INTRODUCTION

Tenosynovitis of the peroneal tendons is a challenging entity to diagnose and treat. The presence of this disorder is not uncommon and it is frequently misdiagnosed as an ankle sprain. It can sometimes present subtlety but create chronic nagging pain. Other times it can present with significant destruction of the tendon and then become a true challenge to surgically repair. Unfortunately, even when surgically repaired, the peroneal tendons can be a source of chronic pain and disability depending on the degree of scar tissue and adhesions which can develop postoperatively. In this paper the clinical entity of peroneal tenosynovitis will be reviewed and the author’s experience will be presented with clinical illustrations.

HISTORICAL PERSPECTIVE

Tenosynovitis was first described by De Quervain in 1895 in a paper describing stenosing tenosynovitis of the wrist. In 1907, Hildebrand first described tenosynovitis of the lower extremity. He described stenosing tenosynovitis of the peroneal tendons. Twenty years later, Hackenbroch presented case studies of two patients on whom he performed surgical procedures to remove the constriction of the peroneal tendons.

PERONEAL TENDON ANATOMY

A thorough understanding of the anatomy of the lateral compartment of the leg and the distal insertion of the peroneal tendons is necessary for both conservative and surgical treatment. The peroneal retinaculum is the retaining ligamentous structure for the posterolateral aspect of the ankle and foot and can be directly related to peroneal tendon pathology. It is composed of a superior and inferior component. The superior peroneal retinaculum covers the peroneal tendons at the level of the ankle joint and attaches posteriorly to the posterolateral aspect of the calcaneus and the tendon Achilles fascial structures and anteriorly to the lateral aspect of the peroneal groove. The inferior peroneal retinaculum is continuous with the lateral aspect of the inferior extensor retinaculum. Inferiorly and laterally, the structure covers the peroneus longus tendon, attaches to the peroneal groove and then extends over the peroneus brevis tendon to make attachments to the lateral rim of the sinus tarsi.

The peroneus longus muscle arises from the head of the fibula and the upper one-half to two-thirds of the lateral surface of the shaft of the fibula. In addition, fibers also originate from the anterior and posterior inter-muscular septa, the overlying fascia cruris and from the lateral condyle of the tibia. Sarrafian describes the peroneus longus tendon as being retained by three tunnels and making three turns before reaching its destination. The first tunnel common to both peroneal tendons is retro-malleolar and is formed by the superior peroneal
At the tip of the fibular malleolus, the tendon makes its first turn and is directed distally and anteriorly. It enters the inferior, or second tunnel, formed by the inferior peroneal retinaculum. At the level of the peroneal trochlea of the os calcis, the tendon makes its second turn. It makes its third turn around the lateral border of the foot between the cuboid and the base of the fifth metatarsal and enters the plantar, or third tunnel.

The peroneus brevis muscle arises anterior and deep to the peroneus longus, from the inferior two-thirds of the lateral surface of the fibula and the intermuscular septum. According to Sarrafian, just below the tip of the fibula, the tendon is directed downward, anteriorly and slightly laterally as it passes on the superior aspect of the peroneal tubercle. The peroneus brevis finally inserts onto the base of the fifth metatarsal.

Understanding the anatomic complexity of the peroneal tendon sheaths is key for diagnosis and treatment of peroneal tenosynovitis. As the peroneal muscles extend distally as end tendons, they are located in one common sheath beginning approximately 4 cm above the lateral malleolus. As the tendons continue distally, the tendon sheath bifurcates at the level of the peroneal tubercle of the calcaneus, which anatomically serves to separate the two tendons. The peroneus longus sheath terminates at the level of the groove of the cuboid; however, a second synovial sheath extends from the groove of the cuboid to the base of the first metatarsal.

**CLASSIFICATION OF PERONEAL INJURY**

Gilula et al. classify tenosynovitis into types I-IV based on tenographic findings. The classification is as follows:

- **Type I** - normal tendon.
- **Type II** - involves minimal to marginal irregularities.
- **Type III** - definite synovitis, characterized by moderate to marked marginal irregularity. (Fig. 1)
- **Type IV** - occlusion of the tendon sheath, indicating rupture or stenosing tenosynovitis.

This classification scheme demonstrates that tenogram findings may be correlated with therapeutic guidelines, and thus with prognosis. Their conclusion is that all patients respond to conservative treatment except Type IV.

