Lower Leg, Ankle, & Foot Injuries

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LOWER LEG PAIN

Anatomy
Athletes performing any sport expose themselves to a particular subset of sports specific injuries including injury to the foot and ankle. Approximately 25% of all sports injuries involve the foot and ankle and about 45% of these are simple lateral ankle sprains. In cutting sports such as football, baseball, volleyball and soccer these injuries account for up to 25% of lost playing time. Some sports have a low incidence of foot injuries. Swimming for example has a very low incidence of ankle injuries. Basketball and figure skating have the highest incidence. With respect to foot injuries, football and weight lifting are the lowest risk sports. Hiking and high-speed motor sports have a higher incidence (>50%).

Overall, every sport poses a specific threat to the athletes’ foot and ankle as a result of the activity and the equipment that is used. These injuries may not be benign. Approximately 40% of all simple ankle sprains lead to chronic disabilities and may be the end of the athlete’s career.

This chapter will discuss the diagnosis and treatment of common injuries and conditions of the lower leg and foot that frequently occur in athletes. Anatomic, biomechanical and functional principles of the foot and ankle will be described. We will discuss conservative as well as operative treatments.

MUSCLE STRAINS

Essentials of Diagnosis:
Immediate sharp pain in calf
Hear or feel a pop or a snap
Feeling as if someone “kicked you in the calf”
Inability or pain with calf raise
Swelling, bruising to calf
Palpable gap in the medial or lateral gastrocnemius-soleus (gastroc-soleus) muscles

**Etiology:**

Muscle strains of the gastroc-soleus complex are most frequent injuries in jumping or sprinting athletes. Muscle injuries account for up to 30% of all injuries sustained in sports events. These injuries pose a significant challenge to every sports medicine clinician. The majority of these muscle injuries are caused by contusion or excessive strain of the muscle and they can sideline athletes for a long time.

Those with a prior history of muscle strains have increased risk of further injury. The closer to the previous calf strain the re-injury occurs the higher the risk for a recurrent calf strain. There also seems to be a relationship with age predisposing older athletes to a higher risk for calf strains.

**Prevention:**
Fatigue may play a key role in the occurrence of calf strains since muscle strain injuries seem to occur late in either training sessions or competitive settings. It was shown that the energy absorbed before failure was significantly less in the fatigued muscle than in the controls. Therefore, proper conditioning to reduce or delay fatigue should be part of the prevention strategy.

Calf stretching and warm up should be an integral part of the athletes’ preparation. The musculo-tendinous unit has specific viscoelastic properties that can be influenced by warm up and cyclic stretching. Cyclic stretching up to 50% of the maximum stretch to failure has a beneficial effect on the amount of energy that a muscle can absorb before failure. Stretching past 50% of the maximum stretch reduces the maximal amount of energy that the muscle can absorb. Therefore, the recommendation is to perform light stretching exercises before sports activities. Viscoelasticity is temperature dependent. Therefore, the warm up exercises help to increase the viscoelastic properties of the musculotendinous unit before athletic activity.

Clinical findings:

Symptoms (History)
Calf muscle strains can occur in cutting and jumping sports such as basketball, football, soccer, tennis and racquetball. The athlete is usually not able to return to play after a significant calf muscle strain. It's important to know if the athlete has had a prior, recent, minor injury to the affected calf. If the athlete had a previous injury, the severity of the muscle injury is likely to be greater the second time. Patients report immediate pain in the back of their calf. They may have felt a sudden pop or snap. The feeling “as if someone hit them in back of the calf” is often mentioned.

Signs (physical examination)

It is important to do a thorough physical examination to determine if the injury is in the muscular portion of the medial or lateral head of the gastrocnemius or at the musculotendinous junction of the gastroc-soleus complex. A muscle strain may be palpable as a swelling of the calf. Alternatively, if a disruption of the muscle fibers has occurred a gap may be felt by physical examination before swelling has occurred or after it has subsided. The gap and swelling is usually felt in the muscle substance. A rupture of the medial head of the gastrocnemius is more frequent than the lateral head. The gap is therefore palpable medial or lateral to the midline. The most important differential
diagnosis of a calf strain is the rupture of the Achilles tendon. Therefore, if the gap is palpable directly in the midline and distal to the musculotendinous junction, a rupture of the Achilles tendon rather than a muscle strain should be suspected. The Thompson test or calf squeeze test is helpful to make the differential diagnosis. In order to perform the Thompson test the patient is positioned prone with the foot hanging over the edge of a table. The examiner squeezes the calf muscle and watches for plantar flexion of the foot. If there is plantar flexion the test is negative and identifies an intact musculo-tendon complex. If there is no plantar flexion, either then the gastroc-soleus complex is torn at the myotendinous junction or the Achilles tendon is torn.
Imaging:

The need for imaging of the calf after a calf muscle strain depends on the severity of the injury. If the physical examination suggests a large or complete tear of the medial or lateral gastrocnemius, a magnetic resonance imaging (MRI) may be used to evaluate the extend of the injury. This also aids in surgical planning. MRI may also be useful to monitor the healing of a muscle strain (Fig.1).

Calf muscle strains can also occur in combination with other injuries such as ankle sprains fractures of the fibula or neurovascular injuries in which case the associated injuries dictate the need for imaging modalities.

Treatment:

The treatment for calf muscle strains begins immediately at the sideline according to the RICE (rest, ice, compression, elevation) principle. This initial treatment regimen is designed to avoid large hematoma formation, which may affect the size of the scar tissue formed during the recovery. Initial treatment can be performed for 24-48 hours.

After the initial treatment the subacute treatment protocol calls for rehabilitation as well pain control with
non-steroidal anti-inflammatory drugs (NSAIDs) have been shown not to adversely affect healing in the initial phase of the muscle strain recovery. NSAIDS, however, should not be given long term, past 7-10 days since they may interfere with muscle healing at a later stage. A mainstay for the treatment of muscle strains is rehabilitation. Light stretching and strengthening as well as ultrasound therapy provide significant pain relief and begin to recondition the muscle fibers as well as the scar tissue. The key is not to disrupt healing of the soft tissue. Treatment should be started after a couple of days of rest. Light stretching exercises such as towel stretches, standing calf stretches, and progressive resistive exercises can be performed very early. The exercise should be performed within the pain free range of motion (ROM). After 7-10 days, light strengthening can be performed using standing heel raises, single leg exercises such as hops and gradual return to sports specific exercises involving running, cutting and jumping.

Surgical Treatment:

Injury resulting in tears of the entire muscle mass are more frequent in the abdominal, hamstring or rectus muscles but also occur in the calf. These injuries usually have a very large palpable gap and result in significant
loss of function. Conservative treatment of massive muscle tears results in very large amounts of scar tissue that may preclude return to sport. Primary muscle repair in complete disruptions of the muscle belly may lead to a smaller scar formation and better functional recovery.

Return to play:

Generally, the severity of the muscle strain will determine the period of return to play. It is important to keep in mind that a previous calf strain predisposes the athlete to a more severe calf strain. It may therefore be prudent to let the strain be fully healed before the athlete returns to play. Depending on the severity this may be anywhere between 1 and 4 weeks. We feel that an important criterion for return to play is the isometric muscle strength being within 90% of the opposite side or the pre-injury value. The athlete has to be able to perform all of the cutting maneuvers and specialty maneuvers that are required for the sport without having pain.

