REVIEW ARTICLE

Common Orthopaedic Foot & Ankle Diagnoses Encountered in the Primary Care Setting

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Foot and ankle disorders are commonly encountered in the primary care setting. Many of these disorders can be successfully managed by primary care physicians, allowing for early detection and prompt treatment. However, there are circumstances when patients require a referral to a foot and ankle specialist to decrease potential complications of these disorders. This article will review ten common foot and ankle disorders to aid in the improved understanding of when conservative management is appropriate and when referral to a specialist is necessary.

INTRODUCTION

Foot and ankle disorders are commonly encountered in the primary care setting. Many of these disorders can be successfully managed by primary care physicians, allowing for early detection and prompt treatment. However, there are circumstances when patients require a referral to a foot and ankle specialist to decrease potential complications of these disorders.[1] This article will review ten common foot and ankle disorders to aid in the determination of when conservative management is appropriate and when referral to a specialist is necessary.

ANKLE SPRAINS

Ankle sprains are one of the most commonly encountered orthopaedic injuries, comprising 15-20% of all sports injuries.[2] Ankle sprains are most commonly due to inversion and adduction of the plantar flexed foot, resulting in injury to the lateral ligamentous complex. Medial sprains are less common, resulting from an eversion and abduction force. The anterior talofibular ligament is the most common ligament injured in a lateral ankle sprain, followed by the posterior talofibular and calcaneofibular ligament. In one-third of cases all three ligaments are injured.[3]

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Ankle sprains can be difficult to differentiate from other conditions, including fractures, tendon ruptures and midfoot injuries. Patients may present with bony tenderness to palpation (TTP) if there is an avulsion rather than a mid-substance ligament tear. Ankle stability can be assessed by performing the anterior and posterior drawer tests, the talar tilt test, Kleiger's test and the dorsiflexion torque test.

The anterior and posterior drawer tests are performed by grasping the foot and stabilizing the tibia while applying an anterior or posterior force to the ankle. A positive test is indicated by translation of the ankle joint relative to the uninjured side. The talar tilt test is performed by applying an inversion stress to the ankle in neutral dorsiflexion with the knee flexed to 90.°

Many injuries occur concomitantly with ankle sprains, and for this reason Fallat et al. suggested that these injuries be referred to as "Ankle Sprain Syndrome." Commonly associated injuries include ankle syndesmotic injuries, Achilles tendonitis, peroneal tendonitis, medial and intermediate dorsal cutaneous neuritis, ankle avulsion fractures, and fifth metatarsal base fractures.[3]

Ankle sprains are classified as grade one through three based on increasing severity of injury and stability of the ankle joint, which serves to guide treatment.[2,4] Low grade sprains with a stable ankle joint are managed using functional bracing: taping, elastic bandage, lace-up or semi-rigid ankle braces, which yield superior outcomes as compared to rigid immobilization. Lace-up braces result in less persistent swelling than elastic bandages, but more dermatologic complications. Grade III sprains, those with an unstable ankle joint, may benefit from a short period of immobilization in a short leg cast, removable cast boot, splint, or air-cast, followed by a supervised rehabilitation program. Surgical management, consisting of suture repair or tendon transfers is controversial. There is little evidence supporting routine surgical management of acute ankle sprains, except in cases of chronic recurrent ankle sprains failing non-operative treatment.[3,4]

Like the talofibular and calcaneofibular ligaments, those comprising the ankle syndesmosis can also be sprained or torn as a result of rotational ankle injuries, a so-called "high ankle sprain." The ankle syndesmosis is comprised of the anterior inferior tibiofibular ligament, the posterior inferior tibiofibular ligament, the inferior transverse ligament, the interosseous membrane and the interosseous ligament. The mechanism of injury is usually external rotation and can be purely ligamentous or associated with a fracture. Clinically, patients will present with pain between the tibia and fibula more proximal to the ankle joint. A positive squeeze test, eliciting pain when the proximal tibia and fibula are squeezed together, can aid in the diagnosis. Radiographic evaluation will be discussed in a later section. Isolated syndesmotic sprains rarely result in ankle instability and can be managed nonoperatively with cast immobilization for 2 to 3 weeks followed by progressive weight bearing in a walking boot.[5]

