Metadata of the chapter that will be visualized online

Chapter Title	Achilles Tendon Rupture	
Copyright Year	2015	
Copyright Holder	Springer International Publishing Switzerland	
Corresponding Author	Family Name	**Maffulli**
	Particle	
	Given Name	**Nicola**
	Suffix	
	Division	Department of Musculoskeletal Disorders
	Organization/University	School of Medicine and Surgery, University of Salerno
	City	Salerno
	Country	Italy
	Division	Centre for Sports and Exercise Medicine
	Organization/University	Mile End Hospital, Barts and the London School of Medicine and Dentistry, Mary University of London
	Street	275 Bancroft Road
	City	London
	Postcode	E1 4DG
	Country	England, UK
	Email	n.maffulli@qmul.ac.uk
Author	Family Name	**Giai Via**
	Particle	
	Given Name	**Alessio**
	Suffix	
	Division	Department of Orthopaedic and Traumatology
	Organization/University	School of Medicine, University of Rome “Tor Vergata”
	Street	Viale Oxford 81
	City	Rome

Author's Proof

	Postcode	00133
	Country	Italy
	Email	alessiogiaivia@hotmail.it
Author	Family Name	**Oliva**
	Particle	
	Given Name	**Francesco**
	Suffix	
	Division	Department of Orthopaedic and Traumatology
	Organization/University	School of Medicine, University of Rome "Tor Vergata"
	Street	Viale Oxford 81
	City	Rome
	Postcode	00133
	Country	Italy
	Email	olivafrancesco@hotmail.com

Abstract

The Achilles tendon (AT) is the largest tendon in the human body, but it is also the one that frequently undergoes a complete subcutaneous tear. Men are more frequently affected than women, in particular between 30–40 years old. An AT tear is usually the end result of an asymptomatic process of failed response typical of tendinopathy. The diagnosis of acute tears of the AT is clinical, based on careful history taking and detailed clinical examination. Operative management of acute AT ruptures provides lower re-rupture rate, early functional treatment, less calf atrophy, and better functional results, in particular in athletes. Minimally invasive Achilles tendon repair provides many advantages and should be considered in young and active patients. Simple parameters as single-legged concentric strengthening, range of motion, and calf circumference can be used to predict the ability to return to activity. On the basis of current evidence-based studies, the routine use of PRP to improve tendon AT healing is not recommended.

Keywords (separated by " - ") Achilles tendon - Ruptures - Athletes - Sports - Surgical repair

10 Achilles Tendon Rupture

Nicola Maffulli, Alessio Giai Via, and Francesco Oliva

10.1 Introduction

The Achilles tendon (AT) is the thickest and the strongest tendon in the human body with a tensile strength in the order of 50–100 N/mm [1]. About 15 cm long, it originates in the midcalf and extends distally to insert into the posterior surface of the calcaneus. It is formed from the joining of the two tendons of soleus (dorsally) and gastrocnemius (ventrally). Despite its strength, it is one of the tendons most commonly affected by spontaneous rupture. Most acute ruptures (75 %) occur during recreational activities in men between 30 and 40 years old, in particular in soccer, basketball, tennis, and squash, but 25 % of ruptures may occur in sedentary patients [2]. The incidence rate ranges from 6 to 18 per 100,000 people per year, and it has been steadily increasing during the past few decades [2].

AT rupture can present acutely or as chronic tears (>6 weeks). Management of acute ruptures is still controversial. Recent well-conducted randomized controlled trials showed that conservative treatment with accelerated functional rehabilitation and open surgery management produce, in an unselected population, similar functional results [3, 4]. However, a relatively high re-rupture rate is still reported in patients treated conservatively [4, 5], and healing in a lengthened position may determine loss of calf muscle strength. These results are not acceptable in young patients and athletes. In these patients, operative management provides lower rerupture rate, early functional treatment, less calf atrophy, and better functional performance than nonsurgical treatment.