Leadbetter WB; Soft tissue Athletic Injury in: Sports Injuries Mechanism, Prevention, Treatment; Fu FH, Stone DA (eds). Lippincott Williams & Wilkins, Philadelphia 2001
STRESS FRACTURES and STRESS REACTION

Essentials of Diagnosis:

Pain with activity
Insidious onset
Point tenderness over fracture site
Radiograph initially are not diagnostic but may show sclerosis

Etiology:

Stress fractures of the tibia can commonly be seen in dancers and runners. There is a higher occurrence of tibial stress fractures in female athletes. Stress fractures around the foot and ankle most often occur in the metatarsal bones. The second or third metatarsals are the most common locations followed by the fifth metatarsal and the navicular. Other sites for stress fractures are the calcaneus and the cuboid.

In general, stress fractures are the result of chronic overload. This chronic overload can be due to anatomic predisposition (e.g. anterior bow of the tibia in dancers) or due to sports that elicit extreme deceleration or chronic deceleration forces in the tibia such as the bravura technique in ballet dancers. Stress fractures in the foot are often due to overly heavy gear (e.g. third
metatarsal “marching fracture”), faulty training routines or atypical foot alignment (e. g. short first ray), which may predispose the metatarsals to overload. Alterations in footwear, previous injuries and fractures as well as underlying health problems such as osteopenia, osteoporosis and metabolic disorders can lead to the occurrence of stress fractures around the foot. As preventative measures, the athlete should not overtrain and should use appropriate training technique. In ballet dancers the “Balanchine” technique that requires more fluid motion and very few jumps into “pose” puts the dancer at much less risk than the “bravura” technique.

The best footwear for the sporting activity should be used and may need to be customized in case of anatomic variation (e. g. high arch, flat foot, short first ray etc.). In case of suspected osteopenia or osteoporosis, a bone densitometry analysis should be performed.

Clinical findings:

Symptoms (History)

Athletes will have pain at the site of the stress fracture after exercise. Most commonly this is at the junction of the middle and dital third of the tibia on its medial side. If the stress fracture is in the foot, the athlete may notice swelling in after exercise and some
local point tenderness. For example, for a navicular stress fracture the “N” spot should be palpated. The pain typically appears during exercise. Often they will not be tender after initial activity but after a certain amount of exercise, the pain will set in. In fact, the pain may occur after a repeatable distance or time after exercise begins. Often the athlete is able to continue with his activity, however, with pain at the site of fracture.

Signs (Physical Examination)

It is crucial to do a full physical examination of the tibia and the foot. The tender spots need to be palpated to define the anatomic location of the injury. Navicular stress fractures for example typically hurt in the midportion of the navicular very close to the insertion of the tibialis anterior. In order to differentiate tibialis anterior tendinitis from a navicular stress fracture it is important to examine the tibialis tendon and its function and to directly palpate the outline of the navicular. For metatarsal stress fractures, it is important to inspect the plantar aspect of the foot for plantar calluses that may give a clue about improper force distribution in the forefoot.

Imaging:
A stress fracture is usually a diagnosis based upon clinical suspicion. Very often, the initial radiographs are negative. A stress fracture will be detectable on radiographs once the healing response and sclerosis at the fracture site has begun. This usually occurs 2 to 3 weeks after the onset of the symptoms. Radiographic changes are very subtle and consist of cortical thickening, trabecular sclerosis and possibly cortical defects. In the tibia the dreaded “black line” can be identified at a later stage, once the stress fracture has essentially become a non-union. For further imaging usually an MRI or a bone scan is required. We feel that a bone scan is the most sensitive test. It will show a hot spot right at the fracture site. The advantage is that it will also assess surrounding bones and point out possible other bones that are in danger. MRI scan is an excellent tool to evaluate the surrounding soft tissues as well as the involved bone. Typically, a stress fracture leads to edema at the fracture site, which can easily be visualized by MRI (Fig. 2, 3). Edema typically shows up as very low signal on the T-1 sequences and as a high-density area on the T-2 sequences. Inversion recovery images can be utilized to display more subtle stress reactions in smaller bones such as the anterior process of the calcaneus or the navicular. (Fig. 4, 5)
Treatment:

The treatment of stress fractures around the foot is largely non-operative. Stress fractures generally require immobilization in a hard soled shoe or cast for 6-8 weeks. Stress fractures of the forefoot (i.e. metatarsal fractures) can be treated in a hard soled shoe (e.g. cast shoe) or a removable boot with a rocking sole. Stress fractures of the tarsal bones usually require protected- or non-weight bearing for 6-8 weeks. Once the fracture has healed, a gradual return to activities can be allowed. Precautions should be taken to avoid the training errors or activities that led to the initial stress fracture. Very rarely a stress fracture will go on to non-union. The most notorious stress fracture for going on to a non-union involves the base of the fifth metatarsal fracture, known as the “Jones fracture”. This stress fracture occurs in athletes who jump a lot such as basketball, football or volleyball players. In high-level athletes, we tend to fix fifth metatarsal stress fractures with an intramedullary screw to allow faster return to play. In the average recreational athlete, however, the treatment of choice is activity modification and cast or boot treatment for 4 to 6 weeks.
If a stress fracture proceeds to become a non-union or a mal-union then the same rules apply as for acute fractures. Any rotational misalignment needs to be corrected. A painful non-union needs to be taken down operatively and bone grafted. Rigid fixation with an IM nail, screws or plates is then necessary.

Complications

The biggest complication of a stress fracture is to ignore the telltale signs and not be responsive to the athlete’s complaint. Non-operative treatment has certain risks that are associated with the casting treatment; include deep vein thrombosis and skin injuries. It is important to arrange periodic follow-up after cast treatment to inspect the soft tissues. Operative treatment has surgical risks and may results in painful hardware that may have to be removed once the fracture is healed.


Verma RB, Sherman O. Athletic stress fractures: part II. The lower body. Part III. The upper body with a section on
EXERTIONAL COMPARTMENT SYNDROME

Essentials of Diagnosis:
Dull ache in a tibial muscle compartment (usually antero lateral)
Insidious onset with activity followed by relief once activity is halted
No history of acute trauma
Reproducible onset of pain after a specific duration or intensity of exercise
Pain with passive stretch, numbness, weakness
Measurement of compartment pressures

Etiology:
While athletes, and especially runners, are predisposed to overuse injuries, exertional compartment syndrome must be differentiated from two other conditions. The first of these and the most common is the medial tibial stress syndrome. Typically, the athlete has distinct pain alongside the postero-medial border of the middle to distal third tibia. There are no sensory, motor or vascular anomalies. The distal one third of the tibia is tender
postero medially and the pain can be elicited with a forced plantar flexion against resistance. This syndrome was previously described as the classic “shin splints”. The prevention and therapy of this problem can be addressed with careful cross training, light stretching and a combination of initial rest and subsequent careful strengthening of the weak muscle groups.

The second entity that needs to be ruled out before a diagnosis of exertional compartment syndrome is made is a muscle strain in the medial gastro-soleus. Discussed earlier in this chapter, it may present in a fashion similar to the medial tibial stress syndrome.