A second classification system of tendon injuries about the foot is based upon definition. It includes peritendinitis, tendinosis, chronic tenosynovitis, stenosing tenosynovitis, and hemorrhagic tenosynovitis. The definition of each pathologic entity is beyond the scope of this paper.

**ETIOLOGY OF PERONEAL TENOSYNOVITIS**

A number of mechanisms have been proposed to explain the etiology of peroneal tenosynovitis. Traumatic and biomechanical etiologies are the most common. Traumatic etiologies of peroneal tenosynovitis usually involve inversion ankle sprains. The superior peroneal retinaculum is ruptured and the peroneal tendons are allowed to sublux out of the fibular peroneal groove. The peroneal retinaculum can heal in a lax position and result in chronic subluxation creating eventual damage of the tendons. In the literature this is a leading cause of peroneal tenosynovitis. Often patients are seen in the emergency room after an inversion injury and diagnosed with an “ankle sprain.” Chronic pain following this injury may be due to a misdiagnosis. The patients usually complain of fullness in the lateral aspect of the ankle since the injury.

Peroneal tenosynovitis has been related to abnormal foot mechanics. Excessive subtalar joint pronation during gait allows for midtarsal joint pronation, thereby unlocking the cuboid. This decreases the functional advantage of the peroneus longus tendon and creates increased strain upon the tendon at the level of the peroneal groove. A second biomechanical disadvantage is the pes cavus foot type. The pes cavus foot type is more likely to sustain repetitive lateral ankle sprains, thus compromising the peroneal tendons.

Systemic diseases such as gout and rheumatoid arthritis, as well as infectious diseases such as tuberculosis and gonorrhea have all been implicated in the etiology of tenosynovitis. The use of ciprofloxacin and concomitant tenosynovitis has also been described. The authors have even seen stenosing peroneal tenosynovitis following the use of an improperly designed new pair of custom molded functional orthoses.
The most common anatomic factor as a cause of peroneal tenosynovitis is described as a congenital or acquired hypertrophy of the peroneal tubercle creating a stenotic fibro-osseous tunnel. The hypertrophied tubercle may also lead to longitudinal tears along the peroneal tendons. Less frequently cited anatomic etiologies include inflammatory arthritis, presence of a peroneus quartus muscle, or a low-lying peroneus brevis muscle belly.

Another etiology of peroneal tenosynovitis is entrapment of the peroneal tendon sheaths following a calcaneal fracture with a lateral wall blowout. The tendon sheaths and tendons become adhered to the calcaneal wall. This can be the source of chronic pain and is very difficult to treat both conservatively and surgically.

**CLINICAL PRESENTATION**

According to Thompson et al., there appears to be a bimodal incidence of patients that suffer from disorders of the peroneal tendons. The first group includes late middle-aged and elderly patients with attritional ruptures of the peroneus longus around the level of the cuboid tunnel. This rupture has been associated with an os peroneum. The second group includes young athletes and “weekend warriors” with recurrent plantigrade inversion injuries. The occurrence of ruptures and tenosynovitis is especially high in feet with cavus or high-normal medial longitudinal arches.

Proper history taking may reveal the cause of the patient’s discomfort. Often, the patient will describe pain after performing a repetitive motion over and over again, such as after playing tennis. The presenting symptoms of tenosynovitis usually include swelling, tenderness, grating, and crepitus just posterior to the lateral malleolus, which may extend distally to the base of the fifth metatarsal. The patient will often indicate chronic pain, which increases with exertion, along the course of the peroneal longus tendon.

Physical examination of the lateral foot and ankle may reveal palpable fusiform swelling along the course of the peroneal tendons distally to the base of the fifth metatarsal. Manual muscle testing will reveal weakness of the myotendinous unit, and pain will be elicited with passive inversion and plantarflexion or active eversion and dorsiflexion of the foot. Rarely, peroneal tenosynovitis may present as a pseudotumor in the form of a soft-tissue mass along the lateral aspect of the foot secondary to cystic and fibrotic degeneration of the tendon sheath.

**Diagnosis**

Diagnosis of peroneal tenosynovitis can be made by physical examination by the trained practitioner. However, multiple imaging studies exist to aid in making or confirming a diagnosis. Plain radiographs are important to rule out ankle fractures or small avulsion fractures from the fibula. A medial oblique view of the foot may demonstrate an os peroneum at the peroneal groove of the cuboid. Additionally, a calcaneal axial view is important to identify the presence of a hypertrophied peroneal tubercle. Stress tests of the ankle including inversion and anterior drawer can be used to help rule out ankle instability.

