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Review Article

Persistent pain after lateral ankle sprain: A Diagnostic and treatment Dilemma A Review Article

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Abstract

A number of patients continue to experience prolonged pain, swelling, instability and disability after a lateral ankle sprain which is sufficient to limit the activity. There are several conditions which are misdiagnosed as chronic lateral ligament injury. In this review we highlight the common conditions that can mimic chronic lateral ligament injury and can cause persistent ankle pain after lateral ankle sprain. An outline to diagnose and treat these pathologies is also discussed, to resolve the diagnostic and treatment dilemma in most of the patients, but this problem still remains unresolved in a few patients.

Introduction

In an orthopedic or foot and ankle clinic, it is not uncommon to see a patient with persistent ankle pain following an ankle sprain. Most of the patients often remember their affected ankle being forcefully inverted and adducted during the injury leading to lateral ankle sprain. Lateral ankle sprain accounts for 80% of ankle sprains [1,2] and most of injuries are due to sports [3], but in some patients this injury may be due to falls, slips, or tripping during the routine daily activities [4].

Lateral Ligament Injury

Acute lateral ligament injury of the ankle vary in its presentation with respect to the amount of pain, swelling, haematoma discolouration, range of motion limitation and loss of function. Although tenderness may have low specificity in lateral ligament injury, a combination of tenderness, hematoma discoloration, and anterior drawer test in the subacute phase (5 days after injury) has demonstrated a sensitivity of 96% and a specificity of 84% [5].

In most of these patients there is rapid decrease in pain and improvement in function after two weeks of the injury. However, 5 to 33% of patients continue to have pain at 1 year or longer follow-up with 5 to 25% still experiencing pain after 3years [6]. Residual problems included pain (30%), instability (20%), crepitus (18%), weakness(17%), stiffness (15%), and swelling (14%) [7]. A severe grade of injury is more likely to produce persistent symptoms, which has been well demonstrated by using an expanded classification [8]. Symptoms often persist, when athletes return to sports without consulting a medical care provider [9]. Apart from the severity of the disease and treatment neglect the other factors like varus heel at heel strike of gait, increased talar curvature, anterior positioning of talus, achillis tendon tightness, and generalised and lateral ligament laxity can contribute to the persistence of symptoms [10-17]. Lateral ligament healing after an injury occurs in three different phases (1) inflammatory phase (until 10 days after trauma), (2) the proliferation phase (4^{th}-8^{th} week) and (3) the remodelling or maturation phase (until 1 year after trauma). The duration of the different phases may individually vary [18].

After inflammatory phase most of the swelling subsides, but there is no significant improvement in ROM deficit [19]. Decreased ankle dorsiflexion leads to abnormal gait pattern due to decreases in step and single leg support time [20]. In addition, proprioceptive

function might be deficient because mechanoreceptors are damaged by lateral ankle sprain. In addition to decreased range of movement proprioceptive [21] and postural control [22] deficits have been found in acute ankle sprains. A lateral ankle sprain may result in injuries to the lateral musculotendinous structures, peroneus longus and brevis, resulting in tendon tearing, intra-muscular strain, or tendon subluxation [23]. The tibialis anterior and extensor digitorum longus and brevis, thought to eccentrically control ankle plantar flexion may be affected. Both peripheral and central reactions of a muscle response are likely too slow to protect against a sudden inversion force [24]. While the ligament is healing after lateral ankle sprain, the ligamentous collagen sequence change leading to the looseness of the ligament [25].

The treatment of the acute phase is implemented with an aim to reduce swelling, improve range of movements, and allowing weight bearing as tolerated. The use of external supports like lace-up braces or semi-rigid braces with functional weight bearing significantly reduce time to work and sport and decrease the incidence of subjective instability [26]. Once the acute phase is over the treatment is directed towards progressive loading and sensory motor training to improve mobility strength, coordination and postural control. An inappropriate treatment, or no treatment, and more severe injuries are likely to cause persistent pain. These patients are likely to have recurrent sprains which cause persistent symptoms. 56% to 74% patients are reported to have recurrent sprains [27-30]. Patients with recurrent sprains present with pain and crepitus in the ankle and with four or more recurrences have an unstable feeling in the ankle [27].