Exertional compartment syndrome results from over-compression of the calf muscles during strenuous physical exercise. There are multiple muscle compartments within the calf. We consider it to have four major muscle compartments: anterior, lateral, posterior and a deep posterior compartment. Others separate the calf into as many as seven compartments. During exercise, the muscle compartment can undergo a normal volume increase of up to 20%. Most often, the involved compartment is the anterior compartment followed in frequency by the deep posterior compartment. Fascial membranes make up the compartment and provide the anatomic casing for the muscles lying within
its confines. As the muscle volume increases with exercise, it expands against the fascia, yet the fascia does not yield. Henceforth the intramuscular (intra-compartmental) pressure will rise. As long as this pressure stays short of a threshold that will not compromise blood flow and soft tissue integrity, the muscle functions within its physiologic capabilities and can recover. If the pressure rises beyond this physiologic threshold, the soft tissues are compromised. There is no preventive program. Clinical findings:

Symptoms (History)

Exertional compartment syndrome typically presents a dull ache or pain in the involved compartment. Patients typically describe the onset after a very specific duration or type of exercise. This onset is so reproducible that it has been given the eponym “third lap syndrome”. In 75-90% of patients, symptoms are bilateral usually with one leg being worse than the other is. The dull ache and discomfort typically remains for a certain duration after the exercise (minutes to hours) before it dissipates. In some patients, this may be accompanied by weakness, numbness or paraesthesias.

Signs (physical examination)
Immediately after exercise, the affected compartment may be tender or may feel significantly swollen. This, however, is usually only helpful if only one side is affected. In some patients muscle herniations can occur and be palpated. The finding of these herniations, although frequently present in patients with compartment syndrome, is usually incidental and has no diagnostic value.

Imaging

Imaging modalities can aid in ruling out the diagnoses of medial tibial stress syndrome, medial gastrocnemius rupture and stress fracture of the tibia. Radiographs in two orthogonal plains (i.e. AP and lateral) will usually show the periosteal stress reaction in posteromedial stress syndrome. It may or may not show a stress fracture. MRI scanning is very sensitive in evaluating edema around the calf. In case of a muscle injury, it will show a significant signal change with a high intensity in the T2 weighted image. A chronic compartment syndrome may show chronic scarring in the affected compartment. A bone scan will not be able to diagnose chronic exertional compartment syndrome but it may rule out a stress reaction or stress fracture.

Compartment Pressure Measurements:
The most helpful diagnostic tool for the diagnosis of chronic exertional compartment syndrome is the direct measurement of compartment pressures immediately after exercise in combination with the clinical findings. There are multiple different techniques to measure compartment pressures including a handheld pressure measurement device to the utilization of an arterial line and arterial pressure monitor.

The criteria that we use to diagnose a chronic exertional compartment syndrome are if any one of three criteria are met:

Pre-exercise compartment pressure equal or higher than 15 mmHg.

One-minute-after-exercise pressures above 50 mm Hg

5-minutes-post-exercise pressures above 15 mm Hg.

These measurements are not affected by age but may be affected by position. The correct position during the test is with the patient supine and the foot vertical.

Treatment:

Conservative treatment of a chronic exertional compartment syndrome is generally unsatisfactory. Attempts can be made with the use of NSAIDS and rest but usually the symptoms will not get better unless the athlete is willing to completely stop the activity that brings on the
symptoms. If the athlete wishes to continue the activity, operative intervention is the treatment of choice.

Surgical Compartment Release:

All involved compartments need to be surgically released with great care and adequate homeostasis. There are multiple different techniques described through a single incision or a two-incision technique. Care must be taken to identify the peroneal and saphenous nerve as well as the saphenous vein.

The results of operative compartment release have been consistently good, 90% have a complete recovery with no residual symptoms.

Surgical Complications:

Making the correct diagnosis of exertional compartment syndrome in the athlete with lower leg pain can be difficult. Successful treatment, of course depends on the correct diagnosis. Then, failure of treatment largely results from excessive scarring or incomplete compartment release, especially of the deep compartment. This may happen when the surgeon makes the skin incisions too small for cosmetic reasons. We explain to the patient that this is not a cosmetically pleasant procedure and cannot be done through small incisions. Five to 10% of patients have residual symptoms after surgery. Failure can also result
infection, scarring, nerve and vessel damage as well as recurrence of symptoms despite adequate compartment release.

Return to play:

After surgical treatment, the patient can start gradual strengthening and aerobic training as soon as the incisions have healed. The athlete should be able to return to a full exercise program 8-12 weeks after surgery.


ANKLE PAIN

Ankle injuries are among the most common injuries in athletes. Soft tissue sprains and strains as well as ligament ruptures make up the vast majority of all ankle injuries. Ankle sprains and ligament tears usually can be treated successfully with non-operative management and athletes recover quickly from these injuries. Some
pathologic conditions, however, can lead to chronic irritation and inflammation of the ankle and pose substantial problems over a prolonged period.

POSTERIOR TIBIAL TENDONITIS

Essentials of Diagnosis:
- Pain of the medial aspect of the ankle
- Pain with weight bearing exercise
- Usually follows an injury to the medial side of the ankle
- Pain is exacerbated with active inversion and eversion of the subtalar joint
- Often associated with a flat foot deformity

Etiology:

Posterior tibial tendinitis is a very rare occurrence in athletes under the age of 30. The vast majority of posterior tibial tendon problems occur in middle-aged athletes and particularly women. Posterior tibial tendon injuries rarely develop acutely. Usually there is a precipitating incident and subsequent slowly developing pain along the course of the posterior tibial tendon. This makes the tendinitis and subsequent rupture of the posterior tibial tendon a chronic disease process. Despite it being a rather rare problem for athletes it is
devastating should the tendon rupture since the therapeutic options are not ideal and usually lead to a dysfunction.

The posterior tibial tendon is predisposed to injury due to its critical zone of local hypovascularity combined with great mechanical stress acting on the tendon. The large distance between the posterior tibial tendon insertion and the axis of the subtalar joint provides a large lever arm that magnifies the stress on the tendon. Rapid changes in direction (i.e. cutting) and jumping activities place the highest stress on the tendon. Sports such as basketball, tennis, ice hockey and soccer predispose the athlete to posterior tibial tendon injuries.

Clinical findings:

Symptoms (History)

Athletes complain of medial-sided ankle pain, worse with activity. Night- or morning-pain indicates more severe injury.

Signs (physical examination)

Physical examination reveals medial-sided point tenderness just inferior and posterior to the medial malleolus. This pain is usually exacerbated by forced inversion or eversion against resistance. A single leg toe raise is usually not possible. Commonly, the athlete will have a flat foot. Athletes usually show up early in the
course of this disease because it is debilitating and usually not well tolerated for longer periods of athletic activity.

Imaging:

Posterior tibial tendinitis is a clinical diagnosis and does not require imaging to be diagnosed. However, it is useful to obtain an MRI for the purpose of documentation and to evaluate the integrity of the tendon and the success of treatment (Fig. 6). X-rays of the foot and ankle are utilized to rule out other pathologic entities such as an accessory navicular, stress fractures, degenerative joint disease, anterior tibiotalar impingement etc.

Ultrasonography is equally accurate in diagnosing tendinitis as well as a rupture of the tibialis posterior tendon and can be easily done in an office setting.

