon.

In most cases RF tendinopathy is usually a self-limiting condition, responding well to conservative measures; the majority of patients find adequate relief from conservative treatment, and for this reason, it is not necessary to undergo a surgical intervention. Surgical management could be an effective option in carefully selected patients who have failed 3–6 months of conservative therapy.

Only in the cases of complete rupture of the tendon, surgical repair should be proposed even if there is no definitive treatment protocol to recommend; in fact there are few data published in the literature.

In these cases the tendon stump is inserted via a bony trough prepared in the AIIS, through an anterior (Smith-Petersen) approach, and the sutures are tied over bony tunnels [44].

In painful calcific tendinopathies, arthroscopic excision of tendon calcification yields satisfying results with few risks to the patient, rapid recovery, and satisfactory short-term outcomes.

As stated by Zini et al., it can be removed using a 5.5 mm bur through two standard portals (anterolateral and mid-anterior), after achieving the complete exposure of the calcification [45].

In refractory cases or when a large bone formation occurs after traumatic injuries (i.e., avulsion fracture of AIIS), a surgical excision of the calcification may be necessary, traditionally performed through an anterior approach.

Furthermore, hip arthroscopy gives the chance to treat this pathology providing the opportunity to address concomitant lesions, such as labral tear, femoroacetabular impingement, and cartilage lesions with minimal damage of the soft tissues surrounding the hip area [46].

4.9 Conclusions

Overuse disorders of tendons, bones, and joints represent a widespread problem in sport and a challenge to sports physicians. Even if rectus femoris tendinopathies are not so often observed in the clinical practice of sport medicine, sometimes the strong eccentric loading, like during sprinting and kicking, could lead to a tendinopathy.

Athletes with rectus femoris tendinopathy could refer a gradual and progressive onset of pain and tenderness at the anterior aspect of the hip during sport activity. Pain may be reproduced when attempting a straight leg raise or trying to lift the knee up against resistance. Pain and stiffness may be worse first thing in the morning or after periods of rest.

A thorough clinical history and specific physical examination are essential to make the appropriate diagnosis and facilitate a specific treatment plan. Rectus femoris direct tendon can be easily appreciated with ultrasound using sagittal scans performed taking as anatomical landmark (the AIIS) that can be readily identified by palpation. Chronic conditions could lead to a calcific tendinitis; in this case, clinical manifestations include severe localized pain, swelling, tenderness, and loss of function. In calcific tendinopathies MRI shows on conventional spin echo (SE) T1- and T2-weighted images lower signal intensity in the tendon; however, the calcifications are not always easy to detect with MR imaging; an X-ray could be more appropriate in order to identify a calcification of the tendon.

The differential diagnoses are many and often difficult to reach; a rectus femoris tendinopathy should be distinguished from hip injuries as acute hip joint arthritis, periarticular infections, labral tears, degenerative changes and chondral lesions of acetabulum, stress fractures of femoral neck, and femoroacetabular impingement.

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Chapter 5
Iliopsoas Tendinopathy

Andrea Foglia, Achim Veuhoff, Cesare Bartolucci, Gianni Secchiari, and Gian Nicola Bisciotti

Abstract Iliopsoas tendinopathy represents a 'grey area' in the sports-related biomedical literature, from the terminology used to describe it to its aetiopathogenesis, from conservative management to surgical treatment. Not much has been written about it, and the opinions expressed, despite their being 'evidence based' in some cases, are often conflicting.

In this chapter, the authors review and synthesise information about the different clinical scenarios of iliopsoas tendinopathy.

The authors propose a 'best practice' approach, where daily clinical decisions result from the integration of the health professional's experience, competence and skill with the use of the best available scientific evidence.

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5.1 Introduction

Iliopsoas tendinopathy represents a 'grey area' in the sports-related biomedical literature. Not much has been written about it, and the opinions expressed, despite their being 'evidence based' in some cases, are often conflicting. These discrepancies affect all aspects of the condition: topographic anatomy, functional considerations, nomenclature used, the location of the presumed pathoanatomical changes, aetiopathogenesis and conservative or surgical management.

In this chapter, we shall attempt to propose a 'best practice' approach, where daily clinical decisions result from the integration of the health professional's experience, competence and skill with the conscientious, explicit and judicious use of the best available scientific evidence, combined with the preferences, expectations and needs of the patient/athlete.

5.2 Epidemiology and Aetiopathogenesis

The clinical manifestations affecting the iliopsoas tendon and its associated structures are many and varied. Analysis of the different definitions reported in the literature reveals conflicting descriptions and therefore evident terminological confusion.

Listed below are some of these clinical presentations:

1. Iliopectineal/iliopsoas bursitis and friction bursitis [1, 2].
2. Distal iliopsoas strain with partial or total tendon tears [3].
3. Iliopsoas syndrome, represented by the combination of two different pathophysiological phenomena: iliopectineal bursitis and iliopsoas tendinopathy. The two structures – tendon and bursa – influence each other because of their close proximity [4, 5].
4. Snapping hip syndrome [6–8], which deserves special attention.
   'Snapping' or 'clunking' originates from three distinct situations:

   - Intra-articular factors (least common)
   - External factors, i.e. from the iliotibial band (most common)
   - Internal factors

Whereas intra-articular and external problems are *not* related to the *iliopsoas tendon*, internal factors consist of irregularities in its movement: the tendon may meet an obstacle, a hindrance on the iliopectineal eminence, anterior-inferior iliac spine or on the femur head. There may be paralabral cysts acting as an anterior barrier to the passage of the tendon. With regard to the tendon itself, part of the muscle belly may be caught by its own tendon, and there have been reports of a bifid tendon or stenosing tenosynovitis [9, 10]. A snapping phenomenon coming from the iliopsoas tendon is also considered to represent the true 'snapping hip' [11].

A snapping hip produced by the iliopsoas tendon does not necessarily cause pain or require treatment.

Leaving aside the topic of strain with partial or complete tear of the muscle-tendon complex, usually the outcome of a particularly violent event, we will deal with the remaining causes of tendinopathy: snapping related to iliopsoas bursitis. A further remark about the connection between the bursa and the tendon, a structure whose integrity has been damaged by long-lasting inflammatory processes, will be more susceptible to inciting events.

Turning to the risk factors and mechanisms of injury, a crucial passage in identifying the aetiology is obtaining information about two variables [12]. Risk factors are classified into intrinsic and extrinsic, modifiable and non-modifiable. A few *intrinsic risk factors*, which are predominantly non-modifiable (or poorly modifiable), can be identified for iliopsoas tendon disorders: age between 15 and 40 years; female sex (predominant in the reported series); hip hypermobility [13]; coxa vara, a bony crest above the lesser trochanter (very rare); previous strains; previous surgical procedures that have modified the mechanical axis of the lower limb; and caused exuberant scar tissue in the area of passage [14, 15]. In hypermobile subjects the periarticular muscular structures have to work more intensely to control the excess of movement [13]. As regards strength (a factor closely related to neuromuscular control), instead, reduced capacity of the gluteal musculature, combined with hypermobility, could constitute a further risk factor for the development of iliopsoas tendinopathy [13].

5.3 Clinical Examination

In the physical examination, the examiner's ability to distinguish between 'normal' (clinically irrelevant and not related to the athlete's reported complaint) and abnormal (clinically relevant and potentially related to the patient's clinical condition) is crucial. Through the physical examination, the examiner aims to:

- Appreciate and recognise '*relevant clinical signs*'.
- Relate the clinical signs to the athlete's reported symptoms.

Possible signs and symptoms during palpation are:

- Pain on passive, active or active-resisted hip flexion
- Pain on internal rotation or with passive hyperextension
- Tenderness to palpation over the femoral triangle

Another aspect to be considered is hypermobility on account of its role as an intrinsic risk factor. Hypermobility is evaluated with the Beighton score or with the questionnaire developed by Hakim and Grahame [16].

We believe it useful to suggest specific manoeuvres for internal snapping hip and a checklist with additional items that may lend support to a suspicion of bursitis. At the same time, however, given their doubtful diagnostic power, these tests should be taken as merely provocative tests to help orient the diagnosis.

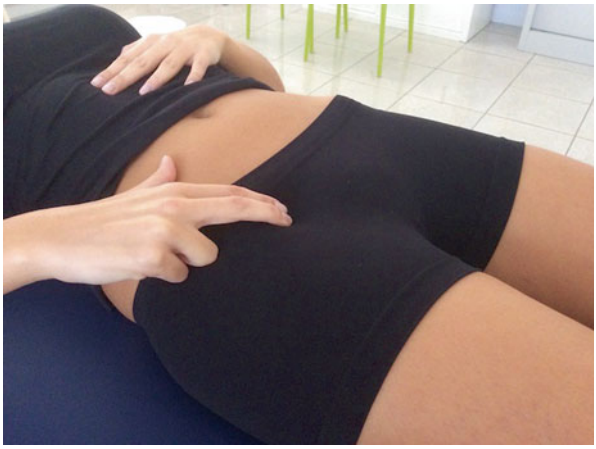

Fig. 5.1 An important point is to ask to the patient to point the area where the snapping is felt

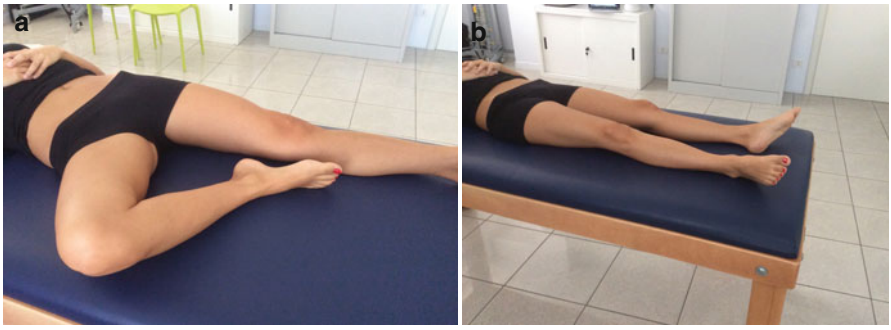

Fig. 5.2 (**a**, **b**) When we ask to the patient if he/she can reproduce the snapping, an audible snap and/or palpable snap is considered as a positive test

It is important to ask to the patient to point the area where the snapping is felt (Fig. 5.1) and ask the patient if he/she can reproduce the snapping. As stated, the snapping can often be reproduced only by the subjects themselves, who are used to the combination of movements required to produce it. After these two primary steps, the snapping might also be induced passively. This is a manoeuvre for eliciting internal hip snapping.

The dynamic manoeuvre needed to elicit the snap phenomenon may vary considerably within the affected population. Also different is the starting position: the patient may be seated, standing or lying down. Among all these variables, there is one constant: the passage from flexion to extension. An audible snap and/or palpable snap is considered a positive test (Fig. 5.2). The snapping can often be eliminated or significantly lessened by applying pressure over the iliopsoas tendon.

Byrd invites us not to interpret the snapping phenomenon as an intra-articular problem, stating that the medical history combined with the physical examination is the most valuable instrument available to the clinician. Snapping of the iliotibial band (external cause), very similar in pain and sound produced, is easier to differentiate from articular injury because of its lateral location [14]. Three more interesting tests to assess iliopsoas disorder are shown in Figs. 5.3, 5.4 and 5.5.

Fig. 5.3 Functional muscle testing of the iliopsoas muscle the subject lies supine. The test leg is flexed maximally in the hip and knee joint. The examiner tries to extend the flexed hip by pulling it with two hands wrapped around the femur just proximal to the knee. Pain is recorded as 'yes' or 'no'. Strength is assessed by the examiner as 'strong', 'intermediate' or 'weak'

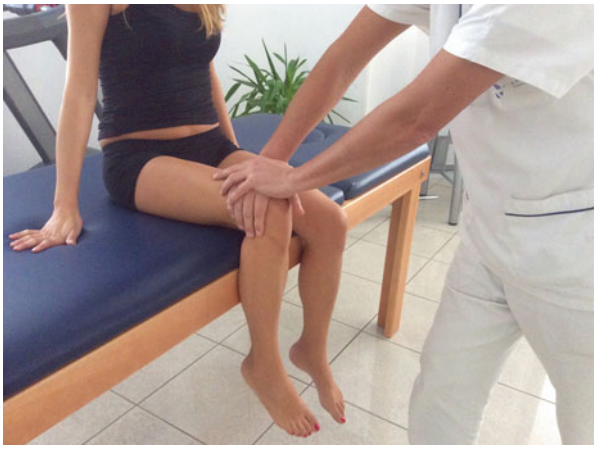

Fig. 5.4 Iliopsoas-related pain or iliopsoas weakness may be also exacerbated by testing hip flexion strength while the patient is seated. The same items (pain and strength) will be investigated and recorded

The previously mentioned 'psoas syndrome', on the other hand, consists of the simultaneous presence of an internal snapping hip and an iliopectineal bursitis [15]. As regards 'symptomatic' snapping phenomenon, once the clinician has considered the athlete's clinical history, the symptoms elicited by the provocation tests and the snapping reproduced by the procedure reported above, the diagnosis can be considered probable.

5.4 Imaging in Iliopsoas Tendinopathy

The presence of numerous, often coexisting, anatomical and mechanical factors predisposing to the development of similar signs and symptoms, coupled with the difficulty discriminating between external and internal factors on the basis of the patient's medical history and clinical examination, calls for an integration with imaging techniques to help establish the diagnosis. In the presence of the

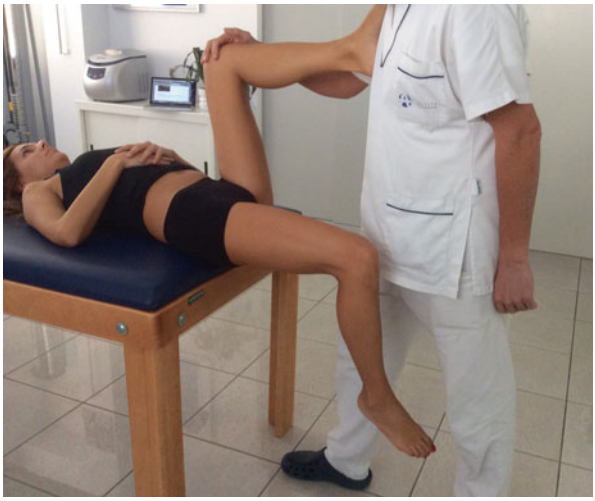

Fig. 5.5 Modified Thomas Test. When the patient holds their knee to their chest, the following thing should become immediately apparent: if the psoas is of normal length, then the dependent thigh should be free to hang down 45° below the plane of the table. In this picture, the patient's right psoas is significantly shortened. A positive Thomas test can also be an indicator of damage to the bursa, which leads to our next point. Bursitis of the iliopectineal bursa (of the iliopsoas) may be suspected if items from the following checklist are added [17, 18]

'*pathognomonic*' signs and symptoms of a snapping hip syndrome, obtained from the medical history and physical examination, ultrasound proves to be a frequently useful, rapid, economically sustainable, first-choice procedure for the static and dynamic study of the iliopsoas tendon and related tendinous structures [19]. A dynamic ultrasound study allows assessment of the function of the tendon along its entire course: the examiner can therefore identify possible 'obstacles' and appreciate the resulting abnormalities in tendon movement [20] with considerable discriminatory precision [21]. In brief, ultrasound can help to detect, characterise and stage iliopsoas tendinopathy.

The particular topographical location of the iliopsoas tendon requires the use of sonographic probes of varying frequency, ranging between 3.5 and 18 MHz [9]. The advantages and disadvantages of ultrasound imaging in general also apply to the study of the iliopsoas muscle. Advantages include its noninvasiveness, the absence of ionising radiation, widespread availability and the possibility of performing scans also during muscle contraction. Disadvantages are selective imaging documentation and operator dependence.

In order to identify the iliopsoas tendon, two scanning planes are commonly used. The first is a transverse/oblique plane along the iliopubic line [9]. The second is a pure transverse plane passing below the first plane, just beneath the anteroinferior iliac spine [9].

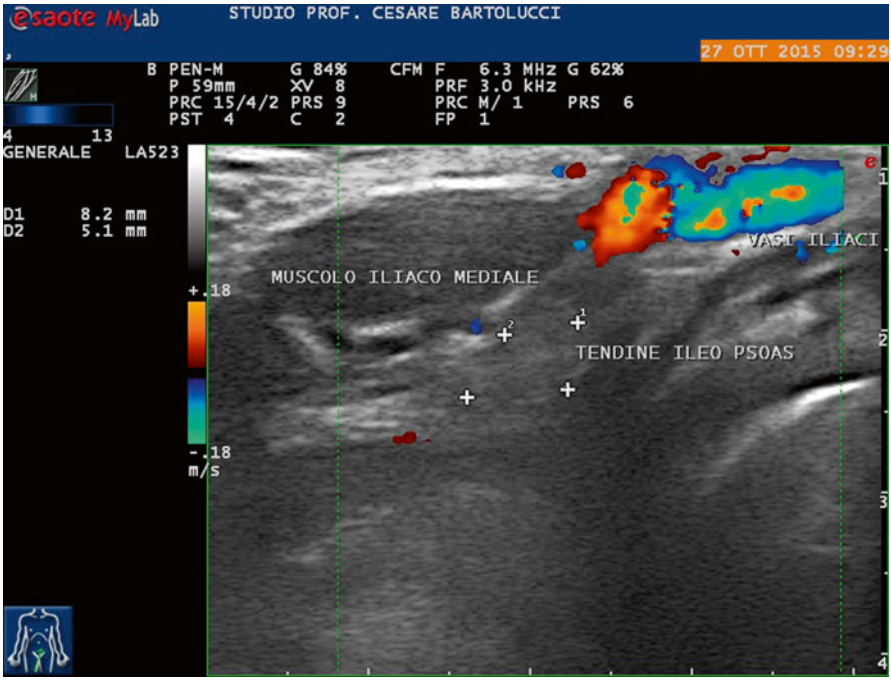

Fig. 5.6 Sonographic view of iliopsoas tendon

The field of view of the first scanning plane, with an axial oblique section above the anteroinferior iliac spine, includes the lateral and medial portion of the iliac muscle (separated by the intramuscular fascia of the iliac muscle), the superior pubic ramus, the iliopubic eminence and the femoral vessels, which can be optimally studied with the aid of colour Doppler imaging [22, 23]. The iliopsoas tendon can be distinguished below the femoral vessels and the lateral and medial fascia of the iliac muscle (Fig. 5.6).

The field of view of the second scanning plane, with a transverse section below the anteroinferior iliac spine, includes the sartorius muscle, the rectus femoris, the infratrochanteric portion of the iliac muscle and the medial and lateral portions of the iliac muscle with their intramuscular fascia. The iliopsoas tendon can be distinguished in contact with the acetabular rim (Fig. 5.7).

On ultrasound, it is sometimes possible to detect the rare anatomical variant of a bifid tendon [24–26]. Partial or complete divisions have also been reported that are not depicted by ultrasound but can be seen on magnetic resonance imaging (MRI).

Before insertion of the tendon on the posterior aspect of the lesser trochanter, a synovial bursa is visualised. The bursa is only seen when it contains fluid. The iliopsoas bursa is an area of natural expansion of the hip joint cavity. Bursal distension is commonly seen in trauma; in inflammatory, infectious or

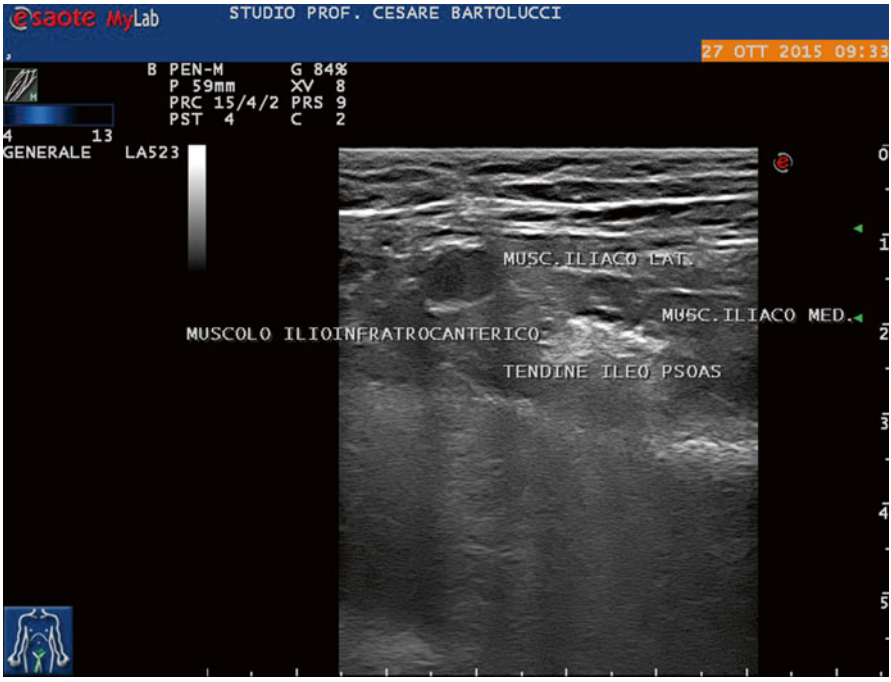

Fig. 5.7 Sonographic view of iliopsoas tendon in contact with the acetabular rim

degenerative arthritis; in villonodular synovitis; and in overuse and impingement syndromes [27].

Ultrasound has a significant role in the differential diagnosis between a large bursal distension and an inguinal or pelvic mass such as lymphadenopathies, hernias or tumours [22].

MRI is successfully used to obtain well-defined images of the overdistended iliopectineal bursa [2, 28], of the components of the iliopsoas muscle and its musculotendinous continuum. In the distal portion, close to the insertion on the lesser trochanter, MRI allows detection of possible tendon avulsion. Completion of the assessment with MRI allows differentiation between tumours and other types of lesions.

The various causes of iliopsoas bursitis include those secondary to femoral pathology such as avascular necrosis of the femoral head. In particular, MRI is used in distal tendon tears in elderly patients with avulsion of the insertion on the lesser trochanter [29, 30]. The anatomy of the iliopsoas tendon is in fact fairly complex, especially at its insertion on the lesser trochanter, where fibres also arrive directly from the lateral portion of the iliac muscle as well as from a very thin tendon separated from the iliopsoas by a thin cleft of fat. Even in iliopsoas tendinopathies affecting endurance athletes, MRI proves to be superior to ultrasound in the differential diagnosis of inguinal pain which may be produced by partial tears or tears involving only a few fibres.

5.5 Conservative Treatment of Iliopsoas Tendinopathy

Snapping hip syndrome in general (internal or external) may be treated conservatively in a similar manner to other tendinopathies: by suspension, reduction or modification of athletic activity or any other activity that causes pain, application of physical agents (ice), oral administration of drugs (NSAIDs) [31], local injection of NSAIDs or corticosteroids [32, 33] – even though this last approach is poorly supported by scientific evidence [34, 35] – and not better specified 'sports rehabilitation' [14, 15, 36].

As regards manual therapy for the iliopsoas muscle-tendon complex, and in particular transverse friction massage, we believe that this technique is unlikely to be effective. Deep transverse friction massage administered to other more superficial, and thus more accessible, anatomical structures and in similar pathoanatomical settings has not been shown to modify clinically relevant outcomes [37].

The most commonly described exercises for the treatment of tendinopathies are eccentric exercises. In *eccentric* contraction, the tension generated is sufficient to exceed the external load on the muscle, and the muscle fibres lengthen despite there being a contraction.

Although this type of contraction has been used for many years, its mechanism of action on both the micro- and macrostructure of the tendon and on pain reduction has yet to be elucidated [38]. As regards the loading dose, speed of execution, number of series and repetitions of an exercise, there is currently no consensus or a high level of evidence or strength of the recommendations, as these parameters are still being studied.

In addition to these *general* indications, the literature offers no other proven therapeutic solutions for the clinical presentations discussed in this chapter. We will briefly mention the medical treatment of iliopsoas bursitis.

Treatment of iliopsoas bursitis differs depending on the severity of the clinical presentation and the presence of possible complications. If the bursitis is mild, it is usually sufficient to apply an ice pack, observe a period of rest (absolute or relative) and administer an anti-inflammatory agent to reduce inflammation and pain. In some cases, however, it may be necessary to aspirate the liquid contained in the inflamed bursa (ultrasound-guided aspiration) and sometimes inject corticosteroids and/or anaesthetic directly into the bursa so as to resolve the inflammation and reduce the risk of recurrence. In addition to using drugs to control inflammation and pain, the treatment of bursitis may also include application of local physical therapies (such as cryotherapy) [39].

There is good evidence of the clinical effectiveness of *conservative medical treatment* in the presence both of bursitis and of iliopsoas tendinopathy (evidence level 1a/1b).

The literature indicates that the conservative medical treatment for the two structures is very similar [40].

In the presence of pathological conditions affecting the tendon and its associated structures, multimodal interventions are generally adopted: therapeutic exercise

associated with manual therapy (e.g. transverse friction massage, Cyriax), instrumental therapy (ultrasound, shockwave, low-level laser, iontophoresis with NSAIDs) and the use of taping [41]. Much of the literature on the subject consists of expert opinion with the lowest level of evidence.

Further research is needed to find out which treatment strategy combined with therapeutic exercise will provide the best results in the rehabilitation of tendinopathy in general [41].

5.6 Surgical Treatment of Iliopsoas Tendinopathy

Surgical treatment represents the last resort for resolving the patient's symptoms and is used when conservative treatment has failed. With regard to the iliopsoas bursa, various surgical procedures have been proposed: a plain bursectomy or a capsulectomy to gain access to the bursa in the case of bursitis not responding to conservative treatment. Another frequently used procedure is arthroscopic synovectomy, the surgical removal of a portion of the synovial membrane, combined, in selected cases, with iliopsoas tendon release (see below) [40, 42]. Review of the literature on the subject has revealed a fair level of evidence.

For snapping hip syndromes in general, the surgical approach consists in resecting, lengthening and/or relaxing the involved tight tendon, in order to reduce the impingement and eliminate the symptomatic snapping. The surgical approach comprises several possible procedures depending on the cause of the snapping hip syndrome [14, 43, 44].

For external causes, various types of iliotibial tract resections may be performed. For each type of operation, different approaches exist (e.g. iliotibial tract resections may be partial, Z shaped or elliptical shaped), and to date no significant differences in terms of clinical outcome have been found among the various techniques proposed.

For the less common intra-articular disorders, on the other hand, the primary causes may be very different: there may be partial tears of the acetabular labrum, synovial osteochondromatosis or loose bodies inside the joint (e.g. from osteochondral fractures) [43, 45]. The surgical approaches will vary based on the specific diagnosis.

For internal causes the tendon of the iliopsoas muscle may be lengthened [46], or a complete arthroscopic release may be performed. Endoscopic release provides good results, also supported by midterm follow-up [47].