ANKLE FRACTURES

Fractures of the malleoli typically occur as a result of rotational forces to a planted foot, most commonly an external rotation force applied about a supinated foot. Two-thirds of ankle fractures are unimalleolar, with bimalleolar and trimalleolar fractures resulting from increasingly higher energy injuries. Radiographs of the ankle typically include AP, lateral, and mortise views. Additionally, full length x-rays of the tibia and fibula should be obtained due to possible proximal fibula fractures occurring in conjunction with ankle fractures.[6] Particularly in cases of tenderness to palpation over the proximal lateral calf.

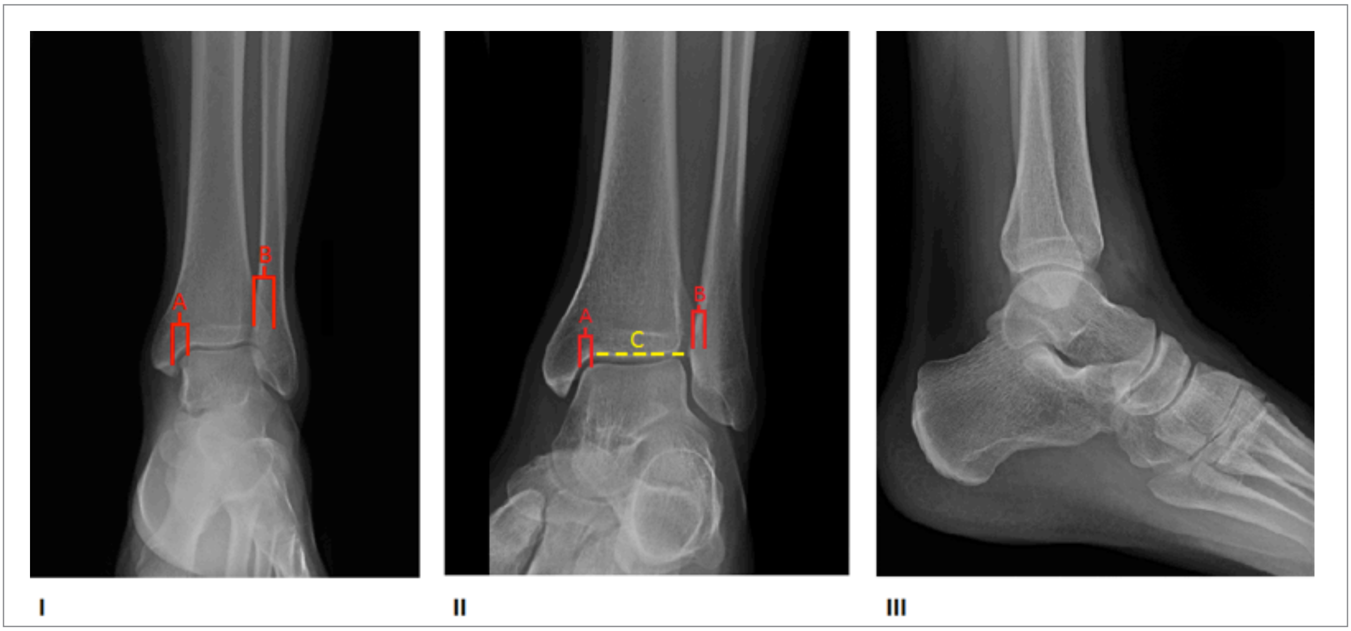
Management of ankle fractures depends on the stability of the injury, which can be determined by a number of radiographic parameters, which are illustrated in Figure 1. The medial clear space (A) between the talus and medial malleolus should equal the tibiotalar joint space (Normal is <6mm) on AP or mortise views.[5] Medial clear space widening is indicative of deltoid ligament injury, seen in bimalleolar and trimalleolar equivalent fractures (injuries with a lateral and posterior malleolar fracture and medial deltoid disruption). Also on AP and mortise x-rays, the tibiofibular overlap (B) should not be less than 10mm, as this is indicative of injury to the tibiofibular syndesmosis.[5]