Treatment:

Non-operative treatment is successful in the majority of patients with posterior tibial tendinitis. The initial treatment follows the earlier described RICE principle. A reduction in the current training program as well NSAIDs is usually successful. In some athletes, a medial arch support may be helpful, particularly if they have a pronounced flat foot deformity. This treatment should be tried for 6 weeks.
If this is not successful then an immobilization in a cast or cast-boot should be tried for 4-6 weeks.

If no improvement of symptoms can be obtained by 4-6 months surgical, treatment options should to be entertained.

Surgical Treatment:

Operative treatment for posterior tibial tendinitis includes tendon inspection and a tenosynovectomy. The adjacent structures such as the anterior deltoid ligament and the immediately adjacent spring ligament need to be inspected for fraying and tears. If torn, they should be repaired. For more severe injuries, a tendon reconstruction using the flexor hallicus longus or the flexor digitorum longus tendons may be necessary. For severe and chronic injuries with a flexible hind foot, a medial slide osteotomy of the calcaneus may be needed in addition to the tendon reconstruction to rebuild the medial arch, however, this rare in athletes.

Return to play:

Return to play after non-operative treatment is guided by the absence of pain. The process takes usually 2-4 months before full athletic activity can be resumed. Post operatively a full ROM needs to be achieved and the return to 80% or more inversion strength and toe raise strength
should be obtained before return to full activities. This takes between 4 and 12 months depending on the magnitude of surgery done.

ANTERIOR TIBIOTALAR IMPINGMENT

Essentials of Diagnosis:

Differentiate between anterior and antero-lateral tibiotalar impingement

Usually insidious onset anterior ankle pain

Pain with forced dorsiflexion

Usually only present with activity, no rest pain

Local tenderness over anterior or antero-lateral ankle

Anterior tibiotalar impingement needs to be clearly divided into two separate entities:

- Anterior tibiotalar impingement
- Antero-lateral tibiotalar impingement

ANTERIOR TIBIOTALAR IMPINGMENT:

Etiology:

True anterior tibiotalar impingement was first described in 1943 as the “athlete’s ankle” and was subsequently described as “footballer’s ankle” in 1950. It was also described as “impingement exostosis of the tibia
and talus” in 1954. Athletes that perform sports requiring repetitive forced dorsiflexion of the ankle (i.e. soccer, football, dance, and gymnastics) report repetitive small sprains to the anterior ankle that they sustain in full dorsiflexion of their foot. These repetitive injuries lead to chronic sprains of the anterior ankle capsule and microtrauma to the anterior cartilage cap of the distal tibia. These micro-injuries lead to a continuous cycle of microtrauma, inflammation, scarring of the capsule, subsequent calcification and finally formation of bone spurs. Once the bone spurs grow large, they can directly impinge on each other and cause limited dorsiflexion. They may also fracture and cause the formation of loose bodies in the ankle.

Prevention:

Both anterior and antero-lateral tibiotalar impingement are chronic conditions that are the result of repetitive microtrauma and therefore are very difficult to prevent. Anterior shin guards extending over the span of the ankle have been tried on soccer players; however, the acceptance has been very low since these guards tend to interfere with the soft touch that these athletes require while handling the ball. Taping the ankle against maximally forced dorsiflexion as well as stretching and strengthening
exercises should be routine measures in any competitive athlete. The athlete may use local anti-inflammatory measures such as cryo-therapy if the ankle is sore or he/she has suffered a minor ankle sprain. This may prevent the vicious circle of chronic inflammation and scar formation early in the process.

Clinical findings:

Symptoms (History)

Patients most often present with a history of anterior ankle or midfoot pain radiating towards the lateral aspect of the ankle joint or the fibula. Initially this pain occurs after vigorous activity and dissipates soon after the activity is stopped. Gradually these symptoms, however, may appear with light or even daily activity and may not dissipate after the activity is stopped. Patients typically report difficulties with climbing stairs and squatting. They report stiffness of the ankle.

Signs (physical examination)

Physical examination may reveal marked tenderness over the anterior border of the tibia and sometimes over the dorsum of the talus when the foot is plantarflexed. A ridge may be palpable over the dorsum of the talus. Patients usually display reduced dorsiflexion and a tight heel
chord. The ligamentous examination of the ankle is usually within normal limits and does not show ligamentous injury. 

Imaging

Radiographs of the ankle show that the anterior margin of the tibia has lost its round contour. Sometimes a bone spur can be seen on the dorsal surface of the neck of the talus. These spurs can be fragmented and can be a source of loose bodies.

Treatment:

The initial treatment is non-operative with rest and NSAIDs. If this fails immobilization in a cast or cast-boot for 4-6 weeks should be tried. If the symptoms do not dissipate with modified activities and conservative treatment then an operative procedure should be considered.

Operative treatment addresses the bone spurs and aims at removing the anterior osteophyte on the tibia as well as the dorsal bone spur on the neck of the talus. This can be done in an arthroscopic or a mini-open technique. The arthroscopic technique may yield a faster return to activities and enables the surgeon to inspect the ankle joint and thus recognize concomitant pathologies such as osteochondral defects, loose bodies or scar tissue formation. Particularly for anterolateral impingement, we prefer the arthroscopic technique.
Post-surgical rehabilitation aims at restoration of motion and muscle strengthening. Once the surgical incisions have healed a gradual return to activities can be started. Full ROM of the ankle and 80%-100% of inversion / eversion and plantar flexion dorsiflexion strengths should be regained before return to competitive athletic activity.

ANTERO-LATERAL TIBIOTALAR IMPINGEMENT:

**Etiology:**

Antero-lateral tibiotalar impingement has a different underlying pathology. Diagnosed much less frequently antero-lateral tibiotalar impingement follows repetitive ankle sprains or chronic overuse during pivoting sports. Antero-lateral tibiotalar impingement can also follow non-displaced fibula fractures or ligamentous avulsions of the fibula. The underlying pathology is believed to be a chronic synovitis and thickening of the distal most portion of the anterior inferior tibio-fibular ligament complex (e.g. anterior syndesmotic band) as result of repetitive inversion injuries to the ankle. This scar formation was first described as a “meniscoid band” in 1950. Subsequently, a separate band was described as being within the distal aspect of the anteroinferior tibiofibular ligament that is separated, through a fatty-fibrous layer
to the rest of the ligament and can impinge on the talar
dome in maximal dorsiflexion of the ankle.

Prevention:

Prevention of antero-lateral tibitalar impingement
should follow the general principles of preventing ankle sprains, particularly inversion trauma to the ankle.
Taping as well as off-the-shelf or custom lace-up braces to provide restraints against forced inversion of the ankle as well as proper strengthening and stretching exercises are the best preventative measures for chronic ankle sprains.
High top athletic shoe-wear particularly for basketball players is another means of trying to protect the ankle against inversion injury.

Clinical findings:
Symptoms (History)

Antero-lateral anterior impingement is a chronic condition that needs to be considered in patients that have severe antero-lateral point tenderness and soreness for a prolonged period of time. Most importantly it needs to be differentiated from chronic instability symptoms. It usually is a diagnosis of exclusion and should not be made before all non-operative treatment options such as NSAIDS,
rest, ice, rehabilitation and modalities have been exhausted.

Signs (Physical Examination)

Patients typically have anterolateral pain with dorsiflexion and sometimes clicking with motion of the ankle. Antero-lateral impingement does not cause instability of the ankle.