Surgical reduction of the tendon diameter is compensated for by a regeneration of '*tendinous*' tissue over an average period of 20 months: the tendon diameter returns to 80 % of its initial value. In current endoscopic resection techniques, surgeons try to preserve a ratio of 60 % muscle to 40 % muscle tendon in the remaining tissue, and it is very likely that the preserved continuity of muscle fibres provided by this procedure prevents retraction of the tendon ends, thereby contributing to partial regeneration. In addition to the regrowth, the iliopsoas muscle fibres continue

to run parallel to the iliopsoas tendon, and after the operation this bundle of remaining muscle fibres will insert directly onto the femur without a tendon, thereby contributing to overall strength which some authors believe is fully restored (to presurgery levels).

5.7 Conclusions

Iliopsoas tendinopathy remains a poorly investigated subject characterised by many grey areas. There is no consensus in the literature on the terminology used, and there are no experimental studies of good methodological quality. Moreover, the number of epidemiological studies (aetiological, observational, cohort studies, etc.) is insufficient to enable the precise identification of risk factors. Finally, there is a lack of valid and reliable diagnostic tests to be combined with imaging to be able to establish a firm diagnosis. All this translates into clear limitations for our study and our clinical practice, and in particular, the inability to derive significant data from the publications available today for our reference models [48, 49].

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Chapter 6
Quadriceps Tendinopathy

Stefano Respizzi, M.C. d'Agostino, E. Tibalt, and L. Castagnetti

Abstract The quadriceps can be considered a relative strong structure of the extensor apparatus of the knee; nevertheless, it can be affected and weakened by degenerative changes, under the influence of local and systemic factors. In some cases, progression of tendinopathy can lead also to spontaneous, partial, or complete rupture. For this reason, prevention and treatment of predisposing factors, as well as early recognition, objective grading, and monitoring of structural alterations, can be helpful in identifying those ones more susceptible to rupture. Symptomatic enthesopathy is rarely the unique cause of bilateral simultaneous and complete rupture of the quadriceps.

Quadriceps tendon tear is a not so common injury, usually occurring in patients older than 40 years of age, frequently due to a fall during sudden quadriceps muscle contraction. Early recognition of this injury can be strategic to ensure a more rapid healing, as well as a better recovery and prognosis.

For complete quadriceps tendon rupture, surgery is the treatment of choice, while in partial tears, an accurate assessment of the degree of the lesion and disability, or loss of function, can aid the clinician in deciding surgical versus conservative treatment. Conservative treatment of partial tears of the quadriceps tendon consists in a full extension brace for 6 weeks, followed by a protected range of motion exercises, that can guarantee a good outcome and regain of function in the follow-up. When surgical treatment is required, 6 weeks of postoperative immobilization with intense rehabilitation are indicated, for obtaining the best functional outcomes as well.

All grades of tendinosis are usually treated conservatively as first-line therapy. Among conservative treatments, biophysical stimulations (extracorporeal shock waves and some so-called physical therapies), as well as autologous growth factors, surely represent some innovative, safe, and effective therapeutic strategies that are

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indicated in conjunction with rehabilitative programs, in order to obtain, besides anti-inflammatory and analgesic effects, a positive action on tendon tissue and its remodeling processes, for improving recovery.

6.1 Etiopathogenesis and Clinics

Quadriceps tendon injuries include a spectrum of diseases, ranging from tendinosis to partial-thickness tears to complete tendon rupture. Relatively uncommon, between the injuries of the knee, nevertheless they can be considered the second most common ones, after patellar fracture [1]. Moreover, in clinical practice, quadriceps enthesopathy is less common than the patellar one [2].

It has been described in the literature also the possibility of a "calcific tendonitis" of the quadriceps, although an uncommon condition. It has to be considered, in any case, not a "benign" condition, and an awareness of this tendinopathy and the potential complication of quadriceps tendon damage are important, when investigating acute symptoms associated with the extensor mechanism of the knee [3].

Quadriceps tendinopathy has been described to be more frequent in the elderly population, practicing regular sports activities, as well as in weight lifters, due to the fact that the quadriceps tendon is particularly subjected to load, especially in the deep squat [4].

In the older patient population, quadriceps tendon tears usually occur in the setting of a slip and fall. In most of the cases, there are preexisting degenerative changes within the tendon, thought to be due to degeneration or repetitive microtrauma. In younger patients, acute tears are usually sports related, due to sudden acceleration forces or extreme stress [1].

From a general point of view, it manifests with pain along the top of the knee; also it shows resistance during contraction of the quadriceps [4].

Extensor mechanism disruptions of the knee are relatively uncommon but serious injuries. They are clinically diagnosed by the triad of swelling around the knee, palpable defect, and inability to perform straight leg raise. According to Kumar et al., the most common form of disruption would be patellar fracture, followed by rupture of the patellar tendon and the quadriceps tendon. A simultaneous rupture of a patellar tendon and the opposite quadriceps tendon is extremely rare, and different mechanical, systemic, and local factors are involved. The quadriceps rupture is more common in older subjects (>50 years) with associated systemic factors, such as obesity, gout, and local degenerative changes [5].

According to Hardy et al., the predictive value of a spur for rupture of the quadriceps tendon should be considered, in the light of the obvious selection bias of their cohort of patients chosen for operative repair. Although the prevalence of a patellar spur in the normal population is unknown, nevertheless it is as uncommon as rupture of the quadriceps tendon. Thus, the presence of a radiographically identified spur should alert the examining doctor, to the possibility of such an injury in patients with traumatic failure of the extensor mechanism [6].

Bilateral traumatic ruptures of extensor tendons are being increasingly reported, and it is important to consider a bilateral pathology, in all those cases presenting with traumatic rupture on one side. The commonest cause of bilateral simultaneous rupture would appear to be a sudden violent contraction of the quadriceps mechanism, with the knees slightly flexed and feet firmly planted on the ground. It is important to feel for tenderness and defects in both the suprapatellar and infrapatellar regions, as both ruptures can occur simultaneously in spite of having two different pathogeneses [5].

As already mentioned, chronic tendinopathy may evolve in many cases toward progressive tendon degeneration, with possible rupture. This type of injury is usually the consequence of a forced contraction of the quadriceps, with the knee flexed and the foot fixed to the ground, but the pathogenesis has to be considered as multifactorial, as correlated with some general disorders like renal insufficiency (especially with hemodialysis substitutive therapy), primary or secondary hyperparathyroidism, and some other conditions, which impair and weaken the osteotendinous junction [2, 7–10].

Other comorbidities that may cause spontaneous rupture are represented by diabetes, rheumatoid arthritis, and gout, use of quinolones, corticosteroid injections, anabolic steroids, statins, and obesity [8–10]. According to some of the authors, there would be also a genetical predisposition, especially when bilateral rupture of the quadriceps tendon occurs [11]. From a general point of view, some histopathological changes of tendon structure (as abnormal collagen distribution and increased production of type III collagen fibers by tenocytes) seem to be correlated with spontaneous rupture, generally due to minor trauma [8].

Classically, the quadriceps tendon rupture frequently occurs in the attempt to recover balance, in order to avoid a fall. A considerable tension on the tendon occurs, when there is an important eccentric contraction of the quadriceps femoris and the knee is in a half-bent position.

Pain, loss of extension, and suprapatellar gap are the clinical signs of the quadriceps tendon rupture. The clinical assessment can be difficult in this case, due to tenderness and swelling, that increase the risk of missed diagnoses and/or diagnostic delay [9].

Extensor mechanism ruptures might be easily overlooked and misdiagnosed, and delayed diagnosis of quadriceps tendon rupture is not so rare. In these cases, the literature recommends early surgical repair within 72 h [12].

Patients suffering from this injury complain of a painful and swollen knee. They are unable to walk without some sort of assistance and usually hold their leg as straight as possible. Despite these clinical signs, misdiagnosis is frequent, ranging from 39 to 67 %. A gap in the tendon may not be convincingly palpable, and the patient's ability to raise his/her leg straight with the use of the medial and lateral patellar retinaculum or iliotibial band may convince the inexperienced observer that only a few fibers of the tendon have been ruptured. However, the results of the repair may be compromised if the delay to surgery exceeds a few days and no clinical diagnostic test has been described to date [12]. The high rate of misdiagnosis did not decrease with the introduction of ultrasound and MRI investigations. In addition, these instrumental investi-

gations can be considered costly and time consuming in an emergency department. The reasons for delayed diagnosis are probably multifactorial, and the consequences can be considered important, as the outcome could be severely affected [12].

Inspired by the O'Brien test for complete rupture of the Achilles tendon, Jolles et al. proposed in the literature a new minimally invasive test that directly can determine the integrity of the quadriceps tendon in its five distal centimeters [12].

With the patient lying in the supine position, in aseptic condition, a 25-gauge needle is inserted at a right angle through the skin of the thigh, at a midline point, 5 cm proximal to the superior border of the patella. The needle is inserted gently through the skin until further resistance is felt, so that the needle's tip is just within the substance of the quadriceps tendon but without transfixing it. A passive knee flexion and extension movement is then performed, and the movement of the hub of the needle is observed. Two distinct types of response may occur. One response (negative test) is swiveling of the needle about its pivot point in the skin which indicates that the tendon is intact throughout its distal 5 cm. If no swiveling of the needle is observed (positive test), this indicates loss of continuity of the extensor apparatus (quadriceps tendon between its insertion and the position of the needle) [12].

Only few cases of bilateral simultaneous complete quadriceps rupture, in patients with symptomatic enthesopathy, have been previously reported. Arumilli et al. stressed the importance of warning patients of the risk of developing complete tendon rupture, when they present with an enthesopathy around the knee [13].

6.2 Imaging

Although many cases of quadriceps rupture can be diagnosed clinically, partial tendon tears may be more difficult to assess clinically, as some degree of function can be maintained in any case. When tendon tears are acute, soft tissue swelling and associated hematoma may obscure the tendon defect on physical examination and may limit the clinical evaluation. With delayed presentation of a tear, the tendon defect may not be evident due to scar tissue formation. There may also be partial return of quadriceps function after several weeks, with the risk of confusing the diagnosis [1].

Therefore, accurate imaging can aid diagnosis and directly affect patient treatment. Radiographs are usually not diagnostic for quadriceps tendon tear. The use of magnetic resonance imaging for the diagnosis of quadriceps tendon tears has been advocated, but its accuracy has not been defined. Bianchi et al. described the usefulness of sonography for complete quadriceps tendon tears with surgical correlation [1].

6.2.1 Quadriceps Tendinopathy

In normal conditions, the quadriceps tendon is visible at ultrasound examination with anechogenic and "multistratus" appearance, in relation to the presence of 2–4 tissue layers. The most frequent anatomical situation is the presence of three

layers: the more superficial stratus comes from the more distal part of the rectus femoris, the deeper one derives from the front fascia of the vastus intermedius, and the intermediate one directly from the fascia between the three vastus muscles (intermedius, lateralis, and medialis) [14]. The quadriceps tendon has an area of major weakness, at its distal end, and it is described as the area where tendinopathies arise more frequently. Moreover, in clinical practice, it is possible to observe occasionally some small changes of tendon structure, even in asymptomatic subjects [15].

The ultrasound images of quadriceps tendinopathy can be extremely heterogeneous, from some degenerative alterations limited to a single layer to larger ones engaging the whole tendon. In the first case, tendinopathy can be recognized as a hypoechoic oval image, while in the second one, a wider hypoechogenicity of the different collagen layers will be described, together with thickening of the different layers and loosening of the fibrillar structure, as pathognomonic signs of degenerative tendon disease [15].

Frequent etiologies of quadriceps tendinopathy can be represented by a chronic overload, related to professional activity or overweight, as well as seronegative spondyloarthropathies [15]. In all these conditions, it is possible to demonstrate, through ultrasound examination, the presence of calcifications. Moreover, in seronegative spondylopathy, some areas of bony erosion and soft tissue swelling can be detected, while in calcific overload, enthesopathy calcifications on the tendon surface or in the intermediate layers may be visible [16].

Generally, when a localized tenderness and thickening along the course of the tendon are clinically visible, under sonographic examination, they correspond to a hypoechoic area and pathologic hypervascularization phenomenon. This ultrasound picture, as already mentioned, is sometimes present even in asymptomatic individuals. Some of the authors emphasize that the presence of echographic and structural alterations, in the absence of symptoms, could represent a risk factor for the development of painful symptoms in the future [17].

6.2.2 Tendon Injuries

The obliteration of the "quadriceps shadow," the presence of a mass in the suprapatellar soft tissues secondary to tendon retraction, and the avulsion of a bony segment from the proximal pole of the patella are all typical radiographic features. Another feature that can be considered as suggestive of quadriceps tendon rupture is the so-called "low-riding" patella or patella baja. By the ratio between the length of the patellar tendon and the length of the patella in the lateral projection, it is possible to calculate the index of Insall–Salvati, which is useful for identifying a low patella, if the value is lower than zero [18].

Compared to traditional radiography, ultrasound examination is a more effective method for diagnosing the rupture of the quadriceps tendon [10]. Foley et al. studied the ability of sonography in detecting those quadriceps tendon tears that require surgical treatment (high-grade partial tears and complete ruptures). After having examined 239 consecutive sonographic cases, they were able to demonstrate that

sonography is an effective tool for identifying quadriceps tendon tears requiring surgical treatment (high-grade partial tears and complete ruptures) [19].

Magnetic resonance imaging (MRI) is another important instrumental method for tendon evaluation, especially in case of injury. Some main different situations can be described. In case of partial rupture, at least one of the layers is still intact. The superficial layer is the most common site of injury, followed by the intermediate one [20]. In this case, the size of the lesion does not increase with distal end traction of the patella. Finally, we must consider that if the peritendinous envelope is intact, the diagnosis may be difficult. When a total breakage occurred, there is an interruption of all the tendon layers, with a possible hematoma in the area of the lesion. By the traction of the patella, it is possible to observe an increase in the interval between the two tendon stumps. The framework of MRI in complete breaking of the quadriceps tendon demonstrates a retracted proximal stump and a wavy look of the patellar tendon, caused by the lack of tensile force applied to it [2].

Already in 1994, Bianchi S. et al. confirmed that sonography should be considered in the diagnostic workup and treatment planning of patients with suspected tears of the quadriceps tendon, for evaluating quadriceps tendon rupture, due to its noninvasiveness and high degree of sensitivity and specificity [21]. In the following years, ultrasound has been extensively used as a diagnostic tool before surgery, for diagnosing acute traumatic tears of the patellar and quadriceps tendons; these ones are rare injuries, requiring immediate repair to reestablish knee extensor continuity and allow early motion. More recently, Swamy et al. concluded that ultrasonography is not a reliable method in establishing the diagnosis of acute injuries to the extensor mechanism of the knee, especially for the quadriceps tendon ruptures in obese and very muscular patients. When a certain clinical ambiguity is present, MRI scan is a recommended investigation tool, to be taken before any surgical treatment [22].

6.3 Conservative Treatment

The conservative therapeutic approaches to quadriceps tendinopathy are various, and among them, some programs of physiokinesitherapy can be considered, besides manual medicine and different manipulative approaches on soft tissues. From a general point of view, quadriceps tendinopathy is responsive to physiotherapeutic treatments, according to the same general principles of patellar tendinopathy [23]. Abnormal fascial tensions of the thigh frequently hesitate in an uncoordinated contraction of the quadriceps that ultimately hesitates in tendinopathy of the extensor apparatus of the knee. For this reason, an effective approach, in terms of tenderness reduction, can be obtained by treating the fascia of the quadriceps femoris [24].

Eccentric exercises, as much as for some other tendinopathies, play surely a key role also in the conservative and rehabilitative treatment of quadriceps tendinopathy. Even for those tendons subjected to autologous growth factor (platelet-rich plasma or PRP) injections, the need for a standardized rehabilitative program is emphasized, based on a progressive submaximal eccentric work [25].

Dimitrios et al. in 2012 demonstrated that the combination of eccentric training and quadriceps static stretching exercises would be superior to eccentric training alone, in order to reduce pain and improve function, both at the end of treatment and at the end of follow-up as well [26]. Nevertheless, sometimes, the eccentric work can be more painful to be completed; moreover, if performed during the competitive season, it has, in addition to worsening pain, poorer immediate benefits, resulting in poor adhesion to the rehabilitative program [27]. A more immediate effect in controlling pain relief in tendinopathies seems to be obtainable by isometric exercises, with sustained effect for at least 45 min, as well as by an increase in quadriceps strength, through increased voluntary isometric contraction [28].

Setting a timely and proper rehabilitation treatment is also very crucial after surgery, along with the need of a protected load. The rehabilitation program will be developed with exercises aimed to recover range of motion and strengthen the quadriceps. Patients who undergo delayed repair are at risk for a compromised result, due to loss of full knee flexion and decreased quadriceps strength, although a functional extensor mechanism is likely to be reestablished [29].

Many studies demonstrated that treatment involving eccentric training can be effective in the conservative management of chronic tendinosis, although the mechanisms of action are still under study. In particular, Kubo et al. in 11 healthy volunteers were able to demonstrate some changes in blood circulation of the Achilles tendon, during and after repeated eccentric contractions, being more remarkable than those during and after repeated concentric ones [30].

From a general point of view, intact tendons adapt slowly to changes in mechanical loading, whereas in healing tendons, the effect of mechanical loading or its absence is dramatic. Numerous animal studies show that immobilization of healing tendon compromises the healing process [31–33], and many other studies show that tendons adapt to changes in mechanical loading [34]. Nevertheless, some authors hypothesized and demonstrated that tendon tissue would have a "memory" of loading episodes, thus allowing short loadings being enough to elicit improved healing, while suggesting that patients might be allowed early short loading episodes, following a tendon lesion, in order to obtain a better outcome [35]. However, some details about amplitude, frequency, and duration of mechanical stimulation are still partly unknown, as well as the appropriate time point during the repair process to start loading and how to avoid overload. Furthermore, in animal models as well as in humans, there is a great individual variation in tendon response to mechanical stimulation [35].

Moreover, tendon healing/recovering is a complex and highly regulated process that is initiated, sustained, and eventually concluded by a large number and variety of molecules. Growth factors represent one of the most important classes of molecular families involved in tendon healing, and many studies have elucidated their functions, especially in more recent years [36].

Nowadays, the use of PRP has become increasingly popular in tendon and muscle repair. The scientific rationale for its application is based on the fact that it contains a wide variety of growth factors as insulin-like growth factor 1 (IGF-1), transforming growth factor β (TGFβ), vascular endothelial growth factor (VEGF), platelet-derived growth factor (PDGF), and basic fibroblast growth factor (bFGF),

and also in vivo, at the site of a tendon injury, platelets are activated and release their factors [37].

Although in the last years the use of PRP in clinical practice is increasing, literature concerning PRP applications is still under debate. In any case, many studies already demonstrated that PRP use for tendon repair can have a positive effect, probably due to an enhancement of the reparative processes. For these reasons, it can be taken into consideration in case of chronic tendinopathy not responsive to conservative treatment [38].

From a general point of view, tendinopathy can be considered as the failure of a chronic healing response, associated with both chronic overloaded or unloaded states. Although several conservative therapeutic options have been proposed in the literature, very few of them are supported by randomized controlled trials. The use of injectable substances such as PRP, autologous blood, polidocanol, and corticosteroids in and around tendons is not supported by strong clinical evidence. Further randomized controlled trials are necessary to define the best conservative management of tendinopathy [39].

If, nowadays, therapeutical exercise represents one of the most important strategic approaches for tendinopathies, nevertheless, some biophysical regenerative therapies can be applied successfully, in order to reduce inflammation and pain and to positively interfere with a local altered turnover of tendon tissue. From this point of view, it has been recently underlined, for example, the efficacy of laser therapy in the field of tendinopathies, despite the need for more studies about this topic. The mechanisms of action would seem related to reduction of inflammation and increased collagen synthesis [40].

Among *biophysical therapies*, nowadays, extracorporeal shock wave therapy represents a valid tool for treating tendinopathies, including quadriceps tendinopathy, while considering this specific anatomic region as part of the extensor compartment of the knee, for which this mechanotherapy has been already demonstrated to be a valid tool. Interestingly, when applied in the musculoskeletal and, in general, in non-urological fields, ESWT has no pure mechanical effect of disruption, but a real biological action on cells and tissues (due to mechanotransduction), with a success rate, according to the literature, up to 91 % (for jumper's knee, Achilles tendinitis, plantar fasciitis, rotator cuff "tendinitis," and greater trochanter pain syndrome) [41–44]. More in details, some clinical experiences would indicate this therapy as effective in treating tendon pathologies, especially when some other conservative treatments have failed, although the mechanisms of action remain still under study [41–43, 45, 46].

From the experimental point of view, tendon and tenocytes have been extensively studied in the field of shock wave (SW) mechanobiology. Some of the authors described, for example, that an optimal SW treatment would promote the healing of a "collagenase" – Achilles tendinitis – by inducing TGF-beta1 and IGF-1 secretion, with the effect of reducing edema, swelling, and inflammatory cell infiltration in experimentally injured tendons. Moreover, in this condition, the authors were able to demonstrate an intensive tenocyte proliferation and progressive tendon tissue regeneration, related to the expression of cell nuclear antigen (PCNA), besides intensive

TGF-beta1 and IGF-1 expression, quite similar to what happens in the early stage of tendon healing. On the basis of these results, it has been hypothesized that physical SW stimulation could increase the mitogenic responses of tendons [47].

Moreover, in the literature, there are many other evidences of a possible direct action of SW on tendon cells, thus implying also a possible future involvement in tendon healing and regenerative therapies. These effects can be so summarized [48–54]:

1. Increased expression of lubricin
2. In vitro enhancing of functional activities of ruptured tendon-derived tenocytes (proliferation and migration)
3. Ehancement of cell vitality and proliferation, besides expression of typical tendon markers and anti-inflammatory cytokines
4. Reduced expression of several metalloproteinases and interleukins (MMPs and ILs)

Regarding the effects of SW on tendons, very recently the biochemical responses of tendons after SW stimulation have been described by microdialysis, in humans; moreover, it was hypothesized that this type of mechanical stimulation could be able to improve tendon remodeling in tendinopathies, by promoting the inflammatory and catabolic processes, that are associated with removal of "pathological" matrix constituents, thus suggesting a potential scientific background for future studies and clinical applications [54].

6.4 Surgical Treatment

From a general point of view, in complete quadriceps tendon rupture, surgery is indicated, whereas tendinosis is usually treated conservatively. According to the extension of the tear and the resulting loss of function, partial tears may be treated surgically [3, 55]. Accurate assessment of the degree of partial tears and the degree of disability can aid the clinician, in determining surgical versus conservative treatment [1]. Conservative treatment of partial tears of the quadriceps tendon consists in a full extension brace for 6 weeks, followed by a protected range of motion exercises, allowing good outcomes and regain of function [3].

Biomechanical studies have demonstrated that normal quadriceps tendon, if subjected to longitudinal tensional strength, can withstand loads up to 30 kg/mm before going to meet at break. It is also estimated that it needs a force equal to three times the body weight, resulting in a fall in knee flexion and maximal contraction of the quadriceps muscle, to lead a tendon rupture. According to what is described, it is understandable that quadriceps tendon tear is an uncommon injury. Nevertheless, the most common modality of rupture is a minor trauma (such as a simple fall or an eccentric contraction of the quadriceps muscle), and usually it represents the end stage of a long process of chronic degeneration and overuse [55, 56]. As already mentioned, in most of the cases, rupture is a spontaneous event, sometimes bilat-

eral, and occurs preferably in older patients, especially if affected by metabolic diseases [57]. Structural tendon alterations are related to muscle atrophy and tendon structure degeneration, but also to modification of the microcirculation. Rupture often occurs in those tendons suffering from fatty, cystic, or myxoid degeneration or characterized by the deposition of calcium salts; all these conditions can modify the tendon architecture and, consequently, its biochemical properties and resistance capacity [58–61]. It has also been suggested that the presence of a patellar spur, a bony prominence at the quadriceps tendon insertion point of the proximal pole, may be related to the rupture itself [62]. Rarely ruptures occur in high-level athletes, and they usually result from an eccentric load on the extensor mechanism. Unfortunately, prodromal symptoms and predisposing factors are almost always absent, and even respecting timing and surgical procedure, a low rate of return to play is described [63, 64]. In athletic population, in contrast with the older ones, a different process involving repetitive microtrauma is thought to occur in the quadriceps tendon, which can be subclinical and eventually lead to tendon ruptures. Ipsilateral tendinopathy of the extensor apparatus may be found in patients who subsequently suffer a quadriceps tendon tear [58]. There are also some iatrogenic conditions (such as total knee replacement, lateral release, and steroid injections) that can modify the normal structure of the quadriceps tendon, with the risk of tendon rupture [55]. The most common rupture site of the quadriceps tendon is located between 1 and 2 cm from the superior pole of the patella, while, in older people, rupture most probably occurs at the osteotendinous junction. This area is less vascularized, thus suggesting that a vascular imbalance may contribute to the pathogenesis of the rupture itself [55]. The vascular supply to the quadriceps tendon is usually sustained by three systems: the medial arcade (that supplies the medial border of the tendon), the lateral arcade (that supplies the lateral border), and the peripatellar vascular ring [59]. In relation to the vascular supply of the quadriceps tendon, some authors proposed that the articular side of the tendon, the deepest one, may be exposed to a relative compressive strength, produced by the bone femoral condyles, thus involving an impairment of the vascular supply [59]. From a general point of view, the viscoelastic properties of the tendon lead fairly quickly to a retraction after rupture, thus making it necessary to proceed surgically as soon as possible, in order to avoid pathological muscle retraction and fibrosis [55–57, 60, 64].

The most widely accepted method for repairing the quadriceps tendon when an acute rupture occurs is the *primary suture repair*, consisting in continuous interlocking sutures, passed through the distal end of the tendon and then secured through some drill holes made into the patella. Regarding the transosseous technique, it is usually used a drill to create a small groove in the upper pole of the patella, whereas tendon sutures are passed through the tendon with Krackow, Kessler, or an equivalent technique. Regarding the transosseous technique, it is usually used a drill to create some small grooves in the upper pole of the patella, whereas tendon sutures are passed through the tendon with Krackow, Kessler, or an equivalent technique. In this way, strong transosseous sutures are placed through the patella, thus securing the distal part of the quadriceps tendon to bone [60, 64, 65].

In the literature, a variety of reinforcement techniques have been described, in order to add an extra support to the primary repairs. These techniques are especially useful, and in case of poor quality of the tendon tissue, or if retraction of torn ends is present, they include wire augmentation, autografts, allografts, and synthetic materials. With all recent advances in technology, numerous studies describe the use of suture anchors for fixation of the quadriceps tendon [66]. This technique can be performed with a smaller incision while preventing potential violation of the patella – tendon junction. Moreover, as another advantage, pilot holes for anchor placement are not transosseous, thus decreasing the potential complication of patella loosening [67, 68]. Human cadaveric studies demonstrate that quadriceps tendon repair with suture anchors yields significantly better biomechanical results than transosseous sutures. These data support a more consistent load-to-failure strength and allow an early partial weight bearing, immediately after surgery. With respect to traditional, primary transosseous technique, there is a much higher cost [69]. Sooner after surgery, the knee is protected with a brace that holds the joint in an extended position for a time varying from 2 to 6 weeks, before starting with progressive knee flexion exercises, according to the surgical technique [70].