The location of the lateral malleolus facture also assists in determining stability. Lateral malleolus fractures at or above the level of the tibial plafond, the distal tibial articular surface (C), are more likely to have associated tibiofibular syndesmotic injury than those below the level of the plafond. Any injury to the medial side of the ankle including medial malleolus fractures, bimalleolar and trimalleolar fractures with deltoid ligament disruption indicates an unstable injury requiring operative management.[5,7]

Dislocation of the ankle will require prompt reduction and immobilization to minimize swelling and soft tissue damage.[7] Isolated lateral malleolus fractures below or at the tibial plafond, including lateral malleolus avulsion fractures, with no medial clear space widening, talar shift or medial tenderness can be managed nonoperatively with an ankle brace and full weight bearing as tolerated. Isolated lateral malleolus fractures at or above the tibial plafond without medial clear space widening and without talar shift can

FIGURE 1:

AP (I), Mortise (II), and Lateral (III) radiographs of a left ankle. Medial clear space is demonstrated by "A." Tibiofibular overlap is demonstrated by "B." The tibial plafond is demonstrated by "C."



also be managed nonoperatively, with immobilization and non-weight bearing. Isolated medial malleolar fractures, including avulsion fractures, can be treated conservatively with immobilization if there are no radiographic indicators of ankle instability. Additional consideration of stress radiographs may be indicated in suspected unstable injuries. Suspicion of an unstable ankle fracture pattern is an indication to refer to an orthopedic specialist to further evaluate and stress an ankle for instability, as these may require operative intervention. Bimalleolar and trimalleolar fractures, and any fracture-dislocations are unstable injuries requiring operative management, following reduction and splinting. The goal of operative fixation is the restoration of fibular length and the congruity of the tibiotalar joint. Injury to the syndesmosis, which may be found intraoperatively, requires additional fixation; however, the exact technique for syndesmotic fixation, remains controversial.[6,8] Ankle fractures in skeletally immature patients should generally be referred to a pediatric foot and ankle orthopaedic specialist as these injuries frequently involve the physis.

ANKLE ARTHRITIS

Approximately 1% of the population suffers from ankle osteoarthritis (OA). Compared to the hip and knee, the ankle is rarely subjected to primary OA, with most cases being post-traumatic. Brown et al found that 79.5% of patients with ankle OA had a history of at least one joint injury, versus 1.6% and 9.8% in hip and knee OA, respectively. Ankle fractures have been shown to be the primary cause of ankle post-traumatic osteoarthritis (PTOA), followed by ligamentous ankle injuries.[9,10]

There are two underlying mechanisms involved in PTOA of the ankle, direct damage to the chondral surfaces from the initial injury, and the resultant chronic abnormal overloading of the joint secondary to incongruity of the articular surfaces and instability. Risk factors for PTOA include lateral malleolus fractures above the tibial plafond, medial malleolus fracture, dislocation, increasing BMI, age, time since surgery and severity of chondral damage at the time of injury.[10,11]

Patients will present with progressively worsening deep ankle pain. Early on, pain occurs during the day with activities that load the ankle joint, whereas advanced ankle OA will cause pain at rest and at night. With mid-stage and end-stage disease, significant lower extremity muscle atrophy can be seen. Reduced ankle ROM is the earliest physical exam finding, which can be attributed to many factors including joint incongruity, cartilage loss, soft tissue contractures or muscle spasm, along with osteophytes or loose bodies. Over time, crepitus, joint effusion, joint line tenderness and hindfoot instability can develop.[10,11]

