Achilles Tendon Pathology

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95.1 Preface

Although Achilles tendon is the strongest in the human body, it is one of the most frequently injured one [11, 15, 20].

There is no consensus on the best method for management of the Achilles tendon pathologies. Individual preferences, drawn from experience and study, determine whether treatment is operative or nonoperative.

95.2 Disorders of Achilles Tendon Insertion

95.2.1 Achilles Tendon Enthesis

Achilles tendon, as many other different tendons, approaches its distal attachment site obliquely, and during ankle plantar flexion, the tendon comes in contact with the bone (heel) (Fig. 95.1a, b), which puts stress dissipation at the enthesis itself [4]. The retrocalcaneal bursa allows for free movement between the tendon and the bone. The fat pad protrudes into the bursa during plantar flexion and is retracted during dorsiflexion. The Kager's fat pad has three distinct regions that are associated with all three borders of Kager's triangle: flexor hallucis longus part (which is responsible for moving the bursal wedge during plantar flexion), bursal part (which minimises pressure changes in the bursa) and Achilles part (which protects blood vessels that are entering the tendon) [25].

95.2.2 Insertional Achilles Tendinopathy

In 1928, Haglund described a single case of a clinical condition of painful hindfoot caused by an enlarged posterosuperior border of the os calcis and the wearing of rigid low-back shoes [8]. Haglund syndrome was defined as a complex of symptoms involving superolateral calcaneal prominence, retrocalcaneal bursitis and superficial Achilles tendon bursitis [26, 27]. However, one must distinguish Haglund syndrome with retrocalcaneal bursitis from Achilles tendinopathy. Unfortunately, these two pathologies may coexist, especially if insertional tendinopathy is considered (Fig. 95.2).

The confusion regarding terminology of distal Achilles tendon problems has a historical background. In the times when those conditions were first described, doctors were establishing their diagnosis almost solely based on clinical symptoms, and these may be very similar in different pathologies. The lack of adequate radiological examination and histopathological evaluation leaded to misuse, not fully understood, terms.

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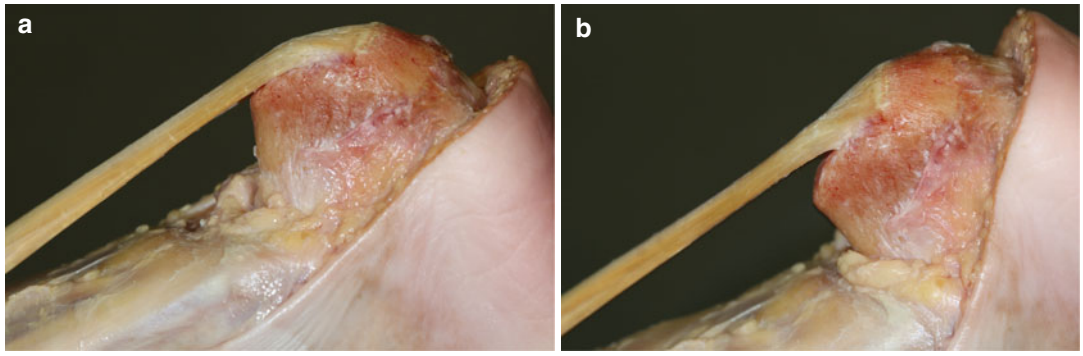

Fig. 95.1 (**a**, **b**) Cadaveric dissection of distal attachment of the Achilles tendon. Notice the way Achilles tendon approaches the calcaneus and gets in contact with the bone. This is the area where many Achilles tendon pathologies take place

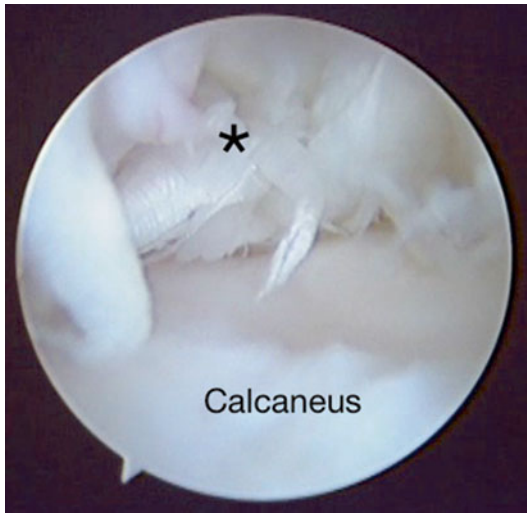

Fig. 95.2 Endoscopy of retrocalcaneal bursa. (*) Partial rupture of distal attachment of bursal side of the Achilles tendon

That is why Niek van Dijk et al. [27] proposed orderliness in the terminology and use of the following terms: insertional Achilles tendinopathy, retrocalcaneal bursitis and superficial calcaneal bursitis (see description in Table 95.1).