Delayed quadriceps tendon rupture requires some more complex surgical techniques, such as the *Scuderi* and *Codivilla* techniques. The *Scuderi* technique consists of a direct repair of the quadriceps tear, reinforced by a tendon partial-thickness triangular flap, turned down and sutured on the superficial aspect of the patella. Nevertheless, the Codivilla method, if presence of significant retraction of quadriceps, can be used: a full-thickness inverted-V-shaped incision is made in the tendon. The base of tissue triangle obtained in the tendon can be moved distally to allow the tear to be approximated and repaired. The proximal aspect of the inverted V can be then repaired side to side and the remaining flap turned down and used as augmentation [56].

Complications are relatively uncommon and include deep venous thrombosis or pulmonary embolism, heterotopic ossifications, superficial and deep infection, and reruptures. Quadriceps muscular atrophy and muscular strength deficit can be present in most of the cases, but generally this does not have any influence on the satisfaction of the patient. No significant differences in order to final results among the surgical techniques used in primary repair are reported, as generally, functional and clinical outcomes seem to be very good; on the contrary, a delay in surgical repair can have an adverse influence on the outcome [56].

Over the years, the repair techniques have progressed from simple suture with catgut or silk to wire-reinforced repair, pullout suture fixation through the patella, suture anchor fixation, tendon lengthening repair, Scuderi technique, allograft, autograft, and synthetic materials. Recently, some authors reported their results using arthroscopy [71]. The purpose of reporting spontaneous quadriceps tendon rupture cases was to describe a new surgical procedure using arthroscopy, which indicated the positive effect of stable fixation followed by early range of motion exercise on the result of treatment [71]. Many investigators reported that the shorter the period between injury and surgery, the better will be the clinical outcome, regardless of surgery method, age, or body mass index (BMI), in patients with quadriceps tendon with no underlying diseases. Delayed diagnosis leads to unsatisfactory clinical

outcomes; early diagnosis and early restoration of function are important for preventing disability [71].

Regarding postoperative rehabilitation, it has been reported that prolonged knee immobilization for 6–8 weeks after primary suture was necessary to allow complete healing of the repair and to ensure an acceptable outcome. In addition, postoperative rehabilitation should be determined according to the presence of the underlying diseases, influence of body weight, and period between injury and surgery, and surgical methods with arthroscopic repair of quadriceps tendon rupture can provide excellent results [71].

Adequate repair of the quadriceps and patellar tendons to the patella is essential for a functional extensor mechanism. Too often, these injuries are missed as a result of inadequate suspicion of lesion or confusion with other potential knee injuries. A careful examination, combined with imaging, can detect these injuries, which should be repaired promptly, if the patient is a suitable surgical candidate, in order to provide the most optimal outcomes. In the case of delayed presentation, retraction of the quadriceps tendon yields a challenging reconstruction problem, often requiring the use of allograft and advanced reconstruction techniques, with less predictable results. Every effort should be made to avoid missing this diagnosis [72].

6.5 Synthesis and Therapeutic Indications

Quadriceps tendon is a very strong structure contributing to the extensor mechanism of the knee. The structural and biomechanical properties of the quadriceps tendon allow it to sustain very high loads without rupture. Nevertheless, it can be affected and weakened by degenerative changes, due to some local and systemic factors, although less frequently than some other anatomic districts. Many degenerative age-related changes of the tendon tissue have been described, such as fatty and myxoid degeneration, tendon fibrosis, decrease in quantity, and change in type and cross-links of collagen fibers, microangioblastic dysplasia, and calcifications [73]. Several clinical conditions have been reported to induce tendon degenerative changes: for example, renal diseases, long period of hemodialysis, and uremia are associated with spontaneous tendon rupture, affecting maturation of collagen and quadriceps muscle fiber atrophy [74]. In addition, hyperparathyroidism causes dystrophic calcifications and subperiosteal bone resorption and can weaken the osteotendinous junction between the quadriceps tendon and patella [74]. Diabetes mellitus has also been reported to cause fibrinous necrosis and vascular damage, as observed in quadriceps tendon rupture specimens [73]. Other metabolic and rheumatic diseases such as gout, pseudogout, rheumatoid arthritis, systemic lupus erythematosus, obesity, and steroid intake have been reported to induce tendon degenerative changes [74].

Progression of tendinopathy can lead in some cases to spontaneous partial or complete rupture. For this reason, early recognition and preventing of predisposing

factors, as well as objective grading and monitoring of quadriceps structural alterations, will be helpful in identifying those ones most at risk of rupture. Symptomatic enthesopathy can be a rare and unique cause of bilateral simultaneous complete quadriceps rupture [13].

From a general point of view, quadriceps tendinopathy treatment is mainly conservative, and it is based on rehabilitation, biophysical therapies, and autologous growth factors (PRP). Among biophysical therapies, nowadays, those ones which seem to induce greater therapeutic efficacy on degenerative tendon pathology can be considered laser and extracorporeal shock wave therapy (ESWT) [40, 43]. ESWT represents a new therapeutic frontier, by its ability not only to induce an anti-inflammatory effect but also a possible trophic effect on tendon remodeling as well [43].

Rehabilitation is an important basis for functional recovery of quadriceps tendon, and it is based on the general principles applied to patellar tendinopathy [23–28, 30].

The use of innovative techniques such as PRP represents an interesting method, although current studies are not yet fully explaining, while requiring further scientific and clinical trials [36–39].

The tendon tissue response to load consists of three phases: linear deformation, elastic deformation, and plastic deformation. The first one depends only on flattening of the crimp pattern of collagen fibers (2 % of the strain), the second one is due its behavior to intramolecular sliding of collagen triple helices in which the fibers become aligned in parallel fashion (4 % of the strain), and the third one is characterized by a plastic deformation and a microscopic failure of tissue. Approximately at 10 % of strain, macroscopic failure arises. Sudden and eccentric contractions of quadriceps, as occurring during a fall in attempt to maintain balance, can exceed the phase of plastic deformity and may lead to an incomplete or complete rupture [75].

Quadriceps tendon tear is a not so common and often misunderstood injury, usually occurring in patients older than 40 years of age, due to a fall with sudden quadriceps muscle contraction. The earlier the injury is diagnosed right away, the better the prognosis will be. Surgical treatment is required, and at least 6 weeks of postoperative immobilization with intense rehabilitation are needed to obtain the best functional outcomes [76].

The viscoelastic properties of tendons and muscles determine the degree of retraction during the rupture process. Unless the original length is restored, the retracted quadriceps become too short to function properly, and, as demonstrated in neglected ruptures, only inadequate muscle function can be expected. Furthermore, early motion and tensile stresses appear to promote the organization and remodeling of collagen fibers, decrease scar tissue, and increase strength [76].

Surgical techniques for quadriceps tendon repair are different. Generally, for mid-substance ruptures, end-to-end sutures are used, while for ruptures closed to the osteotendinous junction, patellar drill holes or anchors are largely employed. Augmentation techniques are applied in presence of poor quality of tendon tissue or in case of delayed surgery, whenever a quadriceps retraction is present. Technical evolution of surgical materials has enabled an improvement in terms of results and biocompatibility [66–71].

After surgery, according to the biological processes of tendon healing and some clinical evidences, 6 weeks seems to be the most appropriate period of immobilization; after this period, it is essential to plan a proper and customized rehabilitative pathway for functional recovery of the knee, in order to prevent and/or avoid joint stiffness and functional impairment [23–28, 30].

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Chapter 7
Iliotibial Band Syndrome (ITBS)

Marco Merlo and Sergio Migliorini

Abstract Iliotibial band syndrome (ITBS) is an overuse injury caused by ripetitive friction of the iliotibial band and underlying bursa or lateral synovial recess across the lateral femoral epicondyle. ITBS is a well known cause of lateral knee pain in endurance sports and particularly in long distance runners. Friction occurs near foot strike, predominantly in the foot contact phase at, or slightly below, 30° of knee flexion. The development of ITBS is usually caused by a combination of intrinsic and extrinsic factors, and it must be evaluated also the biomechanics of running and the training program. Imaging (MRI, sonography) is not usually required to confirm the diagnosis of ITBS. The conservative treatment includes activity modification, soft tissue therapy, electrotherapeutic modalities, corticosteroid injection and correction of the biomechanical abnormalities. The personal ITB surgical release, performed by the authors, may be indicated if the conservative management fails and it is a safe procedure to return to practice sports at the same level.

7.1 Introduction

Iliotibial band syndrome (ITBS) is an overuse injury caused by repetitive friction of the iliotibial band (ITB) and underlying bursa across the lateral femoral epicondyle. ITBS is the most common causes of lateral knee pain in runners, with 12 %

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incidence of all the overuse injuries related to running [1, 2]. Renne, in 1975, first described the ITBS in marines, who underwent to heavy endurance training [3]. Considering the success of road endurance races in these years, the ITBS diagnosis is now really frequent. Other sports disciplines are involved in that pathology, such as triathlon, football, and cycling.

7.2 Etiopathogenesis

The ITB is considered a continuation of the tendinous portion of the tensor fascia latae muscle and is indirectly attached to parts of the gluteus medius, gluteus maximus, and the vastus lateralis muscles. The intermuscular septum connects the ITB to the linea aspera femoris until just proximal to the femoral lateral epicondyle. Distally, the ITB spans out and inserts on the lateral border of the patella, the lateral patellar retinaculum, and Gerdy tibial tubercle. The ITB is only free from bony attachment between the superior aspect on the lateral femoral epicondyle and Gerdy tubercle [4, 5]. A bursa, like synovial tissue, insinuates under the ITB and acts as an interface between ITB and lateral femoral epicondyle [6].

The ITB assists the tensor fascia latae as it abducts the thigh and controls and decelerates adduction of the thigh.

With knee in full extension and until 20–30° flexion, the ITB lies anteriorly to the femoral lateral epicondyle and acts as an active knee extensor. From 20° to 30° of knee flexion, the ITB is posterior to the femoral lateral epicondyle and acts like an active knee flexor [7]. During walking and running, the ITB is an important stabilizer of the lower limb.

A biomechanical study of runners found that the posterior edge of the band impinges against the lateral epicondyle just after footstrike in the gait cycle, and the friction occurs at or at slightly less than 30° of knee flexion. Orchard [8] suggests that the impingement, in runners who had ITBS, occurs in the foot contact phase at an average 21.4° ± 4.3° angle knee flexion, at or slightly below the 30° of flexion traditionally described in the literature (Fig. 7.1).

As in the other overuse injuries, the ITBS has intrinsic and extrinsic causes [9] (Table 7.1).

The most important extrinsic factors are the training errors (sudden increases in mileage or intensity, hill running and time trial training, lack of warm-up, insufficient muscle stretching execution, and bad shoes) inadequate to the running biomechanics. In a triathlon, the transition from cycling to running, with the change from concentric muscular contractions of cycling to eccentric contraction of running, and from unloaded cycling phase to the load state of running is an extremely delicate phase in which some kilometers are required to regain neuromuscular efficiency and elasticity indispensable for proper running style [10]. In this phase the inability to dissipate the load forces of the locomotor apparatus by the lower limb can favor the transmission of stress to the knee. The runner's training at lower pace and the high impact running style induce the friction of ITB with less than 30° knee flexion at the footstrike.

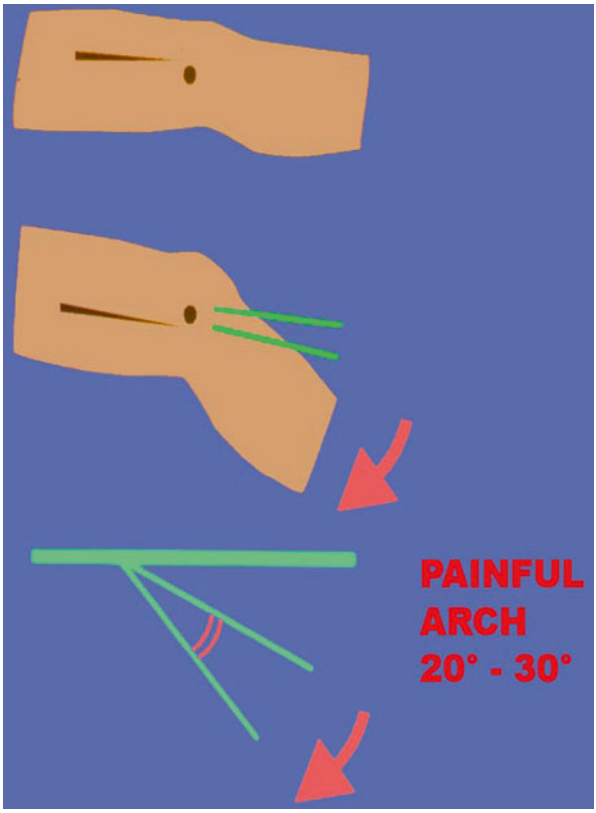

Fig. 7.1 During running the friction between ITB and lateral femoral epicondyle occurs at 20–30° of knee flexion

Table 7.1 ITBS intrinsic and extrinsic factors

Intrinsic	Extrinsic
Varus knee	High impact running styles
Internal tibial rotation	Training errors
Cavus-varus foot	Hill running
Femoral antetorsion	Insufficient muscle stretching
Knee lateral laxity	Bad or inadequate running shoes
Excessive foot pronation	Incorrect cleat pedal and saddle alignment
Hip abductors weakness	Cycling to running transition in triathlon
Lower leg discrepancies	

In cycling the abnormal lateral knee stress is most often the result of incorrect cleat pedal alignments, particularly with varus knee or external tibial rotation greater than 20°, when riding with internally rotated cleats. Generally this injury has increased since the introduction of rigid clip less pedal systems in 1985. Also a too high saddle results in the knee extension behind 150° causing the distal ITB to abrade across the lateral femoral epicondyle.

The surfaces of activity can also contribute to the development of ITBS in runners: running on surfaces with excessive camber can put excess strain on the lateral aspect of the knee. Downhill running tends to be worse because of the decrease in knee flexion that is present at the time of footstrike, thus increasing the force experienced by the knee within the Orchard impingement zone [8].

> Anatomic factors that contribute to ITBS include excessive varus knee, excessive internal tibial torsion, foot pronation, hip abductor weakness, lower leg length discrepancies, cavus foot, femoral antetorsion. Fredericson [11] found that runners with ITBS had significant weakness in the hip abductors of their affected limb; the fatigued runner or those who have a weak gluteus medius are prone to increase thigh adduction and internal rotation at midstance, leading to an increased valgus vector at the knee. These condition increases tension on the ITB, making it more prone to impingement to the lateral femoral epicondyle, especially during the foot contact, when maximal deceleration absorbs ground reaction forces. In leg length discrepancy, the ITBS is more frequent in the lower leg due to the increasing of varus forefoot and increased knee Q angle [12].

Sixty five percent of athletes with ITBS are male, due to greater training volume and more number of practitioners [1]. In women the incidence of ITBS is less frequent than men because of anatomical factors: valgus knee, medial knee laxity, femoral lateral epicondyle less prominent.

Also the lack of muscular elasticity of the tensor fascia latae, knee, and hip flexors is an injury risk factor, particularly in the triathletes that compete in a triathlon after years of cycling competitions [10].

Other authors sustain a different pathogenesis of ITBS. Fairclough [13] suggests that ITB overuse injuries may be more likely to be associated with fat compression beneath the ITB rather than with repetitive friction as the knee flexes and extends. Ekman [14] and Hariri [15] consider that the inflammation of the bursa underlying the ITB is the only cause of ITBS.

7.3 Patient Evaluation

The main symptom of the ITBS is sharp pain or burning on the lateral aspect of the knee. The patient typically localizes the pain to the region of the distal ITB between the lateral femoral condyle and its insertion on the Gerdy tubercle. Runners often note that they start running pain-free but develop symptoms after a reproducible time or distance. If ITBS progresses, pain can persist even during walking, particularly when the patients ascent or descent stairs. In some cases the runner with ITBS can perform painless other sports activities, like skiing, basketball, and football.

The knee examination is typically negative except for local tenderness and, occasionally, swelling over the distal ITB where the band moves over the lateral femoral epicondyle.

Some provocative tests are commonly used in the assessment of ITBS and ITB function. Application of the direct pressure to the distal fibers across the lateral femoral epicondyle as the athletes flexed and extended the knee from 90° to 180° usually

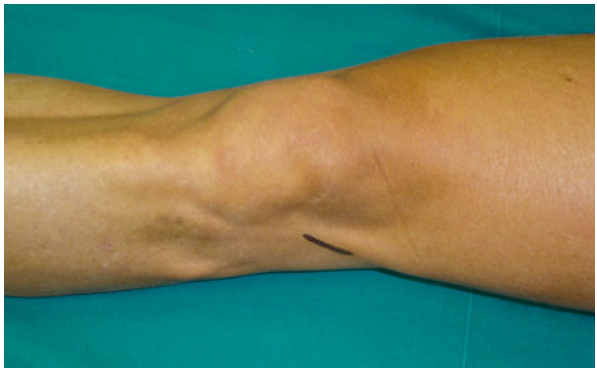

Fig. 7.2 Migliorini-Merlo sign. With the fully extended knee, it can observe an evident sulcus between the ITB and the lateral femoral epicondyle

elicited a very positive response (Holmes test). The Noble's test is performed with the patient lying supine; beginning with the affected knee flexed at 90°, the leg is extended with direct pressure over the lateral femoral epicondyle, with reproducible pain near 30° of knee flexion. The Thomas test is used to determine the tightness of the iliopsoas muscle, rectus femoris muscle, and ITB. The patient is instructed to lie supine at the edge of the examination table with both knees held to the chest. The patient holds the unaffected leg to the chest, and the affected leg is extended and lowered. A positive test results if the patient cannot completely extend and lower the affected leg to horizontal. Useful is also the Migliorini-Merlo sign: with the patient lying supine and the completely extended knee, it can be observed in the case of ITBS an evident sulcus between the ITB and the lateral femoral epicondyle (Fig. 7.2).

In particular cases it's important to perform an evaluation of biomechanics of running, evaluation of triathletes and cyclists on their bicycles, bikes, running shoes, orthosis, and shock absorbers review.

The differential diagnosis includes: lateral meniscal pathology, popliteal or biceps femoris tendinopathy, patellar syndrome, common peroneal nerve injury, lateral myofascial thigh pain, and osteochondral lesion of lateral femoral condyle.

7.4 Imaging

Although not routinely required, radiography imaging can be used to supplement the physical examination. Routine radiographs of the knee, including AP, lateral, and sunrise view can be used as a diagnostic adjunct to rule out other possible causes of the lateral knee pain, such as lateral joint space narrowing from degenerative disease, patellar maltracking, and stress fractures.

Ultrasonography of the knee lateral compartment is the first radiological evaluation considering low cost and availability of this modality compared with MRI. Usually ultrasonography performs a study to measure ITB thickness and the underlying bursa.

All athletes were examined with MRI to confirm the diagnosis because ITBS may be confused with other derangement of the knee, such as lateral meniscal tear, popliteal tendinopathy, lateral collateral ligament strain, hamstring strain, lateral femoral condyle osteochondral lesion, or transchondral fracture. Some authors [14, 16] reported the presence of high intensity signal, representing a fluid-filled collection, over the lateral epicondyle deep to the ITB as well as a marked thickening of the distal ITB. Eckman [14] demonstrated a significantly thicker ITB over the lateral femoral epicondyle. Thickness of the ITB in the disease group was 5.49 ± 2.12 mm, as opposed to 2.52 ± 1.56 mm in the control group.

7.5 Conservative Treatment

Nonsurgical management is the main stay of treatment of symptomatic ITBS.

In the acute phase the therapy must reduce the pain, and we suggest to rest from the inciting activities, such as running or cycling, for 2–3 weeks, until pain has resolved. Swimming with a pool buoy, without using lower extremities, normally is the only sport activity allowed in acute phase. Daily cryotherapy is useful in this phase and should be incorporated into the physical therapy program in an effort to reduce the inflammatory component of the ITBS.

Oral nonsteroidal anti-inflammatory drugs (NSAIDs) can be used to reduce the acute inflammatory response, and these alone have not been found to be effective in providing symptom relief. On the contrary local corticosteroid injections can relieve pain as well as aid in the diagnosis of ITBS, after 3 days of pain and, particularly, infiltrating the underlying bursa. Local corticosteroid infiltrations are effective and safe in the early (first 14 days) treatment of recent onset ITBS [17].

Therapy with platelet-rich plasma (PRP) should be an opportunity, but actually it needs more studies to determine the real efficacy.

Physical therapy is an important component of the nonsurgical management of ITBS. Typical regimens consist of specific stretching exercises focused on the ITB, tensor fascia latae, hamstrings, and gluteus medius. Stretching is indicated also for the iliopsoas, rectus femoris, and gastrocnemius and soleus muscles when restrictions are noted on physical examination. Stretching exercises are started after acute inflammation subsides. Additionally, the patient can use a foam roller as a myofascial release tool to break up soft-tissue adhesions in the ITB. In the subacute phase, it's important for the treatment of fascia lata and vastus lateralis trigger points with deep stripping massage, compression, fibrolysis, and mesotherapy. Shock waves should show good results if applied to the ITB and to the trigger points in the musculotendinous junction of the distal vastus lateralis. Manual therapy that consists of soft-tissue and medial patella mobilization may also contribute to lengthening the ITB.

Once the patient is capable of performing the stretching regimen without pain, strengthening is added to the rehabilitation program. Attention is paid to proximal strengthening of the hip abductors (gluteus medius) and the core muscles to stabi-

lize the pelvis to prevent excessive adduction of the hip [7]. The initial exercise is side-lying leg lifts to help the patient learn to isolate the gluteus medius muscle. All strength exercises, should start with one set of 20 repetitions and build up to free sets of repetition daily. Because training is specific to limb position, it is essential that the patients progress to weight-bearing exercises. Initially the patients are asked to perform a step-down exercise; once this is mustered patients are taught to perform the pelvic drop exercises [11].

During the subacute phase, the athlete can return to cycling, easy pedaling (80 rpm or less) with a little resistance at a pain-free cadence on flat terrains. Return to running depends on the severity of the condition and patient's premorbid function. We recommend running, every 2 days, starting with easy sprints on level ground over slow jogging; biomechanical studies have shown that faster-paced running is less likely to aggravate the ITBS [8]. We suggest gradual increase in distance running and frequency, avoiding hill training and intensive training (too much, too soon). Most of the patients have symptomatic relief without surgery within 6–8 weeks and are capable of returning to their athletic activities with no long-term sequelae.

Great attention should be focused on sport equipment, considering running shoes and biomechanics of the foot contact on the ground, the bike saddle position (avoiding knee extension beyond 150°), and the incorrect cleat pedal alignment (varus knee or external tibial rotation more then 20 % causes a significant stress on distal ITB when riding with internally rotated cleats). The hyperpronated runner should use stable running shoes and change the shoes every 500–600 miles, considering that shoes lose almost 60 % of their capacity to absorb ground reaction forces [18].

Length discrepancies of the lower legs should be corrected with orthosis for the running shoes or with thickness between cycling shoes and cleat pedals. In some cases it is useful putting inside the shoes the shock absorbers, in order to increase the knee flexion at the footstrike.

7.6 Surgical Treatment

Surgical treatment is indicated only after extensive nonoperative measures have failed to relieve symptoms (3–6 months).

Holmes [19] suggests two procedures: percutaneous release and the open release excision. The percutaneous incision is performed under local anesthetic with the knee held at 90° so that the posterior fibers are free from the lateral femoral epicondyle. The open release surgery consists of the excision of an ellipse tissue measuring 4 cm at the base and 2 cm at the apex.

Martens [20] resected from the posterior part of the ITB a triangular portion 2-cm wide at the posterior base and 1.5-cm in height. The ITB Z-lengthening is also an option described by Richards [9]. Hariri [15] suggests an open ITB bursectomy. Michels [21] described an arthroscopic technique to treat refractory ITBS using a synovial shaver to resect the lateral synovial recess accessed through an accessory superolateral portal.

Our personal surgical procedure, usually in local anesthesia and in day surgery hospital, is performed by a small access (less than 2 cm) and consists of a subtotal vertical tenotomy of the ITB with the knee held at 70° of flexion. We don't remove the bursa underlying the ITB because of the high risk of hematoma and persistent synovial reaction. The ITB release is sufficient to remove the ITB impingement with the lateral femoral epicondyle and consequent reduction of the bursitis. Compression bandage and cryotherapy is mandatory at the end of the procedure. In some cases we perform an arthroscopic evaluation of the knee joint to rule out other associated articular lesions, particularly involving the lateral meniscus (Fig. 7.3).

Postoperative protocol began immediately after surgery. Patients are allowed to be weight-bearing as tolerated with bilateral crutches until their gait normalized. Typically they are able to walk without crutches between 3 and 5 days postoperatively. Some athletes develop small seromas or hematomas near the incision during the 2nd postoperative week. These resolved spontaneously in 3–5 days with intermittent icing, local compresses, or aspiration. Passive range of motion for the hip and knee began on postoperative day 1 with full knee extension achieved by day 3 and full hip and knee flexion by the end of week 2. Patellofemoral joint mobilizations are also performed with specific emphasis on medial glides of the patella. A gentle massage and range of motion for the ITB are initiated in week 1 and progressed to gradual stretching in week 2.