Imaging:

Radiographs need to be obtained to rule out stress fractures or fracture of the lateral process of the talus. An MRI can be helpful in identifying the thickened synovial band in the anterolateral recess of the ankle joint. MRI can be helpful in differentiating antero-lateral impingement from chronic ruptures of the anterior talofibular ligament (ATFL). The “gold standard” for this diagnosis is ankle arthroscopy which shows a thickening of the anterolateral inferior band of the syndesmosis and a synovial fold in the antero-lateral recess. Sometimes there can even be a small meniscus causing the symptoms.

Treatment:

The initial non-operative treatment is identical to the treatment of anterior tibio-talar impingement. Operative treatment is an arthroscopic debridement of the synovial fold and the inferior anterior band of the
syndesmosis. Once this is resected patients usually have excellent pain relief.

Return to play:

Ambulation should be allowed immediately after surgery. Most importantly these patients need to undergo a vigorous rehabilitation program to regain their ROM. Since many of these patients have been deconditioned for a long period of time the coordination and strength of the ankle flexors and extensors as well as pronators and supinators needs to be trained and proprioceptive exercises should be part of the rehabilitation process. Return to play should be possible after a 6 week rehabilitation period.


ANKLE INSTABILITY

Injuries to the ankle are among the most common lower extremity injuries in sports. Overall there are as many as 23,000 ankle sprain in the United States each day. Women have slightly higher risk of suffering a grade 1 ankle sprain than men on a collegiate level. The recurrence rate after a lateral ankle sprain is high. In high demand sports such as basketball, the recurrence may be as high as 70%.

Etiology:

Lateral ankle sprains most commonly occur due to excessive supination of the rearfoot about an externally rotated lower leg soon after initial contact of the rearfoot during gait or landing from a jump. Excessive inversion and internal rotation of the rearfoot, coupled with external rotation of the lower leg, results in strain to the lateral ankle ligaments. If the strain in any of the ligaments exceeds the tensile strength of the tissues, ligamentous damage occurs. Increased plantar flexion at initial contact appears to increase the likelihood of suffering a lateral ankle sprain.

The ATFL is the first ligament to be damaged during a lateral ankle sprain, followed most often by the calcaneofibular ligament (CFL). After the ATFL is ruptured, the amount of transverse-plane motion (internal rotation)
of the rearfoot increases substantially, thus further stressing the remaining intact ligaments. This phenomenon has been described as “rotational instability” of the ankle and is often overlooked when considering laxity patterns in the sprained ankle. Concurrent damage to the talocrural joint capsule and the ligamentous stabilizers of the subtalar joint is also common with lateral ankle sprains. The incidence of subtalar joint injury has been reported to be as high as 80% among patients suffering acute lateral ankle sprains.

The cause of lateral ankle sprain may be an increased supination moment at the subtalar joint. An increased supination moment about the ankle moment could thus cause excessive inversion and internal rotation of the rearfoot in the closed kinetic chain and potentially lead to injury of the lateral ligaments. Individuals with a rigid supinated foot would be expected to have a more laterally deviated subtalar axis of rotation and a calcaneal varus (inverted rearfoot) malalignment, which could predispose those with a rigid supinated foot to lateral ankle sprains.

Some have questioned whether the peroneal muscles are able to respond quickly enough to protect the lateral ligaments from being injured once the ankle begins rapid inversion. The peroneal muscles are active before initial
foot contact during stair descent and landing after a jump. This preparatory activity, along with similar activity in the other muscle groups that cross the ankle, is likely to create stiffness in tendons before initial foot contact with the ground. If the peroneal muscles are to protect against unexpected inversion of the rearfoot, preparatory muscle activation before foot contact with the ground is necessary.

Structural predispositions to first-time ankle sprains include increased tibial varum and non-pathologic talar tilt, whereas functional predispositions included poor postural-control performance, impaired proprioception, and higher eversion-to-inversion and plantar flexion-to-dorsiflexion strength ratios. Further research into prevention programs based on these predisposing factors is clearly warranted.

Clinical Findings:
Symptoms (History)

Typically an inversion injury has occurred that can be associated with an audible “pop” or a click. The ankle typically becomes swollen, tender, and painful with movement and full weight bearing.

Signs (Physical Examination)
It is important that the examiner delineates the extent of the injury to determine if the patient injured one or multiple ligaments, tendons, bone or even nerves. Systematically the examiner should palpate the ATFL, the CFL and the posterior tibiofibular ligament (PTFL). The syndesmosis needs to be examined as well as the medial aspect of the ankle, the deltoid ligament and medial malleolus. The lateral malleolus should be palpated at its posterior border and tested for tenderness. Peroneal tendons and the base of the fifth metatarsal also need to be palpated. The clinician should stress the ATFL by performing an anterior drawer test. This may or may not be possible in the acute setting depending on the pain level. The test is performed with the patient sitting and the lower leg hanging freely. The examiner holds the heel and positions the foot in slight plantar flexion. Then the heel is directed anterior while the other hand pushes the tibia posterior. This test is compared to the uninjured side. A difference is positive and is considered pathologic. The ankle inversion test can be used to differentiate between the ATFL and the CFL. A forced ankle inversion is performed and a difference to the opposite side is recorded. The inversion tested in plantar flexion evaluates the ATFL. Tested in dorsiflexion the CFL is tested.
To examine the syndesmosis the Hopkinson’s syndesmotic squeeze test can be performed or a forced external rotation of the tibia can be performed. Pain with squeezing the fibula and tibia together approximately 10 cm above the joint and pain with forced external rotation of the tibia versus the talus (Keigler test) is suspicious. Tenderness at the inferior syndesmotic band in any of these tests is to be regarded as a syndesmotic injury until proven otherwise.

Imaging:

Radiographs are used to rule out a fibular fracture, anterior process of the calcaneus fracture, lateral or posterior process of the talus fractures, midtarsal fractures, osteochondral lesions of the talus or disruptions of the ankle mortise indicating a syndesmotic injury (“high ankle sprain”). MRI can be useful to evaluate for bone contusions and the ligament injury. (Fig. 7)

Treatment:

The initial treatment of an ankle sprain consists of the RICE principle. Additional modalities such as electrical stimulation or iontophoresis may be helpful adjuncts to reduce pain and swelling. Provided the injury does not involve the syndesmosis and there is no fracture, rehabilitation of the ankle sprain should be started as
soon as pain control has been achieved. The rehabilitation has to address three different aspects: ROM, strength and proprioception. Once this has been established and the athlete is pain-free with exercises the third phase is designed to bring the athlete back to sports specific drills and maneuvers such as cutting, jumping, running etc. When the patient returns to athletic activities a protective, lace-up ankle brace should be worn to reduce recurrence.

If patients report recurrent sprains and continue to be painful for a long period of time and continue to have swelling and instability, surgical treatment options may need to be considered.

Surgical Treatment:

A myriad of different techniques have been described for the repair of the AFTL and CFL. The most common operative technique today is probably the Gould modification of the Brostrom technique. This technique essentially repairs the ATFL and CFL directly and imbricates the extensor retinaculum over top of the direct ligament repair thus serving as a reinforcement of the ATFL and CFL repair. This technique has lead to excellent results in high-level athletes and dancers. Using this technique there is no need to harvest any other tendon
around the ankle as is required for many of the other ATFL and CFL reconstruction techniques. The Rehabilitation after the modified Brostrom procedure requires a cast or cast boot for about 6 weeks followed by ROM exercises and the formal rehabilitation program as outlined above.

