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Original Article

Novel surgical management of tibialis anterior tendinosis using an anchor augment

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Abstract

Objective: The objective of this paper was to describe a surgical technique for the management of TA tendinosis: Debridement, repair, and augmentation with a suture anchor without tendon transfer.

Methods: This is a retrospective case series including five patients managed surgically for TA tendinosis. If ≥ 30% of healthy tendon remains after debridement, the tendon is augmented with a suture anchor, with the suture incorporating the healthy tendon as a checkrein. Patient outcomes were assessed using the AOFAS midfoot, VAS Pain, and SEFAS score.

Results: The mean AOFAS improved preoperatively from 37.0 (range 18–51) to 97.6 (range 95–100), the VAS pain score from 8.0 (range 7–9) to 0.8 (range 0–1), and the mean SEFAS score at final follow-up was 43.8 (range 41–48).

Conclusions: Tibialis anterior tendinosis is an uncommon degenerative process. Early diagnosis and appropriate management can prevent tendon rupture. This case series of debridement, repair, and augmentation with a suture anchor showed high patient satisfaction and good clinical outcomes without needing a tendon transfer.

Level of Evidence III; Therapeutic Studies - Investigating the Results of Treatment; Retrospective Comparative Study

Keywords: Tendinopathy; Tendons transfer; Suture techniques.

Introduction

Tibialis anterior (TA) tendinosis is a rare condition often missed or diagnosed late as a tendon rupture, causing significant morbidity to the patient. Literature regarding TA tendinosis is scarce, unlike TA rupture, commonly described dating back to 1905[1-4]. Despite the TA having a well-vascularized posterior surface, an avascular zone exists where the tendon runs under the superior and inferior extensor retinacula. This results in an area with decreased blood flow and an increased risk of rupture, extending up to 1–3 cm from the tendon insertion[5-8].

A failed healing response may occur due to overuse or ongoing mechanical forces on the tendon in the area of poor blood supply. This results in the deposition of a disorganized matrix of hypercellular and hypervascular tissue, weakening the tendon and resulting in pain[9]. The TA is active for 54% of the gait cycle during walking and 73% during running while maintaining the longitudinal arch under load[10,11]. This heavy workload interferes with the normal healing response.

Tibialis anterior tendinosis commonly presents in 50- to 70-year-old overweight females. The most common presenting symptom is burning pain over the dorsal medial midfoot[5,12]. This burning pain is worse at night, affecting their sleep pattern. The increased tension from the foot in equinus at night could be a reason for this pain. Burning night pain is not common for tendinopathies and more associated with neurogenic, infectious, or malignant pathologies. As a result, the physician can misdiagnose this pathology. The authors have found that increased pain over the medial midfoot with

Study performed at the Wilgers MRI Department (Magnetic Resonance Imaging), Pretoria, South Africa.

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the patient's heel walking is diagnostic of TA tendinosis. Point tenderness exists over the TA insertion at the medial cuneiform, often associated with local swelling and edema[12]. Beischer et al.[13] described the tibialis anterior passive stretch (TAPS) test, which has a 90% sensitivity and 95% specificity for diagnosing TA tendinosis. The test is performed by plantarflexing the ankle, everting the hindfoot, and abducting and pronating the midfoot, thereby passively stretching the TA. The test is positive if the patient reports reproduction or increased pain over the TA insertion[5,13].

Ultrasound and magnetic resonance imaging (MRI) are the gold standard for diagnosing TA tendinosis by showing characteristic edematous tendon changes, fluid collections, and features of tendon degeneration (including tears)[13,14].

Untreated TA tendinosis can result in tendon rupture, causing a drop foot gait[13,14]. If TA tendinosis is managed appropriately early, it can decrease the patient morbidity from rupture. Management of TA ruptures and tendinosis reported in the literature often describes augmentation with an extensor hallucis longus (EHL) tendon transfer. Beischer et al. managed severe TA tendinosis with debridement, repair, and augmentation with EHL tendon transfer[13]. Cignetti et al.[12] performed only debridement in mild cases and augmentation with extensor digitorum longus tendon, plantaris tendon, or both in moderate to severe cases. Transferring the EHL comes with inherent risks and comorbidities. The authors prefer to avoid tendon transfers by debriding, repairing, and augmenting the tendon with an anchor.