There are a variety of different procedures for treatment of retrocalcaneal bursitis and Haglund syndrome. Conservative treatment includes activity modification, avoiding rigid heel counters in shoes, NSAID, inlays, padding and/or physiotherapy. Some doctors also prefer local injections of steroidal drugs; however, there are a number of reports proving that it might be a risk factor of subsequent Achilles tendon rupture [2, 6, 22] (Fig. 95.3). Local steroid injections give rather quick pain relief in a short time, but rarely permanently solve the problem of underlying pathology. Nowadays for surgical treatment, endoscopic surgery (Fig. 95.4), first proposed by Niek van Dijk – as the one that offers the advantages of reduced morbidity and reduced postoperative pain and allows for early rehabilitation [7, 26] – should be the treatment of choice.

95.3 Achilles Tendinopathy

Terminology used to describe the painful condition of Achilles tendon is often confusing and most often does not reflect the underlying pathology. According to Khan et al. [13], the term "tendinopathy" might be defined as painful condition of the Achilles tendon, which is rather a general description of clinical symptoms than accurate and precise diagnosis.

95.3.1 Paratendinopathy

Paratendinopathy may be defined as local inflammation of the paratendon (Fig. 95.5a–c). It may be a separate pathology or an accompanying tendinopathy. Many believe that tendinopathy often starts with paratendinopathy. Clinical symptoms might not be different from tendinopathy or

Table 95.1 Terminology of distal Achilles tendon problems proposed by van Dijk et al. [27]

	Anatomic location	Symptoms	Clinical findings	Histopathology
Insertional Achilles tendinopathy	Distal insertion	Pain, stiffness, swelling	Painful tendon insertion in the mid-portion of the calcaneus, swelling, bony spur	Ossification within tendon insertion, tendon degeneration, micro-tears at the tendon attachment
Retrocalcaneal bursitis	Bursa between anteroinferior side of Achilles tendon and posterosuperior aspect of the calcaneus	Painful swelling superior to the calcaneus	Painful soft tissue swelling, medial and lateral to the Achilles tendon at the level of the posterosuperior calcaneus	Fibrocartilaginous bursal walls show degeneration and/or calcification, with hypertrophy of the synovial infoldings and accumulation of fluid in the bursa
Superficial calcaneal bursitis	Bursa located between calcaneal prominence or the Achilles tendon and the skin	Swelling of posterolateral calcaneus (often associated with rigid shoes)	Solid swelling (sometimes discoloration) often located at the posterolateral calcaneus	Inflamed bursa, lined by hypertrophic synovial tissue and fluid

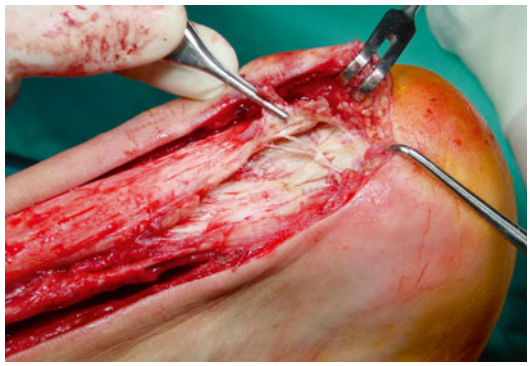

Fig. 95.3 A 28-year-old soccer player with a history of several local steroid injections presents with a sub-complete rupture of Achilles tendon distal insertion

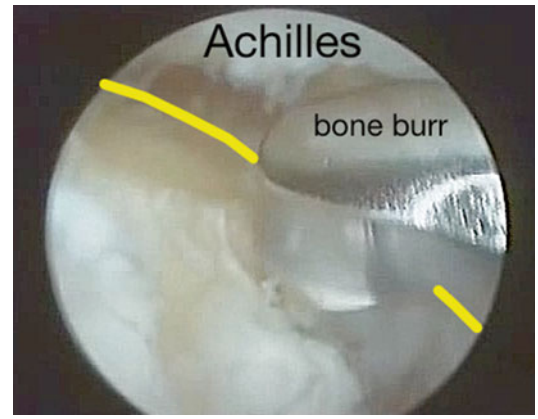

Fig. 95.4 Endoscopic calcaneoplasty

partial rupture, and therefore the need for radiologic evaluation occurs.