Syndesmotic Injury:

If an injury to the syndesmosis is suspected, it is imperative that the syndesmotic integrity (i.e. ankle mortise) is scrutinized. Any increase in medial joint space, disruption of the mortise or widened gap between the fibula and the tibia is suspicious for a syndesmotic injury. These “high” ankle sprains are not uncommon. Up to 10% of all ankle sprains also have an involvement of the syndesmosis. They occur more frequently in high energy collision sports such as ice hockey, football and soccer.

If the athlete suffered a syndesmotic sprain the initial treatment is a cast or cast boot for a minimum of 2-4 weeks followed by a re-examination. If the tenderness at the anterior syndesmotic band persists, the cast treatment needs to be continued for an additional 2 weeks. Once the anterior syndesmotic tenderness has subsided, the rehabilitation protocol can be begun. It is important to know that athletes with a “high” ankle sprain will be sidelined significantly longer than athletes with a simple
ankle sprain. If the syndesmosis is disrupted a surgical repair and syndesmotic screw needs to be performed. This will require a cast and cast boot for 6-9 weeks followed by rehabilitation.

Return to play:

Simple ankle sprains can be treated with RICE and can return as soon as the athletes have no pain with their sports specific activities. ATFL tears should undergo formal rehabilitation and can return after the third phase of the rehabilitation has been concluded successfully. This usually takes 3-6 weeks. Patients with “high” ankle sprains will be sidelined for 4-12 weeks depending on the treatment necessary.


FOOT PAIN

ACHILLES TENDONITIS

Essentials of Diagnosis:
Onset of tenderness and swelling approximately 2–6 cm above the insertion of the tendo Achilles (TA)

Initial pain after activity, in later stage onset of pain during activity

Diffuse pain with palpation of TA

Pain over the insertion of TA with pressure and during night (insertional tendinitis)

Decreased dorsiflexion of ankle with tight heel chord

Etiology:

There are three different inflammatory entities of the TA that are closely related and require the same initial treatment regime:

• Achilles peritendinitis: inflammation of the para-tenon with or without degeneration of the TA

• Achilles tendinosis: inflammation and degeneration of the TA without involvement of the para-tenon

Insertional Achilles Tendinitis: inflammation and degeneration at the TA insertion with or without calcifications and
bone spur formation

A predominantly sedentary lifestyle followed by a sudden increase of physical activity involving walking, jogging and running in the mid ages (40-60 years of age) in combination with tight heel chords and decreased ROM of the ankle leads to TA injury. As a general precaution stretching of the TA combined with gradual increase in activities for elder athletes helps to avoid TA injury. Achilles tendinitis in high-level athletes is usually a sign of faulty training, improper running techniques, or overuse.

Clinical findings:

Symptoms (History)

Most patients complain of gradual onset of pain in the posterior calf approximately 2-6 cm above the insertion of the TA. Often this pain is accompanied with swelling. Initially the pain will appear after physical activity. This can change and the athlete may experience pain during physical activity usually indicating a worsening of the pathology. Insertional TA tendinitis present similarly with the exception that it often presents as night pain when the athlete rests the foot on the back of his heel while sleeping.
Signs (Physical Examination)

The diagnosis of achilles peritendinitis versus tendinosis can theoretically be made by evaluating the location of the point of maximal tenderness. In peritendinitis the entire paratenon is inflamed and therefore will not be affected by ankle ROM during the examination. TA tendinosis is a localized inflammation in which case ROM will lead to a migration of the point of maximal tenderness throughout the examination. It is important to rule out a tear of the Achilles tendon that can be done with the Thompson-test as described in the chapter on muscle strains.

Imaging:

Standard radiographs of the ankle may show some calcification along the TA. There may be a thickened soft tissue shadow visible. In case of an insertional tendinitis there may be calcifications anterior to the insertion of the TA (Fig.8). An MRI may be helpful to differentiate tendinitis from tendinosis. Tendinitis shows significant fluid retention within the tendon without hypertrophy of the tendinous tissue. This is an acute finding and can usually be treated with anti-inflammatory medication and RICE successfully. Significant hypertrophy of the tendon indicates replacement of tendon tissue with a fibrous scar
(Fig.9). This tendinosis can predispose the patient to a rupture of the TA.

Treatment:

All three achilles tendon injuries initially are treated equally. Initial treatment consists of non-operative management including NSAIDS and rehabilitation exercises such as stretching of the TA, strengthening of the TA and gastroc-soleus complex. If the patient has significant hindfoot varus or valgus a correcting orthosis may need to be issued. Modalities such as iontophoresis, electrotherapy are not proven in their efficacy but may be employed if pain relief can be obtained. Usually it is not necessary to treat patients in a cast immobilization. In rare cases this may be necessary for recalcitrant pain.

Steroid injections should not be given into the tendon or the tendon insertion since these can lead to an early rupture of the tendon making a primary repair difficult, if not impossible secondary to the degeneration that the tendon undergoes in response to the steroid injection.

Surgical Treatment:

Recalcitrant cases that have not responded to treatment in over 6 months may need a surgical debridement of the paratenon and the tendinosis. Using a slightly medially based skin incision the paratenon is incised and
debrided and the thickened tendon is thoroughly debrided. If more than 50% of the tendon is involved the plantaris tendon may be utilized to weave it into the defect to strengthen the repair. The tendon is repaired as well as the paratenon. In order not to tighten the paratenon too much, it can be released carefully on its anterior aspect thus allowing posterior closure without undue tension. A lateral approach is used for insertional tendinitis and the calcaneal bursa is excised. In some cases there is a prominent bony ridge of the posterior calcaneus (i.e. “Haglund’s deformity“) that may abut the tendon insertion. This bony ridge needs to be removed with an osteotome. Post-operatively the patient is put into a cast or cast-boot for 4-6 weeks. Weight bearing is usually allowed between 2 and 4 weeks and is followed by rehabilitation for 6 weeks.

Return to play:

Once the symptoms have subsided a gradual return to the former activities can be allowed. If symptoms return, the eliciting activity should be stopped immediately and a more gradual return has to be tried. After operative treatment the rehabilitation protocol is similar after the initial postoperative casting period.


HEEL PAIN

Essentials of Diagnosis:

Very common in runners and overweight athletes

Usually stabbing pain in the morning for first couple of steps

Medial calcaneal pain

Often associated with tight heel chords

Takes 1-2 years to resolve completely

Etiology:

Plantar heel pain has many medical names such as plantar fasciitis, runner’s heel, policemen’s heel, calcaneodynia or heel pain syndrome. It is one of the most
common problems in athletes. The differential diagnosis of plantar heel pain is often difficult and has to address various different anatomic sites. The spectrum ranges from systemic conditions such as Reiter’s syndrome or ankylosing spondylitis or rheumatoid arthritis to medial plantar nerve entrapment or plantar fibromatosis. Most commonly, however, it is the running athlete who presents with complaints about plantar heel pain. Higher intensity of training sessions, weight gain and return of overweight athletes to previous training schedules can be a reason. Furthermore there are certain risk factors that include high impact aerobics or prolonged daily walking on hard surfaces (i.e. construction workers, orthopaedic resident’s). In order to understand the underlying pathology one has to understand the anatomy and function of the plantar fascia.