With repeated recurrences patients develop chronic ankle instability, present with repeated episodes of an unstable feeling of giving away. The chronic ankle instability may be mechanical which is caused by ligament laxity or functional which is caused by proprioceptive deficits, neuromuscular deficits, postural control deficits and muscle weakness [31]. Most often both mechanical and functional instabilities co-exist. Mechanical instability leads to increased talocrural anterior translation and internal rotation as compared to the normal side [32-36]. Chronic lateral ankle sprains are often treated as injuries of the lateral ligament of the ankle, without giving due consideration to lateral sub-talar ligaments. Combined ankle and subtalar instability have been well defined and can lead to severe disability [37]. Stress radiography of the ankle including an anterior drawer and talar tilt are useful for determining both subtalar and ankle instability [38,39]. Magnetic resonance imaging can be used to demonstrate the injured ligaments [40]. Mechanical instability of the talocrural and subtalar joints was found in 24% to 68% and 58%and 58% respectively [41-45].

Proprioceptive deficits, neuromuscular deficits, postural control deficits and muscle weakness lead to functional instability. The alteration of three distinct components: joint position sense, kinesthesia, and force sense has been reported to be responsible for proprioceptive deficits. In a meta-analysis, the patients with chronic ankle instability displayed deficits in joint position sense when compared with patients without chronic ankle instability [46]. Garn and Newton demonstrated a significant decrease in kinesthesia in the injured side when compared with the normal side [47]. As far as force sense is concerned, different studies have shown correlation between error in evertor torque and chronic ankle instability [48-50]. A significant delay in the reaction time of peroneus muscles point towards the association of neuromuscular deficits with chronic ankle instability [51]. A significant decrease in static postural control have been suggested in chronic instability [52,53].

Ankle instability is primarily a clinical diagnosis and stress radiography might be more appropriate as an adjunctive test for patients for whom the history and physical examination findings are equivocal. MRI has high sensitivity but low specificity in the evaluation of clinical ankle instability [54]. While MRI has value as a screening tool for concomitant ankle pathology, it should not be considered diagnostic in terms of lateral ankle instability [55].

A course of physical therapy is indicated as the first line of treatment to treat chronic lateral ligament instability, whether functional or mechanical. When conservative management is not successful, surgical intervention can be employed successfully with high, predictable rates [56]. The lateral ligament repair is widely accepted as the primary operative treatment for chronic lateral ankle instability. Both arthroscopic and non-arthroscopic techniques used for repairing have yielded good results. Arthroscopy is usually done at the time of lateral ligament repair to rule out any intra- articular pathology, however in the patient without pain and negative MRI arthroscopy may not be necessary [57]. The traditional Brostrom and modified Brostrom-Gould repair are two historically reliable procedures known to provide sufficient biomechanical stability [58-62]. Various techniques like trans-osseous sutures, single and double suture anchors and direct suture to fibular periosteum have been found effective in reattachment of the lateral ligament [63,64]. Recently arthroscopic repair is becoming popular and reattachment of ligament and capsule performed with one or two anchors have shown excellent results [65-68]. These procedures are less invasive and allow faster return to normal and sports activities. In a recent systematic review comparing open and arthroscopic lateral ligament repair for chronic ankle stability both procedures were found to be equally reproducible and safe [69]. Whether the repair is done arthroscopically or by open method, arthroscopy at the time of repair is usually done to rule out associated intra-articular lesions often found in 63-95% of the cases [70-74]. In order to accelerate the recovery process and provide extra stability particularly in high level athletes augmentation of the repair by transferring of half of the peroneus brevis to the fibula [75], or an internal brace has been recommended [76].

In certain subgroups such as heavier athletes, revision cases and patients with ligamentous laxity, hindfoot varus, or inadequate residual ligament for direct repair, ligament repair is not possible or

inefficient. Historically several non-anatomic techniques requiring sacrifice of local tissues or peroneal tendons, like Evans [77], the Chrisman-Snook [78], the Watson-Jones [79] and the Castaing procedure [80] have been used in these situations. Nonanatomic procedures have largely been abandoned because of excessive peroneal weakness, stiffness of the subtalar joint and ankle degeneration [71,81]. Recently anatomic ligament reconstruction using free autograft or allograft tendons has become popular in these circumstances. This reconstruction re-creates the anatomic orientation of anterior talofibular ligament and calcaneofibular ligament and provides the required ankle stability [82-85].