Weight loss, activity modification and restoration of articular surfaces in ankle fractures are key to prevention of PTOA. Conservative treatment options include NSAIDs, orthotics, and physical therapy. Viscosupplementation with hyaluronic acid is controversial and has limited evidence supporting its efficacy in ankle OA. Steroids are marginally more effective and longer lasting than NSAIDS, which are often first-line treatment of arthritis. Orthotics and bracing can be used to help unload the effected side of the ankle and provide stability, but their effectiveness is limited by patient compliance. Finally, physical therapy can play a role in prevention of OA progression, as well as preserving independent ambulation.[10-12]

Surgical treatment varies in scope and effectiveness. Osteophytes, loose bodies, and chondral defects can be treated with arthroscopy. Osteotomies can be performed in early OA to correct bony deformities. Arthrodesis in neutral dorsiflexion with roughly 5 degrees of external rotation is reserved for end-stage ankle OA to allow for near normal gait and pain relief. Total ankle arthroplasty is an emerging option for severe ankle OA, resulting in improved pain relief, gait and patient satisfaction, but potentially has a higher reoperation rate when compared to arthrodesis.[13-15]

ACHILLES TENDON INJURIES

Despite being the largest and strongest tendon in the body, the Achilles tendon is the most commonly injured tendon in the lower extremity. Achilles tendon disorders exist along a spectrum ranging from tendinosis to acute tendon rupture. Achilles tendinosis is a non-inflammatory degeneration secondary to repetitive microtrauma and aging. Patients are often asymptomatic, however some patients with partial ruptures of the tendon can experience focal tenderness over the area of rupture. Areas of tendinosis can be visualized as hypoechoic lesions on ultrasound or as areas of altered signal on MRI. Treatment of Achilles tendinosis is largely conservative, consisting of rest, anti-inflammatories, heel lifts and activity modifications. Those with severe pain may respond well to a period of immobilization followed by physical therapy consisting of eccentric heel stretching and calf strengthening exercises.[16,17]

Achilles tendon ruptures occur most commonly in males in their fourth or fifth decade. Sixty-eight percent are sports-related, resulting from an eccentric contraction of the triceps surae, especially in episodic athletes (“weekend warriors”). Approximately 75% of ruptures occur 2-6cm from the calcaneal insertion due to a vascular watershed area here. Other common sites of rupture include the distal insertion (10-20%) and the musculotendinous junction (5-15%).[17,18]

Corticosteroids, flouroquinolone antibiotics, and chronic tendinopathy such as from a Haglund’s deformity, an enlargement of the posterosuperior calcaneal tuberosity, are also associated with Achilles tendon ruptures. Patients will describe feeling a “pop” or a sensation of being hit in the back of the leg following resisted plantar flexion. Patients may also complain of inability to bear weight and plantar flexion weakness, but often will not complain of pain after the acute episode.[17,18]

Diagnosis of acute ruptures requires at least two physical exam findings: a positive Thompson Test, decreased plantar flexion strength, decreased resting plantarflexion of the ankle, a palpable defect over the tendon, or increased passive dorsiflexion. Intact plantar flexion does not rule out an Achilles rupture, as the tibialis posterior, long flexor tendons of the hallux and toes and plantaris muscles can still weakly plantarflex the foot. Ultrasound, radiography, and MRI are not necessary to confirm the diagnosis, but can be beneficial in the management of chronic ruptures, and to differentiate partial and complete tears.[17]

Patients should be immobilized in a posterior splint in plantar flexion, until a definitive treatment course is determined. Recent studies has shown when nonoperative management includes an early functional rehab program, re-rupture rates and plantar flexion strength appear to be equivocal between operative and nonoperative management. Operative management does however carry surgical risks including infection, adhesions, nerve injury, and wound problems, therefore one must carefully consider pursuing operative management in patients at risk for poor wound healing.[18,19]