The objective of this paper is to describe a surgical technique for the management of TA tendinosis: Debridement, repair, and augmentation with a suture anchor without tendon transfer. We also report on the functional outcome of a small case series using this technique.

Methods

This retrospective case series includes five patients managed surgically for TA tendinosis. This case series was approved by the university's Human Research Ethics Committee (M220489). Patients with TA tendinosis are initially given a trial of conservative management for up to 12 weeks, which includes immobilization in a moon boot with a forefoot wedge and avoiding all impact sports to prevent tendon rupture. If a patient has more than 50% tendon damage reported on the initial ultrasound, surgical intervention is recommended due to the increased risk of rupture, but conservative management is also explained so that the patient can make an informed decision. All patients older than 18 who had failed conservative management requiring surgical management were included. There were two males and three females in this case series, with a mean age of 68 (range 62–74).

Clinical assessment, surgical intervention, and follow-up were performed by the surgeon and recorded. Demographic data was recorded, including age, gender, body mass index (BMI), recreational activities, and sport participation. Comorbidities, such as inflammatory arthritis, diabetes, hypertension, gout, HIV, and immune suppressive medications, were also recorded. Diagnostic imaging included weight-bearing foot radiographs, ultrasound and/or MRI. The MRI was assessed by a musculoskeletal trained radiologist.

During surgery, the tendon was assessed macroscopically. The length of the longitudinal tear(s), thickness, and general appearance was recorded. Concomitant surgery and postoperative complications were also recorded.

Patient outcomes were assessed using The American Orthopaedic Foot and Ankle Society (AOFAS) midfoot scale and Visual analog pain scale (VAS) preoperatively and at the final follow-up. Patients completed a self-reported foot and ankle score (SEFAS) at the final follow-up. The mean follow-up was 39.8 (range 6.4–74.2) months.

Surgical technique

Patients were operated under general anesthesia, with a regional nerve block for pain control. A curvilinear incision was made 5 mm lateral to the course of the anterior tibial tendon, extending from the extensor retinaculum to its insertion on the medial cuneiform. The tendon sheath was opened, and all synovitis debrided. The tendon was examined and partially detached from the medial cuneiform so that any osteophytes could be removed using a rongeur (Figure 1). All devitalized tendon tissue was excised. If ≥ 30% of healthy tendon remains, the tendon was repaired using Vicryl 2-0 (Figure 2). The medial cuneiform was then drilled using a 2 mm drill bit to allow marrow elements into the tendon attachment area (Figure 3). A suture anchor was placed into the insertion point on the medial cuneiform. The suture anchor was run up

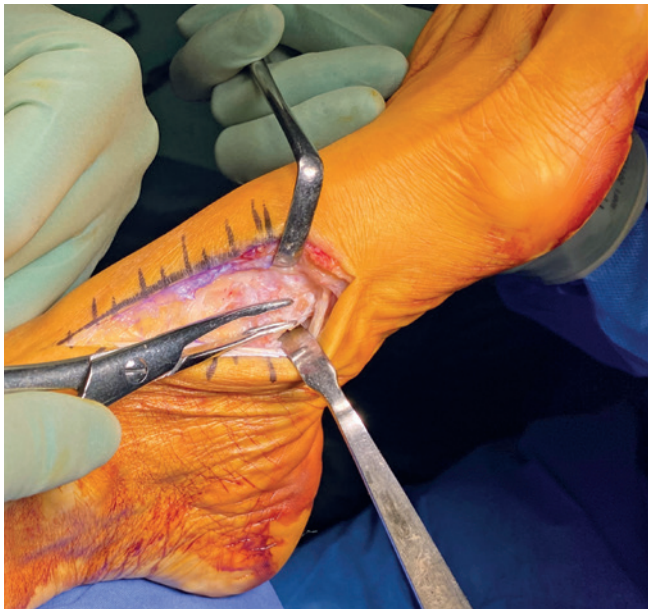

Figure 1. Bone osteophytes were removed from around the insertion site of the tibialis anterior tendon on the medial cuneiform.