Magnetic resonance imaging of the affected lower extremity may be used to demonstrate tendon pathology. Tendons have a low signal intensity at all sequences due to low water content. T1-weighted images provide contrast between the dark signal of tendons and the bright signal of surrounding fat. T2-weighted images depict the increased signal associated with pathologic processes such as tendinosis and tenosynovitis with high signal intensity. Tendon ruptures are demonstrated by the absence of a portion of the tendon, whereas partial or linear tears are represented by areas of high signal intensity in the tendon substance on T1-weighted images and by fluid and edema in the tendon sheath on T2-weighted images. Tenosynovitis is demonstrated by a high signal intensity on T2-weighted images due to the fluid contained within the peroneal tendon sheath. (Fig. 2)
Peroneal tenography allows for anatomic definition of the peroneal tendon sheaths. It allows for visualization of normal anatomy, stenosis or entrapment of the peroneal tendons. Pre-procedural AP and oblique ankle radiographs are taken for comparison. The patient is then aligned with the leg internally rotated on the radiographic table. Following local anesthesia under sterile conditions, a #21 butterfly needle is then inserted into the sheaths of the peroneal tendons approximately 2 to 6 centimeters proximal to the lateral malleolus, following the course of the tendons. The needle may either be inserted until it strikes fibula and then pulled slightly back into the sheath, or the needle may be inserted until the tendons are palpated. Next, approximately 1cc of radio-opaque dye such as isovue is infiltrated into the tendon sheaths. The dye is infiltrated under fluoroscopic control. If the dye runs proximally up the tendon sheaths toward the muscle bellies, or extravasates into the surrounding tissue, then most likely the needle is not within the peroneal tendon sheaths. One should visualize the dye extending distally outlining the peroneus longus and brevis tendons. Once it is determined that the dye is running along the course of the peroneal tendons an additional 3cc’s of dye is infiltrated to obtain a better image. The foot and ankle are then put through a range of motion. Multiple fluoroscopic films are taken as the dye extends distally. A stenosis in the sheaths is identified by a partial or complete obstruction of the flow of the contrast medium distal to the site of primary pathology. If stenosis is seen, injection of 1-2cc of corticosteroid into the tendon sheaths may aid in diagnosis as well as treatment of the pain. The injection of corticosteroid following accurate diagnosis with a peroneal tenogram insures the accurate placement of the steroid within the tendon sheath (Fig. 3).

Mizel et al., have suggested a method to aid in diagnosis of peroneal tendon injury utilizing fluoroscopy. They inject contrast dye with bupivacaine under fluoroscopic control. They state that the radio-opaque material is not used as a diagnostic tool; it is used to insure the accurate and specific placement of the anaesthetic agent.

Computed Tomography (CT) aids in the identification of a hypertrophied peroneal tubercle or any other bony abnormality. Although CT is mainly used for osseous pathology, Rosenberg et al., have described its usefulness in identifying peroneal tendon injuries. The soft tissue resolution of computed tomography allows direct visualization of tendons and enables tendons to be differentiated from adjacent soft tissue and osseous structures. According to Rosenberg et al., with peroneal tenosynovitis, the peritendinous fat planes are obliterated, with the tendons almost completely engulfed in a soft tissue mass.

Figure 3A. Clinical picture of a peroneal tenogram. The butterfly needle has been placed approximately 2cm proximal to the ankle joint.

Figure 3B. Tenogram reveals stenosing tenosynovitis of the peroneus brevis.
Ultrasonography is useful for evaluating superficial muscle masses. It has limited use in the lower extremity, however, it can be used to define tendon pathology at the level of the ankle. Tendonitis is manifested as thickening of the tendon, with hypoechoic and nodular inhomogeneous regions; rare calcifications may be seen in chronic tendonitis. Complete ruptures may also be visualized by a lack of signal or discontinuities within the tendon proper. Although it is non-invasive and relatively simple, ultrasonography has very little role in the diagnosis and treatment of peroneal tendon pathology.

Treatment

Treatment of peroneal tenosynovitis usually begins with conservative modalities. Rest, ice, nonsteroidal anti-inflammatories, corticosteroid injections or immobilization are attempted at the acute phase, or upon initial presentation of chronic tenosynovitis. Physical therapy may also be of assistance by stretching and strengthening the peroneals. Other authors have advocated the use of a lateral heel wedge or AFO to decrease the load of the peroneals during gait. Acute peroneal tenosynovitis usually responds within 4 to 6 weeks of conservative therapy.