Other Ligamentous Injuries

Chronic lateral ankle sprains are often treated as injuries of the lateral ligament of the ankle, without giving due consideration to the other ligaments around ankle like lateral sub-talar ligaments, syndesmotic ligaments and deltoid ligament. Combined lateral ankle ligament and syndesmotic Injuries do occur when an external rotation injury accompanies an ankle inversion, although external rotation remains the most common mechanism associated with syndesmotic injury [86]. It has been reported that a high-grade injury to the syndesmosis is typically associated with minimally traumatized lateral ankle ligaments, where as a lowgrade injury may be associated with concurrent low- and high-grade lateral ligament sprains [87]. In a cross-sectional MRI study 20.3% athletes referred for MRI after suffering an acute ankle sprain had evidence of syndesmotic injury regardless of lateral ligament involvement [88]. In the presence of supra-malleolar edema and pain with passive dorsiflexion and external rotation of the ankle syndesmotic injury should be suspected. A chronic, symptomatic and an unstable syndesmosis on stress tests and/or arthroscopy needs syndesmotic reduction and fixation, by screws, suture buttons, or a combination of the two [86]. In the presence of frank diastasis open debridement and reconstruction using fixation or reconstruction of ligaments using hamstring autografts or allografts, or peroneus longus had been suggested [89-91]. In neglected cases with severe incongruity, or a recurrence of diastasis after removal of fixation arthrodesis of the distal tibiofibular joint remains another option, however ankle arthritis and pain following arthrodesis remains a genuine concern [92-94].

Because of the internal talar rotation related with lateral ankle instability, many athletes with significant lateral ligament injury have been found to have deltoid ligament abnormality on arthroscopy and MR imaging [95,96]. In patients with combination of chronic ankle instability and medial and lateral ankle pain 40% of patients are found to have deltoid injury [97]. Tibiocalcaneal component of the deltoid complex is most commonly involved, but in some patients additional avulsion of the deep anterior tibiotalar component is reported [97]. Ankle repair of both sides of the ankle is required in these cases to provide adequate stability.

Impingement Syndromes

Following an inversion injury of the ankle a combination of lateral ligament injury and anterior inferior tibiofibular ligament may lead to inflammation in proximity of the healing ligaments and subsequent synovitis and scar tissue formation [98]. The entrapment of this hypertrophic soft tissue or torn and inflamed ligaments in the lateral gutter and anterolateral ankle joint produces ankle pain due to anterolateral ankle impingement [99]. Anterolateral impingement is suspected in the presence of tenderness at the joint line lateral to peroneus tertius [100] and can be further assessed by MR imaging which can detect the presence of anterolateral soft tissue pathology as well as rule out associated pathologies like osteochondral lesions, loose bodies and stress fractures [101-103]. Currently open resection for anterolateral impingement has been replaced by arthroscopic resection due to complications as low as 3.5% with arthroscopy [104,105].

Peroneal Tendon Injuries

Following an excessive ankle inversion apart from injuries to lateral ligament, subtalar, medial and syndesmotic structures, injuries to peroneal tendons can occur. A variety of peroneal tendon pathologies like tenosynovitis, fraying, longitudinal fissuring, partial and full-thickness tears, ruptures, and peroneal tendons subluxations and dislocations have been reported [106]. Persistent posterolateral or posterior hindfoot pain, swelling, tenderness and thickening along the peroneal tendons should prompt the surgeon to consider peroneal tendon pathology as a possible contributor to pain [107]. Pain with resisted eversion, passive inversion stretches and resisted plantar flexion of first metatarsal is present on clinical examination [108]. In the presence of peroneal tendon instability patients report a snapping and popping or giving way in the ankle and subluxation can be recreated via forced dorsiflexionor resisted plantar flexion and inversion [109]. Longitudinal and acute tears of the peroneal tendons require debridement, tubularization, or end to end repair [107]. When one of the peroneal tendons is irreparable the surgical options include tenodesis to the adjacent functional tendon or bridging the defect using allografts or autografts, and the salvage of both irreparable tendons may require allografts or autografts, or tendon transfer [110]. A variety of surgical methods available for peroneal tendon subluxation or dislocation include enhanced repair of the superior peroneal retinaculum, transposition of soft tissues, tendon rearrangement, bone blocking and groove deepening [111-115].