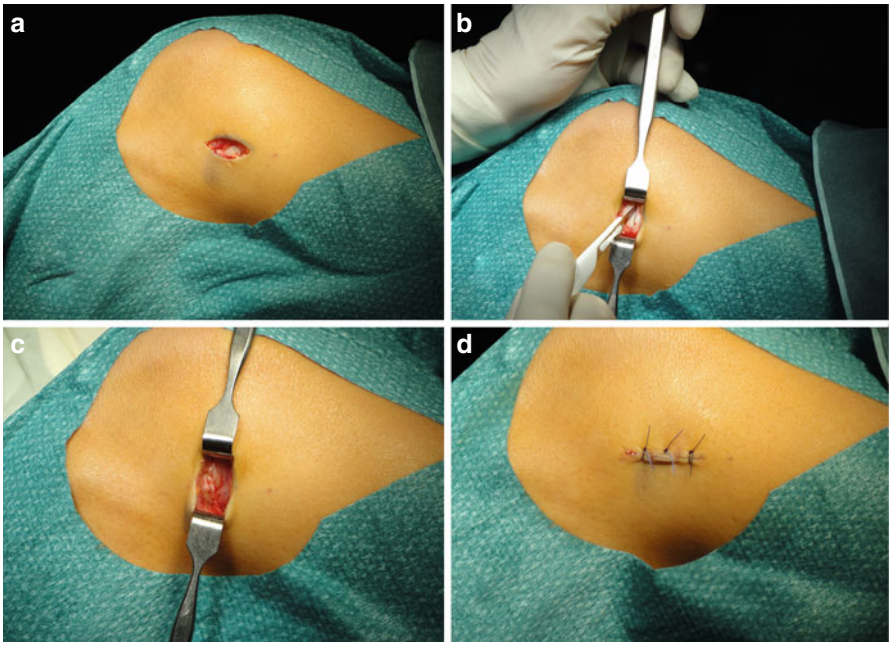

Fig. 7.3 Our personal procedure. (**a**) Approach to ITB; (**b**) vertical incision of ITB; (**c**) complete release of ITB; (**d**) skin suture

Isometric exercises for the hip abductors are performed during week 1 and progressed to active range of motion and progressive resisted isotonic exercises during week 2–4. The initial exercise is side-lying leg lifts to help the patient learn to isolate the gluteus medius muscle. For all strength exercises, the patients start with one set of 20 repetitions and build up to 3 sets of 20 repetitions daily. Because training is specific to limb position, it is essential that patients progress to weight-bearing exercises. Initially, athletes perform step-down exercises. Once this is mastered, patients are taught to perform the pelvic drop exercises. Neuromuscular electrical stimulation is applied to the quadriceps to prevent the effects of postoperative inhibition. The patients are revaluated by the operating surgeon at week 4 and cleared to begin a gradual progression of swimming and cycling as tolerated with a return to running between 6 and 8 weeks postoperatively. As a general rule, athletes can return to running once they can perform all strength exercises without pain. We recommend running every other day for the first 4 weeks, starting with easy sprints on level ground. Biomechanical studies [8] have shown that faster-paced running is less likely to aggravate ITBS because at footstrike the knee is flexed beyond the angles that cause friction. In our experience, according to Evans [4], the ITB release does not affect the biomechanics of the hip and knee.

7.7 Conclusions

ITBS is a common cause of lateral knee pain in the athletic patients' population, particularly in endurance sports. We focus the importance of the exact etiology underlying the condition that it is different for every athlete considering the intrinsic and extrinsic factors. Most patients with ITBS improve with aggressive protocol rehabilitation and activity modification. Surgery release of the ITB is indicated only after extensive nonoperative measures have failed to relieve symptoms. Between a variety of surgical management options, our personal surgical procedure reported good results and return athletes to pain-free athletic activity.

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Chapter 8
Hamstring Syndrome

Gian Nicola Bisciotti, L. Pulici, A. Quaglia, A. Orgiani, L. Balzarini, P. Felisaz, and Piero Volpi

Abstract The hamstring syndrome is classically considered an insertional tendinopathy concerning the proximal hamstrings. However, we must not forget its possible posttraumatic cause. In effect, its association with previous proximal hamstring lesion is well described in literature. It is a typical sport pathology that mainly affects sprinters, distance runners, soccer players, and nordic skiers. Its greatest incidence is between 29 and 37 years old. The first-choice treatment is conservative and the surgical treatment can be considered only in the case of a failure of conservative treatment.

8.1 Introduction

The hamstring syndrome (HS) was described for the first time from Puranen and Orawa in 1988 [1] and is considered an insertional tendinopathy of proximal hamstrings. Usually HS is associated to a previous proximal hamstring lesion with the production of fibrotic tissue or to a degenerative process that causes a widening of the proximal hamstring tendon which causes an irritation of the near sciatic nerve [1, 3]. The association with previous proximal hamstring lesion is well described [1, 3–7] and it represents from 19 to 76 % of cases [2, 5, 8–12]. At the beginning the HS

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was described as a sprinter's pathology [1], but our current knowledge is that this pathology is much frequent in distance runners, soccer players, and nordic skiers [1, 2, 5, 7]. The higher incidence is between 29 and 37 years old [5, 11, 13].

8.2 Clinical Diagnosis

Most patients with HS had an elective pain, with different intensity, when palpating proximal hamstring insertion [1, 2, 5, 7, 11], while manual tests of hamstrings or gluteal muscles can be normal [3, 8]. Also neurological tests are negative [5, 8]. Usually the patient refers a discomfort at proximal hamstring insertion during the test of flexibility, but these tests couldn't show a significant deficit [1, 5]. The hamstring-stretch test (HST) (Fig. 8.1) is positive if it evokes pain at proximal hamstring insertion [7]. Other similar tests are the Puranen-Orawa test (Fig. 8.2) and the bent-knee stretch test (Fig. 8.3). Since it often involves a hypotrophy of the abductor hip muscles [14], the Trendelenburg test could be positive at the affected site [2]. According to the involvement of sciatic nerve, patients can show a different grade

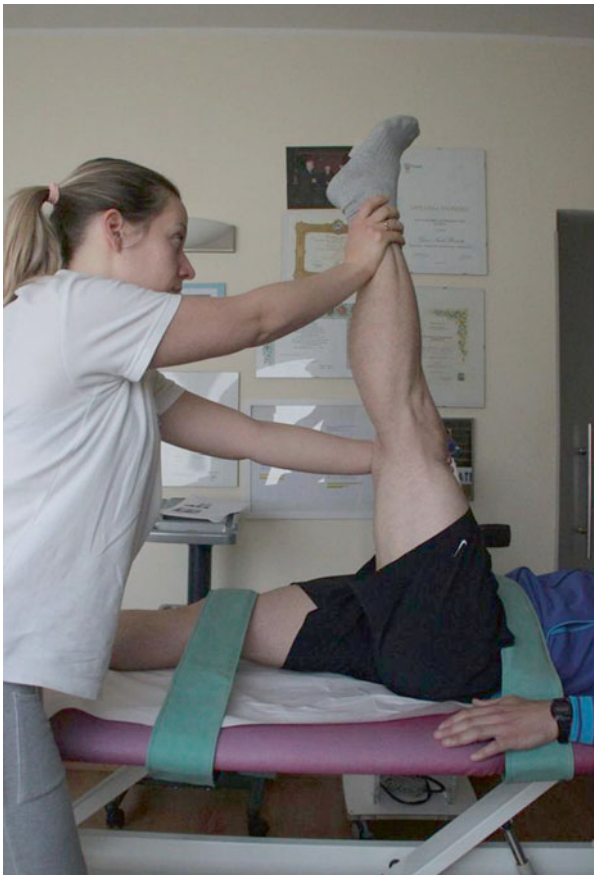

Fig. 8.1 In the hamstring-stretch test, the operator makes a rapid hip flexion followed immediately by an equally rapid extension of the knee. It is positive if it evokes pain at proximal hamstring insertion

Fig. 8.2 In the Puranen-Orawa test, the patient makes an active stretching of hamstrings during stance. It is positive if it evokes pain at proximal hamstring insertion

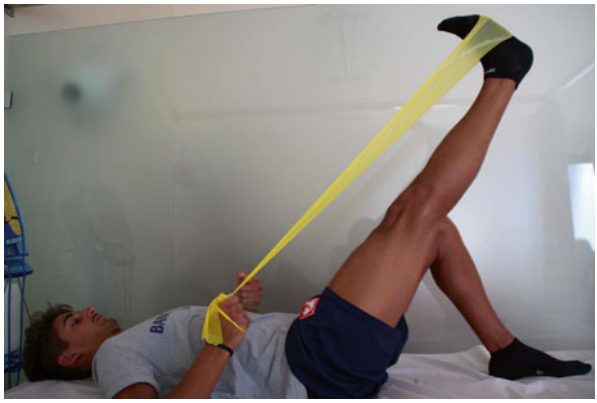

Fig. 8.3 In the bent-knee stretch test, the patient makes, using a band, a stretching of hamstring in supine position. It is positive if it evokes pain at proximal hamstring insertion

of radicular symptoms. Generally HS does not interfere with running but limits it greatly. Usually pain is evoked by running uphill and downhill and during sprinting, remaining "low" during running at low velocity and on flat ground. Other symptom referred is pain at ischiatic tuberosity when patients maintain sitting position especially on hard surfaces [2]. Differential diagnosis should consider [15]:

- Indirect lesions of hamstrings
- Piriform syndrome
- Ischiatic bursitis
- Sciatica
- Chronic compartment syndrome of the thigh
- Deep hematomas
- Soft tissue neoplasia

8.3 Imaging

Pathology of the hamstring complex may affect the cancellous bone and the cortex of the ischial tuberosity, the tendons, the myotendinous junction, the perimysial fascia, the muscle, and finally the sciatic nerve.

The diagnostic imaging approach requires high tissue contrast resolution, safety, and favorable cost/benefit ratio modalities.

Today MRI is considered the gold standard in diagnostic imaging.

Conventional plain films may play a role in detecting cortical bone changes and soft tissue calcifications adjacent to the ischial tuberosity, which are typical signs of chronic enthesopathy, typically seen in elderly patients. Conventional radiography allows also visualization of avulsed bone fragments in posttraumatic injuries.

CT should be limited to selected cases because of the poor soft tissue contrast and the high radiation dose on the pelvis. Nonetheless it may be required to better define the anatomy of an avulsed bone fragment.

Ultrasound is a safe technique, sensitive for the study of superficial structures such as the tendons, but do not provide information on the cancellous bone. Furthermore it suffers of inter-operator variability and hence skilled operators are required.

The imaging technique that can overview all such pathologic changes is MRI, with adequate detection of bone marrow edema, tendons or muscle strains or tears, and scar tissue formation.

MRI is a safe, repeatable examination with low inter-operator variability and allows great definition of the anatomy for the surgical repair.

On the coronal plane, the ischial tuberosity presents two facets; the superolateral or oblique facet is the side of attachment of the semimembranosus, while the inferomedial or horizontal facet is the side where the biceps femoris and the semitendinosus tendons attach as a conjoint tendon. Sometimes these two tendons are difficult to separate, reflecting anatomic variation. The tendon attachments are best seen in the coronal plane but verification of the findings in the axial plane is very important. The radiologist must be aware of the attachment of the adductor magnum tendon at the inferomedial facet, which is intimately associated with the conjoint tendon, just adjacent to it but slight medially and anteriorly located, which may be a source of misdiagnosis if confused.

The type of injury is most easily defined with MRI, which in most cases does not require contrast injection. Hamstring injuries may be classified in strains (or partial tears) and avulsion (or complete tears).

Most strains occur in the region of the myotendinous junction (MTJ) [16], which is the weakest area linked to the muscle. However, the MTJ is not a distinct area but a 10–12-cm zone of transition in which muscle fibrils intersect with the tendon origin or ligamentous insertion. Hyperintensity in T2 fat suppressed or STIR images is typical of edema, fluid, and blood products that are commonly displayed between intact muscle fibers, creating a feathered appearance. Hemorrhage may be more important with major muscle strain; it is typically hyperintense in the acute setting and may track around the sciatic nerve.

Hamstring avulsion is defined by the detachment of one or more proximal tendons of the hamstring from the flat bony surfaces of the ischial tuberosity. In adults complete avulsion usually involves the tendons but not the bone. Tendon avulsion must be recognized with specification of the degree of retraction because it necessitates prompt surgical repair. Complete avulsion almost always involves the conjoint tendon (biceps femoris and semitendinosus) and most frequently a partial avulsion of the semimembranosus. The avulsion of all the three tendons is also common, while avulsion at one or two attachments is less frequent [17].

Acute injuries in pediatric patients often result with a bone fragment avulsion, detectable on plain films and by US.

In the immature skeleton, the apophysis forms the weakest link in the musculotendinous unit due to its incomplete ossification; these injuries sometimes lead to a large bone formation because of the typical great vascularity of the avulsed ossification center [18]. It is important to determine whether the attachment side is displaced more than 2 cm [19].

Chronic tears of the hamstring complex can be investigated with MRI to evaluate scar tissue formation. This scar tissue has low signal intensity with all pulse sequences.

On MRI long-standing injuries lead to disuse changes such as atrophy of the muscles with fatty replacement, best demonstrated on T1-weighted MR images. The sciatic nerve should be routinely assessed to demonstrate potential encasement, compression, or tethering.

On US the scar tissue is characterized by irregular shape and heterogeneous echotexture. Diffuse and focal thickening with hypoechoic change is characteristic of chronic tendinopathy. US combines good spatial resolution, with the ability of dynamic assessment that may provide additional useful information about the tendon integrity. Fluid collections around the tendons may be easily demonstrated. Contralateral evaluation is useful for comparison.

MTJ tears can be subtle at MR imaging and even more at US, because of the low contrast between the muscle and the interfascicular edema. Nevertheless, US is a sensitive imaging modality in the presence of blood products and edema, which increase with the conspicuity of muscle disruption. Color Doppler US can be used to assess neovascularization, inflammation, and healing, while elastography may theoretically assess the degree of muscle edema or connective tissue substitution.

8.4 Conservative Treatment

Conservative treatment is the first approach for HS; it should consider these points:

- A pelvic alignment
- Soft tissue mobilization
- Active and passive stretching of hamstring muscles
- Eccentric training of hamstring muscles
- Core stability

The extracorporeal shock wave therapy has limited evidence even if a recent study [7] showed possible benefit. Local injection of corticosteroids has a positive outcome in little more than 20 %, and the symptomatic relief is not more than 6 months [13]. Regenerative therapy, based on platelet-rich plasma, nowadays didn't show sufficient scientific evidence [20].

The eccentric training of hamstring muscles is the focus of the rehabilitation program [21–26]. None of the authors cited suggest the elimination of concentric phase during the exercises proposed, but they underline the importance of improving the eccentric phase with a reduction of the concentric one. According to our

experience, eccentric isokinetic exercises should be completed with isotonic exercises promoting eccentric phase.

We propose some exercises very helpful into the conservative treatment of HS (Figs. 8.4, 8.5, 8.6, and 8.7). This kind of exercise can be introduced as soon as the

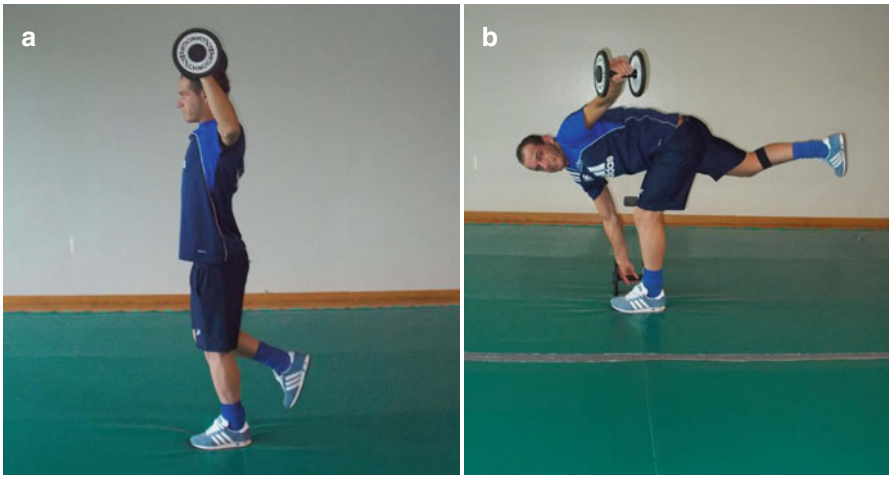

Fig. 8.4 Single-limb balance windmill touches with dumbbells: begin in (**a**) single-limb stance position with dumbbells overhead and (**b**) perform windmill motion under control with end position of touching dumbbell to the floor

Fig. 8.5 Eccentric backward steps. The subject resists the clinician attempt to push him/her backward. The injured leg is held back

Fig. 8.6 Eccentric forward pulls. The subject resists forward pull by the clinician

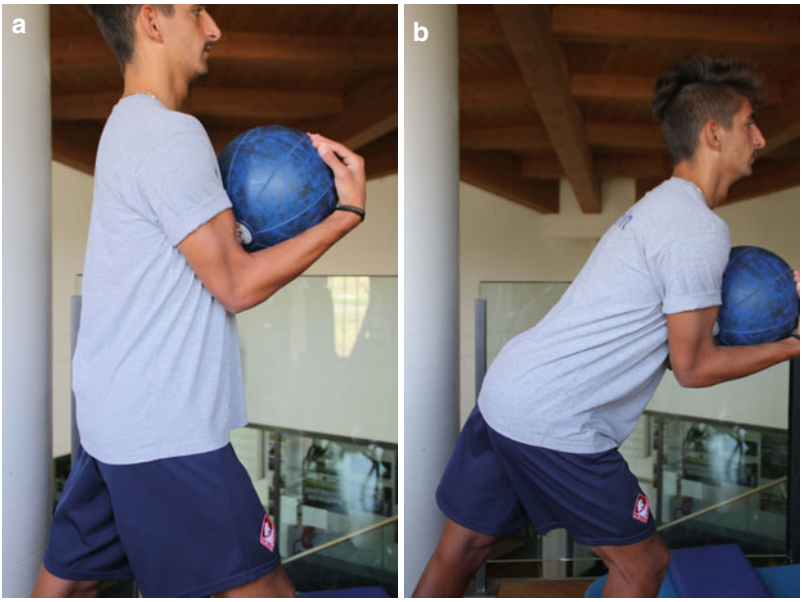

Fig. 8.7 Split stance exercise also called the "good morning" exercise. The subject is in a split stance position with the weight placed anteriorly to increase the lever arm on the hamstrings (**a**). The subject then leans forward through flexion at the hip (**b**)

first part of the conservative program, based on weight-bearing exercises made at low velocity, is tolerated from patients without discomfort [27].

Further the exercises described above, we find very interesting the introduction of a workout based on whole-body vibrations (WBV) (Figs. 8.8, 8.9, 8.10, 8.11, and 8.12). There are some studies that show how the WBV are able to improve elongation capacity of hamstrings [28, 29]. This effect is attributable to the myorelaxant effect induced by vibration at particular frequency (18–20 Hz) [30]. According to this aspect of WBV, we recommend the introduction of this program [31, 32] into the conservative treatment of HS.

8.5 Surgical Treatment

A surgical approach to HS is not often easy to choose. Obviously this choice is limited to those cases where the conservative treatment has failed [5, 11]. In the preoperative evaluation, the close proximity between the proximal insertion of hamstrings and the sciatic nerve should be taken into account, because an edematous or thickened tendon can compress the sciatic nerve or could form adhesion between these structures [5].

Fig. 8.8 Stretching of posterior kinetic chain. The subject must make a flexion/extension of the patella, forcing an extension to increase the stretch of the muscle tendon complex
Frequency, from 18 to 20 Hz
Vibration amplitude, from 2 to 3 mm
Acceleration peak, between 25.5 m.s−2 (2.6 g) and 47 m.s−2 (4.8 g)
Set, from 3 to 5
Working time, 1′
Rest time, 2′

Fig. 8.9 Stretching of paravertebral muscle with Freeman platform, combined with vibrations. It's important to combine a full expiration to the muscular spine stretching
Frequency, from 18 to 20 Hz
Vibration amplitude, from 2 to 3 mm
Acceleration peak, between 25.5 m.s−2 (2.6 g) and 47 m.s−2 (4.8 g)
Set, from 3 to 5; working time, 1′
Rest time, 2′

Fig. 8.10 Stretching exercise for back chain muscle
Frequency, from 18 to 20 Hz
Vibration amplitude, from 2 to 3 mm
Acceleration peak, between 25.5 m.s−2 (2.6 g) and 47 m.s−2 (4.8 g)
Set, from 3 to 5; working time, 1′
Rest time, 2′

Fig. 8.11 Stretching exercise of piriformis muscle and sacrotuberous ligament
Frequency, from 18 to 20 Hz
Vibration amplitude, from 2 to 3 mm
Acceleration peak, between 25.5 m.s−2 (2.6 g) and 47 m.s−2 (4.8 g)
Set, from 3 to 5; working time, 1′
Rest time, 2′

Fig. 8.12 Prolonged stretching exercise of posterior kinetic chain
Frequency, from 18 to 20 Hz
Vibration amplitude, from 2 to 3 mm
Acceleration peak, between 25.5 m.s−2 (2.6 g) and 47 m.s−2 (4.8 g); set, from 3 to 5
Working time, 1′
Rest time, 2′

The patient is positioned in the prone position. As surgical approach, a transverse incision along the gluteal fold or a posterior longitudinal approach can be used. To identify the proximal insertion of the hamstrings, the lower edge of the gluteus maximum should be moved proximally, after identification of the posterior femoral cutaneous nerve. At this point a transverse tenotomy of semimembranosus muscle at about 3–4 cm from its origin can be performed, and the biceps femoris and semitendinosus muscles were left intact. The tendon sectioned is fixed to biceps femoris to avoid excessive retraction. Once the tendon is sutured, the sciatic nerve is explored and it is released from any adhesions.

Postoperative treatment consists of an elastic bandage for about 2 weeks and a progressive load until reaching the full weight bearing in about 2 weeks. Swimming is allowed in 2 weeks from surgery while isometric exercises and cycling at 4 weeks. Running and more intensive workouts can be performed after 2 months. The return to sport is after at least 2 months.

The rehabilitation period after surgery lasts between 60 and 90 days, and the follow-up (2–10 years) report results defined as "excellent" or "good" between 77 and 88 % of the cases considered.

8.6 Conclusion

The HS diagnosis is not easy, and for this reason it can often be misunderstood. Often a HS framework is interpreted as a simple tendinopathy or an indirect muscle injury; as consequence the therapeutic process is based on a misdiagnosis. This leads to a lengthening of recovery times and especially to the risk of a disease chronicity. In this case a careful evaluation of the patient's medical history can help the clinician in identifying the correct diagnosis. Upon reaching a definitive diagnosis (that is possible only after the exclusion of all differential diagnoses), the patient must be routed, as first choice, to a conservative treatment. In any case the clinic and the patient should be aware that the conservative treatment involves a long and complex pathway. The surgical treatment is limited only when any type of conservative treatment is completely failed.

Note The authors thank Dr. Jose Manuel Sanchez (Centro de Alto Rendimiento de Barcelona) for the use of the Figs. 8.8, 8.9, 8.10, 8.11, and 8.12.

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Chapter 9
Pes Anserine Tendinopathy

S. Lupo and Gian Nicola Bisciotti

Abstract Pes anserine tendinopathy can hit sartorius, gracilis, and semitendinosus muscle tendons in their insertion area. Overweight, biomechanical overburden, and improper posture could predispose to this pathology. In fact they provoke excessive friction on the common tendon insertion and underlying bursa that may cause overload work for these muscles and lead to inflammation. This pathology occurs mainly to long-distance runners, young athletes (because of their premature beginning of the sport practice), and especially in women. Symptoms consist in strong pain and burning sensation underneath and inside the knee, where pes anserine tendons insert. In most cases, tendinopathy is provoked by continual mechanical stress causing repeated microtraumas. Therapy consists in rest and appropriate therapeutic and physiotherapeutic treatments.

9.1 Introduction

The pes anserine (PA) or goose's foot is formed by the tendinous insertion of the sartorius (SA), gracilis (GR), and semitendinosus (ST) muscles (Fig. 9.1). This relative strange name of goose's foot is due to the fact that the three muscles attaching to the medial side of tibia form a shape reminiscent of a goose's foot. PA is a structure clinically important in the reconstructive surgery; in fact PA tendons are commonly used as autografts in ligamentous reconstruction of the

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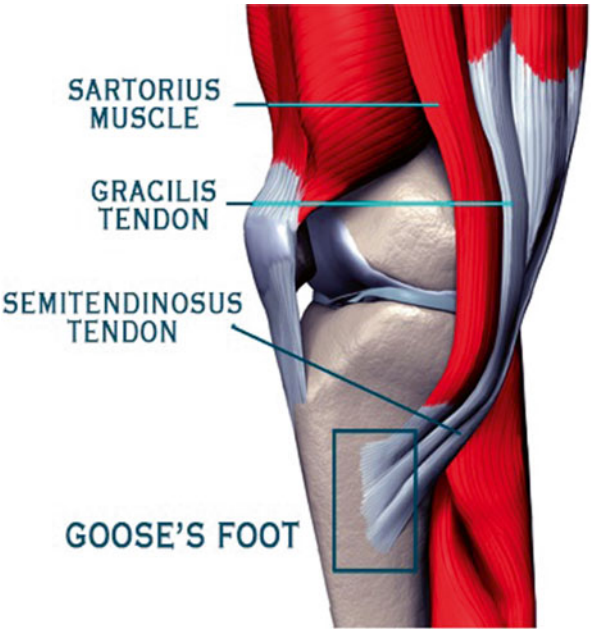

Fig. 9.1 The insertion of sartorius, gracilis, and semitendinosus muscles in the medial area of the knee creates a structure that looks like a goose's foot (after whom it is named)

knees [1, 2]. Studies on cadavers have shown that the SA tendon fascia is intimately attached to the superficial fascial layer; on the contrary the GR and ST tendons are located in the deep surface of the superficial fascial layer over the medial aspect of the tibia [3]. PA presents a bursa, the anserine bursa (AB), that is located at the upper medial aspect of the tibia, in correspondence of the insertion of the conjoined tendon of the PA muscles. Some study [1] reported the presence of accessory bands in gracilis and semitendinosus tendons. The muscle forming PA are main flexors and internally rotators of the knee [4]. In particular SA muscle flexes, abducts, and externally rotates the thigh, while in knee-flexed position flexes and internally rotates the leg (Fig. 9.2). The ST muscle flexes and internally rotates the leg (in a knee-flexed position), extends, and abducts the thigh (Fig. 9.3). The GA muscle adducts and flexes the thigh and flexes and internally rotates the leg (Fig. 9.4).