PERONEAL TENDON INJURIES

Peroneal tendon injuries are often overlooked as a source of ankle pain, and commonly misdiagnosed as ankle sprains. The peroneus brevis and longus tendons pass posterior to the lateral malleolus within the retromalleolar sulcus, beneath the inferior and superior peroneal retinaculum (SPR) to insert at varying sites on the foot. The peroneal tendons are subject to two types of acute injuries, tendon subluxation and tendon tears. Peroneal tendon subluxation occurs with a tear or defect in the SPR, allowing the tendons to subluxate out of the retromalleolar sulcus.[20,21]

Following a dorsiflexion or inversion injury, patients will complain of a "popping" sensation, lateral ankle pain, and possible instability. Patients will have tenderness and swelling behind the lateral malleolus and along the lateral calcaneus, weakness and pain with resisted eversion, and pain with passive inversion stretching. Also, with resisted eversion and active dorsiflexion, patients may feel apprehension or physical subluxation of the peroneal tendons.[21,22]

Standard foot and ankle radiographs should be obtained to rule out fracture or other osseous abnormalities. Ultrasound can visualize dynamic subluxation of the tendons, as well as tears and fluid collections associated with peroneal tendonitis.[23] MRI can be used as an adjunct to evaluate for variations in normal anatomy of the peroneal muscles and tendons, as well as determination of whether tendon tears are isolated or combined injuries.[20]

Treatment of peroneal tendon subluxation depends on the acuity of the injury. Acute subluxations can be treated nonoperatively with a short leg cast in a plantar flexion and inversion for approximately 6 weeks, after reduction of the tendons. With chronic subluxation (greater than 4 weeks), nonoperative treatment has a poor success rate. Chronic subluxations have multiple surgical options, including direct SPR repair, retromalleolar groove deepening, soft tissue transfers, and fibular osteotomies. Operative management is indicated in athletes wishing a rapid return to sport, with direct SPR repair the most common surgical treatment.[20,22]

Isolated peroneus brevis tears can be treated conservatively with NSAIDS, cast or walking boot, and activity modification, however the success rate is low and surgical management should be strongly considered. Isolated peroneus longus tears are rare, occurring in patients with predisposing conditions including diabetes, hyperparathyroidism, rheumatoid arthritis, and psoriasis.[20] Patients with full function and minimal symptoms can be managed non-operatively, while those with more severe or recalcitrant symptoms can be managed operatively, similar to isolated peroneus brevis tears. With concomitant tears of both tendons, surgical management is indicated, ranging from tenodesis to repair, depending on the condition of each tendon. Most patients return to full activity following operative treatment.[21]

Peroneal tendinopathy is a more chronic degenerative condition affecting the peroneal tendons, resulting from repetitive microtrauma in patients such as runners and dancers, or patients with chronic ankle sprains or high arch feet. Patients present similarly to those with tendon tears, with pain and tenderness along the length of the peroneal tendons. Ultrasound and MRI can be effective in visualizing fluid surrounding the tendon sheath as well as tendon thickening. Treatment is generally conservative, with NSAIDS, cast or walking boot and activity modification. If patients fail 3-6 months of conservative therapy, operative tendon debridement and tenosynovectomy can be effective.[22]

POSTERIOR TIBIALIS TENDON DYSFUNCTION

The tibialis posterior is the most important dynamic stabilizer of the arch of the foot. Contraction causes elevation of the medial longitudinal arch, locking the midfoot and hindfoot, thus increasing rigidity during gait. More commonly, laxity of the hind and midfoot results in gradual degeneration of the subtalar, calcaneocuboid, talonavicular, and tibiotalar joints. Posterior tibialis tendon dysfunction (PTTD) is the most common cause of acquired flat foot deformity in adults, occurring most commonly in middle-aged, obese women. Other risk factors include hypertension, diabetes, local steroid injection and seronegative arthropathies, such as ankylosing spondylitis and psoriatic arthritis.[25] The pathophysiology of PTTD is a tendinosis resulting from a tenuous blood supply to the tendon as it passes posterior to the medial malleolus in addition to chronic stresses. Direct rupture of the tendon is rare.