N. Maffulli, MD, PhD, FRCP, FRCS (✉)
Department of Musculoskeletal Disorders, School of Medicine and Surgery, University of Salerno, Salerno, Italy

Centre for Sports and Exercise Medicine, Mile End Hospital, Barts and the London School of Medicine and Dentistry, Mary University of London, 275 Bancroft Road, London E1 4DG, England, UK
e-mail: n.maffulli@qmul.ac.uk

A. Giai Via, MD • F. Oliva, MD, PhD
Department of Orthopaedic and Traumatology, School of Medicine, University of Rome "Tor Vergata", Viale Oxford 81, Rome 00133, Italy
e-mail: alessiogiaivia@hotmail.it; olivafrancesco@hotmail.com

10.2 Etiopathogenesis and Injury Mechanism

Acute AT rupture is a serious injury for high-level athletes. Tendon fibers begin to disrupt after a length increase of 3–4 % and rupture after an increase of 8 %. Ruptures usually occur between 2 and 6 cm of its insertion into the superior surface of the calcaneus. The tendon is at the greatest risk of rupture when it is obliquely loaded, the muscle is contracting maximally, and the tendon length is short [6]. The most common mechanism of injury is pushing off with the weight-bearing forefoot while extending the knee. Sudden unexpected dorsiflexion of the ankle or violent dorsiflexion of a plantar flexed foot may also result in ruptures [6]. Men are more frequently affected than women, in particular in their fourth decade [6].

Even though the rupture seemingly occurs as consequence of a traumatic insult on a nevertheless healthy tendon, in reality it is the end result of a single eccentric contraction on a tendon in which a tendinopathic process had been present, albeit not symptomatic [7]. The etiopathogenesis of AT rupture is still unknown, but histological evidence of failed healing response is relatively common. Both tendinopathic and ruptured tendons have a greater degree of histological evidence of tendinopathy compared with normal tendons, and the degree of degeneration in the ruptured group is statistically greater than in the tendinopathic group [7]. Corticosteroids, fluoroquinolone use, rheumatoid arthritis, and renal transplantation have been associated with AT rupture [6, 8]. More recent researches show that metabolic disease and endocrine disorders such as diabetes mellitus, hypothyroidism, hypercholesterolemia, and obesity could predispose to tendinopathies and tendon tears [9–12].

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_10,

10.3 Clinical and Diagnostic Examination

Acute AT rupture is usually a clinical diagnosis based on a careful history and detailed clinical examination. Patients often give a history of feeling a blow to the posterior aspect of the leg and may describe an audible snap followed by pain and inability to walk. A gap in the Achilles tendon is usually palpable. With increased time after the tear, the gap may be obliterated by edema, which makes palpation unreliable, while in the early stages, edema and bruising may not be apparent. Active plantar flexion of the foot is usually preserved given the action of the tibialis posterior, the long toe flexors, and the peroneus muscles. Numerous clinical tests have been described to aid in the diagnosis of Achilles tendon tears, and palpation, calf squeeze test, Matles test, O'Brien test, and Copeland test have been used [13]. All the tests described in the literature may be used to correctly diagnose a subcutaneous Achilles tendon tear with a high degree of certainty.

A retrospective study showed that if two or more of these tests are positive, the diagnosis of an AT tear is certain [13]. As the Copeland and the O'Brien tests may cause discomfort, the diagnosis of a subcutaneous tear of the Achilles tendon may be reliably made on the basis of the calf squeeze and Matles tests. The calf squeeze test, first described by Simmonds in 1957 [14] but often credited to Thompson, is performed with the patient prone and the ankles clear of the table. The examiner squeezes the fleshy part of the calf, causing deformation of the soleus and resulting in plantar flexion of the foot if the Achilles tendon is intact. The affected leg should be compared to the contralateral leg. The Matles test or knee flexion test is performed with the patient prone and the ankles clear of the table [15]. The patient is asked to actively flex the knee to 90°. During this movement, the foot on the affected side falls into neutral or dorsiflexion, and a rupture of the Achilles tendon can be diagnosed (Fig. 10.1).

Fig. 10.1 The Matles test or the knee flexion test: the foot on the affected side falls into neutral or dorsiflexion

Sometimes diagnostic imaging may be required to verify a clinical suspicion or for chronic injuries. Plain lateral radiographs may reveal an irregular configuration of the fat-filled triangular space anterior to the Achilles tendon and between the posterior aspect of the tibia and the superior aspect of the calcaneus. It is also helpful to exclude bone injuries in case of acute trauma. Ultrasound (US) and magnetic resonance imaging (MRI) are widely used, even if there is no clear evidence that they improve the rate of correct diagnosis. According to the AAOS guidelines for acute AT rupture, there is not enough evidence to recommend for or against the routine use of US and MRI to confirm the diagnosis [16]. A recent study showed that physical examination, including an abnormal calf squeeze test, a palpable defect, and decreased resting tension, is more sensitive in diagnosing an acute complete AT rupture than MRI (sensitivity 100 % vs 90.9 %) [17]. Moreover MRI is time consuming and expensive and can lead to a delay in treatment (mean time to surgery 5.6 days vs 12.4 day in MRI group). The authors concluded recommending careful evaluation and judicious use of advanced imaging as needed.