the tendon in a whip stitch fashion incorporating at least 2 cm of the normal proximal tendon (Figure 4). The suture anchor was used as a checkrein to protect the damaged portion of the tendon while it heals. The tendon sheath was repaired using Vicryl 3-0 (Figure 5). The wound was closed in layers using Vicryl and Monocryl. The leg was placed in a below-knee plaster cast with the ankle in 10 degrees dorsiflexion. The patient was instructed to non-weight-bearing in the cast for four weeks. At four weeks, the patient was placed in a moon boot with progressive weight-bearing as tolerated. The patient transitions into supportive shoes at eight weeks. Physiotherapy started at four weeks focusing on edema control, scar management, tendon gliding, and strengthening. The patient was allowed to cycle and swim after six weeks, but no sports were allowed until week 12.

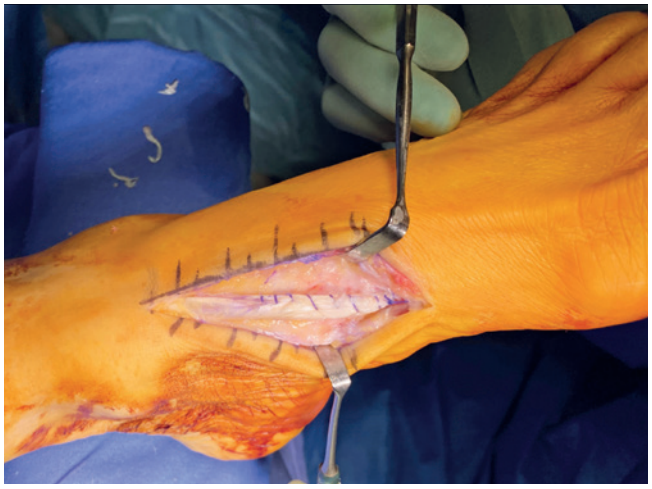

Figure 2. The remaining healthy tibialis anterior tendon was repaired using Vicryl 2-0.

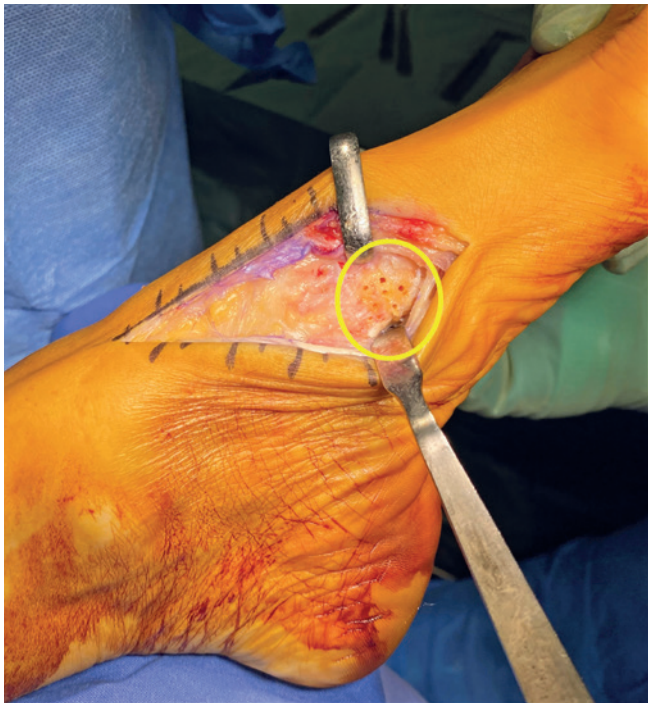

Figure 3. The insertion site on the medial cuneiform was perforated using a 2 mm drill bit to allow marrow elements to aid healing.