95.3.2 Architectonical Structure of the Achilles

In order to fully understand this pathology, one must understand the architectonical structure of the Achilles tendon. Sixty-five to 75 % of tendon consists of collagen (mostly type I) that is responsible for its mechanical strength. Two percent of tendon dry mass is elastin, responsible for recovery of the wavy configuration of the collagen fibres after stretch [5]. Type I collagen molecules have a unique ability to form microfibrils, as well as larger unit fibrils and fibres, that finally create the Achilles tendon (Fig. 95.6).

95.3.3 Metabolism of Tendon Cells

The level of oxygen consumption of a tendon is relatively low comparing to other tissues, like muscles or the liver. However, keeping in mind that the cell mass in tendon is only 1–3 % (while in the muscle almost 95 %) and calculating this ratio "per cell", not "per the dry mass of the tissue", this

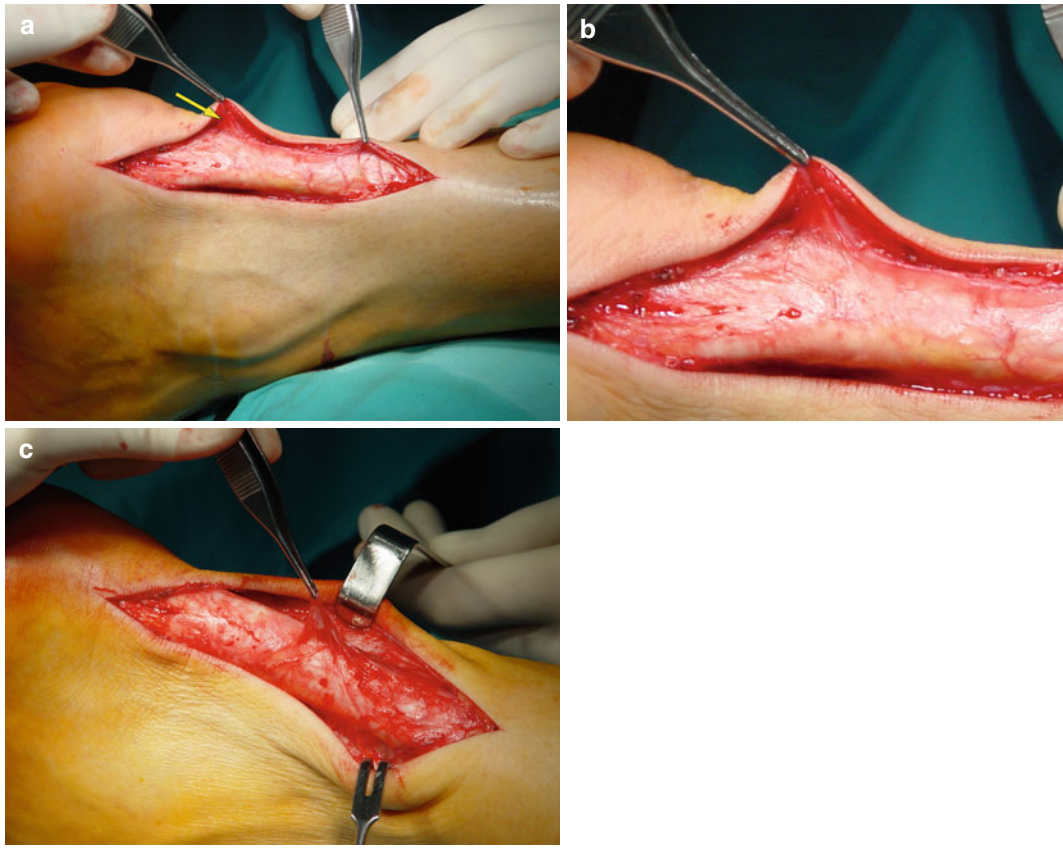

Fig. 95.5 (**a**) A 43-year-old female with chronic paratendonitis and tendinopathy, who did not respond to conservative treatment. Notice (marked with *yellow arrow*) adhesions between the paratendon, Achilles tendon and subcutaneous tissue. This may cause pain and crepitations. Some physiotherapist tries to break those adhesions with intensive manual therapy. (**b**) Same patient – zoom in on the adhesions between the paratendon and surrounding tissues. (**c**) Different patient, similar appearance: 38-year-old male, amateur runner with chronic paratendonitis