10.4 Treatment Strategy

The consensus for athletes is surgery [2], as it provides early functional treatment, less calf atrophy, and the best functional performance with a lower re-rupture rate. Open, percutaneous, or minimally invasive procedures have been successfully used. Open surgery provides good strength to the repair, low re-rupture rates, and reliably good endurance and power to the gastrocnemius-Achilles tendon complex. However, open surgical approaches resulted in high risk of infection and morbidity. Review articles and meta-analysis showed high costs and a 20-fold higher rate of complications in open procedures than conservative treatment [13]. Therefore, minimally invasive procedures have been successfully used to avoid these complications. Minimally invasive AT repair provides many advantages. Major advantages are less iatrogenic damage to normal tissues, lower postoperative pain, accurate opposition of the tendon ends minimizing surgical incisions, and improved cosmetics. A recent systematic review reported a rate of superficial infections of 0.5 and 4.3 % after minimally invasive and open surgery, respectively [18]. Shorter hospitalization time and average time to return to working activities was also shown. Functional outcomes were not significantly different between minimally invasive and open surgery. Although sural nerve injury has been reported as a potential complication of this kind of surgery, new techniques have minimized the risk of sural nerve damage [18].

10.5 Percutaneous Achilles Tendon Repair: Surgical Technique

A 1 cm transverse incision is made over the defect using a size 11 blade. Four longitudinal stab incisions are made lateral and medial to the tendon 6 cm proximal to the palpable defect. Two further longitudinal incisions on either side of the tendon are made 4–6 cm distal to the palpable defect. Forceps are then used to mobilize the tendon from beneath the subcutaneous tissues. A 9 cm Mayo needle is threaded with two double loops of Number 1 Maxon, and this is passed transversely between the proximal stab incisions through the bulk of the tendon (Fig. 10.2). The bulk of the tendon is surprisingly superficial. The loose ends are held with a clip. In turn, each of the ends is then passed distally from just proximal to the transverse Maxon passage through the bulk of the tendon to pass out of the diagonally opposing stab incision. A subsequent diagonal pass is then made to the transverse incision over the ruptured tendon. To prevent entanglement, both ends of the Maxon are held in separate clips. This suture is then tested for security by pulling with both ends of the Maxon distally. Another double loop of Maxon is then passed between the distal stab incisions through the tendon (Fig. 10.3) and in turn through the tendon and out of the transverse incision starting distal to the transverse passage (Fig. 10.4). The ankle is held in full plantar flexion, and in turn the opposing ends of the Maxon thread are tied together with a double-throw knot, and then three further throws before being buried using the forceps (Fig. 10.5). A clip is used to hold the first throw of the lateral side to maintain the tension of the suture. We use 3-0 Vicryl suture to close the transverse incision and Steri-Strips close the stab incisions. A nonadherent dressing is applied. A full plaster cast is applied in the operating room with the ankle in physiologic equinus. The cast is split on both medial and lateral sides to allow for swelling. The patient is discharged on the same day of the operation.

Fig. 10.2 A 9 cm Mayo is threaded with two double loops of Number 1 Maxon, and this is passed transversely between the proximal stab incisions through the bulk of the tendon

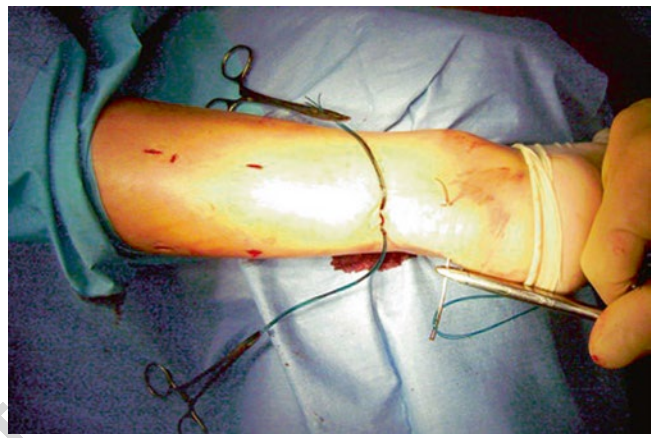

Fig. 10.3 Another double loop of Maxon is then passed between the distal stab incisions through the tendon