Swelling and TTP along the tendon may be noted posterior to the medial malleolus. In PTTD, hindfoot valgus and forefoot abduction results in flattening of the medial longitudinal arch leading to the "too many toes sign" as compared to the normal.[25] The ability to perform a single-leg heel rise is the most commonly used functional test in PTTD; patients are unable to perform the normal 5-10 repetitions of a single-leg heel rise. Alternatively patients can be asked to walk a short distance on their toes. Imaging in early stages should be limited to standard ankle and foot radiographs to rule out other causes of ankle pain. In later stages, radiographs and CT scan can reveal arthrosis in the hind and midfoot joints. MRI is useful for evaluating abnormal pathology along the tendon course.[25]

Conservative treatment of early PTTD consists of weight bearing in a cast or fracture boot for 4-weeks, followed by arch support and medial heel wedge orthotics. Physical therapy involves strengthening the tibialis posterior. Initial surgical management may entail tendon transfers and osteotomies that can be used to correct flat foot deformities failing conservative management. At this stage preserving remaining motion is still possible. In late stages, involving the subtalar, calcaneocuboid, and talonavicular joints, a triple arthrodesis can be considered to correct deformity and relieve pain, with subsequent loss of motion. Finally, with advanced disease involving the tibiotalar joint, a pan-talar arthrodesis can be performed as a salvage procedure.[25]

METATARSAL FRACTURES

Metatarsal fractures comprise approximately five to six percent of all fractures in the primary care setting.[26] In all cases suspicious for fracture, standard AP, lateral and oblique radiographs of the foot should be obtained *(Figure 2, page 16)*.

FIGURE 2:

AP (I), Lateral (II), and Oblique (III) radiographs of a left foot.

Metatarsal shaft fractures result from twisting injuries or direct blows. These fractures are generally stabilized by adjacent metatarsals, and the majority can be treated nonoperatively with reduction and immobilization using a compressive wrap, posterior splint or stiff-soled shoe with weight-bearing as tolerated. Unstable fractures will require operative fixation. Metatarsal base fractures can be categorized into first through fourth metatarsal base fractures and fifth metatarsal base fractures.[26]

Fractures of the base of the first through fourth metatarsals are usually caused by crush injuries, direct blows or axial loads to a plantarflexed foot. These fractures rarely occur in isolation as adjacent structural ligaments may also be damaged and frequently require surgical intervention.[26] Of note, special attention should be paid to fractures at the base of the second metatarsal as these can be fractured in LisFranc joint injuries, a disruption of the articulation of between the base of the second metatarsal and the medial cuneiform. One must have a high suspicion for LisFranc injuries in any patient presenting with forefoot pain following a hyper-plantarflexion injury, as these injuries are highly unstable and routinely require operative management. Weight bearing foot radiographs will aid the primary care physician in making this diagnosis.

Fifth metatarsal base fractures are more common, and result from inversion injuries to the ankle, often being misdiagnosed as ankle sprains. These fractures are grouped into zones, depending on location, as illustrated in figure 3 (Figure 3). There is typically a high level of confusion in regards to these injuries with the main differentiation between a Zone 1 and 2 injury. Zone 1 (pseudo Jones fractures) or tuberosity fractures can be treated with protected weight bearing in a soft dressing or hard-soled shoe, or a splint initially if the patient has significant pain.[27] Zone 2 or metaphyseal-diaphyseal junction fractures occur at the fourth-fifth metatarsal articulation, and are commonly referred to as "Jones Fractures." This zone coincides with the avascular watershed area, increasing the risk of nonunion by disrupting the already tenuous vascular supply. Zone 3 or diaphyseal stress fractures occur distal to the fourth-fifth metatarsal articulation. Zone 2 and 3 fractures may be treated with 6-8 weeks of nonweightbearing in a short leg cast.[27]

In higher level athletes or individuals consideration for surgical intervention of Zone 2 and 3 injuries should be discussed. It is important to differentiate Zone I fractures in adolescents from Iselin's disease, traction apophysitis of the base of the fifth metatarsal, which is the result of repetitive traction by the lateral plantar aponeurosis and can be treated with rest, ice and activity modification.