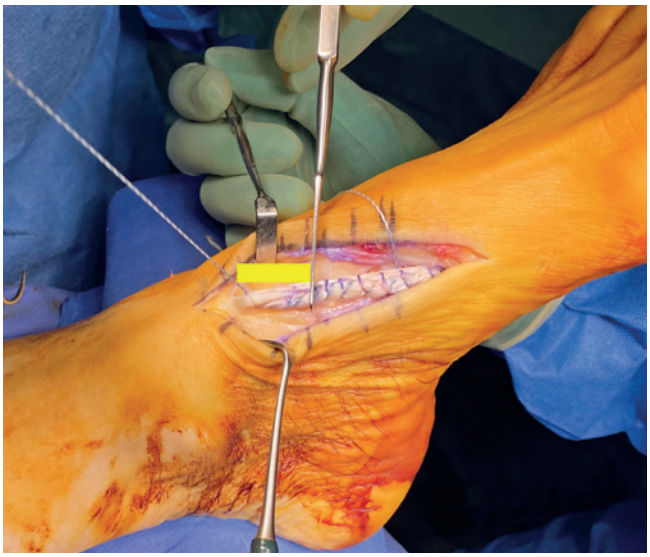

Figure 4. The suture anchor was run up the tendon in a whip stitch fashion incorporating at least 2 cm of the normal proximal tendon (Yellow bar indicates the portion of a normal tendon).

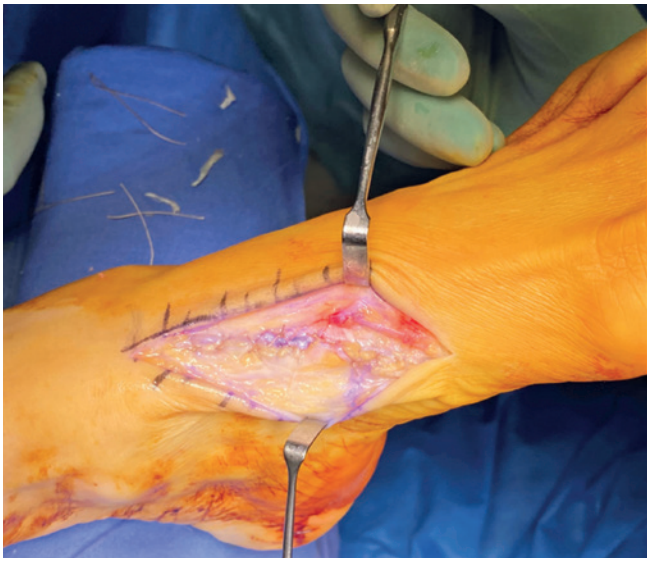

Figure 5. The tendon sheath was repaired using Vicryl 3-0.

Results

The average BMI was 31.8 (range 26–39) kg/m^2, with two being overweight, one obese, and one morbidly obese. None of the patients were smokers. Four of the patients had hypertension, and one suffered from gout. All five patients were physically active, and their recreational activities consisted of walking, cycling, going to the gym, and playing tennis.

All patients presented with a history of pain over the dorsomedial midfoot area, aggravated by impact activities, and present at night. The patient's symptoms were reproduced by asking them to walk on their heels.

All patients had ultrasound examinations reporting inflammation and edema surrounding the TA tendon. There was degeneration and/or a longitudinal tear visible from the insertion of the TA extending up to 40 mm proximally in various degrees of severity in all patients. Two patients had an MRI. Both showed tendinitis over the distal portion of the TA, with degeneration and possible partial tear.

Macroscopically, the pathology ranged from having degenerative changes of the tendon only (in one case) to longitudinal tears from the insertion to between 4 and 6 cm proximally. No complications occurred, and no concomitant surgery was required.

The mean AOFAS preoperatively was 37.0 (range 18–51) and postoperatively 97.6 (range 95–100). The mean VAS pain score improved from 8.0 (range 7–9) to 0.8 (range 0–1). The mean SEFAS score at the final follow-up was 43.8 (range 41–48), with all patients having excellent outcome scores (Table 1). All patients returned to their previous activity level and were satisfied with the result, and would recommend the surgery.