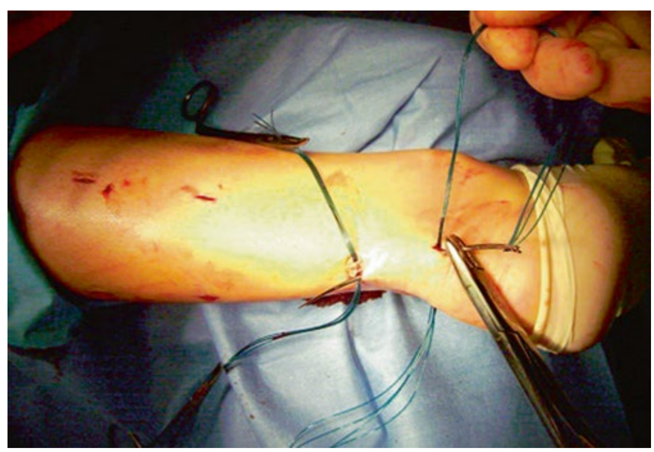

Fig. 10.4 The double loop of Maxon is passed in turn through the tendon and out of the transverse incision starting distal to the transverse passage

10.6 Rehabilitation and Return to Play

Following percutaneous repair, patients are encouraged to bear weight on the operated limb as soon as possible as tolerated. The cast is removed at 2 weeks postoperatively, and a boot with the ankle in a plantigrade position is used. Removal of the boot under supervision of a physiotherapist allows the ankle to be plantar flexed fully but not dorsiflexed. These

Fig. 10.5 The two tendon stumps are sutured together with the ankle in full plantar flexion

exercises are performed against manual resistance. At 6 weeks postoperatively, the boot is removed, and the patient referred to physiotherapy for active mobilization. At 12 weeks postoperatively, patients are assessed as to whether they were able to undertake more vigorous physiotherapy and are encouraged to gradually return to their normal activities. Progressive activities are incorporated as strength allows, with the aim to return to unrestricted activities by 6 months following surgery. Patients are reviewed at 3-month intervals and discharged at 9 or 12 months after the operation once they are able to perform at least five toe raises unaided on the operated leg and after they returned to their normal activities.

10.7 Discussion

AT ruptures are common in athletes. Surgical repair provides good results in young and active people. Open, percutaneous, or minimally invasive procedures have been successfully used. Open surgery provides good strength to the repair, low re-rupture rates, and reliably good endurance and power to the gastrocnemius-Achilles tendon complex. However, open surgical repair may result in high risk of infection and morbidity. Review articles and meta-analysis showed high costs and a 20-fold higher rate of complications in open procedures than conservative treatment [6]. Therefore, minimally invasive procedures have been successfully used to avoid these complications. Minimally invasive Achilles tendon repair provides many advantages. Major advantages are less iatrogenic damage to normal tissues, lower postoperative pain, accurate opposition of the tendon ends minimizing surgical incisions, shorter hospitalization time, lower rate of

Table 10.1 Parameters used in assessing the time frame of patients undergoing Achilles tendon surgery return to activity (RTA)

Parameters predicting the ability to RTA	
Concentric strengthening	Ability to perform 5 sets of 25 single-legged heel raises
Muscle girth	Calf circumference: 5 mm or less difference measured 10 cm distal to the tibial tuberosity of the operative limb as compared with the nonoperative limb
Range of motion (ROM)	Ankle dorsiflexion and plantar flexion ROM within 5° of the nonoperative limb

postsurgical infections, and improved cosmesis [18]. Excellent results have been reported in 17 elite athletes after percutaneous surgical repair of Achilles tendon ruptures [19]. All patients returned to high-level competition, with an average time to return to full sport participation of 4.8 months (range 3.2–6.5).