9.2 Etiopathogenesis of PA Tendinopathy

The first description about PA tendinopathy that we can find in literature dates back to 1937, in that year Moschcowitz [5] described medial knee pain in a female population. The observed patients reported pain when going downstairs

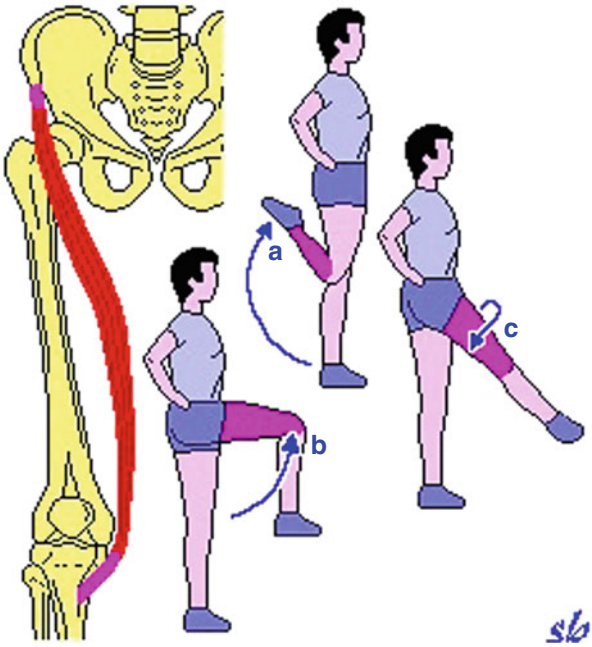

Fig. 9.2 Sartorius muscle flexes (**a**), abducts (**b**), and externally rotates the thigh (**c**), while in knee-flexed position flexes and internally rotates the leg

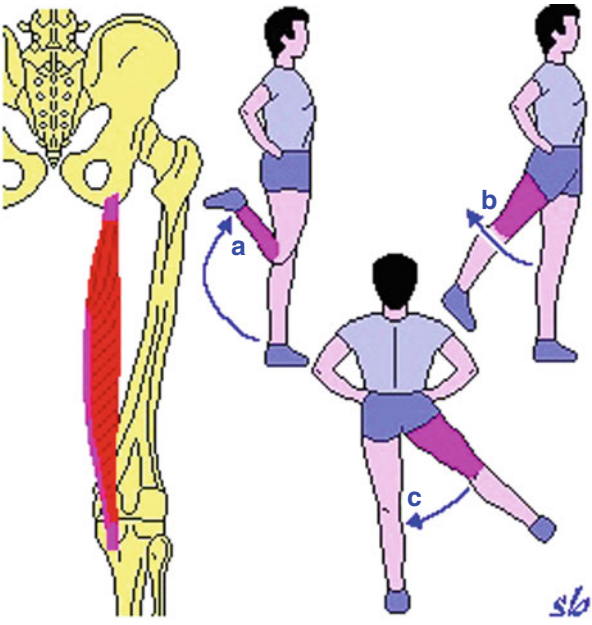

Fig. 9.3 Semitendinosus muscle flexes (**a**) and internally rotates the leg (**b**) (in a knee-flexed position) and extends and abducts the thigh (**c**)

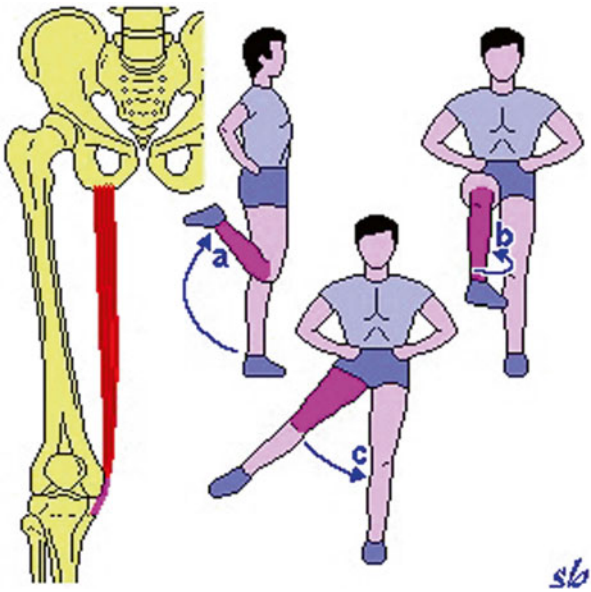

Fig. 9.4 The gracilis muscle adducts and flexes the thigh and flexes and internally rotates the leg

or upstairs, upon rising from a chair, or referred difficulty when flexing the knee. PA tendinopathy is a degenerative condition that can hit these tendons in their insertion area, and it is often linked with AB bursitis. The causes of onset of this pathology may be of intrinsic and extrinsic origin; between the intrinsic causes, we can remember the overweight, a malalignment of the knee, in particular a valgus knee, flatfoot, and postural alteration especially of the pelvis. All these condition may cause an excessive friction on the common tendon insertion [6, 7]. Also diabetes mellitus is known as a predisposing factor [8]. Cases of chronic bursitis concerning AB are well documented in patients with rheumatoid arthritis and osteoarthritis [6, 9, 10]. The intrinsic causes include retraction of posterior thigh muscles, bone exostosis, irritation of the suprapatellar plica, damage to the medial meniscus, and infection [11]. The extrinsic causes are mainly represented by an increase of overuse or trauma as often happens in sport [7]. This pathology occurs mainly in long-distance runners or to young athletes, in this case probably because of their premature beginning of the sport practice [12]. Female gender seems to be more likely to develop PA tendinopathy. This greater predisposition in women is probably due to the fact that women have a wider pelvis, resulting in greater knee Q angle value which leads to more pressure in the area of insertion of the PA [13–15]. In effect, AB bursitis is more common in overweight females with osteoarthritis of the knees [13–15]. In literature we can find several studies based on different imaging modalities focused on the identification if the patients affected by PA tendinopathy suffer only for tendinopathy or from bursitis of AB or both [16–18]. In effect little is known regarding the anatomical

structural defect that may be responsible for this pathology. For this reason there is in literature a lack of consensus and some controversies that have led some authors to suggest the term "anserine syndrome" for this specific condition [9]. As of our knowledge until today, no prospective study based on determination whether these patients really suffer from tendinopathy or bursitis or both has been undertaken.

9.3 Clinical Evaluation

Usually the inflammation often comes out after biomechanical overuse, with pain and burning sensation, in the lower internal part of the knee, where PA tendons insert. Symptoms may be particularly evident also in daily leaving activities as going up and down the stairs, or sometimes the pain appears strongly after the patient has been seated a lot and he stands up. Rarely it is possible that an edema appears on PA level, especially if simultaneously there is an AB bursitis. The inflammatory pain persists even at rest; sometimes burning sensation appears during the night, impeding the patient from falling asleep [9]. Diagnosis is imminently clinical also if the history of the patient can be of great help. Patient's history, who complains pain in the medial part of the knee in case of sedentary subject, especially if they are overweight, is often correlated with signs of degenerative joint disease. On the contrary patient's history in case of athlete is often correlated with an overload situation. The clinical evaluation is based on the deep palpation of the affected area; also it is important to note that 30 % of asymptomatic individuals experience pain during deep palpation of the medial area of the knee [7]. Differential diagnosis is based on lesions at the medial meniscus, L3-L4 radiculopathy, lesions at medial collateral ligament, panniculitis, suprapatellar bursitis, prepatellar bursitis, synovial osteochondromatosis, malignant tumors (fibrous histiocytoma, liposarcoma, and synovial sarcoma), varicose popliteal vein, popliteal aneurysm, infectious complication of surgical procedure, osteonecrosis affecting the medial tibial plateau, patellofemoral syndrome, patellar chondromalacia, recurring patellar subluxation, and direct trauma. Frequently, pain in the anserine region is part of the fibromyalgia syndrome [19]. Another differential diagnosis to consider is represented by semimembranosus bursitis (SB). SB, also called semimembranosus tibial collateral ligament bursitis, is an inflammatory process of the semimembranosus bursa that is within the superficial and deep layers of the medial collateral ligament and involves the anterosuperior margin of the semimembranosus tendon [20]. In the same manner, we must also consider iliotibial bursitis (IB) as differential diagnosis. The ITB is an inflammatory process regarding the iliotibial bursa. The iliotibial bursa is located between the distal portion of the iliotibial band (near its insertion in the Gerdy tuberculum) and the adjacent tibial surface. The ITB can be associated with iliotibial tendinitis and it is usually due to overload and varus stress [21].

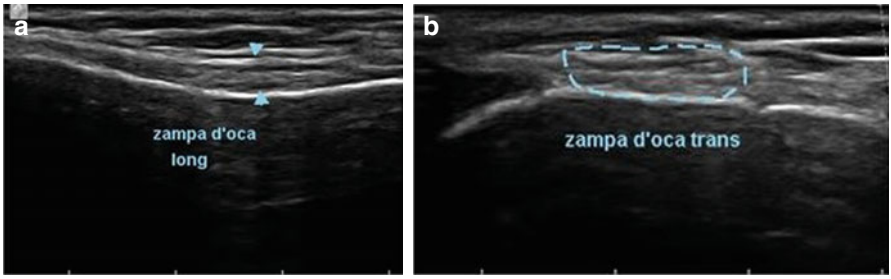

Fig. 9.5 Ultrasound longitudinal examination (**a**) and axial examination (**b**) of pes anserine in normal anatomical situation

9.4 Imaging Assessment

Also if the diagnosis is imminently clinical, this latter can be corroborated by imaging exams. In this regard it is important to remember that relatively few studies using imaging exams assessed the morphological characteristics of the PA tendons insertion and respective AB in a patient population that has clinical diagnosis of anserine syndrome [6, 16, 17, 22–25]. These studies showed substantially that only a minority of patients with clinical diagnosis of anserine syndrome have compatible anatomical ultrasound changes. Then, since the ultrasound might not detect the abnormalities produced by a PA tendinopathy and/or AB bursitis, MRI might present a contribution. In any case the current literature shows that ultrasound, CT, and MRI findings usually do not allow the identification of anatomical structure responsible for the symptoms in PA tendinopathy and/or AB bursitis. In Fig. 9.5 we report a PA ultrasound normal anatomical situation.

9.5 Conservative Treatment

The treatment starts, first of all, with an initial period of rest and abstinence from the daily activities potentially dangerous. In overweight subjects it is very important to lose weight. The initial treatment should include resting and cryotherapy. The use of a pillow between the thighs at nighttimes is strongly recommended. Physiotherapy has an important role in the treatment of PA tendinopathy and AB bursitis. The use of ultrasound has been documented as effective in the reduction of the inflammatory process [26]. Stretching of SA, GR, and ST is strongly recommended. In effect an improvement in flexibility obtainable through a systematic program of passive and active stretching can promote an important reduction in the tension on the anserine bursa [27]. The medical treatment involves the use of FANS. Another possible option is represented by the injection of local anesthetic associated with corticosteroids in the AB in cases of proven AB bursitis. Some authors suggest the use of 20–40 mg of methylprednisolone [26, 28, 29]; triamcinolone, 20–40 mg; or betamethasone, 6 mg [30]. In any case no more than three infiltrations should be done

over a 1-year period; furthermore the interval time between infiltrations should be greater than 1 month. It is important to note that rarely the patients who do not show any positive response to an initial infiltration respond to repeated infiltrations. Some studies have demonstrated that infiltration with corticosteroids represent a valid good option in well-selected patients [9, 31, 32]. However, it is important to note that cortisone injections are not without risk that is represented by atrophy of the subcutaneous tissue, skin depigmentation, and tendon rupture [30, 33].

9.6 Surgical Treatment

Surgical treatment is rare and obviously is indicated in case of failure of the conservative treatment. In the case of AB bursitis, it consists in an incision followed by drainage of the distended bursa; generally this simple surgical act can provide improvement of symptom [9, 26, 28, 34].

In cases of exostosis, the surgery includes excision of the bursa and the removal of any bony exostosis [34]. However, in literature we can find the description of the cases in which the removal of the bursa was necessary [11]. In case of important tendinopathy associated with AB bursitis, arthroscopic debridement can be considered [35].

9.7 Conclusions

Many patients that show pain in the medial compartment of the knee receive the diagnosis of PA tendinopathy, AB bursitis, or both these conditions. However, the diagnosis is based only on the presence of pain on palpation in the anatomic site, and when patients are submitted to imaging exams these last do not confirm the clinical diagnosis. Very often in effect, especially in certain patients (i.e., female patients in overweight), a panniculitis of the fat tissue of the medial compartment can be exchanged with PA tendinopathy or AB bursitis. Instead in the case of osteoarthrosis, it is very important to underline that both PA tendinopathy and AB bursitis must be considered as secondary or associated pathology. It is therefore very likely that a true PA tendinopathy and AB bursitis (i.e., that can be considered as primary pathology) can be found only in athletes.

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Chapter 10
Achilles Tendinopathy

Nicola Maffulli, Alessio Giai Via, and Francesco Oliva

Abstract Achilles tendinopathy is a common cause of disability. Despite the economic and social relevance of the problem, the causes and mechanisms of Achilles tendinopathy remain unclear. Tendon vascularity, gastrocnemius-soleus dysfunction, age, sex, body weight and height, pes cavus, and lateral ankle instability are considered common risk factors. Currently, intratendinous degenerative changes are considered responsible for tendinopathy and symptoms. Although Achilles tendinopathy has been extensively studied, there is a clear lack of properly conducted scientific research to clarify the optimal management option. The management of Achilles tendinopathy lacks evidence-based support, and patients are at risk of long-term morbidity with unpredictable clinical outcome. Most patients respond to conservative management. Eccentric exercises and shockwave provide excellent clinical results both in athletic and sedentary patients, with no reported adverse effects. However, in about 20–45 % of cases, patients do not respond well to conservative treatment, and they need surgery. Both minimally invasive and open surgeries have been described with similar results.

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10.1 Introduction

Achilles tendinopathy (AT) is characterized by pain, swelling, impaired performance ability, and pathologic changes in and around the tendon. In chronic AT, symptoms have been present longer than 6 months [1]. The incidence has risen in the past three decades as a result of greater participation in sporting activities. AT is estimated to occur in 7–9 % of top-level runners. A tenfold increase in Achilles tendon injuries has been reported in runners compared to age-matched controls [2]. AT is also common in individuals participating in racquet sports, track and field, volleyball, and soccer. However, the condition is not confined to athletes because 30 % of patients have a sedentary lifestyle [3].

10.2 Etiopathogenesis of Achilles Tendinopathy

The etiopathogenesis of AT remains unclear. It is currently considered multifactorial, and the interaction between extrinsic and intrinsic factors has been postulated [1]. Extrinsic factors such as increased frequency and duration of training have been associated with AT. Changes in training pattern, poor technique, previous injuries, footwear, and environmental factors such as training on hard, slippery, or slanting surfaces may predispose athletes to tendinopathy [4]. However, a retrospective study showed that tendinopathy was not necessarily associated with the level of physical activity [5]. Some drugs and antibiotics such as fluoroquinolones and corticosteroids have been recognized as risk factors of tendinopathy and Achilles tendon ruptures. Ciprofloxacin causes enhanced interleukin-1b-mediated MMP3 release, inhibits tenocytes proliferation, and, like corticosteroids, reduces collagen and matrix synthesis [6].

Intrinsic factors include lower limb anatomy, biomechanics, previous injuries, gender, age, metabolic disorders, and systemic diseases. Lower limb alignment and biomechanical faults are claimed to play a causative role in two-thirds of athletes with Achilles tendon disorders. Malalignment and hyperpronation impose excessive strain on the Achilles tendon, and hyperpronation of the foot has been linked with an increased incidence of AT [7].

The incidence of tendinopathy is lower in women than men, and estrogen levels might play an important role in tendon homeostasis [6]. Women showed a lower risk of tendinopathy during premenopausal years than man, but, after menopause, this risk increases [8]. Postmenopausal estrogen deficiency seems to downregulate collagen turnover and to decrease elasticity in tendon. An animal study showed poorer Achilles tendon healing in estrogen-deficient rats compared to controls [9], and Cook et al. reported that the Achilles tendon health of women using hormone replacement therapy was better than the controls [10]. However, little is known about the effect of estrogen on tenocytes. A recent in vitro study showed that proliferation and tenocytes biosynthesis are negatively affected by age and estrogen

deficiency [11]. Tenocytes from ovariectomized and old rats showed a significantly lower proliferation rate, a decreased collagen type I synthesis, and overexpression of MMPs compared to young controls, showing that aging and, more significantly, estrogen levels may affect tendon metabolism and healing.

Connective tissue aging is associated with compromised tissue function, increased susceptibility to injury, and reduced healing capacity. This has been partly attributed to collagen cross-linking by advanced glycation end products (AGEs) that accumulate with both age and chronic diseases, in particular diabetes mellitus [12]. Protein glycation is a spontaneous reaction depending on the degree and duration of hyperglycemia, the half-life of the protein, and permeability of the tissue to free glucose. Glycated proteins can undergo further reactions giving rise to poorly characterized structures called advanced glycation end products (AGEs). AGEs are complex, heterogeneous molecules that cause protein cross-linking, which alter physical characteristics of collagen fibers, and they may affect the tendon properties [13]. The effects of AGEs on the mechanical properties of tendon collagen fiber have been recently studied in a rat model [14]. The formation of AGEs would change the way tendon reacts to loading, in particular significantly reducing collagen fiber sliding. On the other hand, tendons try to compensate this loss of function by increasing collagen fiber stretch which may have potentially important implications for predisposing collagen fibrils to damage during everyday use. Tissue stiffness does not appear to be significantly affected. Therefore, physiological loads in aged and diabetic tendons could involve fiber "overstretching" that leads to accelerated accumulation of damage.

The relationship between thyroid disorders and joint pain has been suspected since the late 1920s [15], but it has not been systematically investigated. Thyroid hormones (THs) play an essential role in the development and metabolism of many tissues. Thyroxine (T4) is important for both collagen synthesis and extracellular matrix (ECM) metabolism. A recent study demonstrates that TH nuclear receptors are present on tenocytes and that, in vitro, THs enhance tenocytes growth and counteract apoptosis in healthy tenocytes isolated from tendon in a dose- and time-dependent manner [16]. This may be reason why the incidence of tendon tears is higher in patients affected by hypothyroidism. [17].

The physiopathology of tendinopathy in obese patients has yet to be understood, but some studies show that obesity may affect tendon health and reduce its healing ability [18]. Many authors consider obesity as a risk factor for tendons injury [19]. Anatomic studies show that Achilles tendons are significantly thicker in obese individuals than in a control group [20], and ultrasonography showed thicker and hypoechogenic tendons in obese subjects compared to normal individuals [21]. Histological changes have been observed in animal studies. Lipid drops accumulate in the extracellular matrix [22], and disorganized collagen fibrils are observed at transmission electron microscopy in the tendon ECM of obese animals [23]. Low levels of glycosaminoglycans (chondroitin and dermatan sulfate), which play an important role in the regulation of the ECM and collagen fibrillogenesis, have been detected, and they may be responsible for the inadequate deposition and organization of collagen fibrils [24]. Finally, obesity is frequently associated with other

pathologies, such as diabetes mellitus and insulin resistance, which may also play a role in tendon pathology.

Many studies currently advocate the importance of extracellular matrix (ECM) for the homeostasis of connective tissue, and its physiological and pathological modifications seem the most important intrinsic factors involved in tendinopathies and tendons ruptures [25]. The turnover of ECM in normal tendons is mediated by matrix metalloproteinases (MMPs) [26], MMP-1, MMP-2, and MMP-3 [27]. They are able to denature collagen type I. After tendon rupture, the activity of MMP-1 increases, while a reduction of MMP-2 and MMP-3 has been showed [28]. An increase in MMP-1 activity and degradation of the collagen type I network is a potential cause of the weakening of the tendon, and it may contribute to tendinopathy and rupture. These findings may represent a failure of the normal matrix remodeling process.

Transglutaminases (TGs) are also implicated in the formation of hard tissue development, matrix maturation, and mineralization [29]. Nine different TGs have been found in mammalian. TG2, also known as tissue transglutaminase, is widely distributed within many connective tissues, and it is implicated in organogenesis, tissue repair, and tissue stabilization. An animal model showed a reduction of TG2 protein expression in tendinopathic supraspinatus tendons [30]. TGs are important in maintaining the structural integrity of tendons thanks to their mechanical or cross-linking function in normal condition, and the fall of TG2 may mean the exhaustion of the reparative capabilities of the tendon.

10.3 Achilles Tendinopathy: Why Painful?

Although significant advances have been made in understanding its pathogenesis, relatively little is known about the source of pain associated with AT. Intratendinous degenerative changes are believed to be the responsible of symptoms. However, intratendinous degenerative changes often occur without symptoms. This is the reason why Achilles tendon rupture often occurs without previous symptoms: histopathological changes alone cannot be the only reason for pain. Recent research identified the peripheral and central pain processing pathways as an important factor in the pathogenesis of painful human tendinopathy, and changes in the peripheral neuronal phenotype may be the primary source of pain [31]. Peripheral neuronal phenotype refers to specific characteristics of the peripheral nervous system including nerves, neuronal mediators, and receptors.

Healthy tendons are relatively aneuronal. The neural supply to the Achilles tendon and the surrounding paratenon is provided by nerves from the attaching muscles and by small fasciculi from cutaneous nerves, in particular the sural nerve [32], but they do not have a rich nervous supply [33]. Indeed, chronic painful tendons present ingrowth of new nerve fibers accompanying the peritendinous neovascularization from the paratenon into the tendon proper [33]. Alfredson et al. demonstrated increased neural ingrowth in AT versus control [34]. The authors found

strong evidence of an upregulation of the glutaminergic system in painful human tendinopathy, and weaker, but still suggestive, evidence that changes in the peripheral neuronal phenotype were related to variations in pain among patients. Glutamate is a key metabolite and neurotransmitter involved in the transmission of pain. Glutamate NMDAR1 receptors have been frequently noted in morphologically altered tenocytes in tendon tissue proper and in the peritendinous connective tissue [35]. There is also some evidence for upregulation of the substance P/CGRP system, which has been linked with increased nociception in both animal models and human disease [31]. Substance P is also associated with vasodilation and stimulates the proliferation of fibroblasts [36], possibly causing some of the morphologic changes in AT.

10.4 Clinical Aspects of Achilles Tendinopathy

Clinical history and examination are essential for diagnosis. A detailed history helps to identify the onset and possible contributing factors. Pain is the main symptom. Pain is usually referred 2–6 cm proximally to the tendon insertion [33]. Initially, pain occurs at the beginning and a short while after the end of a training session with a period of diminished discomfort in between. Runner typically report pain at the beginning ad at the end of the training session, with a pain-free period in the central part [37], but as the pathological process progresses, pain may occur during the entire exercise session. In severe cases, it may interfere with activities of daily living. A good correlation between severity of symptoms and morning stiffness has been reported [38].

On examination, both lower limbs should be exposed, and the patent should be examined standing and prone. The lower limb should be inspected for malalignment, deformities, muscular hypotrophy, and previous scars. The Achilles tendon should be palpated for tenderness, increased local temperature, thickening, and crepitation (Fig. 10.1).

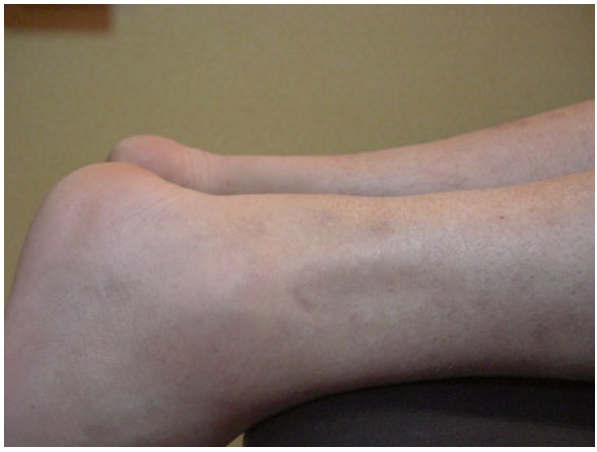

Fig. 10.1 Tendinopathy of the right Achilles tendon

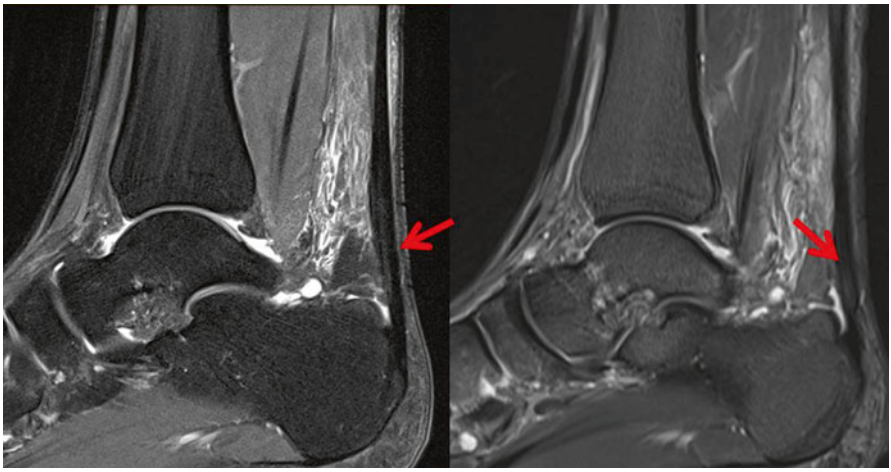

Fig. 10.2 MRI imaging of Achilles tendinopathy (*arrow*)

Plain radiography can be used to diagnose associated bony abnormalities or a calcific tendinopathy [39]. Ultrasound, despite being operator dependent, correlates well with the histopathologic findings, and it is an effective imaging method [40]. MRI provides extensive information on the internal morphology of the tendon and the surrounding structures (Fig. 10.2) and is useful in evaluating the various stages of AT and in differentiating between paratendinopathy and tendinopathy. Areas of mucoid degeneration are shown on MRI as a zone of high signal intensity on T1- and T2-weighted images [41]. Thickening of the Achilles tendon can be easily demonstrated at both MR and ultrasound.

10.5 Prevention of Achilles Tendinopathy

As the management of AT lacks evidence-based support, patients are often at risk of long-term morbidity with unpredictable clinical outcome. The rehabilitation protocols and treatments are long and time consuming. For these reasons, exercise programs which aim to prevent AT are important. However, even if various preventive interventions protocols have been described, there is still little evidence on their effectiveness. A recent systematic review of literature tried to report the current evidence about preventive exercise programs for tendinopathies [42]. Stretching exercise is an empirically accepted and commonly used method to prevent sports injuries and tendinopathy. However, there is no evidence for a positive effect of stretching exercises alone [42–44]. But, when stretching exercises were used together with exercise programs, a significant reduction in the incidence of tendinopathy was reported. Kraemer and Knobloch found that a program that included soccer-specific balance training can significantly reduce the incidence of patellar and Achilles tendinopathy [45]. They also found a dose-effect relationship between duration of balance training and injury incidence.

In addition to stretch and exercise interventions, shoe adaptations such as orthoses are used in the prevention and treatment of lower extremity tendinopathies. Three articles focused on the use of orthoses are available in current literature [46–48], but only House and colleagues found a statistically significant difference with the use of shock-absorbing insoles on the incidence of AT in a military population [49].

Cook et al. [10] studied the effects of activity levels and hormonal status on Achilles tendon structure in asymptomatic postmenopausal women. They found that active postmenopausal women had a greater prevalence of tendon abnormalities and thicker Achilles tendons than inactive women and that hormone replacement therapy seems to reduce the risk for structural Achilles tendon changes in this group of women.

10.6 Conservative Treatment of Achilles Tendinopathy

Many different conservative treatments as physical therapy, rest, training modification, splintage, taping, cryotherapy, electrotherapy, shock wave therapy, hyperthermia, pharmaceutical agents such as NSAIDs, and various peritendinous injections have all been proposed. Managements that have been investigated with randomized controlled trials include nonsteroidal anti-inflammatory medication, eccentric exercise, glyceryl trinitrate patches, electrotherapy (microcurrent and microwave), sclerosing injections, and shock wave treatment [49].