Continuation of conservative therapy in patients with partial or complete stenosis of the tendon sheath usually proves futile. Peroneal tenography may be employed at this point as both diagnostic and therapeutic. It can be used to identify the presence and location of any stenosis. Additionally, the increased force of the added dye may help release the constrictor. The addition of a corticosteroid into the tendon sheath may also help to decrease the inflammatory response around the stenosis and to limit the degree of the tenosynovitis.

Surgical intervention is aimed at excising all synovitis, releasing any constrictions, repairing any partial tears in the tendons, and removing any existing bony pathology. A curvilinear incision is placed overlying the peroneal tendons from the tip of the fibula distally to just proximal to the base of the fifth metatarsal. The incision is carried down to the deep fascia while avoiding the sural nerve. Often there is effusion of the peroneal tendon sheath especially in the area of the peroneal tubercle. The peroneal tendon sheath is then incised, with care being taken to avoid laceration of the peroneal tendons. The synovial fluid bathing the tendons may be clear to yellowish in nature, or it may be hemorrhagic due to a tear in one of the tendons. Several authors advocate taking cultures of the fluid to rule out bacterial or fungal causes of the tenosynovitis.

The inner lining of the synovial sheath is then inspected and any synovitis is excised. The tendons are inspected for any partial or complete tears. If tears are present, the necrotic ends are debrided and the tears are repaired with absorbable or non-absorbable suture. If a hypertrophied peroneal tubercle is present, the excess bone may be excised at this time. Care must be taken to reapproximate tissue between the peroneus longus and brevis tendons at the level of the peroneal tubercle. If the hypertrophied tubercle has been excised, a soft tissue anchor between the tendons re-enveloping them in separate synovial sheaths may be used. The wound is then closed in anatomic layers. The postoperative course consists of 3 weeks of non-weightbearing followed by physical therapy and strength training.

CASE HISTORY

A 60-year-old female slipped on spilled water in the grocery store and suffered an inversion ankle injury. The injury was diagnosed as an ankle sprain in the emergency room and she was treated with rest, elevation and compression with an ace wrap. She was treated by a local physician for several months with immobilization, physical therapy, and NSAIDs but developed chronic lateral ankle and leg pain. She was referred to author A.P., at which time an MRI was ordered revealing synovitis and tears of both peroneal tendons. The patient was eventually brought to the operating room for excision of the synovitis and repair of the tendon 18 months following the original injury.

An incision was made beginning at the posterolateral aspect of the ankle joint and coursed along the peroneal tendons to the base of the fifth metatarsal. Dissection was carried down to the peroneal retinaculum, where the deep fascia layer was incised and serosanguinous fluid exuded, exposing the underlying synovitis. (Fig. 4A) Full exposure of the tendons was then obtained and the acute synovitis was debrided and removed from the wound. Severe damage was noted along the peroneal brevis tendon as it coursed along the peroneal groove to the superior aspect of the peroneal trochlea.
damage was carefully examined and the frayed irreparable portions of the tendon were debrided. (Fig. 4B) The larger portions were repaired with a 5-0 absorbable suture utilizing a running buried stitch. A relatively small tear of the peroneus longus tendon was noted at the level of the peroneal groove. The peroneal retinaculum which was lax due to the original injury was then augmented and tightened via interrupted "pants-over-vest" suture technique. (Fig. 4C)

The wound was then closed and the patient immobilized in a cast for three weeks. She was then placed in a walking cast and began passive and active range of motion of the foot and the ankle for another three weeks. This was followed using an ankle air brace for 3 months and a gradual return to shoe gear. Her symptoms had dramatically reduced at three months postoperative. She did have a recurrence of symptoms at one year postoperative and was diagnosed with adhesions within the tendon sheath. She was treated with tenography and injection of 1cc of celestone. She is now four years postoperative and asymptomatic.

CONCLUSION

The clinical entity of peroneal tenosynovitis has been presented. The etiology, clinical evaluation and treatment options have been reviewed. One case was presented involving a typical peroneal tenosynovitis secondary to trauma. The authors recommend a suspicion of peroneal tenosynovitis when evaluating a patient for chronic ankle and leg pain.

REFERENCES