Rehabilitation of the Achilles tendon is complex and often nonstandardized. Detailed postoperative physical therapy programs for the AT often vary. Return to activity (RTA) can be defined as the time in which patients initiate their desired activity or sport that was limiting them. Evidence-based study of physical therapy regimens with regard to the foot and ankle is very limited. Modalities that have been rigorously studied have shown little benefit, including ultrasound, massage, and injections [20, 21]. Both eccentric exercises and extracorporeal shockwave therapy (ESWT) have been studied with a wide range of results. When patients can return to sports reducing the risk of further injuries is a big question, in particular for athletes because physicians are often faced with the pressing requirements of the athlete himself, the coach, and the team. Recurrence of AT tendinopathy and reinjury risk has been reported to be higher after short recovery periods [22]. Saxena et al. reported that simple parameters such as single-legged concentric strength, range of motion, and muscle girth can predict the ability to RTA [21]. If patients meet all 3 of these criteria, they are allowed to return to sport (Table 10.1), and the mean time to RTA after AT surgical repair was 21.8 ± 4.0 weeks. Females were more likely to have a delay in RTA.

Restoring the normal structure and function of injured tendons is a great challenge, so several strategies have been proposed to enhance tendon healing. Recently research focused on regenerative therapies such as growth factors (GFs) and plasma-rich platelet (PRP), but this is still controversial. The use of PRPs has expanded to meet multiple medical problems where current treatment options were judged suboptimal. It is currently a common treatment for the tendon injuries because of the autologous source, safety profile, and minimal manipulation [23]. In vitro studies showed that the addition of PRP to human tenocytes resulted in cell proliferation, collagen deposition, well-ordered angiogenesis, and improved gene expression for matrix-degrading enzymes and endogenous growth factors [24, 25]. More recently, two studies demonstrated that PRP induced in vitro tendon mesenchymal stem cell (T-MSC) differentiation into active tenocytes and that PRP has an anti-inflammatory function by suppressing the levels of prostaglandin (PGE) biosynthetic pathway components (COX-1, COX-2, and mPGES-1 expression) and PGE2 production [26]. These results have important clinical implications because high levels of PGE2 cause pain, decrease cell proliferation and collagen production, and induce degenerative changes in rabbit tendons [27]. The same authors also reported that even though PRP is able to induce the differentiation of T-MSCs into tenocytes under regular culture conditions, PRP injection in routine clinical practice may not be able to effectively reverse the degenerative conditions of late-stage tendinopathy [28]. Currently, many studies are published in literature with conflicting results. In fact, although a recent study suggests that vascular endothelial growth factor-111 (VEGF-111) could have a potential positive effect on the healing of AT lesions in rats [29], another animal study shows that a single injection of PRP did not influence tendon healing [30]. Well-conducted clinical studies do not report any substantial benefit using PRP, and its routine use is thus not recommended [31].

Conclusion

AT rupture is a serious injury not only in high-level athletes. The management should take into account the age, occupation, and level of sporting activity of the patient. Open surgery is frequently associated with higher risk of superficial skin breakdown and wound problems, which can be prevented by performing percutaneous repair. Percutaneous repair followed by early functional rehabilitation is becoming increasingly common and may be considered in athletes.