Osteochondral Lesions

Osteochondral lesion of the ankle is another common injury following an ankle sprain, caused by rotation of talus in the ankle mortise during inversion injury [116]. An ankle positioned in inversion and dorsiflexion predisposes for an osteochondral lesion on the lateral side, while medial lesions occur mostly with the ankle positioned in inversion and plantar flexion [117]. Osteochondral lesions associated with chronic lateral ankle instability are larger (150mm2 or larger) in ankles without chronic lateral ankle

instability, and additional chondral lesions at the tip of the medial malleolus and the tibia plafond [118]. Osteochondral lesion of the ankle should be suspected when symptoms like dull and deep ankle pain, swelling, restriction of ankle movements, locking or crepitus persist following treatment of ankle inversion injuries. Because clinical findings are mostly non-specific imaging is often required to establish the diagnosis and accurately describe the anatomy of the lesion. Radiography alone is insufficient to detect all lesions and cannot provide the necessary details required to plan the treatment. Most often CT Scan, or MRI or a combination of both is required to establish the precise characteristics of the lesion and classify it. CT Scan provides information about the size shape and displacement of bony injury and MRI is used to evaluate the cartilage [119,120]. MRI can detect articular injury with morphological abnormality, but degenerative changes without morphological change are missed even in MR imaging. Arthroscopy allows the surgeon to diagnose such lesions by probing the articular surface and feeling for fissures [121]. In a study sensitivity and specificity for detecting an OCD with arthroscopy were 100 and 97%, respectively, and sensitivity and specificity values for MRI were 96 and 96%, and 81 and 99 % for CT [122]. Size, location, type, stability, displacement, and containment of the lesion are important factors in deciding the treatment [123].

Asymptomatic lesions or the lesions detected incidentally are kept under observation. Minimally symptomatic lesions may be treated non-operatively with rest, ice and temporarily reduced weight bearing [124]. For lesions up to 15mm in diameter reparative strategy by marrow stimulation using microfracture technique is recommended which is often performed arthroscopically [125-127]. Large cystic lesions with more or less intact cartilage are amenable to retrograde drilling to induce subchondral revascularization and stimulate new bone formation, and when lesions are difficult to approach due to their location antegrade transmalleolar drilling may be used [127-130]. Choi et al. [131] assessed the effect of the size of the lesion on the clinical outcomes and patients with lesions more than 15mm in diameter had worst outcomes [131]. A number of replacement surgeries which are done arthroscopically or performed through open surgery, depending upon the size and location of the lesion are available for larger lesions. These options include osteochondral allograft transplantation [132], osteochondral autograft transplantation [133], autologous chondrocyte implantation [134], matrix induced chondrocyte implantation [134,135], juvenile articular cartilage transplantation [134,136], particulated autograft cartilage implantation [137].

Osseous Injuries

During inversion injury of the ankle anterior talofibular ligament is mostly ruptures in its mid substance but in some cases this mechanism of injury can lead to avulsion fractures of distal fibula or talus [138]. Haraguchi et al. [139] noted avulsion fractures in 26% of the patients with severe inversion injury, with a 36% non-union rate after conservative treatment, but comparable between avulsion fracture groups and ligament rupture groups [139]. Large gaps in avulsion fracture of lateral malleolar tip may cause chronic pain and instability [140]. Symptomatic ossicles without instability may require excision [141]. In the presence of instability the symptomatic ossicles need excision and concomitant repair of lateral ankle ligaments [142]. In a recent study of 10 patients with avulsion fractures of lateral malleolar tip explored arthroscopically after a mean of 7.7 days (range 2-17 days) of injury, anterior talofibular ligament and calcaneofibular ligament were found to be attached to the avulsed fragment. The authors suggested primary fixation with screws and found it to be an efficient treatment to restore function and stability [143].

An inversion injury can cause avulsion fracture of lateral process of talus through lateral talocalcaneal ligament attachment [144]. Up to 40% of lateral process fractures are missed on initial presentation [145]. Tenderness anterior and inferior to the lateral malleolus should raise the suspicion for this fracture. If there is a high index of suspicion, then CT Scan or MRI scan should be done to establish the exact diagnosis. Neglected or insufficiently treated fractures can cause long term disability [146,147]. Complications of fractures of lateral process of talus include severe degenerative subtalar arthritis, nonunion, and impingement on calcaneus and fibula due to overgrowth of the lateral process. Large displaced fractures involving articular surface need open reduction and internal fixation, and non-reconstructible fragments need to be excised [144,148]. In a chronically neglected fracture with arthritis of subtalar joint, subtalar fusion may be required [148].