The plantar fascia is a strong collagenous structure that originates on the anteromedial aspect of the calcaneus and inserts at the base of each proximal phalanx. The fascia is divided such that the flexor tendons can perforate the fascia to reach the toes. This results in 10 individual insertions of the plantar fascia. Overlying the plantar fascia is the plantar fat pad that is approximately 2-3 cm thick.
The biomechanical function of the plantar fascia is a continuation of the Achilles tendon around the calcaneus resulting in the “windlass” mechanism. This mechanism enables the foot to stabilize itself in midstance due to a tightening of the longitudinal foot arch.

The tibial nerve splits into its final branches at the level of the medial malleolus. Particularly the first branch of the lateral plantar nerve, the posterior branch or “Baxter’s nerve” can be a source of pain if it gets trapped between the abductor hallucis and the quadratus planti muscle.

Clinical findings:
Symptoms (History)

Patients will report that they have sharp stabbing pain with the first steps in the morning. The pain eases during the day and towards the evening the entire heel is sore.

Signs (Physical Examination)

Physical examination has to address the underlying pathology and starts with an evaluation of the gastrosoleus and TA complex. Almost all patients with plantar fasciits have a tight heel chord and lack dorsiflexion up to, or past neutral. Furthermore, the forefoot needs to be evaluated. A pronated or plantar flexed first ray can lead
to plantar fasciitis in itself. Typically patients have palpable pain directly anteromedially on the plantar surface of the tuber calcanei. If a nerve entrapment is suspected there should be a positive Tinel’s sign over the medial aspect of the heel just beneath the medial malleolus. Furthermore a careful palpation of the plantar fascia should be performed to rule out single or multiple plantar fibromata.

Imaging:

Radiographs do not usually show the pathology. There may be a “bone spur” along the anterior aspect of the calcaneus, however, this bone spur actually arises within the aponeurosis of the flexor digitorum brevis and has no involvement in the development of plantar fasciitis. An MRI scan can be helpful in detecting abnormalities of the plantar fascia. It will show increased fluid uptake in the T2 weighted images along the anteromedial border of the plantar fascia (Fig.10). It may also show a plantar fibroma or a neuroma of Baxter’s nerve. An MRI scan may also point out occult stress fractures that could be the cause of plantar foot pain such as an anterior process of the calcaneus fracture.

Treatment:
The treatment for plantar fasciitis is focused on addressing the underlying disorder. The patient needs to be counseled so that they understand that this condition is self-limiting and can take up to a year until it is successfully treated. The chronic tightness of the plantar fascia leads to a contracture of the fascia during sleep. The first steps in the morning stretch this contracted fascia and cause micro-tears that subsequently scar during the next rest period until the plantar fascia has eventually elongated to the point that no more micro-tears occur. In order to break this cycle the first line treatment is stretching of the plantar fascia and the Tendo Achilles. A short heel chord leads to an overuse of the windlass mechanism and consequently results in over tightening of the plantar fascia. Patients therefore should be instructed to stretch the TA multiple times during the day. In addition they can be provided with night splints that prevent the contraction of the plantar fascia and keep the foot in neutral dorsiflexion during sleep. Furthermore, heel cups help to cushion the hard impact on the heel. There is some debate about the usefulness of full arch supports. It has been shown that full arch supports work equally as well as simple heel cups as long as they are utilized in conjunction with stretching exercises. In
severe cases of plantar fasciitis it may be helpful to treat the patient with a cast or cast boot for a couple of weeks to rest the plantar fascia before a rigorous stretching program should be started.

We discourage steroid injections into the plantar fascia. The literature provides conflicting data on the success of steroid injections, however, there is clear evidence that steroid injections have a high risk of a tear of the plantar fascia that results in a catastrophic flat foot deformity that is essentially not correctible. If the source of pain is entrapment of Baxter’s nerve an EMG study should be obtained. If the EMG and nerve conduction studies suggest a nerve entrapment the first line of treatment is orthotics that correct any present foot deformity (over-pronation, pes planus, pes planovalgus etc.)

Surgical treatment:

Surgical release of the plantar fascia is reserved for very rare severe cases of plantar fasciitis with intractable pain for more than 1 or more years. Various different techniques have been described to release the plantar fascia. Important is that this has to be done under direct visualization. The medial aspect of the plantar fascia needs to be partially excised. A complete release
will result in a deficiency of the plantar fascia and an uncorrectable flatfoot deformity. If a release of a plantar fascia is performed it is prudent to include a release of baxter’s nerve. This is important for two reasons. First the nerve needs to be visualized in order not to cut it and secondarily there may be an element of both pathologies that can easily be addressed through one approach.

Return to play:

Once the athlete is pain-free they can return to their previous exercise program. Any running athlete is advised to maintain the stretching program in their warm up routine.


TURF TOE

Essentials of Diagnosis:
Mechanism of injury (hyper-plantar / hyper-dorsiflexion of 1st MTP joint)
Tenderness with ROM of 1st MTP joint
Swelling
Pain with weight bearing

Etiology:
Sprains of the first MTP joint have been described as “turf toe” injuries since athletic competition on artificial turf was noted to result in an increased incidence of soft tissue injuries to the great toe. Injuries to the first MTP joint occur in football and soccer but can happen during any athletic activity that forces the first MTP joint into hyper-plantar or hyper-dorsiflexion. Injuries to the first MTP joint are by no means benign and carry a significant short and long term morbidity. Despite the usual perception of a “trivial” injury overall estimates of loss of playing time rank 1st MTP joint injuries at the same level as ankle sprains even though they occur far less frequently.

The mechanism of injury is either a hyper-dorsiflexion injury that usually happens when the foot is planted and the first MTP joint is maximally dorsiflexed. The dorsal edge of the proximal phalanx is cocked against the
articulat

capsuloligamentous structures are maximally stretched in
this position. If the first MTP joint is forced into more
hyper-dorsiflexion it will lead to a structural failure of
either the volar capsule, the collateral ligament or a
fracture of either the dorsal phalanx or the metatarsal
head. The classic situation of this occurring is the
offensive lineman who plants his foot for maximal traction
and another player falls onto his heel forcing his forefoot
into more hyper-dorsiflexion. The other mechanism for turf-
toe is the foot being in maximal plantar flexion with the
first toe pointed and the foot is struck from behind
driving the first MTP joint even more hyper-plantarflexion.

In order to prevent these injuries the first MTP joint
can be taped to limit dorsiflexion and plantarflexion. The
variability in ROM of the 1st MTP joint is great. Normal ROM
can range from 3-43 degrees of dorsiflexion to 40-100
degrees of plantar flexion. Particularly individuals with a
naturally limited ROM are at risk for “turf toe” injuries
and should be taped. There is some evidence that the use of
an orthotic (e.g. Morton extension) or a 0.51-mm spring
steel insert may help prevent these types of injuries.

Clinical findings:
Injuries to the first MTP joint vary widely. The spectrum of injury ranges from a simple sprain to complete avulsions of the dorsal or volar plate with or without associated fractures of the metacarpal head or base of the phalanx. In addition the sesamoid bones can be involved in the injury if the flexor tendons are part of the injury.