difference becomes much lower [17]. During the highest growth rate of a young tendon, all the three pathways of energy production (which means Krebs cycle, pentose phosphate shunt and anaerobic glycolysis) in the tendon are highly active. With increasing age, activity of Krebs cycle and the pentose phosphate shunt decreases, and production of energy changes to anaerobic [9, 12]. In neonate tendon collagen synthesis is high, but reduces drastically with age. Collagen turnover of adult tendon is low, comparable to ligamentous tissues. Metabolically most active collagen is the most newly synthesised [10]. This low metabolic rate of tendon, with well-developed anaerobic energy production, is essential if the tendon is to carry loads and remain in tension for periods of time without the risk of ischaemia and necrosis. However, the inevitable drawback of this low metabolic rate is also a slow recovery rate after activity and healing after injury [29].

95.3.4 Collagen Degeneration

A complete breakdown of collagen requires a number of enzymatic steps. The collagen enzymes make only one cut through each alpha chain, and this results in denaturation of the molecule [3, 23]. The denatured collagen is susceptible to degeneration with other proteolytic enzymes. Collagenases are produced by several tissues and cells, such as synovial cells, leucocytes and macrophages. Additionally, hormones and other substances may play, undefined yet, an

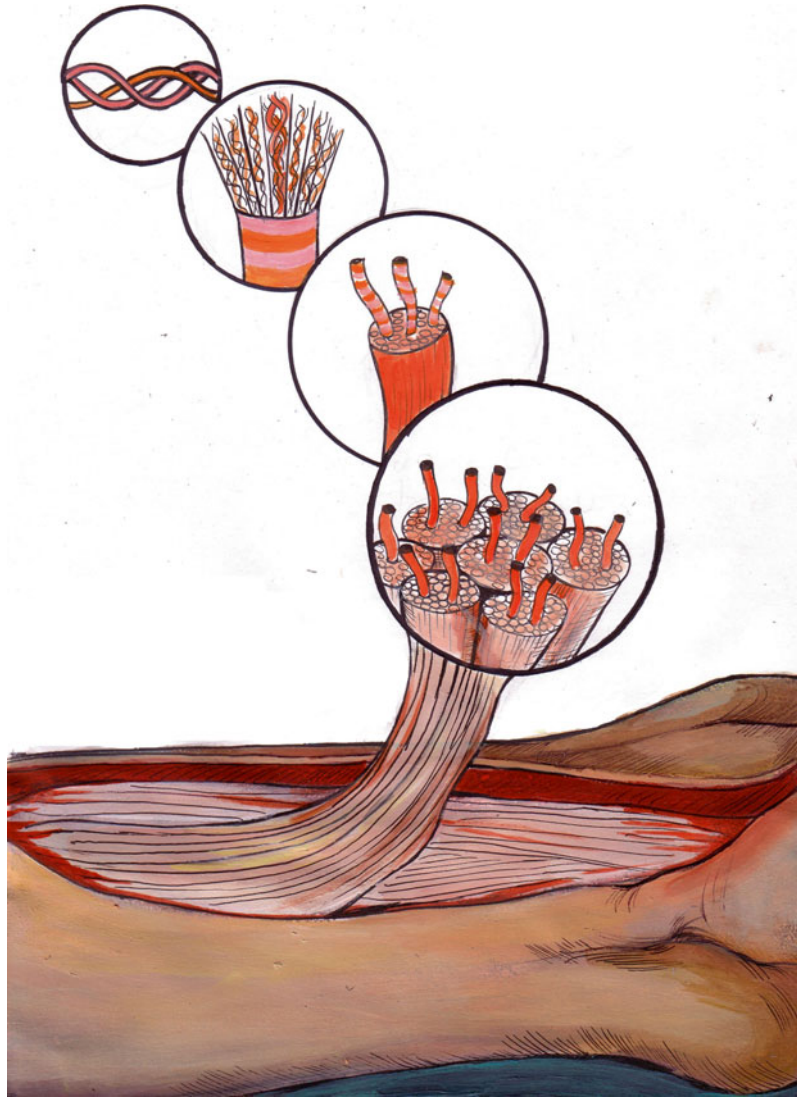

Fig. 95.6 Schematic drawing of Achilles tendon architectonical structure that consists of microfibrils forming bigger and bigger fibrils, fibres and finally Achilles tendon

operational role in the process of collagen degeneration [18].