Discussion

Tibialis anterior tendon pathology has infrequently been reported in the literature. The natural history of this pathology is not well known. Coughlin et al.[5] reported that TA ruptures were caused by a traumatic event in patients with comorbidities, such as diabetes mellitus and inflammatory conditions, without a prodromal phase. The authors believe that TA ruptures result from degenerative tendinosis misdiagnosed in its early stages. Bernstein concluded in his paper on the spontaneous rupture of the TA tendon caused due to an abnormal tendon[15]. Kannus and Józsa[16] reported that 97% of tendons had degenerative changes on visual inspection and were structurally abnormal histologically in their review of 891 spontaneously ruptured tendons. Of note, this study included various tendons (including Achilles, biceps, patella, and TA, amongst others).

Table 1. Functional and outcome scores

Patient number	VAS preop	VAS postop	SEFAS postop	AOFAS preop	AOFAS postop
1	7	1	43	18	98
2	8	0	48	48	100
3	8	1	43	51	99
4	8	1	41	37	96
5	9	1	44	31	95
Mean	8.0	0.8	43.8	37.0	97.6

The cause of tendinosis has been associated with systemic diseases (inflammatory arthropathy, gout, diabetes mellitus), use of immunosuppressive medication, and steroid injections[8,12,17-19]. All five patients in our study group had medical comorbidities, and two were obese, which is also a well-described risk factor[13,14].

Conservative management of TA tendinosis is recommended for three months. Patients are immobilized in a moon boot for the first six weeks, with a forefoot wedge to offload the TA. Physical rehabilitation includes stretching and strengthening. Eccentric exercises also promote new collagen formation and healing[5,14,20,21]. More recent studies suggested treatment modalities include extracorporeal shock wave therapy, platelet-rich plasma, or growth factor injections. However, the effectiveness of these modalities still needs further investigation[5].

The authors feel that if no improvement is found within three months, it is preferable to manage these patients surgically rather than risk the tendon rupturing[12,14]. This study and others showed that prompt diagnosis is important and can lead to more appropriate treatment and negate the need for an EHL transfer[22-24]. The EHL transfer is not innocuous and can have potential complications, including weak hallux extension causing the hallux to catch while walking, and a progressive flexion contracture of the interphalangeal joint requiring a fusion.

Other surgical procedures that have been described for the management of TA tendinosis include decompression, decompressive medial cuneiform exostectomy, and gastrocnemius recession. Decompression of the tendon is limited to the early stages of the disease process before tendon disrepair occurs. De Cock et al.[25] reported excellent results with tendon decompression by releasing the oblique inferomedial band of the inferior extensor retinaculum for cases of distal TA tendinopathy. They consistently reported intraoperative compression and friction on the tendon due to the retinaculum. They highlighted the importance of diagnosing TA tendinosis as this degenerative tendon is at risk of rupturing if left untreated[25]. Gossett et al.[26] presented a case report of bilateral distal TA tendinosis treated successfully with proximal medial gastrocnemius recession only. The antagonizing force of gastrocnemius opposing TA dorsiflexion during the functional arc of ankle motion is decreased with gastrocnemius recession, minimizing tension on the TA tendon. They proposed that gastrocnemius equinus contracture is one of the main driving forces of distal TA tendinosis. As an isolated procedure, this could be limited to early in the disease process but may also be a good adjunct to a reparative procedure if a tight gastrocnemius is

present. None of our patients had a positive Silfverskiold test, but it is important to assess for gastrocnemius contractures when managing these patients. Liang et al. equated distal TA tendinosis to insertional Achilles tendinopathy in that a bone prominence may result in frictional damage of the tendon. They had good results with bone resection of the medial aspect of the medial cuneiform and tendon debridement[21].