Symptoms (History)

The trainer or physician on the sideline must have a close eye on players who come off the field with a limp. Often turf toe injuries are treated as a minor injury by players. This leads to prolonged recovery and problems later on. Patients complain of first MTP joint pain and difficulty pushing off the injured foot.

Signs (Physical Examination)

The physical examination of the first MTP joint should include active and passive ROM. The examination has to be compared to the opposite side and it needs to be noted if either active and passive ROM is painful. The normal examination should be painless. Pain at the extremes of ROM may indicate if the injury is volar or dorsal. Strengths of the FHL as well as the extensor hallucis longus need to be tested to rule out an avulsion injury. Stability of the 1st MTP joint in valgus and varus stress also needs to be evaluated to rule out collateral ligament damage.
Classification

A classification system has been designed that is helpful to assess the severity of the damage.

Grade 1 sprains represent stretch injuries:
Localized pain
Minimal swelling, little ecchymosis
ROM mildly limited with minimal pain
Minimal pain with weight bearing

Grade 2 sprains represent a partial tear of the capsuloligamentous complex:
More intense tenderness and diffuse pain
Moderate swelling with ecchymosis
Moderately decreased ROM
Mild to moderate pain with weight-bearing
Athlete limps clearly

Grade 3 sprains are complete disruptions of the capsuloligamentous complex with or without bony avulsions and osteochondral fractures:
Very intense and diffuse tenderness around entire MTP joint
Sever swelling and ecchymosis
Very limited ROM very painful
Inability to ambulate normally
Once the clinical diagnosis of a “turf toe” injury is made further diagnostic tools may be needed to clearly delineate the severity of the injury. We feel that any turf toe injury that is significant enough to cause pain with ROM should be evaluated with radiographs. Further diagnostic tools should be used if the severity of the injury is suspected to be higher.

Imaging:

Radiographs reveal avulsion fractures, osteochondral incongruencies in the metatarsal head or base of the phalanx, migration of the sesamoids, widening of bipartite sesamoids or subchondral bone resorption that can be seen with chondral injuries. If there is a suspicion for a collateral ligament or a purely ligamentous volar / dorsal plate injury stress radiographs should be performed in valgus, or maximal plantar / dorsiflexion of the MTP joint. If a stress fracture of a sesamoid is suspected a bone scan can be obtained. An MRI can be used for diagnosis of ligament avulsions, particularly of the volar plate (Fig. 11).

Treatment and Return to play:

Treatment of “turf toe” injuries is primarily non-surgical. The initial treatment protocol follows the RICE principle followed by cryo-therapy during the first 48
hours after the injury. The most important factor for the rehabilitation of these injuries is rest until painless ROM is obtained. For simple grade 1 sprains without any structural damage the athlete can usually return to light stretching and functional rehabilitation within the painless ROM. Taping and toe spacers can be used to counter the initial injury mechanism. Once the athlete is pain free return to play is possible.

For more severe grade 2 sprains athletes may miss between 5 and 14 days of training and game time. The treatment follows the same rules as for grade 1. It is imperative that the athlete is pain free before he / she returns to play.

For grade 3 sprains the treatment depends on the anatomy of the injury. The initial treatment is the same as for grade 1 and 2 sprains. Athletes often require crutches for a couple of days to immobilize the toe. After a grade 3 injury, an athlete may need between 4 to 8 weeks to return to play. These injuries can be career ending, so it is not desirable to push a return to play until the athlete is completely pain free throughout all required drills.

Particularly in grade 3 injuries, surgical treatment may be necessary. While capsular avulsions and collateral ligament injuries usually heal with non-operative
treatment, fractures, osteochondral avulsions or non-reducible dislocations need to be addressed surgically. Late sequelae such as osteochondral non-unions, sesamoid non-unions, loose bodies or late acquired deformities such as hallux varus or rigidus almost always require surgical intervention if conservative measures have failed.


Watson TS, Anderson RB, Davis WH. Periarticular injuries to the hallux metatarsophalangeal joint in athletes. Foot Ankle Clin. 2000 Sep;5(3):687-713
Figures:

1. Medial gastrocnemius strain (MRI)
2. Tibial stress fracture (plain film and MRI)
3. Distal tibial stress (MRI)
4. Anterior process of calcaneus and cuboid stress fracture (MRI)
5. Navicular stress fracture (MRI inv. recovery)
6. Posterior tibial tendinitis (MRI)
7. Rupture of ATFL (MRI)
8. Achilles tendonopathy (plain film and MRI)
9. Achilles tendonopathy (MRI)
10. Plantar fasciitis (MRI)
11. Turf toe (MRI)

Figure legends:

Fig.1: T2 weighted MRI showing edema in the medial gastrocnemius (white arrow). This edema is the response to the muscle strain.

Fig. 2: T2 weighted MRI image showing edema in the tibia. The coronal section shows the typical medial location of the stress fracture (left arrow). The axial cut shows the marrow edema that accompanies the fracture and healing response (right arrow).
Fig. 3: T1 and IR weighted MRI image of distal tibia in a female who started high impact aerobics 4 weeks prior to the exam. The distinct edema in the distal tibia (T2) as well as the black line in the IR image indicates the stress fracture. This patient had this finding bilaterally.

Fig. 4: T1 and T2 weighted MRI image of the mid-foot showing a stress fracture of the anterior process of the calcaneus and the cuboid. The left image is the T1 weighted image that shows some blood and cortical irregularity in the anterior calcaneus (arrow). The right image shows the typical edema that accompanies stress fractures in the anterior calcaneus and the cuboid.

Fig. 5: Inversion recovery MRI image of a navicular stress fracture. This may be a subtle finding on a T2-weighted image so special MRI techniques such as inversion recovery may be required.

Fig. 6: T1 weighted MRI images of the hindfoot. The left image shows a normal hindfoot with the posterior tibial tendon, the FDL and the FHL. The tendons appear dark and do not show fraying or degeneration. On the right, there is significant fraying and even a tear in the posterior tibial tendon (arrow). The FDL and FHL look normal but there is
some fatty degeneration inside the tendon sheaths (white areas within tendon sheath).

Fig. 7: Inversion recovery MRI image of the ATFL. On the left, the ATFL (arrow A) is intact. It is visible as a strong dark band. On the left, (arrow B) it is disrupted, disorganized and not visible as a collagenous dark structure.

Fig. 8: The left image shows a plain radiograph with an increased widened soft tissue shadow along the TA (fat arrow). In addition, this patient has calcifications on the TA insertion (dotted arrow) and a Haglund’s deformity (dashed arrow). On the T1-weighted axial MRI image one can easily appreciate a very thick inhomogenous TA (arrow).

Fig. 9: This T1-weighted image of the ankle shows a sagittal image of Achilles tendonopathy. The arrow marks the area of hypertrophic scarring.

Fig. 10: These MRI images show a sagittal image of a T1-weighted image of a normal foot on the left (A). The T1-weighted image in the middle (B) shows a calcification inside the plantar fascia (arrow). The T2-weighted image on the right (C) shows that these calcifications are inside a zone of edema in the plantar fascia signifying plantar fasciitis.
Fig. 11: These T1- and T2-weighted images of a turf toe injury show the disruption of the plantar plate at the first MTP joint (arrows).