95.3.5 Overuse Injury

An overuse injury is a long-standing orthopaedic problem and pain in the musculoskeletal system, which begins during exertion due to repetitive tissue micro-trauma. Repetitive micro-trauma results in microscopic injury. According to current knowledge, "overuse" in tendon injuries implies that the tendon has been strained repeatedly 4–8 % of its original length until it is unable to endure further tension, whereupon injury occurs [19]. In order to heal micro-injuries in otherwise poorly vascularised tendon tissue – neovascularisation is required. The majority of the blood supply to the Achilles tendon comes through the paratendon especially from its anterior surface [1].

VEGF is a growth factor, which stimulates endothelium cells and vessels to grow into the tendon, which is required for healing. But in normal tendon, there is no place for vessels; therefore,

VEGF needs to simulate also some extent of tendon degeneration to prepare this tissue, "making space for new vessels".

Area of neovascularisation within the Achilles tendon should be considered as an area with neovessels and accompanying nerves. And although those nerves are probably the direct cause of pain, nevertheless while treating those patients, one should not "kill" these new vessel formations, because it is a symptom and initial phase of healing.

Tendinopathy (Fig. 95.7) is a result of repetitive micro-trauma and failure of reparative process that may lead to symptomatic or asymptomatic micro-injuries and finally to partial or complete rupture (Fig. 95.8). Majority of complete Achilles tendon rupture patients previously had no symptoms of the Achilles tendon problems (but all of them present histopathologic changes within the tendon). Contrary it is rare that a symptomatic tendinopathy leads to complete rupture. This may be due either to the fact that symptomatic patients, because of pain, limit their activity and therefore stop overusing their tendons or maybe those with complete ruptures had much poorer tissue response and ability for healing.

Fig. 95.7 Schematic drawing picturing the idea of how a number of micro-injuries may cumulate and result in partial injury

95.3.6 Diagnosis and Treatment

In case of clinical symptoms of tendinopathy, the key is to determine the extent and the degree of tissue injury, because that would determine the choice of treatment. In some cases thickening (Fig. 95.9) or thinning might be observed. The severity of process is not proportional to the degree of pain. Therefore, at least one radiological examination, ultrasound and/or magnetic resonance, is required. Unfortunately, both, in inexperienced hands, might give false-positive and false-negative results. Be wise in choosing a good place and good radiologist, and trust your clinical examination.

With the extent of injury, the need to enhance the healing grows.

- With minimal structural changes, rehabilitation might be sufficient. Khan et al. [14] proposed mechanotherapy which turns movement into tissue healing. Immobilisation has not been proven as beneficial in those cases [28].
- With more advanced tendon changes, some healing stimulation incarnated as growth factor and/or stem cells injections, preferably under ultrasound control
- Partial injuries (Figs. 95.10 and 95.11) or failure of conservative treatment might require operative treatment.

95.4 Rupture of the Achilles Tendon

Partial rupture of Achilles was first described by Ljungqvist in 1967 [16]. It is believed that partial rupture results from asymmetric loading of