Eccentric training was first advocated in the management of tendinopathy over two decades ago by Stanish et al. [50]. The concept of eccentric exercises is based on the structural adaptation of the musculotendinous units. However, the mechanism for the efficacy of eccentric loading is largely unknown. A possible mechanism which seems to enhance tendon healing is mechanical conditioning [51]. Mechanotransduction is the process of a cell converting mechanical stimuli into biochemical signals. Tendon responds to mechanical forces by adapting its metabolism, structural, and mechanical properties [52]. Alfredson et al. reported decrease of neovascularization after an eccentric training intervention at color Doppler sonography [53]. Several studies reported good short- and long-term results after eccentric training superior to controls [53–55]. Significant improvement in patient satisfaction and decreased pain were seen in 60–90 % of patients [56] (Fig. 10.3).

Shock wave therapies (SWTs) are widely used for the management of tendon disorders. Two different types of SWs are used in clinical practice, extracorporeal shockwave therapy (ESWT) and “radial shock wave therapy” (RSWT) or “radial pulse therapy” (RPT). ESWTs are concentrated into small focal areas of 2–8 mm diameter. Ultrasound guide is needed to focus the shock wave, and refocusing of the applicator is periodically necessary. RSWTs are not focused, and, once located, the area to treat is included within the wave propagation area, and they do not need periodical refocusing of the applicator. The disadvantage is that they do not have a penetrating effect on tissue but act superficially. Lohrer et al. reported significant pain reduction and increased functionality in patients with AT who

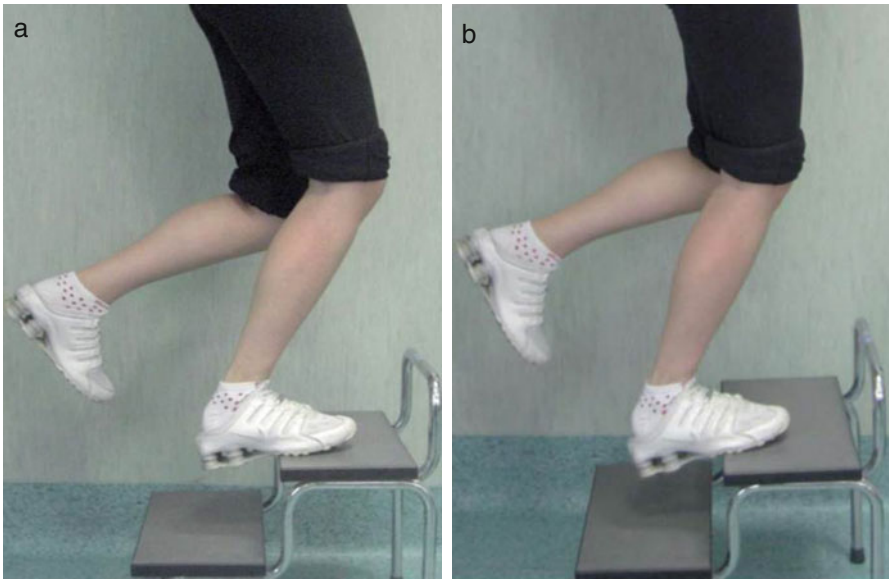

Fig. 10.3 (**a**) Eccentric exercise with the knee straight, to maximize the activation of the gastrocnemius muscle. (**b**) Eccentric exercise with the knee bent, to maximize the activation of the soleus muscle. Please note that, in clinical practice, it is sufficient to undertake the eccentric exercises only with the knee straight

received RSWT [57]. In a small RCT of 39 patients, Peers reported a 77 % success rate at 12-week follow-up [58]. In a RCT, Rompe et al. compared the effectiveness of three management strategies [41]. Group one was treated with eccentric loading exercises, group two was treated with repetitive low energy SWT, and group 3 was treated with a "wait-and-see" approach. At 4 months follow-up, clinical results did not differ significantly between group 1 and 2, but they were significantly better than group 3. A recent systematic review [59] reported some evidence of benefit with high-energy ESWT in midportion AT [60] and lack of benefit of low-dose ESWT [61].

10.7 Minimally Invasive Treatment

Even though open surgery can provide good results, wound complications are frequent. Minimally invasive techniques reduce the risks of infection and wound breakdown and are inexpensive and technically easy to perform. The rationale behind most of these techniques is to break the neovessels and the accompanying nerve supply which may be responsible of pain in most of patients. They can also be associated with other minimally invasive procedures to optimize results, providing great potential for the management of chronic AT.

10.7.1 High-Volume Injections

High-volume image-guided injections (HVIGIs) target the neurovascular bundles growing from the paratenon into the Achilles tendon. Several substances have been injected in and around tendons including normal saline, corticosteroids, and local anesthetics [62, 63]. The injection is performed under ultrasound guidance to avoid intratendinous injections. Patients are allowed to walk on the injected leg immediately, but they are strictly advised to refrain from high-impact activities for 72 h. After this period, they are instructed to restart eccentric loading physiotherapy regime twice daily until they stop their sporting career. Good results have been reported with this technique at short-term follow-up [64]. Platelet-rich plasma injections did not show the expected benefits for AT when administered in randomized controlled trial conditions [65].

10.7.2 Minimally Invasive Achilles Tendon Stripping

The patient is placed prone. Four skin incisions 5 mm length are made, two at the proximal origin of the Achilles tendon, just medial and lateral to the origin of the tendon, and the other two 1 cm distal to the distal end of the tendon insertion on the calcaneus. A mosquito is inserted in the proximal incisions, and the Achilles tendon is freed of the peritendinous adhesions. A number 1 unmounted Ethibond (Ethicon, Somerville, NJ) suture thread is inserted proximally, passing through the two proximal incisions (Fig. 10.4). The Ethibond is retrieved from the distal incisions, over the anterior aspect of the Achilles tendon. Using a gentle seesaw motion, the Ethibond suture thread is made to slide anterior to the tendon (Fig. 10.5), which is stripped and freed from the fat of Kager's triangle (Fig. 10.6).

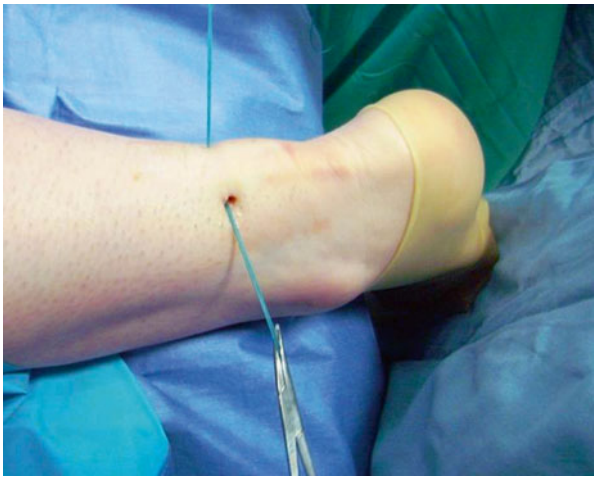

Fig. 10.4 The suture is inserted proximally through the two proximal incisions

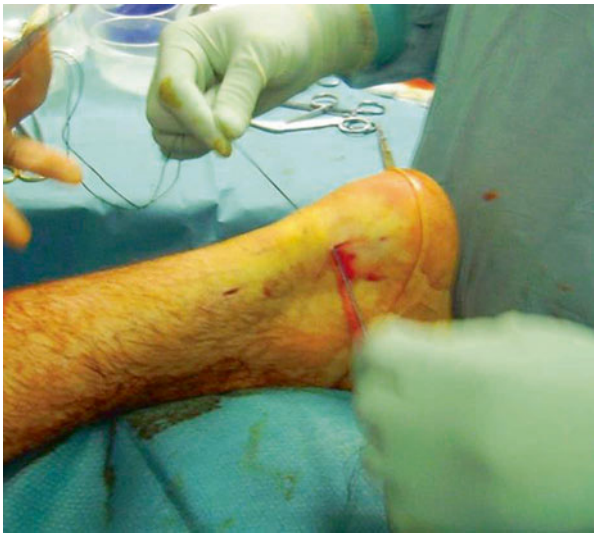

Fig. 10.5 The suture is slid over the anterior aspect of the Achilles tendon with a gentle seesaw motion

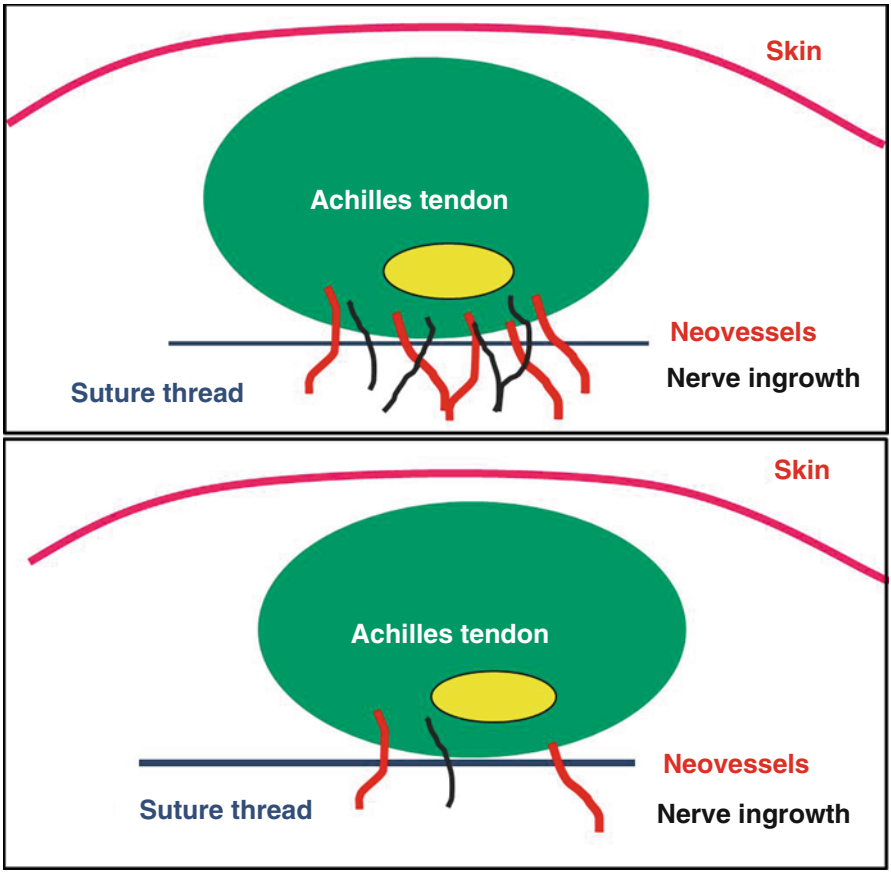

Fig. 10.6 Figure showing the rationale behind minimally invasive Achilles tendon stripping. The suture aim to break vascular and neural ingrowth from the paratenon which is responsible of pain

If necessary, using an 11 blade, longitudinal percutaneous tenotomies parallel to the tendon fibers are made.

Postoperatively, patients are allowed to mobilize fully weight bearing. After 2 weeks, patients start physiotherapy, focusing on proprioception, plantar flexion of the ankle, inversion, and eversion.

10.7.3 Percutaneous Longitudinal Tenotomies

Percutaneous longitudinal tenotomy can be used when there is no paratenon involvement and when the intratendinous lesion is smaller than 2.5 cm. The procedure can be performed under ultrasound guidance [66]. The patient lies prone on the operating table, and a bloodless field is not necessary. The tendon is accurately palpated, and the area of maximum swelling and/or tenderness marked and checked again by US scanning. The skin and the subcutaneous tissues over the Achilles tendon are infiltrated with 10–15 mL of plain 1% lignocaine. A number 11 surgical scalpel blade is inserted parallel to the long axis of the tendon fibers in the marked area in the center of the area of tendinopathy. The cutting edge of the blade points caudally and penetrates the whole thickness of the tendon (Fig. 10.6). Keeping the blade still, a full passive ankle dorsiflexion movement is produced. The scalpel blade is then retracted to the surface of the tendon, inclined 45° on the sagittal axis, and the blade is inserted medially through the original tenotomy (Fig. 10.7). Keeping the blade still, a full passive ankle flexion is produced. The whole procedure is repeated inclining the blade 45° laterally to the original tenotomy, inserting it laterally through the original tenotomy. Keeping the blade still, a full passive ankle flexion is produced. The blade is then partially retracted to the posterior surface of the Achilles tendon, reversed 180°, so that its cutting edge now points cranially, and the whole procedure repeated, taking care to dorsiflex the ankle passively. Preliminary cadaveric studies showed that a tenotomy 2.8 cm long on average is thus obtained through a stab wound in the main body of the tendon [63].

The patient is operated as day case. Early active dorsi- and plantar flexion of the foot are encouraged. On the second postoperative day, patients are allowed to walk

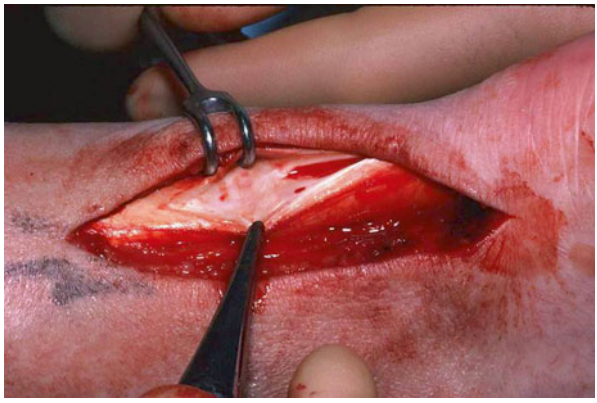

Fig. 10.7 Open excision of degenerated Achilles tendon area

with 2 crutches and weight bearing as able. Stationary bicycling and isometric, concentric, and eccentric strengthening of the calf muscles are started under physiotherapy guidance after 4 weeks. Swimming and water running are encouraged from the second week. Gentle running is started 4–6 weeks after the procedure, and mileage gradually increased.

We reported excellent and good results in 63 % of athletes with unilateral AT treated with ultrasound-guided percutaneous longitudinal tenotomy after failure of conservative management, without experienced significant complications [67].

10.7.4 Open Surgery

Under locoregional or general anesthesia, the patient is placed prone with the ankles clear of the operating table. A tourniquet is applied to the limb to be operated on. The incision is made on the medial side of the tendon to avoid injury to the sural nerve and short saphenous vein. The skin edge of the incision should be handled with extreme care, as wound healing problems are serious. The paratenon is identified and incised. In patients with evidence of coexisting paratendinopathy, the scarred and thickened tissue is generally excised. The tendon is incised sharply in line with the tendon fibers bundles. The tendinopathic tissue can be identified as it generally has lost its shiny appearance, and it frequently contains disorganized fiber bundles that have more of a "crabmeat" appearance (Fig. 10.7). This tissue is sharply excised. The remaining gap can be repaired using a side-to-side repair, but we leave it unsutured. If significant loss of tendon tissue occurs during the débridement, a tendon augmentation or transfer should be consider, even if we rarely undertake this additional procedure. The subcutaneous tissues are sutured with absorbable material; the skin edges are juxtaposed with Steri-Strips and a routine compressive bandage. The limb is immobilized in a below-knee weight-bearing cast with the foot plantigrade. A period of initial splinting and crutch walking allows pain and swelling to subside after surgery. After 14 days, the wound is inspected, and motion exercises are initiated. The patient is encouraged to start daily active and passive ankle range-of-motion exercises. The use of a removable walker boot can be helpful during this phase. Early weight bearing after uncomplicated procedures is encouraged. However, extensive débridements and tendon transfers may require protected weight bearing for 4–6 weeks postoperatively. After 6–8 weeks, more intensive strengthening exercises are started, gradually progressing to plyometrics and eventually running and jumping.

A systematic review of the literature showed successful results in over 70 % of cases [68], but these relatively high success rates are not always observed in clinical practice, probably because of the poor methods scores of many articles. Patients should be informed of the potential failure of the procedure, risk of wound complications, and at times prolonged recovery time. Possible complications of this surgical procedure are wound breakdown (3 %), sural nerve injury (1 %), superficial or deep infection (2.5 %), and deep vein thrombosis [69]. Achilles tendon rupture has been reported after surgery for tendinopathy in a patient performing eccentric loading exercises.

10.8 Synthesis and Therapeutic Indications

Achilles tendinopathy is a common cause of disability both in athletic and sedentary population. The etiopathogenesis is currently considered multifactorial, but precise pathogenetic mechanisms are still largely unclear.

Even though there is a consensus on the importance of preventive exercises programs and different protocols have been proposed, the evidence on their long-term effectiveness is little. Eccentric exercises are realistically effective to manage AT in up to two-thirds of patients.

There is some evidence for benefit for high-energy ESWT in midportion AT. However, conservative management is unsuccessful in 24–45.5 % of patients, and surgery is recommended after 6 months of conservative management. Long-standing AT is associated with poor postoperative results, with a greater rate of reoperation before reaching an acceptable outcome.

Both minimally invasive and open surgical techniques have been described. Good results have been reported with minimally invasive techniques, even though well-conducted level I evidence studies are lacking. They offer many advantages like reduced local morbidity and risks of infection, they are technically easy to perform and relatively inexpensive, and they can be associated with conservative treatment or other minimally invasive procedures to optimize the results.

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Chapter 11
Patellar Tendinopathy

Piero Volpi, E. Prospero, C. Bait, G. Carimati, V.P. Di Francia, P. Felisaz, and L. Balzarini

Abstract Patellar tendon tendinopathies have always been an important and recurring issue in many sports. Even in the case of the patellar tendon, the first hypothesis of inflammatory pathogenesis has been replaced by the degenerative theory, and the term "tendinitis" was soon replaced with that of "tendinopathy." In addition to the hypothesis inflammatory, over the years, several theories have been formulated in the context of its pathogenesis. Conservative treatment proposed by various authors and that is possible to find retrievable in the literature ranges from functional rest, the use of nonsteroidal anti-inflammatory drugs, modification of training techniques, stretching, and eccentric training. However, a not insignificant number of athletes demonstrate nonresponsiveness in respect of conservative therapies, and in front of a long period of persistence of symptoms, a surgical solution must be considered.

11.1 Introduction

Patellar tendinopathy is a common overuse condition of the patellar tendon and represents an important problem for athletes of different sports.

It most commonly affects jumping athletes from young age to adulthood, and it is an acute/chronic injury of patellar tendon.

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Basketball and volleyball are the sports more interested by these pathologies, respectively, with an incidence of 31.9 and 44.6 % [1] mostly because of the mechanics of sport-specific gestures, characterized by high-speed and power demand on the extensor apparatus. This disease was described in 1973 by Blazina as "jumper's knee" and this definition has been preserved until now [2].

Clinically it is characterized by an anterior pain load-dependent at the inferior pole of the patella, to the tendon or at distal insertion of it.

The majority of cases resolve with nonsurgical treatment; rest, cryotherapy, eccentric exercise, extracorporeal shockwave therapy, magnetic fields, and PRP therapy are the most commonly used.

Patellar tendinopathy implies a difficult management by the athlete because of the severe symptomatology and severe impairment of sports performance or because of the long period of stop; more then half of the athletes still have some symptoms after their carrier ended [3–5]

Chronically stressed tendons, at the last stage of the degenerative process, can end in tendon rupture.

In this chapter, we will discuss about the pathology, clinical and instrumental diagnosis, and therapeutic approach.

11.2 Epidemiology and Etiopathogenesis

Patellar tendinopathy is very common among athletes and nonathletes [6].

Mainly it involves people who repeat flexion extension movement under load or, as cited above, sports player with an incidence of up to 40 % in certain sports; an important role is played by overload and reduced loading capacity by the athletes.

Prevalence of patellar tendinopathy ranges from 5 to 45 % in sports players [7–9] with an incidence peak between 15 and 30 years – it is the period in which most patients practice sport activities.

Prevalence is higher in males and affected persons usually are between 15 and 40 years.

Different risk factors for the diseases are described and can be classified as intrinsic and extrinsic.

Intrinsic risk factors cannot be modified and can give information about which groups have to be monitored:

- Gender: the incidence is higher in male than females. This was due to different capacity in generating force and to the ability of the athletic movement [1]. Estrogens may have a protective role on the tendons [10].
- Age: the tendinopathy is considered to be linked to overload, so the greater the age the greater the stress on the tendon at the same level of sport activity.
- Rheumatologic diseases and collagenopathies: they can change the structure of the tendon and decrease the mechanical resistance.

- Injury of the contralateral tendon: patients with a contralateral overload can be more susceptible for developing pathologies of the other tendon.
- Anatomy: there are different opinions about anatomical features predisposing to jumper's knee tendinopathy [11].

Extrinsic risk factors are the most interesting for the professional sportsman and are the once for whom it's possible to apply preventive measures.

- Environment and playground: in volleyball and basketball players, it was shown that a harder ground increases the risk of developing the tendinopathy [1]. In recreational players, no differences were indicated between the incidence of the pathology in athletes playing on natural ground and the incidence in athletes playing on synthetic ground.
- Sport-specific gestures: it is impossible to modify them but it is possible to improve the technique of execution to reduce the load and the frequency of execution in at risk or symptomatic people.
- Equipments: mechanical properties of footwear can change the friction coefficient and the mechanical load on the limb. It is important to choose the best footwear according to the features of the ground, in order to reduce microtrauma from impact with the ground. More often knee bandages are used to reduce the load on the tendon in athletes who have early symptoms, but their benefit is doubtful.
- Exposition level: many hours of training and sport are the most important risk factors for the development of patellar tendon pathology [3, 12]. Some authors have shown a relation between the risk of developing the jumper's knee and 12 h/week training; the same link has been pointed out with 5 h/week training with weights [13, 14]. This is the risk factor more difficult to manage.

11.3 Anatomy and Biomechanics

Patellar tendon is an anatomical structure interposed between the height of patella and the tibial anterior apophysis; the main elements of tendon are collagen, cells, and extracellular matrix. The main task of the collagen is to provide the tensile strength, while tenocytes produce extracellular matrix and procollagen [15].

It is the terminal part of the thigh's extensor apparatus responsible for leg extension, so the tendon supports the work of the muscles and endures a high mechanical stress, mostly at the level of its patellar origin and its tibial insertion. The extensor apparatus can perform both eccentric and concentric work. Patellar tendon is very durable to tensile force and it is proven that a very high strength is required to break a healthy tendon [16, 17]. However, the continuous stress, due to repetitive sport-specific gestures, can lead to an overload pathology.

There are other theories about the pathogenesis of patellar tendinopathy, as mechanical, vascular, or nervous causes [18, 19].

Patellar tendon is stressed in different ways according to the degree of knee flexion: between 45° and 60°, the position of the patellae and the high forces exerted on the tendon result in a higher probability of having injuries, above during eccentric work [20]. The greatest muscle strength, produced in an eccentric way, is 1.5–2 times higher than the greatest isometric force and the higher concentric force, above at high speeds [21]. The force transmitted from the ground varies according to different athletic feats and corresponds to 2.8 times the body weight during long-distance running, to 6 times the body weight during volleyball jumping, and to 10 times the body weight during the takeoff of the long jump [22]. The highest forces of load transmission to the quadriceps occur during the ballistic reaction with the "drop jumps," during which the forces transmitted to the quadriceps are proportional to the reaction force of the ground. So it is reasonable to assume a link between the working model of the quadriceps and the prevalence of the jumper's knee. This assumption seems to be validated by the distribution of the prevalence reported in several studies. This is highest in basketball, volleyball, and athletics and low or negligible in other sports.

11.4 Pathogenesis

The etiology of tendinopathy is not clear and generally has a multifactorial origin; tendinopathy is usually the result of tendon nonhealing and often the affected tendon shows no sign of inflammation but, on the contrary, shows increased new blood vessels, fibroblasts, hypercellularity, and disorganized collagen [23].

Histopathological studies concerning patellar tendinopathy show the separation of the collagen fibers with eventual loss of the normal organization, a most fundamental mucoid substance, morphological changes of tenocytes, fibrocartilaginous metaplasia, and cellular and capillary proliferation [24, 25].

The connection between the conditions of mechanical load and the pathophysiological response is unknown. It was suggested that the mechanical overload can produce partial ruptures in the tendon tissue and the histological results have been interpreted as partial tendon ruptures [8, 9, 14]. The hypoechoic lesions, observed in the proximal patellar tendon, associated to tendinopathy are usually described as a result of the failed healing or as partial tendon ruptures.

Some classical theories suppose that the lack of traction promotes healing and adaptation of tension; a too high traction might prevent healing and promote the accumulation of degenerative tissue. Other authors suggest that an excessive mechanical stretching of the tenocytes may activate some signal pathways able to induce apoptosis. Histological findings, observed during tendinopathy, are compatible with an apoptotic process. It can be possible that, when the mechanical load is higher than the adaptive response of tenocytes, apoptosis is induced [26].

Pain, in tendinopathy, in the past was attributed to inflammation, but new studies and new theories have shown that pain originates from biochemical and mechanical factors [15].

Rupture of the patellar tendon is considered by many authors as the final event of the degenerative disease. These injuries usually occur in more than 40-year-old people and often are associated with metabolic or systemic disease and just occasionally may be bilateral.

11.5 Clinical Examination

Patellar tendinopathy is characterized by pain; it can manifest in an acute, subacute, or in an intermittent way, usually not following local trauma; it can be associated to a change in the type or in the frequency of training, to a work overload. Usually the pain is reported in specific sites, mostly located at the level of insertion near the lower apex of the patella (Fig. 11.1); in the remaining cases, the pain can be localized to the tendon or to the distal insertion of it. For a long time, the clinical classification more commonly followed was the Blazina's one that was improved and divided into six stages according to clinical symptoms (Table 11.1).

In the first Blazina's classification, the last step was related to the catastrophic rupture of the patellar tendon, a rare but possible eventuality. Usually the symptoms described above disappear with a break from sports activities, especially if it is the

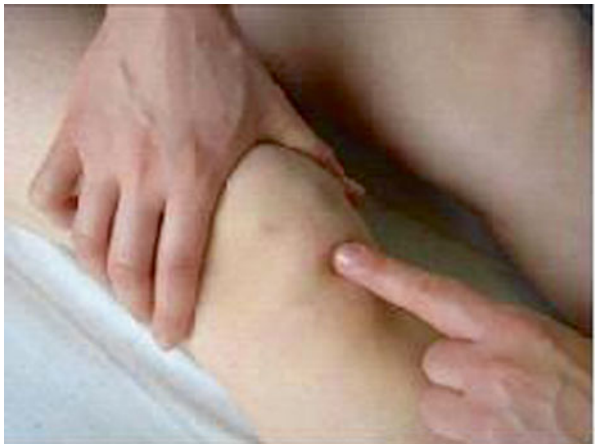

Fig. 11.1 Pain in lower apex of patella

Table 11.1 Classification of patellar tendinopathy according to clinical symptoms

Stage 0	No pain
Stage I	Rare pain with normal performance
Stage II	Moderate pain during sports activities with normal performance
Stage III	Pain during sports activities with initial qualitative or quantitative limitation of performance
Stage IV	Pain during sports activities with an important decrement of performance
Stage V	Pain during daily life and impossibility in practice sport

first episode, but symptoms can recur if the patient resumes physical training too early or with excessive workloads. More attention should be paid to the recovery time of patients with chronic tendinopathy. Concerning patellar tendon rupture, the physician should carefully evaluate the swelling and the hematoma in the patella that can be associated with a hemarthrosis. The more evident and limiting clinical sign is the inability to an active extension of the leg and to lift the extended limb; this makes the gait difficult also using some aids.