In the management of peroneal tendinosis, if ≥ 50% of normal tendon remains after debridement, a direct repair of the tendon is performed. This 50% cut-off as a guide for when to perform augmentation of the diseased tendon dates back to 1924 from a publication by Meyer[27]. Wagner et al.[28] recently reported that peroneal tendons with ≥ 33% of healthy tendon are structurally strong enough for a primary repair. We, the authors, believe that EHL transfer for the management of TA tendinosis should be limited to severe cases. We, therefore, perform an EHL transfer when ≤ 30% of healthy tendon is present after debridement. When ≥ 30% of healthy tendon remained, we augmented the direct repair of the tendon by running the anchor suture up along the tendon until 2 cm of normal tendon is included proximally. Including the normal tendon proximally into the anchor augmentation acts as a checkrein, partially offloading the diseased portion of the tendon while it heals and regenerates.

Lemmens et al.[22] reported on the management of TA tendinosis in ten feet with debridement, direct repair, and anchor reattachment of the tendon onto the medial cuneiform after failed conservative treatment. Only one of the ten TA tendons had a longitudinal tear, therefore, all tendons were mostly intact. Beischer et al.[13] and Grundy et al.[14] debrided TA tendinosis, and if ≥ 50% of healthy tendon remained, repaired the tendon and reattached the tendon to the cuneiform with an anchor. None of these authors extended the suture proximally into the normal tendon, as was done in our series. If ≤ 50% of healthy tendon remained, they augmented it with an EHL transfer. In the Grundy et al.[14] case series, six patients were treated with EHL transfer and six with direct repair. Fifty percent of patients treated with the EHL transfer complained of a symptomatic hallux interphalangeal joint with extensor lag that caused catching the hallux when walking barefoot. One of the six cases treated with debridement, repair, and suture anchor reattachment presented with delayed spontaneous rupture three months postoperatively after they did an EHL transfer. This led the authors to recommend more aggressive reconstruction and use of the EHL augmentation on a routine basis. We believe that incorporating the suture anchor into the normal tendon reduces the risk of rupture[14].

In our small case series, good outcomes have been shown with debridement and augmentation of the TA tendon with a suture anchor incorporating the proximal portion of the healthy tendon. At the final follow-up, patients had a mean AOFAS of 97.6, VAS of 0.8, and SEFAS of 43.8. No complications were related to the surgery, and no additional surgery was performed. This small retrospective case series will hopefully stimulate larger randomized studies.

Conclusion

TA tendinosis is an uncommon degenerative process that the physician needs to pay attention to in older overweight patients complaining of dorsomedial midfoot pain. Early diagnosis and appropriate management can prevent tendon rupture. This small case series of debridement, surgical repair, and augmentation with a suture anchor showed high patient satisfaction and good clinical outcomes without needing a tendon transfer.

Authors' contributions: Each author contributed individually and significantly to the development of this article: JN *(https://orcid.org/0000-0003-1448-4272) and NPS *(https://orcid.org/0000-0002-5566-7588) Wrote the article, interpreted the results of the study, participated in the review process and approved the final version; PNFF *(https://orcid.org/0000-0003-4639-0326) Conceived and planned the activities that led to the paper, interpreted the results achieved and approved the final version. All authors read and approved the final manuscript.*ORCID (Open Researcher and Contributor ID).

References

1. Bruning F. Zwei seltene falle von subkutaner sehnenzerreissung. Munchen Med Wchschr. 1905;52:1928-9.
2. Harkin E, Pinzur M, Schiff A. Treatment of acute and chronic tibialis anterior tendon rupture and tendinopathy. Foot Ankle Clin. 2017;22(4):819-31.
3. Tickner A, Thorng S, Martin M, Marmolejo V. Management of isolated anterior tibial tendon rupture: A systematic review and meta-analysis. J Foot Ankle Surg. 2019;58(2):213-20.
4. Sammarco VJ, Sammarco GJ, Henning C, Chaim S. Surgical repair of acute and chronic tibialis anterior tendon ruptures. J Bone Joint Surg Am. 2009;91(2):325-32.
5. Coughlin MJ, Schon LC. Disorders of tendons. In: Coughlin MJ, Mann RA , Saltzman CL, editors. Mann's surgery of the foot and ankle. 9th ed. Philadelphia: Mosby Elsevier; 2013:1197-1210
6. Petersen W, Stein V, Bobka T. Structure of the human tibialis anterior tendon. J Anat. 2000;197 Pt 4(Pt 4):617-25.
7. Petersen W, Stein V, Tillmann B. Blood supply of the tibialis anterior tendon. Arch Orthop Trauma Surg. 1999;119(7-8): 371-5.
8. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies. Update and implications for clinical management. Sports Med. 1999;27(6):393-408.