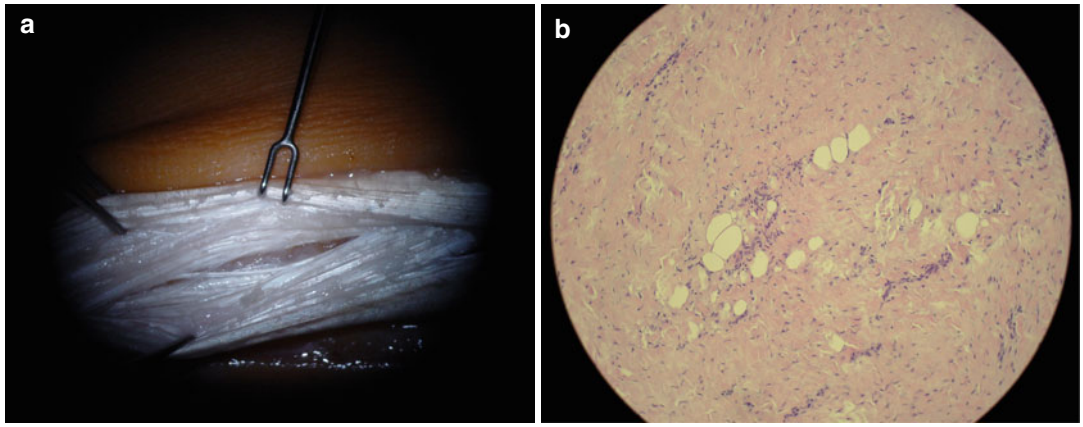

Fig. 95.8 Achilles tendinopathy. (**a**) Intraoperative picture (seen through operative microscope), notice shiny fibres are healthy ones, "frosted" ones – it is where tendinopathy occurs. (**b**) Histopathological view of Achilles tendinopathy: degeneration, old reparative process, disturbance of fibre arrangement

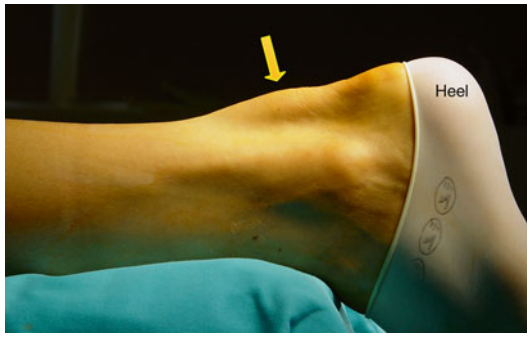

Fig. 95.9 A 36-year-old male with chronic, left Achilles tendinopathy. Notice thickening (marked with *yellow arrow*) of the tendon in typical area of Achilles waist

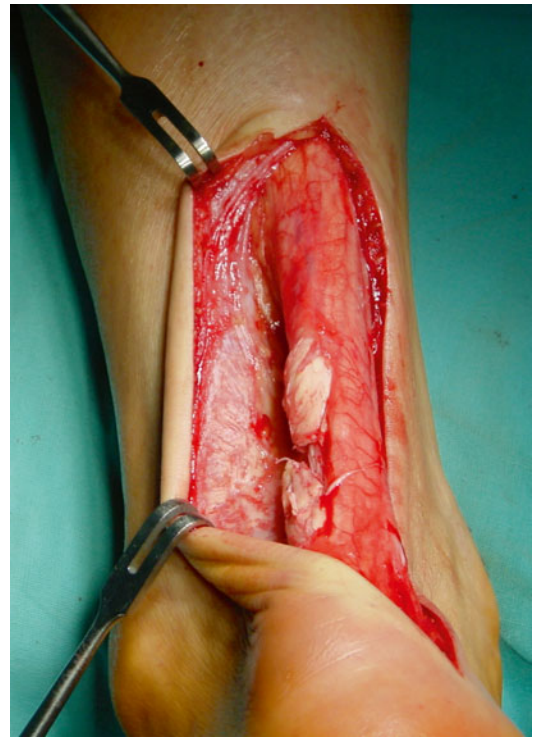

Fig. 95.10 A 27-year-old professional female boxer, with acute, partial rupture (marker with *arrow*) of fibres originating from medial head of the gastrocnemius muscle of left Achilles tendon (type: A-I-GM according to Śmigielski)

Achilles tendon. This asymmetrical loading is possible, because Achilles tendon is not a uniform structure, but consists of three separate bundles: one from medial, one from lateral head of the gastrocnemius muscle and one from soles muscle [20, 21, 24]. The injury occurs in the most loaded bundle. Śmigielski [20] proposed a new classification of partial Achilles tendon injuries, based on both histologic and anatomic appearance. In this classification first part states if this is an acute (A) or chronic (B) case. Second part describes how many Achilles tendon bundles are involved, and the last part describes which bundle is injured: S, fibres originating from soles muscle; GM, fibres from medial head of gastrocnemius muscle; and GL, lateral head of gastrocnemius muscle (e.g. B-I-GM means partial chronic injury of fibres originating from medial head of the gastrocnemius muscle).