References

1. Viidik A (1962) Tensile strength properties of Achilles tendon systems in trained and untrained rabbits. Acta Orthop Scand 10:261–272
2. Longo UG, Petrillo S, Maffulli N et al (2013) Acute Achilles tendon rupture in athletes. Foot Ankle Clin 18:319–338
3. Nilsson-Helander K, Silbernagel KG, Thomee R et al (2010) Acute Achilles tendon rupture: a randomized, controlled study comparing surgical and nonsurgical treatments using validated outcome measures. Am J Sports Med 38:2186–2193
4. Willits K, Amendola A, Bryant D et al (2010) Operative versus nonoperative treatment of acute Achilles tendon ruptures: a multicenter randomized trial using accelerated functional rehabilitation. J Bone Joint Surg Am 92:2767–2775
5. Guillo S, Del Buono A, Dias M et al (2013) Percutaneous repair of acute ruptures of the tendo Achillis. Surgeon 11:14–19
6. Maffulli N (1999) Rupture of the Achilles tendon. J Bone Joint Surg Am 81A:1019–1036
7. Tallon C, Maffulli N, Ewen SW (2001) Ruptured Achilles tendons are significantly more degenerated than tendinopathic tendons. Med Sci Sports Exerc 33:1983–1990
8. Stephenson AL1, Wu W, Cortes D, Rochon PA (2013) Tendon injury and fluoroquinolone use: a systematic review. Drug Saf
9. Bourne JW, Lippell JM, Torzilli PA (2014) Glycation cross-linking induced mechanical-enzymatic cleavage of microscale tendon fibers. Matrix Biol 34:179–184
10. Oliva F, Berardi AC, Misiti S, Maffulli N (2013) Thyroid hormones and tendon: current views and future perspectives. Concise review. Muscles Ligaments Tendons J 3:201–203
11. Beason DP, Abboud JA, Kuntz AF, Bassora R, Soslowsky LJ (2011) Cumulative effects of hypercholesterolemia on tendon biomechanics in a mouse model. J Orthop Res 29:380–383
12. Abate M, Oliva F, Schiavone C, Salini V (2012) Achilles tendinopathy in amateur runners: role of adiposity (Tendinopathies and obesity). Muscles Ligaments Tendons J 2:44–48
13. Maffulli N (1998) The clinical diagnosis of subcutaneous tear of the Achilles tendon. A prospective study in 174 patients. Am J Sports Med 26(2):266–70
14. Simmonds FA (1957) The diagnosis of the ruptured Achilles tendon. Practitioner 179:56–58
15. Matles AL (1975) Rupture of the tendon Achilles. Another diagnostic sign. Bull Hosp Joint Dis 36:48–51
16. Kou J (2010) AAOS clinical practice guideline: acute Achilles tendon rupture. J Am Acad Orthop Surg 18:511–513
17. Garras DN, Raikin SM, Bhat SB, Taweel N, Karanjia H (2012) MRI is unnecessary for diagnosing acute Achilles tendon ruptures: clinical diagnostic criteria. Clin Orthop Relat Res 470:2268–2273
18. Del Buono A, Volpin A, Maffulli N (2014) Minimally invasive versus open surgery for acute Achilles tendon rupture: a systematic review. Br Med Bull 109:45–54
19. Maffulli N, Longo UG, Maffulli GD et al (2011) Achilles tendon ruptures in elite athletes. Foot Ankle Int 32:9–15
20. Park D, Chou L (2006) Stretching for prevention of Achilles tendon injuries: a review of the literature. Foot Ankle Int 27:1086–1095
21. Saxena A, Ewen B, Maffulli N (2011) Rehabilitation of the operated Achilles tendon: parameters for predicting return to activity. J Foot Ankle Surg 50(1):37–40
22. Gajhede-Knudsen M, Ekstrand J, Magnusson H, Maffulli N (2013) Recurrence of Achilles tendon injuries in elite male football players is more common after early return to play: an 11-year follow-up of the UEFA Champions League injury study. Br J Sports Med 47:763–768
23. Andia I (2014) Platelet rich plasma therapies: a great potential to be harnessed. Muscles Ligaments Tendons J 4:1–2
24. Oliva F, Via AG, Maffulli N (2011) Role of growth factors in rotator cuff healing. Sports Med Arthrosc 19:218–226
25. Yuan T, Zhang CQ, Wang JH (2013) Augmenting tendon and ligament repair with platelet-rich plasma (PRP). Muscles Ligaments Tendons J 3:139–149
26. Wang JHC (2014) Can PRP, effectively treat injured tendons? Muscles, Ligaments and Tendons J 4:35–37
27. Khan MH, Li Z, Wang JH (2005) Repeated exposure of tendon to prostaglandin-E2 leads to localized tendon degeneration. Clin J Sport Med 15:27–33
28. Zhang J, Wang JHC (2014) PRP treatment effects on degenerative tendinopathy – an in vitro model study. Muscles, Ligament s and Tendons J 4:10–17
29. Kaux JF, Janssen L, Drion P et al (2014) Vascular Endothelial Growth Factor-111 (VEGF-111) and tendon healing: preliminary results in a rat model of tendon injury. Muscles Ligaments Tendons J 4:24–28
30. Parafioriti A, Armiraglio E, Del Bianco S, Tibalt E, Oliva F, Berardi AC (2011) Single injection of platelet-rich plasma in a rat Achilles tendon tear model. Muscles Ligaments Tend ons J 1:41–47
31. Maffulli N (2013) Autologous blood products in musculoskeletal medicine. BMJ 346:f2979

Author Query

Chapter No.: 10 0002273117

Query	Details Required	Author's Response
AU1	Please update Ref. [8]	