11.6 Instrumental Diagnosis

Patellar tendon injuries can be investigated with conventional radiography, ultrasonography, and MRI.

Standard radiograph could be useful in detecting indirect sign of tendon injuries and may detect abnormalities such as cortex irregularities at the lower pole of the patella or at tibial tuberosity, with insertional or peritendinous calcifications and in case of traumatic cortical sleeve fractures.

Fragmented ossification centers in the Osgood-Schlatter or Sinding-Larsen-Johansson diseases and bipartite patella are common radiological direct sign of pathology.

In the case of patellar tendon rupture, knee radiographs with lateral projections may display proximal dislocation of the patella (high patella or high riding patella) and bone avulsions (Fig. 11.2)

Ultrasonography with linear high-resolution probes allows proper visualization of the tendon structure with high resolution.

It may detect loss of the fibrillar architecture and the presence of intratendinous hypoechoic areas, associated with perilesional edema variably extended.

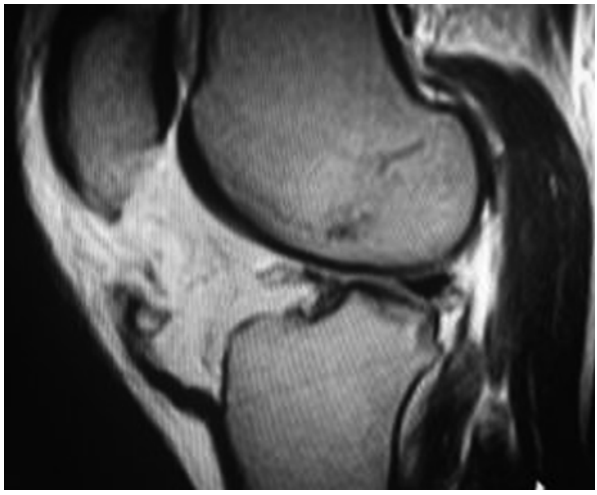

Fig. 11.2 Bone avulsion

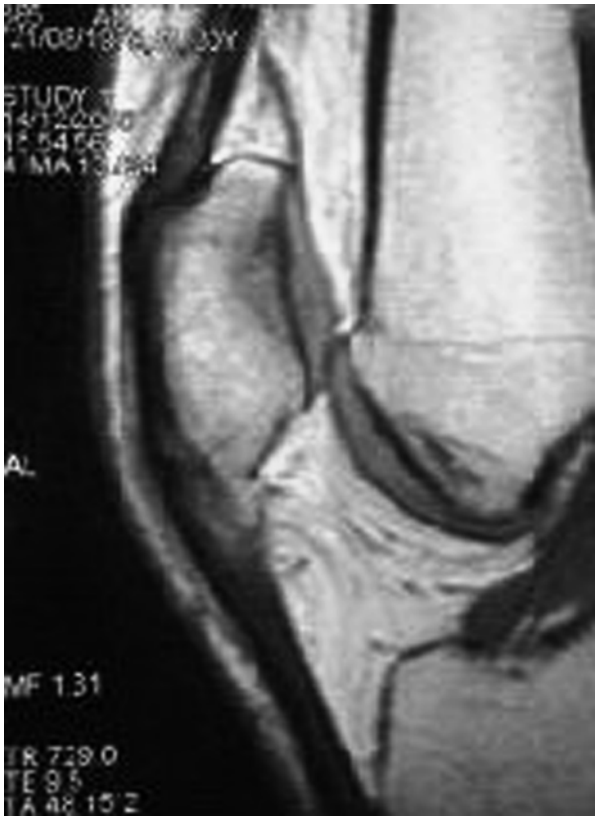

Fig. 11.3 Proximal patellar tendinopathy

Ultrasound is suitable also in patients with patellar fractures, for assessing the hemorrhage, the involvement of the surrounding soft tissues, and the integrity of the patellar tendon.

Ultrasound is a low-cost and safe technique that may be repeated regularly in the follow-up of a patellar tendon injury.

Furthermore, it is limited by the small field of view and inter-operator variability, and hence skilled operators are required. MRI is the most accurate and safe examination for assessing directly patellar tendon pathology, with best demonstration of the morphology (site and extension of fibers solution).

A regular tendon should appear with very low signal in all sequences.

Areas of intratendinous high signal intensity on T1-weighted images depend on tendon degeneration (Fig. 11.3), while hyperintensity in T2-weighted images is referred to edema or hemorrhage.

A common pitfall in patellar tendon MR imaging is the "magic angle effect," a hyperintensity artifact only seen with short TE (time of echo) sequences such as T1W that disappears with long echo time in T2W images [27].

Thickening and deformation of proximal patellar tendon are associated with chronic tendonitis [28, 29].

Swelling of the Hoffa's fat pad and the regional bursae may be present in long-standing tendinopathy or severe tendon injuries.

Bone edema at the enthesis is also a common indirect sign in case of acute-subacute enthesopathy but conspicuous edema may conceal a patellar/tibial sleeve fracture.

So MRI should be done in patients elected for surgical repair to evaluate direct tendon and associated capsular, ligamentous, or meniscal injuries.

11.7 Treatment

Usually the treatment for a sports player affected by patellar tendinopathy is conservative; surgery is often postponed. The treatment should be carefully planned and the physician should modulate and quantify the type, the load and the timing of exercise, the abstention from particular activities, and the rest period. The athlete needs to be informed about what happened and a protocol should be started for the reeducation, rehabilitation, and redistribution of the load and of the intensity of work. The sportsman, during the symptomatic period, should begin a training including all kind of exercises but mostly eccentric ones and stretching. These are suggested to all patients with acute tendinopathy, working mainly on the phase of deceleration and on gradual recovery of elasticity; this treatment can be associated with hydrokinesitherapy and with manual massage to relax quadriceps.

Most important are the modulation and the type of physical activity; if the patient is unable to participate in a usual activity, he or she must find an alternative fitness activity. The patient must promote eccentric exercises. We can divide therapies medically and physically. The medical ones help to solve the clinical symptoms; for this reason, there are many drugs available, above NSAIDs, with systemic and local action. It is important to remember that prolonged use of NSAIDs may have negative effects on long-term tendon healing [30]. There are different opinions about appropriateness of local injections of steroidal anti-inflammatory drugs (SAIDs); these can be useful in reducing symptoms temporarily, but on the other hand these can cause tendon ruptures. In order to improve the clinical symptoms, physical therapies can be performed. Cryotherapy, tens, laser, ultrasound, tecartherapy, shockwave, and magnetic fields are the most used techniques on the tendon to relieve pain. Especially the shockwave seems to play an additional role in stimulating tendon tissue regeneration [31, 32]. Another kind of therapy that has spread in the last decade for the patellar tendinopathy is the injection with autologous platelet-rich plasma (PRP) [33] with the purpose to stimulate healing of the tendon tissue using platelet growth factors. One or more injections can be performed in a few days. Comparing them with shockwave seems that the results of PRP injections are better

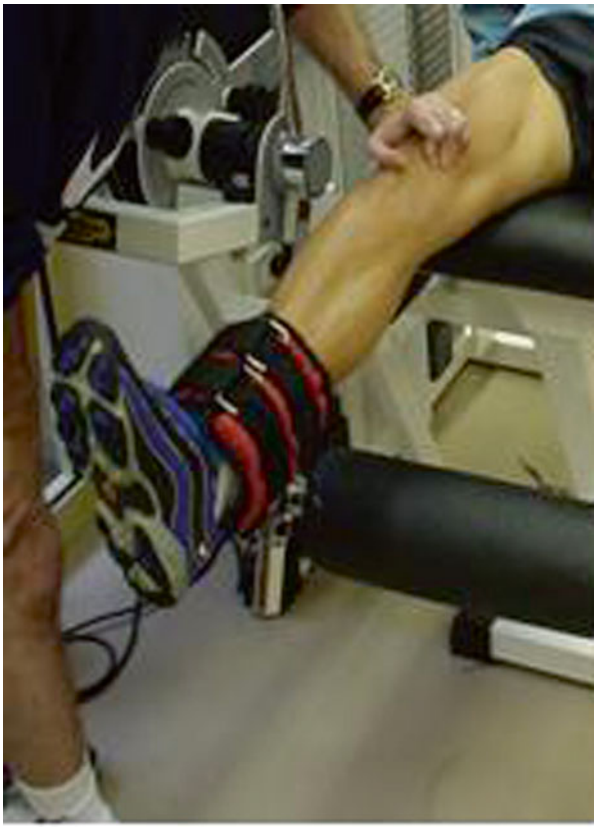

Fig. 11.4 Eccentric exercise

from the biological point of view [34]. Anyway in the literature, the results are controversial, mostly because of the different method of preparation and concentration of the substance to inject [35].

With regard to physical therapy, the most used exercise for the treatment of tendinopathy is based on eccentric work (Fig. 11.4). During those exercises, a lengthening of the muscle-tendon unit with the load with a repetitive loading and unloading in the tendon is observed; this seems to have an important role for the tropism of the tendon and for the reduction of pain [36, 37].

This approach in patients affected by patellar tendinopathy might allow the recovery of the sport activities in a period ranging from a few weeks up to 6 months; if symptoms persists or worsen, surgery should be considered. There are various surgical options but all have as their main purpose, the stimulation and facilitation of the tendon healing. Since in most cases the tendinopathy is proximal, the most used surgical approach consists of some steps: slice longitudinally the tendon for a

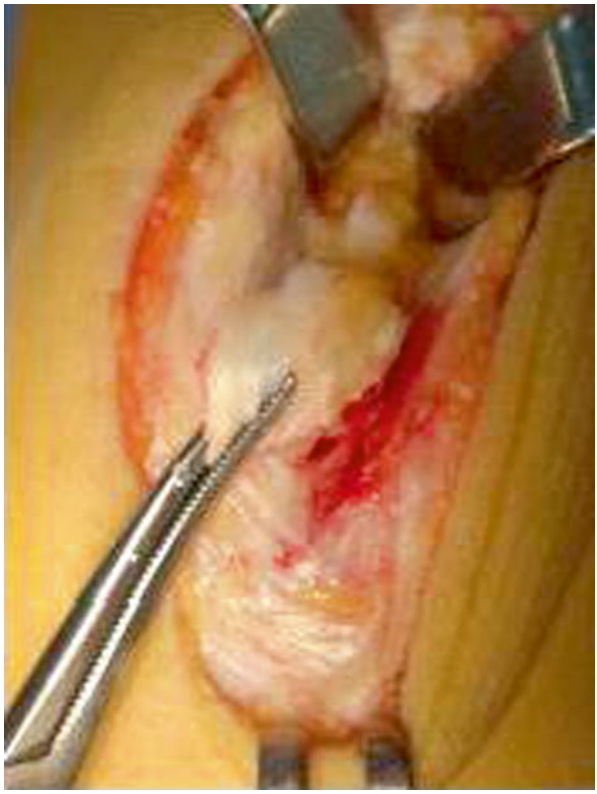

Fig. 11.5 Degenerated tissue removal

few centimeters, in correspondence with the injured area, previously evaluated with MRI; remove the part of possible degenerated tendon tissue (Fig. 11.5); and perform a cruentation of the patellar apex and some small perforations in the apex in order to facilitate the flow of blood in the interested area. It is possible to perform some tendon scarification if the injured area is extended. The same procedure can be used in case of distal tendinopathy, performing scarifications and perforations of the ATA, eventually associated with the removal of the degenerated tissue. During the execution of this surgical technique, it is possible to use PRP gel (Fig. 11.6).

In case of patellar tendon rupture (Fig. 11.7), a discriminating factor able to condition a good outcome of the surgical treatment and of the function recovery is the time since the accident. The injuries treated in the acute phase seem to have a better complete functional recovery. The surgical treatment of the acute rupture of the proximal or distal insertion of the patellar tendon consists in the reinsertion of the tendon to the bone component. It can be made using or transosseous sutures or suture anchors in order to restore the bone-tendon continuity. This first step has to be associated to a biological augmentation (Fig. 11.8) in reabsorbable or not

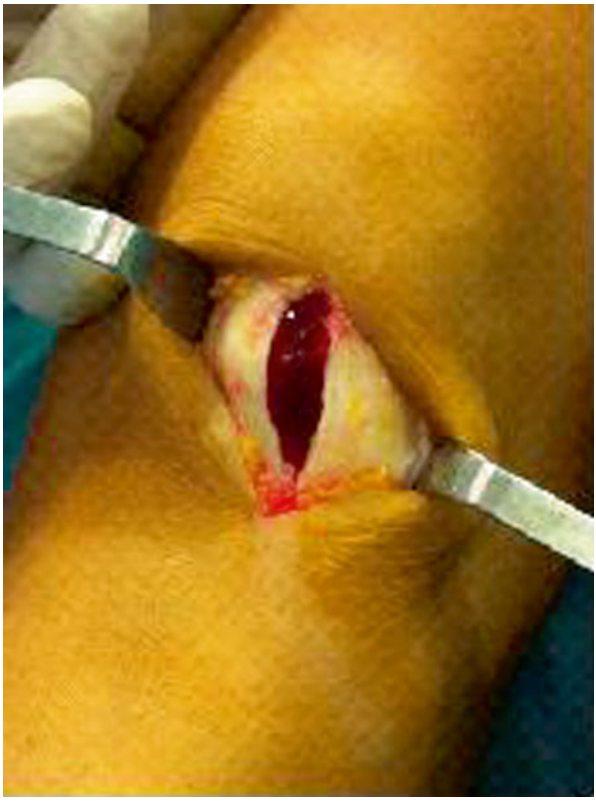

Fig. 11.6 PRP

reabsorbable material [20]. Some author suggests also a transosseous wire augmentation.

In case of ruptures of the central part of the tendon, the continuity of it have to be restored taking back the patella to its original site and eventually associating an augmentation, biological or in reabsorbable material. Also in this case, there are authors who suggest a transosseous wire augmentation. Often if lesions are unrecognized or chronic, perilesional or calcific scar tissue is present associated to anatomical deformity of knee detectable at the physical examination. In this case, the surgical approach is more complex with the possible difficulty to recover the right position of the patella. The treatment consists in the initial removal of the scar tissue and calcifications and in the cruentation of the ends of tendon. Sometimes the gap cannot be filled without a "zed" elongation of the patellar tendon. If the ruptures are inveterate with a considerable ascent of the patella, it is possible to use the technique suggested by Dejour, transplanting the distal part of the extensor apparatus of the contralateral knee [38]. Alternatively tendon allograft or autologous tendon transplantation (semitendinosus and gracilis tendons) can be used to minimize the iatrogenic lesions for the contralateral healthy knee.

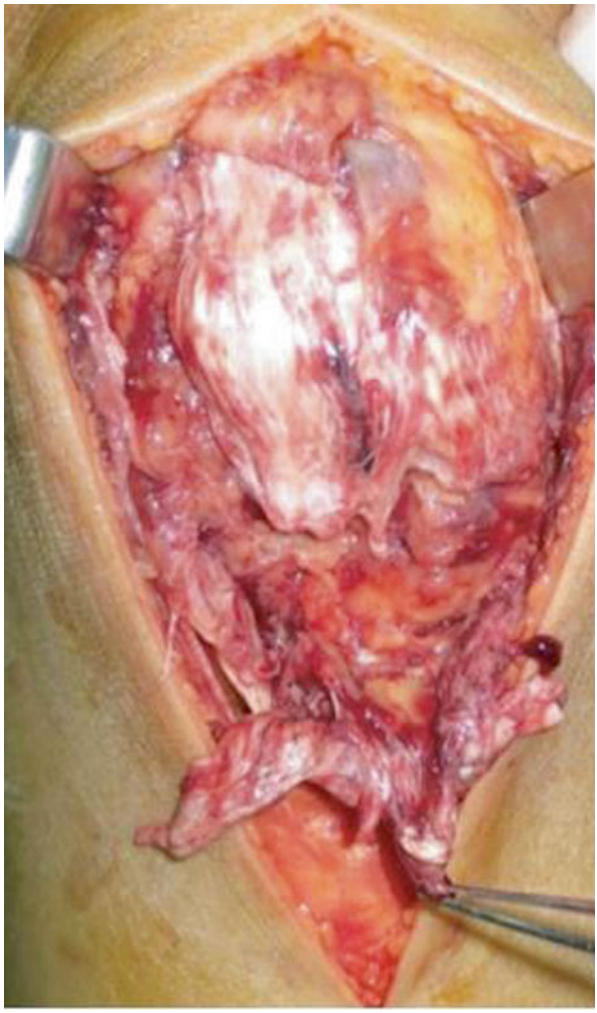

Fig. 11.7 Patellar tendon rupture

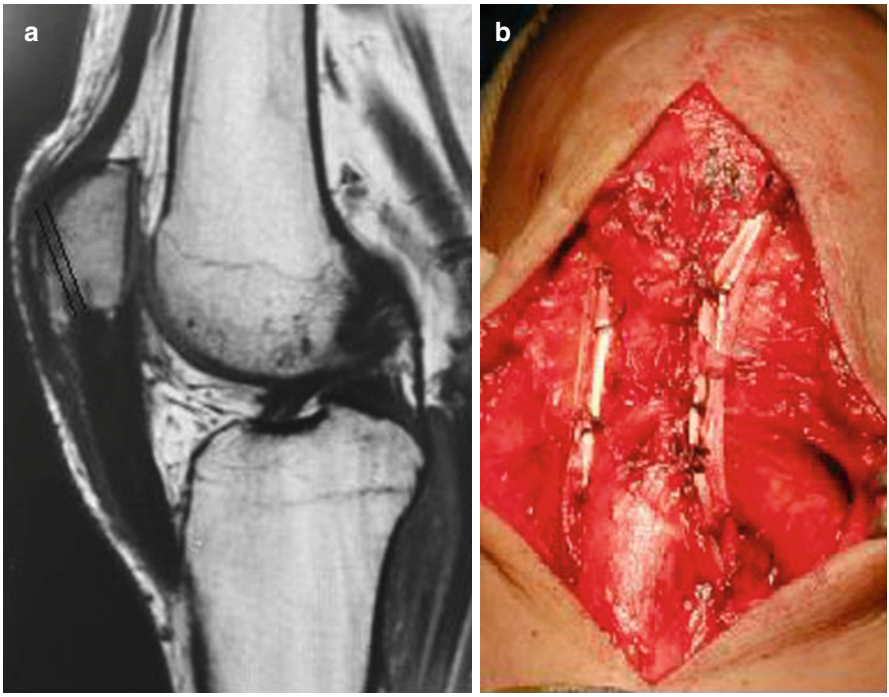

Fig. 11.8 (**a**) Transosseous suture; (**b**) biological augmentation

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Chapter 12
Hindfoot Tendinopathies

Francesco Allegra, Enrico Bonacci, and Francesco Martinelli

Abstract Posttraumatic tendon disorders and their sequelae involve ankle joint and hindfoot in sport and work-related activities much more than other districts. Ankle trauma represented incidence is 37 sprains every 1000 persons, with an increase in progressive tendency. The ankle is the most involved joint in traumatic pathology and represents 10–15 % of all sports injuries. The pathology of the anterior compartment is most prevalent with a progressive involvement of the posterior compartment of no less importance. Numerous risk factors can contribute in hindfoot and posterior ankle disorders development, intrinsic patient related to its physical form and activities, extrinsic equipment, and environmental conditions related to where the activity is practiced. The population with less than 25 years is the most involved in working and sporting activities with the fibular ligament and posterior tendon compartment involvement. Better coordination between muscle and training activities addressed to the perception of balance makes possible to prevent trauma, resulting in improved muscle strength and improved proprioceptive response from the ankle. Conservative treatment is the first one and represents the 85 % of recoveries. About 15 % needs a surgical treatment, performed preferably by arthroscopic procedure.

12.1 Background

Hindfoot is an anatomic district placed in the posterior ankle and lengthened along the superior aspect of calcaneal tuberosity. It is not an articular space, but two joints are posteriorly opened on this, the ankle joint and the subtalar one. Hindfoot can be subdivided into three parts. The first one is anterior in direct relationship with the

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two joints and with the posterior tendons of the calf, both medial and lateral. The second one is the widest on sagittal plane and is represented by the cortical bone of upper calcaneal tuberosity. The third one is limited to the last part of Achilles' tendon and its insertion. The presence of many tendinous structures placed both on medial and lateral of posterior ankle side characterizes the district and its typical pathologies. Most of those has a traumatic or micro-traumatic etiology due to musculoskeletal injuries of the lower limb. When patients present with foot and ankle pain, tendinopathies are often missed and assumed to be evaluated by the family doctor just as an ankle sprain, which can lead to chronic pain. The overuse tendon injuries are the most difficult to diagnose because of the slow progression of the disease and the poorness of symptoms from its beginning. Because the tendinopathies have often evolved in a period of years, the tendon condition is considered a degenerative process, affecting the approach to treatment which varies depending on the stage and the etiology of the disorder.

12.2 Biomechanics and Pathophysiology

The biomechanics is fundamental understanding of the patho-mechanics injury mechanisms. The foot and ankle vary its position on the ground following the gait cycle, from the point of initial contact with the ground and heel strike to the point at which the same foot hits the ground again. This cycle is divided into the stance phase (60 %) when the foot is in direct contact with the ground and the swing phase (40 %) when the foot is not in contact with the ground. At heel strike, the foot is supinated, changing immediately after when the foot begins to pronate, becoming maximally pronated at mid-distance. This pronation is normal as the subtalar joint unlocks so the foot can become flexible, while calcaneal tuberosity is in full contact with the ground allowing for accommodation to its surface. As the body weight shifts forward, the foot begins to return to a neutral position in preparation for incoming heel lift. This occurs at the end of the stance phase when the foot starts to supinate, thanks to the resulting in the formation of a rigid subtalar joint which makes easy, valid, and strong toe off. Every abnormality in this simple gait cycle can develop the risk of tendinopathies, especially in the hindfoot. The word tendinopathy is widely used for overuse tendon injuries describing a spectrum of several diagnoses involving injury to the tendon like tendinitis, peritendinitis, and tendinosis, in the absence of one secure pathological diagnosis [1–3]. These overuse disorders are commonly considered to occur when intensity, duration, and mode of specific physical activity, work, or sport training changes in some way. In the absence of such conditions, also the presence of not completely recovered posterior ankle bony fractures, the presence of ankle ligaments insufficiency and the appearance of initial joint arthritis as consequence of former traumas can induce the development of tendinopathies. Generally a period of recovery and rest is sufficient to reduce the increased demands on the tissues, worsening any symptom in case of inadequate recovery, because it influences the breakdown at the cellular level [4]. When the tendon is injured, normal response consists of inflammation followed by deposition

of collagen matrix within the tendon, accompanied by a weak remodeling response [1]. However, a failed healing response may occur because of ongoing mechanical forces on the tendon, frequently in association with poor blood supply because of both strangulation of small vessels and weak capability of new local angiogenesis. The continuing such mechanical stimulation produces microscopic tendon structure changes, including fibrin deposition, neovascularization, reduction in white blood cells and macrophages, and finally an increase in collagen breakdown and synthesis, consisting of a disorganized matrix tissue with the presence of hypercellularity and hypervascularity. This tissue has the characteristic to be painful and weak [5, 6].

12.3 Clinical Examination

A history of trauma, involvement in a new sport or exercise, or an increase in the intensity of basic physical activity is always present. Checking the biomechanical alignment of the foot and ankle while the patient is standing and throughout the gait cycle represents a fundamental for pointing tendons disorder out. Understanding the relationship between the different bones of the hindfoot is essential for a correct diagnosis of tendinopathies. Hindfoot tendon dysfunction characteristically is a slow onset condition mainly affecting middle-aged, obese women, even the sporters can be affected with. At the patient's visit, the first foot examination must exclude any calcaneal deformity, acquired or congenital. Degenerative changes in hindfoot medial tendons lead to pain and weakness, progressing to deformity of the foot, and degenerative changes in the surrounding joints. Patients will complain of medial foot pain and weakness and can slowly develop a progressive flatfoot deformity, which is always announced and accompanied with continuous local pain, swelling, and difficulties in walking. Patients affected with hindfoot tendinopathy present seldom an insidious onset of pain over the affected tendon which worsens with sustained activity. In the early stages of disease, pain decreases with a warm-up period; on opposite in later stages, pain may be present at rest but worsens with activity [7]. The pain is less severe during prolonged rest periods. Pain is usually described as far and dull at nighttime rest and as sharp as the activity increases during the day life. No systemic symptoms should be present. Observing the affected area, asymmetry, swelling, or muscle atrophy is checked. Palpation may reveal tenderness along the tendon, reproducing the patient's pain and decreasing range of motion of the affected side. Pain in multiple tendons or joints may represent a rheumatologic cause. Risk factors include obesity, hypertension, diabetes, steroid use, and seronegative arthropathies.

12.4 Instrumental Diagnosis

Plain radiography is recommended as the initial imaging study. X-ray plain weight-bearing anteroposterior and lateral scan of both feet, in addition to bilateral weight-bearing ankle views, should be suggested for a first evaluation of the patient.

Changes may not be apparent on X-ray in the early stages, but as the disease progresses, collapse of the medial longitudinal arch and joint degeneration may become sometimes evident [8]. Results are usually normal, but the study may reveal osteoarthritis, loose body, or a calcification of the tendon. Ultrasound is a quick, readily available, low-cost investigation which gives information about the tendon size, degeneration, and presence of fluid. It is useful for obtaining a dynamic examination and should be compared with tendons on the other side to make certain there is no pathological change. In the tendinosis, tendon is thickened, more or less fusiform, and hypoechoic. Gradually, microlesions appear as longitudinal fissures, and finally the tendon wears thin and loses its normal mechanical properties although its continuity seems to be at least partially maintained. In the tenosynovitis suffusion of the sheath, thickening and hyperemia of the synovia on Doppler are variably associated, possibly occurring before, during, or after a rupture. Some segments of the hindfoot tendons are in close proximity to the ankle ligaments and retinacula, and this is probably a source of error, which can be corrected. However the quality of results can be ultrasonographer dependent [9].