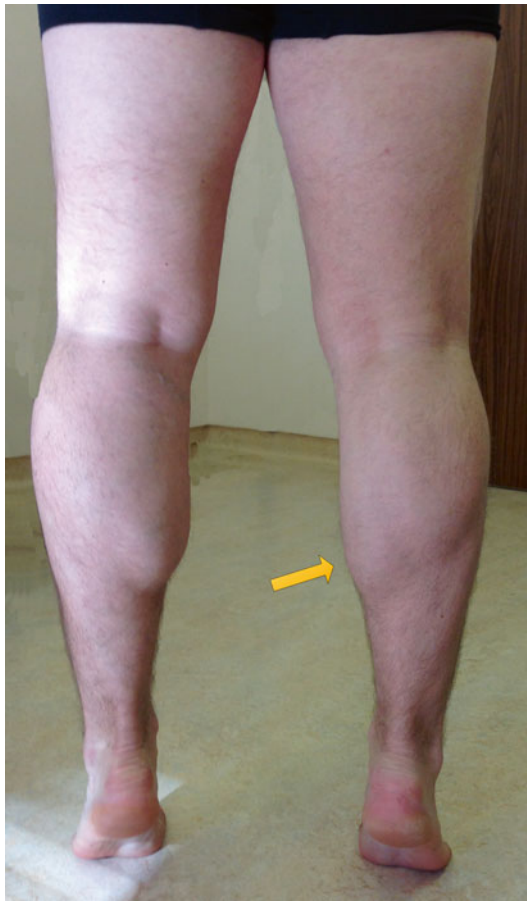

Fig. 95.11 Patient with chronic partial right Achilles tendon rupture. Notice atrophy (marked with *arrow*) of medial head of the gastrocnemius muscle – typical for chronic partial rupture of fibres from medial head of the gastrocnemius muscle. Type: B-I-GM, according to Śmigielski

The treatment of choice for partial ruptures of Achilles tendon is operative reconstruction [20, 21]. Exceptions are patient with general contraindications for operation, patient with rheumatoid diseases and patients with a low level of activity and low expectations, in terms of Achilles tendon function, like running or jumping [20].

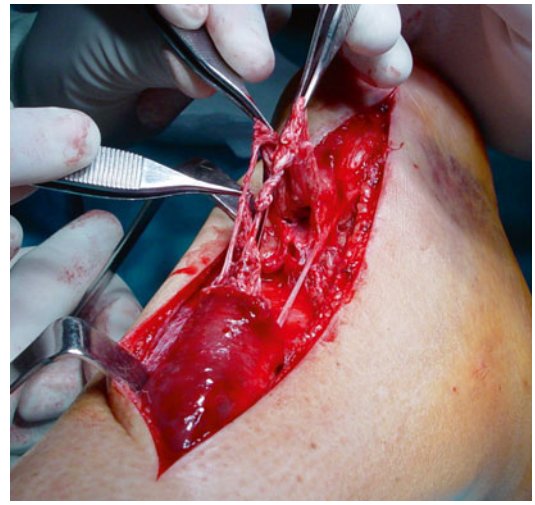

Fig. 95.12 Complete rupture of the Achilles tendon. Some elements of the three bundle structure are still visible

Also in complete ruptures of Achilles tendon, three-bundle structure of Achilles is clearly seen (Fig. 95.12). Those bundles/units depending on the side rotate as left- or right-handed screws – e.g. left Achilles tendon rotates (30–150°) against clockwise [20, 24] (Fig. 95.13a–c). This rotation has significance. It determines Achilles tendon function in terms of jumping and running, and therefore one should try to reconstruct those units and its rotation. On the other hand, in the area of the biggest torsion, there are the highest pressure forces and the poorest blood supply – this area is the most frequent injured one. Also in histopathologic evaluation, we observe much dense fibrocartilage there, for one side reinforcement to sustain those high pressure forces, but in the same time it means poor blood supply.