9. Simpson MR, Howard TM. Tendinopathies of the foot and ankle. Am Fam Physician. 2009;80(10):1107-14.
10. Novacheck TF. The biomechanics of running. Gait Posture. 1998;7(1):77-95.
11. McMinn R. Ankle and foot joints, Lower Limb. In: Last RJ. Last's Anatomy, Regional and Applied. Edinburgh: Churchill Livingstone; 1990.
12. Cignetti C, Peng J, McGee A, Lehtonen E, Abyar E, Patel HA, et al. Tibialis anterior tendinosis: Clinical characterization and surgical treatment. Foot (Edinb). 2019 Jun;39:79-84.
13. Beischer AD, Beamond BM, Jowett AJ, O'Sullivan R. Distal tendinosis of the tibialis anterior tendon. Foot Ankle Int. 2009; 30(11):1053-9.
14. Grundy JR, O'Sullivan RM, Beischer AD. Operative management of distal tibialis anterior tendinopathy. Foot Ankle Int. 2010;31(3): 212-9.
15. Bernstein RM. Spontaneous rupture of the tibialis anterior tendon. Am J Orthop (Belle Mead NJ). 1995;24(4):354-6.
16. Kannus P, Józsa L. Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. J Bone Joint Surg Am. 1991;73(10):1507-25.
17. Levitsky MM, Freibott CE, Greisberg JK, Vosseller JT. Risk Factors for Anterior Tibial Tendon Pathology. Foot Ankle Int. 2021; 42(3):329-332.
18. Bass E. Tendinopathy: why the difference between tendinitis and tendinosis matters. Int J Ther Massage Bodywork. 2012;5(1):14-7.
19. Józsa L, Kannus P. Histopathological findings in spontaneous tendon ruptures. Scand J Med Sci Sports. 1997;7(2):113-8.
20. Levitsky MM, Vosseller JT, Popkin CA. Lace bite: A review of tibialis anterior tendinopathy in ice hockey players. Transl Sports Med. 2020;3(4):296-9.
21. Laing AJ, Carr C. Decompressive Medial Cuneiform Exostectomy for Resistant Tibialis Anterior Insertional Tendinopathy. J Foot Ankle Surg. 2018;57(3):531-6.
22. Lemmens L, van Beek N, Verfaillie S. Postoperative results of distal tibialis anterior tendinosis. Foot Ankle Surg. 2020;26(8):851-4.
23. Markarian GG, Kelikian AS, Brage M, Trainor T, Dias L. Anterior tibialis tendon ruptures: an outcome analysis of operative versus nonoperative treatment. Foot Ankle Int. 1998;19(12):792-802.
24. Ouzounian TJ, Anderson R. Anterior tibial tendon rupture. Foot Ankle Int. 1995;16(7):406-10.
25. De Cock L, Van Eynde E, Vandeputte G. Clinical results of distal anterior tibial tendon decompression. Foot Ankle Surg. 2021;27(7):827-31.
26. Gossett L, Gossett PC, Roberts J, Anderson J. Gastrocnemius recession for the treatment of tibialis anterior tendinopathy. Foot Ankle Orthop. 2019;4(3):2473011419852940.
27. Meyer AW. Further evidence of attrition in the human body. Am J Anat. 1924;34(1):241-67.
28. Wagner E, Wagner P, Solminihac DZ, Ortiz C, Diaz AK, Radkievich R, et al. Peroneal tendon tears: 50% rule, a myth? Biomechanical cadaveric evaluation. Foot Ankle Orthop. 2017; 2(9):24730114175S0000