Metatarsal stress fractures also require special consideration. These injuries occur most commonly in athletes with an acute increase in activity level, or by chronic repetitive overloading. Additionally they can occur in the cohort of patients with osteopenia or osteoporosis with a relatively minor history of trauma. Radiographs are often negative, in which case MRI or bone scan can be useful to confirm diagnosis. Often the only presenting complaint is difficulty with weight bearing and dorsal foot swelling. Stress fractures with no sclerosis at the fracture site are treated nonoperatively with protected weightbearing precautions in a cast or cast boot for 6-8 weeks. When sclerosis is present, operative management is required, consisting of curettage, bone grafting and fixation, followed by six weeks of non-weightbearing.[28] The navicular and cuboid tarsal bones are also subject to stress fractures. These injuries also commonly result from overuse injuries in athletes. These injuries can be treated successfully in most cases with non-weightbearing and cast immobilization.

HALLUX VALGUS

Hallux valgus is the most common cause of forefoot pain in adults. It is a chronic condition characterized by the progressive lateral deviation of the hallux and medial deviation of the first metatarsal, leading to the subluxation of the first metatarsophalangeal (MTP) joint.[30] Predisposing factors to hallux valgus are divided into intrinsic and extrinsic factors. Intrinsic factors include genetic predisposition, female gender, ligamentous laxity, osteoarthritis and rheumatoid arthritis, foot deformities such as pes planus, increased age and neuromuscular disorders such as cerebral palsy. Extrinsic factors include high heeled or narrow toe box shoes, ballet dancing, trauma and excessive weight bearing.[29,31,32]

FIGURE 3:

Oblique radiographs of a left foot. Zone I demonstrated by "A", Zone II is demonstrated by "B", Zone III is demonstrated by "C."

Any factor compromising the medial structures of the first MTP joint can initiate the progression of hallux valgus. When the medial collateral ligament becomes attenuated, the metatarsal head deviates medially and the proximal phalanx is pulled into valgus. This deformity can induce a bursitis over the medial eminence (bunion), and the extensor and flexor hallucis longus tendons bowstring laterally, exaggerating the deformity. Patients will present with pain and varying degrees of deformity at the first MTP joint.[31] Special attention should be paid to the wear patterns of patients' footwear as this can be a large contributing factor to the patient's symptoms.[30] Patients will frequently present early on with callus formation of the first MTP.

Conservative management should be initiated in all patients and particularly is the first-line for pediatric hallux valgus, elderly patients, patients with neuropathy, and other poor surgical candidates. As there is no evidence that conservative treatment can correct deformity, the goal of conservative management is to relieve symptoms while avoiding lifestyle modifications. Avoiding narrow toe box shoes, over the counter cushions and pads, anti-inflammatories and possibly steroid injections can all be attempted. Orthotics have not been shown effective in slowing the progression of hallux valgus.[30] Additionally hallux valgus should not be treated with surgical intervention prophylactically or for cosmetic purposes. Indications for surgery are pain and functional limitations not responding to shoe wear or activity modifications.

