



Case Report

Hypertrophy of the Peroneal Tubercle: A Rare Cause of Peroneal Tendon Synovitis

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Abstract

Hypertrophy of the peroneal tubercle is a rare cause of peroneal tendon synovitis. The peroneal tubercle of the calcaneal bone is an anatomical structure that has both structural and biomechanical functions. When abnormal in shape and size, it can cause friction or impingement of the peroneal tendons, resulting in tenosynovitis and/or tendon tears. Clinically, the patient presents with tenosynovitis and a prominent peroneal tubercle palpable on the lateral calcaneus. Radiographs and ultrasound are sufficient for diagnosis. Additional CT and MRI are often performed to better visualize the bony structures and soft tissues, respectively. Treatment consists of conservative therapy for at least 6 weeks. If conservative therapy fails, or if the tendon is torn, surgery may be required.

Introduction

We describe the case of a 65-year-old woman with peroneal tendon synovitis due to hypertrophy of the peroneal tubercle. The peroneal tubercle is a well-known anatomical structure on the lateral calcaneus. Abnormalities in its shape and/or size can result in tenosynovitis of the peroneal tendons. In this article, we discuss the anatomy and function of the peroneal tubercle. Next, we will highlight the pathogenesis of tubercle hypertrophy and how it can lead to peroneal tendon tenosynovitis. Finally, diagnosis and treatment management are discussed.

Case presentation

A 65-year-old woman with a relevant history of diabetes and diabetic polyneuropathy presented with spontaneous pain and swelling of the right lateral ankle. The pain was present on exertion, with significant pain at night. Clinical examination revealed an ataxic gait, part of the known polyneuropathy. The gait was antalgic on the right side. On inspection, we noted significant pedes cavovari and transversi, more pronounced on the right than the left side (Figure 1). There was visible swelling and pain on palpation of the distal fibula and lateral calcaneus. A bony mass was palpable in the mid lateral third of the calcaneus. Pain was provoked by passive ankle inversion and resisted eversion.

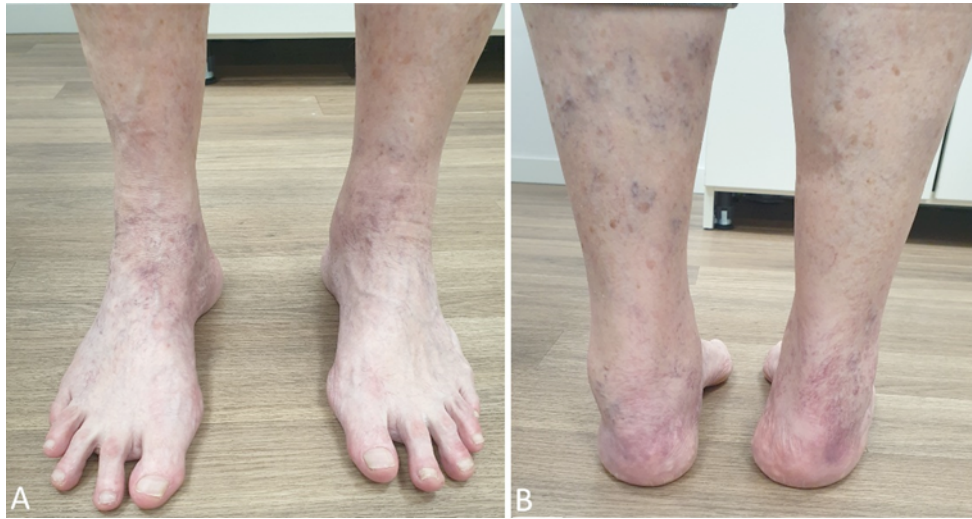


Figure 1: Pedes cavovari with lateral deviation of the subtalar joint axis, metatarsus adductus, hallux valgus and pes transversus. Varus deformity of the calcaneus induces traction on the lateral calcaneus and peroneal tendons.

Based on the clinical findings, tenosynovitis of the peroneus longus and/or brevis was suspected. Ultrasound showed enlarged tendon structures, synovial hyperproliferation and increased fluid in the tendon sheaths of the peroneus longus and brevis muscles with hypervascularisation and peritendinous subcutaneous oedema. These findings supported our clinical diagnosis. We also noted a prominent bony structure at the lateral calcaneus, in close contact with the peroneus longus and brevis tendons. Dynamic evaluation, obtained during ankle inversion and eversion movements, showed reduced tendon gliding over this bony structure (Figure 2).