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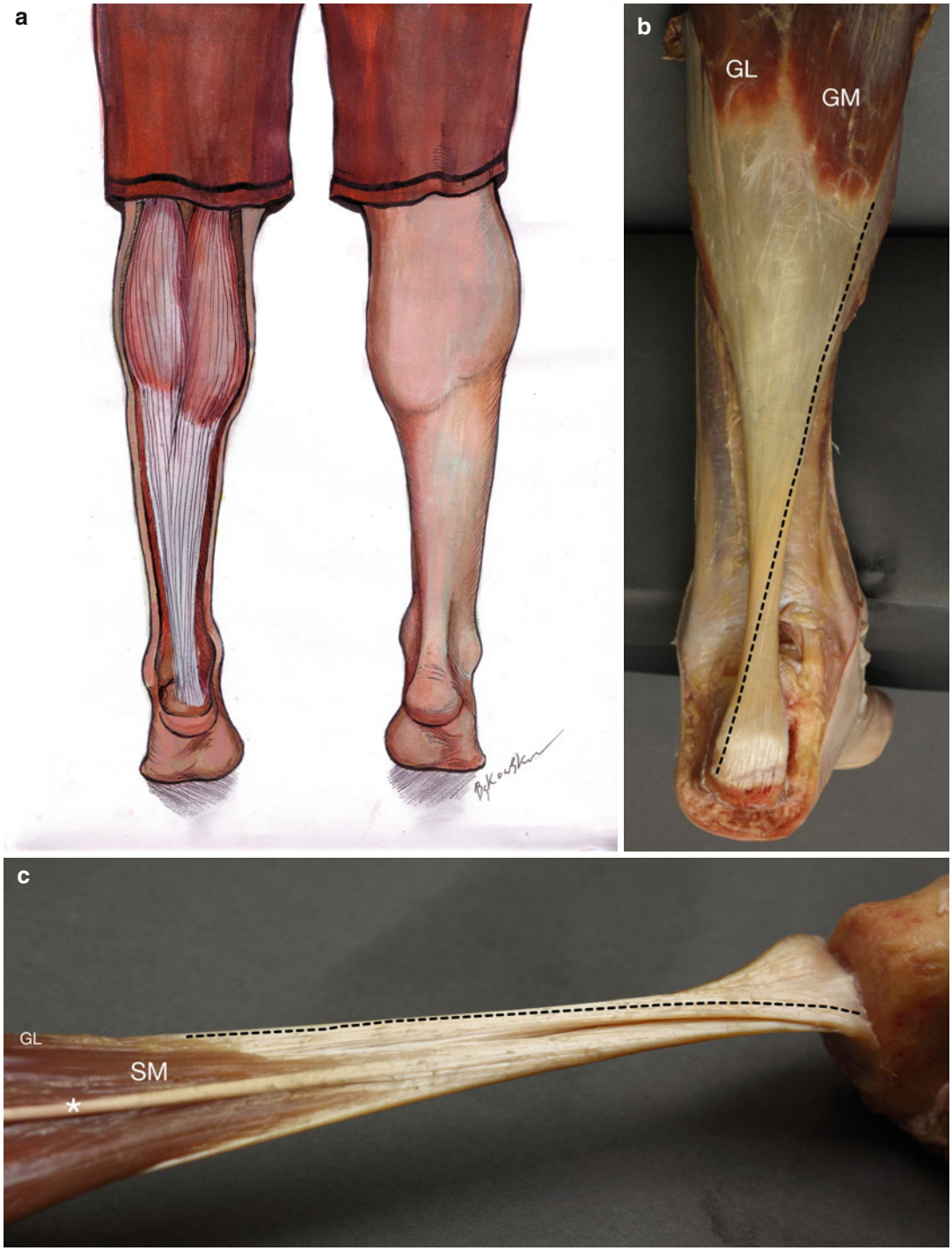

Fig. 95.13 (**a**) Schematic drawing of rotational anatomy of the Achilles tendon. (**b**) Anatomic dissection of the left Achilles tendon. GM – medial head of gastrocnemius muscle. GL – lateral head of gastrocnemius muscle. Notice the rotation of fibres of Achilles tendon and the way fibres originating from medial head of gastrocnemius muscle reaches the calcaneus on the lateral side (marked with *dashed line*). (**c**) Anatomic dissection. Ventral (anterior) part of Achilles tendon. *SM* soleus muscle. GL – lateral head of gastrocnemius muscle. (*) Plantaris tendon. Notice the way fibres from lateral head of the gastrocnemius muscle approaches calcaneus on the medial site (marked with *dashed line*). These are the fibres that get injured the first in cases of prominent posterosuperior edge of the calcaneus, because they are the closest to the bone

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