INTERDIGITAL (MORTON'S) NEUROMA

Interdigital neuroma of the foot is another common cause of adult forefoot pain, occurring primarily in middle-aged women. The most common location is within the third webspace, between the third and fourth metatarsal heads, affecting the third common digital nerve – commonly referred to as a Morton's neuroma. Possible causes include narrow toe box shoes and the inherent anatomy of the third interdigital space predisposing to neuroma formation. Regardless of the cause, the third common digital nerve becomes compressed, leading to a fusiform swelling of the nerve.[32,33]

Patients complain of burning pain between the metatarsal heads radiating to the third and fourth toes, particularly with narrow toe box shoe wear. There is tenderness on the foot plantar surface with standing and walking along with paresthesias, which are reproducible with palpation. A positive Mulder's Sign, eliciting a palpable click in the affected interspace with reproduction of the patient's symptoms when the metatarsal heads are squeezed together, is the most sensitive diagnostic tool. Radiographs can be obtained to rule out osseous conditions, while MRI and ultrasound can be used as an adjunct for diagnosis.[32,33]

Initial management consists of changing to wider toe box shoes, activity modification, NSAIDs and possible steroid injections. Surgical treatment is indicated in those cases failing conservative management. Open or endoscopic release of the transverse intermetatarsal ligament can be performed to remove the mechanical irritation of the common digital nerve. Alternatively, the neuroma and nerve itself can be excised.[33]

PLANTAR FASCIITIS

Plantar fasciitis (PF) is the most common cause of plantar heel pain in adults. It occurs primarily in patients in their 40s and 60s, and with a bimodal distribution in younger patients who are runners and older patients who are relatively sedentary.[34] It is usually unilateral, occurring bilaterally in about one third of cases. The plantar fascia serves as one of the static stabilizers of the longitudinal arch of the foot. PF occurs as a result of repetitive microtrauma and excessive strain to the plantar fascia. Risk factors for the development of PF include Achilles tendon or triceps surae tightness, obesity, chronic weight bearing professions, increased age, poor footwear, over training in athletes, and decreased mobility of the subtalar joint.[34,35]

Patients present with pain that is worst with their first steps in the morning, or after a prolonged period of rest. On exam there will be tenderness at the medial plantar calcaneal tuberosity and along the length of the fascia. Maneuvers that stretch the plantar fascia, including passive dorsiflexion can also elicit pain. The Silfverskiöld test can be used to differentiate between Achilles tendon contracture and gastrocnemius muscles tightness, both of which tension the plantar fascia.[34] To perform the Silfverskiöld test, the patient's ankle dorsiflexion is measured first with the knee extended then flexed to 90.° Decreased dorsiflexion in extension which increases with knee flexion indicates a tight gastrocnemius. If knee flexion and extension does not affect dorsiflexion, the Achilles contracture is the likely cause. The diagnosis of PF is usually clinical, however standard weight bearing radiographs of the foot should be obtained to rule out skeletal causes of heel pain.

Activity modification, rest, stretching, structured physical therapy programs, heel cushions, orthotics, NSAIDs and weight loss are all components of successful conservative management. Night splints are used to help prevent the contracture of the plantar fascia and the triceps surae.[34] Studies have shown steroid injections and platelet rich plasma (PRP) injections into the plantar fascia as equally efficacious, however given the potential risk of fascia atrophy and rupture, PRP is technically a safer alternative, albeit substantially more expensive, requires specialized equipment and typically is not reimbursed through insurance.[36]

Surgical management of PF is usually reserved for recalcitrant cases. Plantar fasciectomy, both open and endoscopic, has been shown to provide relief of symptoms in about two thirds of patients. Open plantar fascia release has the added benefit of releasing the first branch of the lateral plantar nerve, further reducing pain.[35,37] Gastrocnemius recession may also be an option for treatment. Recalcitrant patients should be referred to a foot and ankle specialist to evaluate for additional causes and possible surgical intervention.

CONCLUSION

The ten foot and ankle conditions reviewed in this article are but a small fraction of the myriad of musculoskeletal complaints commonly encountered in the primary care setting. With a better understanding of the disorders and their initial evaluation and management, indications for referral to a musculoskeletal specialists and the need for operative management, primary care physicians can better aid in the diagnosis, care and recovery of their patients afflicted by these common disorders.

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