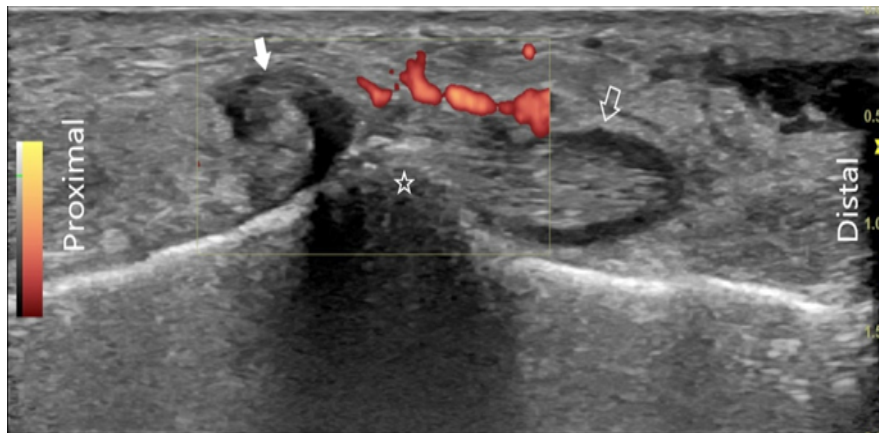


Figure 2: Ultrasound of the lateral calcaneus, longitudinal view. Tenosynovitis of peroneus longus (full arrow) and brevis (hollow arrow) tendons with hyperemia. Between the tendons a prominent osseous structure (star) is visualized.

A radiograph confirmed the presence of a prominent bony structure on the lateral aspect of the calcaneus which was identified as a hypertrophy of the peroneal tubercle (Figure 3).



Figure 3: Normal view (A), anteroposterior view (B) and Harris heel view (C) of the right foot show hypertrophy of the peroneal tubercle (hollow arrow).

The CT scan showed a hypertrophic peroneal tubercle with a hook-shaped appearance (Figure 4). Complementary MRI showed tenosynovitis of the peroneal tendons with bone marrow oedema in the peroneal tubercle (Figure 5).



Figure 4: Axial (A) and coronal (B) CT images of the right ankle show a hook-shaped hypertrophied peroneal tubercle (arrow).



Figure 5: Axial (A) and sagittal (B) MRI images of the right ankle. The peroneus brevis (hollow arrow) and longus (full arrow) tendons are clearly depicted in their course around the peroneal tubercle (star). On the axial images, we see a T2-hyperintense signal around the peroneal tendons indicative of tenosynovitis. The T2-hyperintense signal in the peroneal tubercle indicates bone marrow edema.

Treatment consisted of non-steroidal anti-inflammatory drugs (piroxicam, 20 mg daily) and immobilisation in a walking boot for 2 weeks, with no improvement. Following this, an ultrasound-guided corticosteroid injection into the peroneus longus muscle was performed, together with immobilisation in a walking boot. There was a significant reduction in pain, but symptoms recurred after 3 weeks. The patient was referred for surgical treatment with resection of the peroneal tubercle and synovectomy of the peroneal tendons. After surgery, the patient was kept in a non-weight-bearing short leg cast. At the end of 6 weeks, the cast was removed and the patient was started on a range of motion exercise program. Full weightbearing was encouraged as tolerated. Follow up at 3 months showed the patient to be pain free. To prevent recurrence, the static deformities of the feet were corrected with orthopedic inserts.

Discussion

Anatomy

Peroneus longus and brevis muscles

The peroneus longus and brevis muscles form the lateral compartment of the lower leg. The peroneus longus muscle originates from the lateral condyle of the tibia and from the head of the fibula. The peroneus brevis muscle originates from the middle third of the fibula. The peroneus longus is the most superficial of the muscles that run together along the lateral tibia. The musculotendinous junction of both muscles is located at the distal fibula, proximal to the superior peroneal retinaculum. The latter is a fibrous band that lies between the posterolateral aspect of the fibula and the calcaneus. It prevents subluxation of the peroneal

tendons. In their proximal course, the peroneal tendons share a common tendon sheath. It splits into separate tendon sheaths at the level of the lateral calcaneus, where the peroneal tubercle forms the physical separation. The peroneus brevis continues along the os cuboideum and attaches at the level of the tuberosity of the fifth metatarsal. The peroneus longus extends into the cuboidal groove where it makes a sharp medial turn to attach at the level of the plantar side of the base of the first metatarsal and the medial os cuneiforme. Eversion of the hindfoot and (to a lesser extent) plantar flexion of the ankle are the functions of the peroneus muscles. The peroneus longus muscle also acts as a plantar flexor of the first metatarsal bone [1].