Avulsion fracture of the anterior process of the calcaneus is another supination injury which is often missed in the initial stages [149]. Dorsal attachment of bifurcate ligament can cause this avulsion fracture [150]. Extensor brevis muscle attachment lateral to bifurcate ligament can also cause large wedge or flake shaped avulsion fracture [151]. Most injuries heal uneventfully, and in patients with persistent complaints resection of small avulsion fracture carries a minimal risk and appears to have a significant benefit [150]. Dorsal talar and navicular avulsion fractures are uncommonly seen in patients presenting with lateral ankle sprain [152]. Dorsal talonavicular ligament and the anterior tibiotalar ligament when injured can avulse causing a small bony injury [153]. Most injuries do not warrant any specific treatment but missed diagnosis can be an overly concern that a fracture was missed, which can lead to confusion and anxiety [153,154]. It is important to palpate dorsal surface of talus and navicular in patients presenting with lateral ankle sprain to avoid missing these injuries.

Avulsion fracture of the base of the fifth metatarsal is another common osseous injury associated with inversion injury of the ankle. It is due to forces exerted on the base by the attachment of the peroneus brevis and lateral aponeurosis [155]. Most fractures heal with a walking boot or cast. Neglected or inappropriately treated injuries may lead to painful non-unions, which require excision of the small fragment and repair of the peroneus brevis and fixation

and grafting of the large fragments [156].

Sinus Tarsi Syndrome

Pain directly over the sinus tarsi following an inversion injury of the ankle may be due to sinus tarsi syndrome. Interosseous ligament injury, hypertrophy of the synovium, or hypertrophy of the fat resulting in impingement of the neural plexus are the possible reasons for the pain [144]. Pain relief after an injection of local anaesthetic into the sinus tarsi can be diagnostic as well as therapeutic. Open or arthroscopic debridement of the contents of the sinus tarsi is required sometimes.

Conclusion

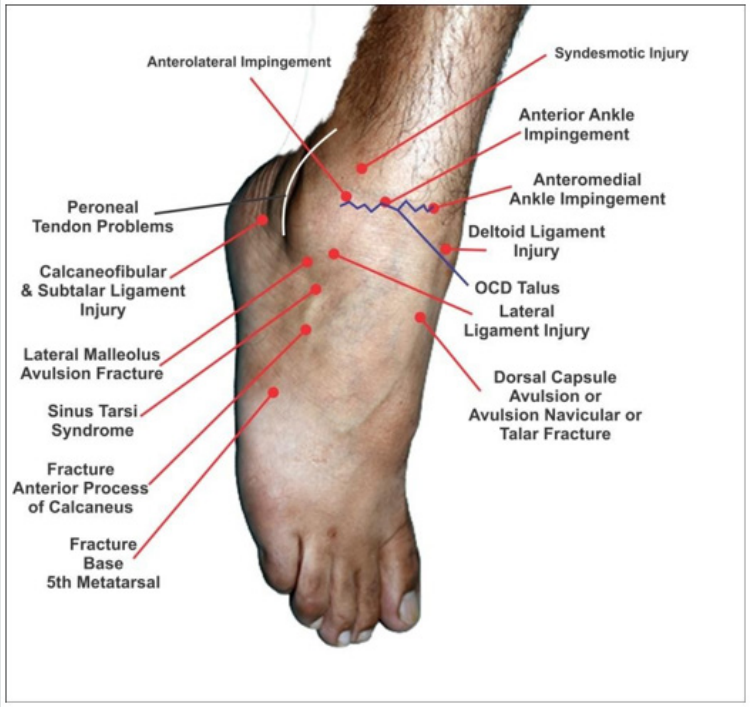

Figure 1: Injuries seen with inversion sprain of the foot and possible sites of tenderness.

An inversion injury of the foot may cause damage to a number of important structures apart from lateral ligament of the ankle. It is essential to have a sound anatomical knowledge of the foot and ankle and a routine examination should include the palpation of various structures as shown in the Figure 1. A structured examination of ligaments, osseous, chondral, muscular and tendinous structures around the lateral malleolus is suggested to locate the exact cause of pain. It is not uncommon to have combined injuries of the above-mentioned structures in one individual. Apart from the systematic examination MRI is a very helpful in reaching the exact diagnosis. In spite of increase in knowledge of the foot and ankle anatomy and advances in imaging techniques the problem of persistent pain after lateral ankle sprain still remains in a few cases.

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