Magnetic resonance imaging (MRI) also provides good images of tendon pathology, especially if surgical evaluation is being considered. There is a significant association with regard to the presence of tendon abnormalities (tendinosis, tenosynovitis, and tearing) in both the peroneal and medial flexor tendons [10]. The complex interconnected system of retaining fascia and retinacula in the distal aspect of the leg, just above and at the level of the ankle, creates distinct tendon compartments. Furthermore, recent evidence has shown that these and other retinacula also play a functional role, as they are not simply static structures for joint stabilization, like ligaments, but rather provide local spatial proprioception during movements of the foot and ankle [11]. A statistical correlation was found between the diameters and sectional areas of most of the ankle and hindfoot tendons, as well as between qualitative abnormalities on MR images of these tendons. This association seems to indicate that abnormalities in one of the tendons may influence the integrity of the other tendons perhaps on the basis of a retinacular and fascial system that connects them [11]. Regarding the posterior tibial tendon dysfunction alone, some authors relieved a confirmation to the statistically significant link between posterior tibial tendon and injuries found on the MRI in key structures for the stabilization of plantar arch, like the spring ligament and tarsal sinus ligaments. Similarly they found a high statistically significant frequency of calcaneal spur and talus peaks in patients affected with same district dysfunction [12].

12.5 Treatment

As considered before, hindfoot tendon injuries are not inflammatory in nature. Medical treatment should be limited to use nonsteroidal anti-inflammatory drugs (NSAIDs) alone to provide short-term pain relief for patients with tendinopathy but do not affect long-term outcomes. Treatment of such disorders ranges from relative rest to surgical debridement. However, many therapies have not been studied in

clinical trials, and it is not known whether the same treatment options can be applied to all tendinopathies. The conservative measures are considered the "golden standard" including protection, relative rest, ice, compression, elevation, local medications, and rehabilitative exercise modalities. At the beginning, patients should be encouraged to reduce their level of physical activity with the immediate aim to reduce and stop repetitive loading on the tendon depending on the injury and the day life activity and the timing of relative rest [13]. Furthermore, any therapy must begin by the medical evaluation of both the extrinsic and intrinsic causes of tendon injury. Extrinsic factors include overuse of the tendon, training errors, smoking, medication abuse, and wearing shoes or other equipments not appropriate for the specific patient's activity. The use of unappropriated type of shoes for the patient's foot type such as motion control, cushioned, or stability must be discouraged . Flexibility and strength of the tendon, patient age, leg length, and vascular supply may also play a role evaluated as intrinsic factors. Although identifying and treating these factors have been the mainstay of treatment over the years, evidence on their effectiveness is lacking [3]. Orthotics, such as inserts or a heel wedge, are sometimes used to help unload, reinforce, and protect the tendon. Eccentric strength training, which involves actively lengthening the muscle, is an effective therapy that helps promote the formation of new collagen. Eccentric exercise has proved beneficial in the treatment of Achilles and patellar tendinosis, and it may be helpful in other tendinopathies as well [5]. Other physical therapy modalities include ultrasound, iontophoresis (electric charge to drive medication into the tissues), and phonophoresis (the use of ultrasound to enhance the delivery of topically applied drugs), but little evidence exists on their effectiveness in treating tendinopathy. Because many of the standard therapies for tendinopathy have failed in time to correct and stop the tendon degenerative process, many new treatments are being developed. These include extracorporeal shock wave therapy [14], radiofrequency ablation, percutaneous tenotomy, autologous blood, or growth factor injection; the effectiveness of these treatments are being currently investigated.

If a comprehensive, custom-made, nonsurgical treatment program of 3–6 months has failed, surgery should be considered only if the patient is unwilling to alter his or her level of physical activity or if rupture of the tendon is evident. The final aim of each surgical treatment is the excision of the tendon release of impinging bony or soft tissue including abnormal scarring and fibrosis tissue.

12.6 Single Site Tendinopathies

12.6.1 Tibialis Posterior Tendon

The function of posterior tibial tendon is to flex plantarly the foot and to activate the inversion of the foot, stabilizing the medial longitudinal arch in the same time. Posterior tibialis tendinopathy is a prevalent musculoskeletal condition often resulting in gait abnormalities along with medial ankle and foot pain. The disorders of this tendon can lead to a painful flatfoot deformity with the consequence of overstress to

the midfoot ligaments in the calcaneonavicular joint and to deltoid ligament, whose elongation can worsen the medial ankle instability in association with flatfoot.

As predisposing condition for posterior tibial tendinopathy is gender, female much more than male and 40 years aged at mean, without previous acute trauma, present in patient's clinical report. The history of a far previous traumatic event to the ankle joint, often misdiagnosed as involving the medial compartment, can be considered the cause of posterior tibial tendon synovitis in the majority of these patients. Symptoms can be referred to a sudden unexpected loss of balance because of slipping of curb or slipping on a stair or stepping in a hole, recovered in a short time but remained painful and dull in any activity. Morphology and vascularization of painful tibialis posterior should be examined, targeting the degenerated tendon. The symptomatic foot can be deformed in an excessive pronation due to the weakness of the posterior tibial muscle and tendon compared with the other side: it is especially evident when patient is requested to maintain a tiptoe standing or walking. When symptoms occur the patient is no more able to single leg and toe raise, associated with the impossibility to have an active and strong plantar flexion of the involved foot. The presence of swelling and foot deformity because of posterior tibial tendinopathy gradually increases in irregular but continuous way, by months to years, progressing symptoms in the opposite side of the ankle joint in the region of sinus tarsi below the fibular malleolus.

Treatment is at beginning conservative and must be initiated promptly because of the risk of progressive deterioration and a nonfunctional tendon. Non-operative treatment can alleviate symptoms and control progression in nearly all stages of the disease. Immobilization in a leg cast or cast boot is suggested for a period of some weeks depending on the symptom recovery. Local injection of hyaluronate can be considered an option because of the effect on inflamed tendon and sheath; furthermore, the use of steroids into synovia are discouraged because of the high rate of tendon rupture secondary to local necrotic effect on tenocytes. Physiotherapy and rehabilitation are considered a valid option. The posterior tibialis muscle is able to perform efficiently at the initial stage by restoring cuboid internal rotation mobility, associated with midtarsal pronation and lower extremity neuromuscular control: activating this rehabilitation in time resolves the chronic tendinopathy and permits the patient to optimum functional ability returning [15]. Despite strengthening and stretching exercises combined with orthosis treatment in a home-based program, which has been evaluated as valid conservative treatment, a moderate-intensity, home-based exercise program is evaluated as minimally effective, in augmenting orthosis wear alone in patients with tibialis posterior tendinopathy disorder stage II [16]. In examining the degenerated tendon by grayscale imaging and Doppler ultrasound at initial and posttreatment stage, to assess the tendon's morphology and signs of neovascularization with functional status and patient pain level, a program of rehabilitation can be lead out: a 10-week tendon-specific eccentric program resulted in improvements in symptoms and function without changes in tendon morphology or neovascularization [17].

If conservative management fails for up to 3 months or if stage 3 flatfoot deformities are diagnosed, the patient should be referred to an orthopedic surgeon. Insufficiency or rupture of the posterior tibial tendon is a frequent cause of adult acquired flatfoot deformity, considered as a common clinical disorder. Its early recognition is essential because natural history is characterized by progressively worsening deformity. Surgical solution should be considered at failure of symptom

control or at prevention of deformity progression. In early stage 1, an arthroscopic surgical procedure should be indicated to explore the tendon and its sheath along (Fig. 12.1), following this structure behind the tibial malleolus: a debridement of the inflamed tissue of synovia or a removal of symptomatic partial tendon tear (see Fig. 12.2) should be performed using two small medial side portal. In stage 1,

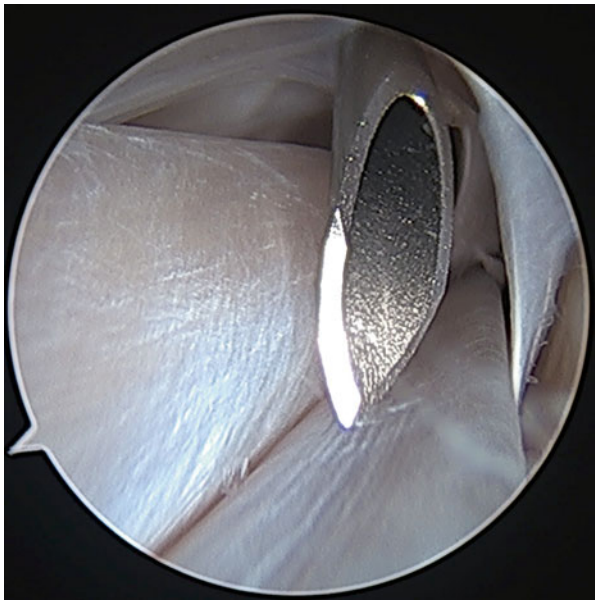

Fig. 12.1 In this arthroscopic image, the posterior tibialis tendon lies below the spinal needle tip, used as a probe in the restricted space of the tendon sheath

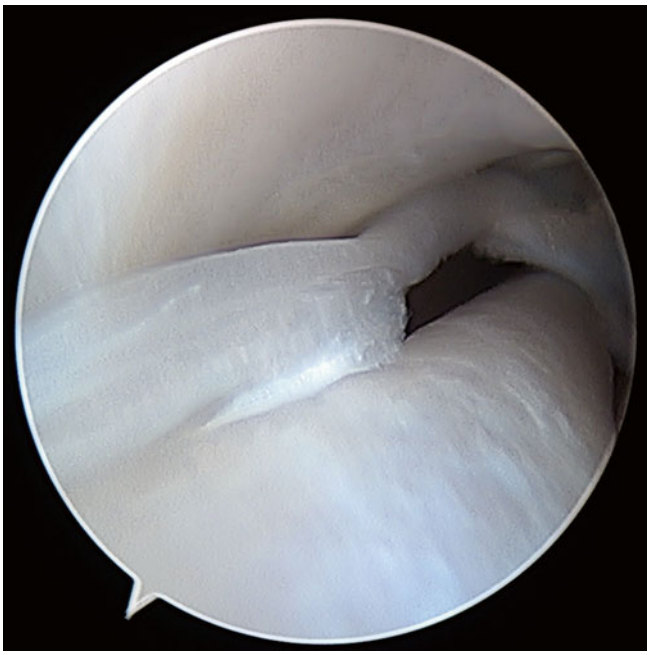

Fig. 12.2 The arthroscopic visualization allows the surgeon to finalize the diagnosis, finding a partial rupture of posterior tibialis tendon easy to be remodeled by shaver introduced in a proximal portal

exploration and debridement with or without flexor digitorum longus (FDL) tendon transfer is a surgical option. In stage 2, the tendon becomes elongated involving a reduction of medial soft tissues. Exploration and debridement of the PTT is performed, but frequently a FDL tendon transfer or side-to-side anastomosis is required performing with an open procedure. In the stage 2b, the treatment addressed on soft tissue alone may fail to resolve patient's symptoms: correct bony deformity should be considered in this stage a valid option to avoid deterioration of results over time even surgery limited only to tendon. Combined procedures, including soft tissue reconstructions to restore PTT function and bony procedures to correct deformity, are considered more safe and durable at outcome. In stage I or early stage II, when the posterior tibial tendon appears intact with minimal degeneration or elongation, a further surgical step to reconstruct the medial column may be considered: an advancement of osteoperiosteal flap based on the tendon insertion is suggested to be combined with selective bony medial column arthrodesis. Although it may be theoretically possible to passively correct hindfoot valgus with these procedures, it seems prudent to limit the indications to patients who have early disease accompanied by an isolated midfoot sag. In more advanced stage II disease, correction of deformity with a tendon transfer alone is incorrect because it is not sufficient to repair the deformity. A combined open procedure addressed to the bone with a medial displacement calcaneal osteotomy or a lateral column lengthening is currently recommended, correcting the deformity and sparing of the hindfoot joints: its advantage is particularly evident in young or active patients. In stage 3, arthrodesis is the procedure of choice because the deformity is fixed and the joints appear to be stiff: the procedure changes depending on the joints affected and the columns of ankle and foot involved. Long-standing results are present when the deformity is approached with isolated talonavicular arthrodesis, correcting all its clinical and radiological aspects; however, in recovering the hindfoot from a complete loss of motion, the appearance of closer joint arthritis is the price to be paid. This clinical situation is not rare, and in a large number of patients, the triple arthrodesis is probably preferred. If residual deformity or instability is present after these procedures, it must be treated. Residual medial column instability may be addressed by adding a selective arthrodesis of the naviculo-cuneiform or first metatarso-cuneiform joint, whereas residual forefoot varus or supination may be addressed with selected midfoot fusions with or without a cuneiform osteotomy.

12.6.2 Peroneal Tendons

Chronic tendinitis, tendinosis, and interstitial tears are more common disorders of peroneal tendon, but complete tear and subluxation at posterior edge of fibular malleolus are also frequent. Often due to ankle joint injury, they are a consequence of lateral ankle instability and are the main cause of the lateral symptoms of the joint [18]. Injuries can be acute as a result of trauma or present as chronic problems, often in patients with predisposing structural components such as hindfoot varus, lateral

ligamentous instability, an enlarged peroneal tubercle, and a symptomatic os peroneum. Because it is a common subjective feeling of ankle giving way, often without sure symptoms of instability, it is very important that the patient should be submitted to a visit of a specialist to evaluate the possibility to stabilize the joint [19]. Persistent lateral ankle swelling, popping, and retro-fibular pain often occurring are typical symptoms of tendinitis. Tendon subluxation is a common disorder characterized by popping and giving way sensation: a positive peroneal tunnel compression test is painful, with active dorsiflexion and eversion of the foot against resistance along the posterior ridge of the fibula. The evidence of subluxation and/or dislocation of the peroneal tendons should be referred to an orthopedist [18]. Also rheumatoid arthritis or a seronegative arthropathy is considered as a cause of symptoms as swelling and tenderness, especially in the absence of increased work or sport activity or trauma. It is generally difficult to refer patient's clinical findings for peroneal pathology and magnetic resonance imaging (MRI), utilized for diagnosing peroneal tendon pathology. However, patients with MRI findings of peroneal tendon pathology should undergo careful clinical examination, as the positive predictive value of MRI for peroneal tendon pathology with actual clinical findings is low [20]. On the other hand, studies demonstrate that peroneal tendon tears are often incidental findings on MRI [21]. Patients with chronic lateral ankle instability also have peroneal tendinopathy often. However, preoperative MRIs of patients affected with peroneal tendon tendinopathy are vague in many cases, especially in those with chronic lateral ankle instability, despite that it is a useful diagnostic tool for detecting such peroneal disorders. Therefore, a thorough delicate physical examination and careful observation is always needed [22].

Conservative treatment is the first to be prescribed to patients. Lateral heel wedges and ankle taping help unload stress on the peroneal tendon, but there is no evidence that they induce healing. Rehabilitation therapy involves ankle range-of-motion exercises, peroneal strengthening, proper warm-up, and proprioception activity. Indications for surgery include failure of conservative management, recurrent peroneal instability, and rupture of the peroneal tendon. Any instable ankle should be stabilized on the surgeon's indication, reconstructing ligaments, or performing surgical procedure to fix injured ligaments or joint stabilizing structures. Also subluxation and/or dislocation of the peroneal tendons should be submitted to an orthopedic surgeon to evaluate the opportunity to reconstruct the posterior fibular retinacula or to treat with a different surgical procedure to stabilize tendons in their own bony groove. Arthroscopic surgery has a reserved space for those peroneal tendons disorders (see Fig. 12.3) limited to symptomatic tendinitis and tendinosis (see Fig. 12.4) or to partial ruptures secondary to posterior ankle soft tissue or bony impingement.

12.6.3 Flexor Hallucis Longus Tendon

Tendinopathy of the flexor hallucis longus (FHL), colloquially referred to as "dancer's tendinitis," is a common condition in dancers and attributed to high demand on this muscle in positions of extreme ankle plantarflexion and

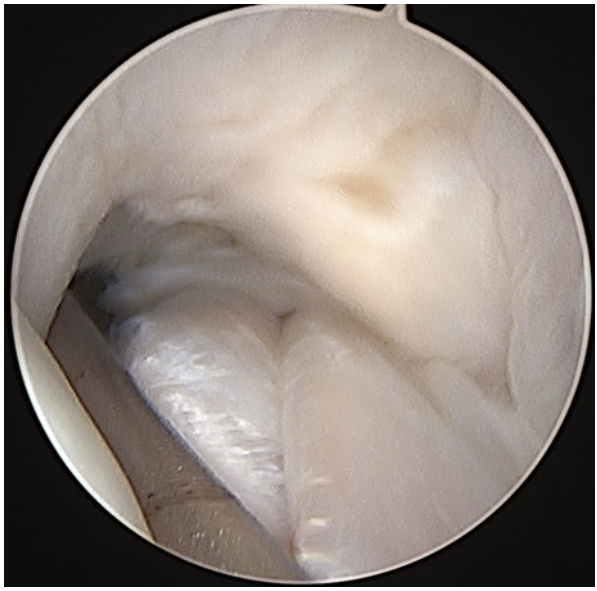

Fig. 12.3 The peroneal tendons appear inside their sheath; place the peroneal brevis close to probe instrument and the peroneal longus closer to internal aspect of peroneal malleolus. The movement of the probe permits an exploration of the tendons under direct visualization of the scope

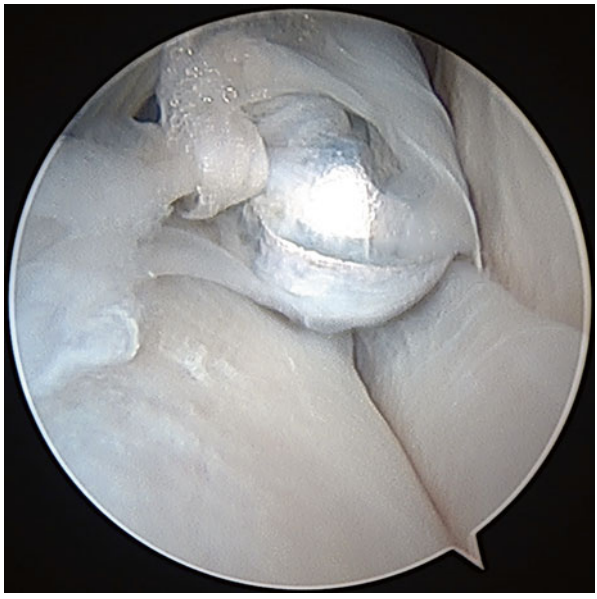

Fig. 12.4 The shaver is approaching to the scope from an anterior and lower portal and is going to debride tendons from the symptomatic synovial tissue

metatarsophalangeal (MTP) flexion and extension. Despite that this injury is the most common lower extremity tendinitis in classical ballet dancers [23], it is also seen in persons who participate in activities requiring frequent push-off maneuvers [24]. Tenosynovitis of the flexor hallucis longus (FHL) tendon is a condition typically found in some sports like in soccer players, related to chronic overuse.

A traumatic cause for this situation, such as an ankle sprain, is considered rare. This particular kind of tendinitis of the hindfoot is almost always caused by the FHL entrapment, like it is realized in the "en pointe" position of dancers, leading to a chronic pain, early arthritis, and fibrosis with progressive decreased range of motion. At the beginning, the onset of symptoms is along the posteromedial aspect of the ankle behind the malleolus or less frequently on the medial side of subtalar joint below the tip of medial malleolus. During the visit, when the doctor asks the patient to plantar flex the great toe against the resistance or to stand and to walk on tiptoe, the pain suddenly appears with an occasional crepitus along the posterior edge of tibial malleolus. A useful clinical test is to compare the passive extension of the first metatarsophalangeal joint with the foot and ankle in the neutral and plantar flexed assessment: when a little or no extension in neutral position is checked, disappearing in plantar flexion, the FHL tendon appears to be entrapped.

Prevention includes reducing turnout of the hip to make the dancer working directly over the foot and avoiding hard floors whenever possible. Strengthening the body's core is one of the main rehabilitation activities to balance strength and to develop the muscles which stabilize and move the trunk of the body such as abdominal, back, and pelvic muscles. The use of firm and well-fitted shoes can be considered a good prevention; however, less possible in dancers whose relatively stiff feet may contribute to the disorder because of the incorrect en pointe position. For the athlete who is specialized in sprint sports (run, soccer, football), it is important to wear appropriate shoes and to correct the starting moment, to avoid overcharging the tendon. For prolonged tendinitis, 2–3 weeks of immobilization in a weight-bearing cast or walking boot is recommended. Efficacy of using modified heel raise task with the toes off the edge of a block as a means to train larger plantarflexors is proposed as therapy and as prevention. Improving interventions for FHL tendinopathy will be impactful for dancers, in whom this condition is highly prevalent and be considered valid for all the workers which are frequently submitted to.

In case of failure of conservative treatment, the tendon can be surgically released, which is usually done through an open procedure. But by years it has been validated also that the arthroscopic procedure, which can release the FHL with a very low surgical aggression, allowed good visualization of the involved structures and yielded good results. This condition can be related to multiple microtraumas, not remaining an exclusive disease of ballet dancers or overuse [25]. Open and arthroscopic techniques have been utilized in the treatment of posterior impingement of the ankle and hindfoot. Because posterior impingement occurs more frequently in patients who repetitively plantarflex the ankle, this population may especially benefit from a procedure that reduces pain and results in maximal range of motion (ROM). Posterior ankle endoscopy allowed for maintenance or restoration of anatomic ROM of the ankle and hindfoot, ability to return to at least previous level of activity, and improvement in objective assessment of pain relief and higher level of function parameters. Complications associated with this procedure were minimal [26]. Hindfoot endoscopic surgery has been described as a minimally

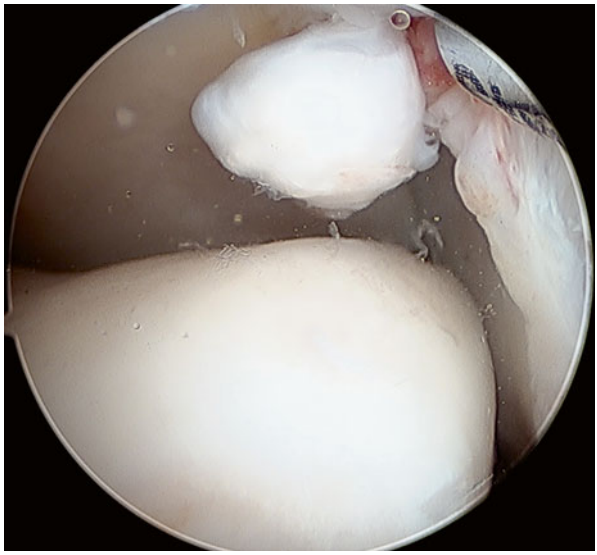

Fig. 12.5 The presence of a cartilaginous loose body can be occasionally cause of hindfoot tendon impingement: its removal releases completely the disorders

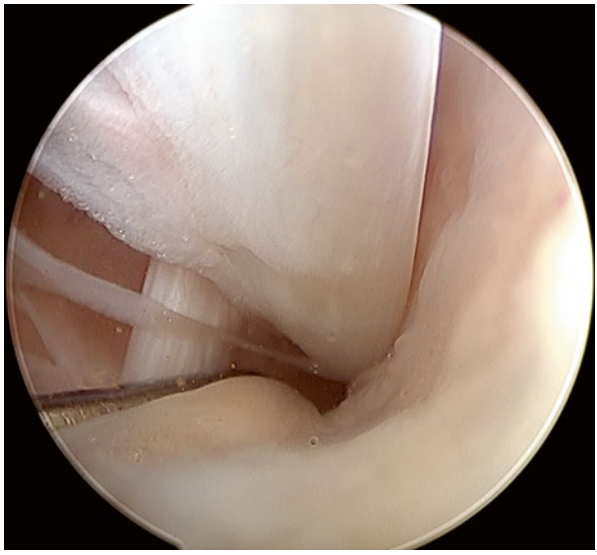

Fig. 12.6 The FHL tendon can also impinge inside its sheath behind the posterior edge of tibial malleolus, where its path curves to become straight, passing into the foot plant

invasive surgical treatment for posterior ankle and FHL impingement syndrome [27] removing the possible causes of impingement as posterior loose body (Fig. 12.5) or removing fibrous tissue from the sheath at its hindfoot origin (Fig. 12.6) until complete tendon release (Fig. 12.7). With this procedure, a systematic approach for identifying relevant hindfoot structures and its abnormalities during hindfoot

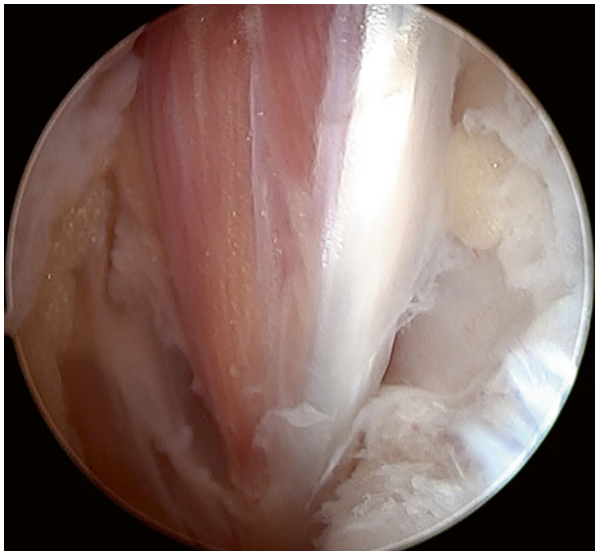

Fig. 12.7 The FHL tendon is completely released and it moves up and down, following the passive movements the surgeon impresses the great toe

exploration is possible, dividing the extra-articular structures of the hindfoot into quadrants as defined by the intermalleolar ligament. Hindfoot arthroscopic surgery is an effective treatment strategy for posterior ankle FHL impingement syndrome [28]. In addition, it allows the patients a rapid return to sporting activities.

12.7 Conclusions

Hindfoot tendinopathies are less frequent and dependent on the patient's activity. Despite the absence of data, possible risk factors have been assumed such as age, duration of symptoms, body mass index, type of tendinopathy, previous therapies, and the presence of associated ankle injuries. At their appearance the right and fast diagnosis leads to the correct treatment, in time to avoid symptoms to become chronic. Patients must be firstly submitted to conservative therapy, remaining the specific indication for surgery only at failure of any other treatments. Advances in foot and ankle arthroscopy have allowed surgeons to diagnose and treat a broadening array of disorders that were previously limited to open procedures. Arthroscopy of the posterior ankle, hindfoot, and tendoscopy can be used to address common ankle ailments, with the potential benefits of decreased pain, fast recovery, and low complication rates. Posterior ankle arthroscopy can be indicated to manage impingement, arthrofibrosis, and synovitis which are the most common causes of hindfoot tendinopathies. Tendoscopy is a minimally invasive alternative for evaluation and debridement of the posterior tibial, flexor hallucis longus, and peroneal tendons.

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