Peroneal tubercle

The peroneal tubercle is an anatomical structure located in the middle third of the lateral calcaneus. It was first described by the Austrian anatomist Josef Hyrtl in 1860 and is present in 90% of heels [2]. The tubercle has three functions: (a) It forms a physical separation between the peroneus longus and brevis tendon sheaths, just distal to the junction of the common tendon sheath. (b) It forms the insertion of the septum of the inferior peroneal retinaculum, which is important for the correct course of the tendons over the lateral calcaneus. (c) From biomechanical point of view, the tubercle enlarges the moment arm of the peroneus muscles and facilitates ankle eversion [3]. The peroneal tubercle is thus a normal anatomical structure with a structural and biomechanical function. In rare cases, variations in size and shape have been described, which may lead to pathology.

Pathogenesis

Abnormalities in the shape and size of the tuberosity can cause mechanical friction on the peroneal tendons, resulting in tendinopathy and/or tenosynovitis. In more severe cases, tendon tears may occur. The friction is usually due to hook and tunnel formation of the hypertrophic tubercle. A case of impingement of the peroneal brevis tendon between the fibula and a hypertrophic tubercle has also been described [4]. The question arises as to how anatomical abnormalities of the tuberosity occur. These can be congenital or acquired. Associations have been described in the literature with static deformities, paralytic feet, calcaneal fractures and the presence of a peroneus quartus [5]. The exact pathogenesis is unknown. One postulated hypothesis is that an increased varus position of the calcaneus reduces the moment arm of the peroneal muscle and thus creates a biomechanical disadvantage. This leads to increased frictional forces at the level of the lateral calcaneus, resulting in reactive hypertrophy of the peroneal tubercle [1].

Diagnosis

Clinically, the patient presents with tenosynovitis of the peroneus longus and/or brevis muscle, characterized by pain and swelling of the lateral ankle. The triad of tendinopathy (painful

palpation, stretching and resistance testing) is often positive. The peroneal tubercle can be felt at the lateral calcaneus, with palpatory pain. Foot statics should always be checked because of the association with static deformity described above. Other causes of tenosynovitis of the peroneal tendons should also be considered. More common causes include lateral ankle instability, hindfoot varus and symptomatic os peroneum. Peroneal tubercle hypertrophy can be visualized by radiography. The most sensitive image is the Harris heel view, in which the x-rays travel from posterior at a 45° angle to horizontal to the calcaneus (Figure 3C). Ultrasound can diagnose tenosynovitis, which is characterized by increased fluid content within the tendon sheath, thickening of the synovial sheath with or without increased vascularity, and peritendinous subcutaneous oedema [6]. The peroneal tubercle can also be visualized and its relationship to the peroneal tendons can be dynamically assessed (Figure 2). Radiographs and ultrasound are usually sufficient for diagnosis. Additional CT and/or MRI scans are often performed, especially if surgical treatment is being considered. CT provides a more detailed bony view of the exact shape and course of the peroneal tubercle (Figure 4). MRI can be used for a more detailed assessment of the tendon structures. Tendinopathy or tenosynovitis, with or without rupture, can be diagnosed (Figure 5). Bone marrow oedema is often seen in the peroneal tubercle, caused by friction of the peroneal tendons [6-8].

Treatment

Conservative treatment consists of rest and immobilization for at least 6 weeks, supplemented by non-steroidal anti-inflammatory drugs or local corticosteroid injections. Orthopedic inserts or footwear can correct static deformities [2]. Surgery is indicated if symptoms persist after conservative treatment or if the tendon ruptures. It consists of resection of the peroneal tubercle, possibly with tendon synovectomy, and suturing of torn tendons. Postoperative management consists of a non-weight-bearing cast for 6 weeks, after which mobilization and progressive weight-bearing can be started as tolerated [3-4,5]. Case reports show good results and rapid return to activity after surgical treatment [3,4]. One case of recurrence after 7 months has been reported in the literature. Incomplete resection was described as the most likely cause. A second resection was successfully performed with no recurrence at 13-month follow-up [9]. The patient was treated with indomethacin postoperatively. The idea was to prevent recurrence, as indomethacin has been shown to prevent heterotopic ossification [10]. Surgical biomechanical correction should be considered for severe static deformities.

Conclusion

The calcaneal peroneal tubercle is an anatomical structure with structural and biomechanical functions. In rare abnormalities of shape and size, this structure can cause friction or impingement

of the peroneal tendons. This can lead to tenosynovitis and/or tendon rupture. The patient presents clinically with tenosynovitis and a prominent peroneal tubercle palpable at the level of the lateral calcaneus. Radiographs and ultrasound are usually sufficient for diagnosis. Additional CT and/or MRI scans are often performed, especially in the case of surgery. Treatment consists of conservative therapy for at least 6 weeks. Surgery is indicated if conservative therapy fails or if the tendon is torn.

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