OSSEOUS PATHOLOGY ABOUT THE ANKLE

D. Richard DiNapoli, D.P.M.

Introduction

The last decade has witnessed an explosion in the number of people involved in athletic activities. The fitness phenomena involves people of all ages and activity levels from the weekend novice to the world class athlete. Paralleling this is a rise in the number of injuries and physician awareness of these injuries. The biggest contributing activity is running. The majority of conditions that affect the runner are secondary to repetitive microtrauma. This is often compounded by errors in training, previous injury, faulty biomechanics, age and hereditary predisposition. The ankle and rearfoot complex represent an important link in the lower extremity system. The limited soft tissue coverage allows for direct palpation of the underlying osseous structures but also predisposes the ankle to blunt trauma.

The focus on this chapter is on osseous conditions that may afflict the runner. Such conditions may develop as primary conditions or secondary to a previous injury. The variety of symptoms, diagnostic strategies, conservative and surgical treatments will be reviewed.

Osteochondral Dome Fractures of the Talus

The talus is almost wholly articular in structure. Consequently its surfaces are extremely vulnerable to injury. The osteochondral or transchondral dome fracture is most descriptive for the articular surface insults sustained by the trochlear surface. This condition enjoys a fair amount of controversy regarding etiology and mechanism of injury.

Kelikian lists a number of synonyms for this condition: flake fracture, scale dome, osteochondral and transchondral fracture. He states that this is a most common fracture of the talus and that it is a totally separate entity from osteochondritis dissecans. He also states that the fracture consists of a sliver of bone coated with cartilage which was precipitated by a traumatic event. The lesion is located at the anterolateral angle of the trochlear surface adjacent to the fibular malleolar articular surface.

This opinion is opposed by the writings of Berndt and Hardy who postulated that all fractures of the talar dome were due to trauma. They described two primary lesions, medial posterior and lateral anterior on the trochlear surface. In addition they performed cadaveric experiments and proposed etiological mechanisms for both lesions. The pathological events included: 1. trauma leading to fracture, 2. development of avascular necrosis of the osseous portion of the fracture fragment while the cartilaginous portion survived via nutrition from the synovial fluid and low metabolic demand, 3. This was followed by union of the fracture via creeping substitution or sequestration and nonunion of the fracture fragment with resulting osteoarthritis of the ankle joint.

Berndt and Hardy described four stages to the injury based or radiographic appearance.

- Stage I. A small area of compression of subchondral bone
- Stage II. A partially detached osteochondral fragment
- Stage III. A completely detached osteochondral fracture fragment remaining in the defect
- Stage IV. A displaced osteochondral fracture fragment

The lateral lesions of the middle one-third of the trochlear surface were believed to be produced by inversion and strong dorsiflexion. Medial lesions involving the posterior one-third of the medial border of the trochlear surface were caused by inversion, plantarflexion of the talus with external rotation of the leg.

Since the publication of Berndt and Hardy in 1959, many other authors have reported difficulty in detecting occult lesions, utilization of specialized diagnostic techniques, conservative and surgical treatment protocols and finally long term results.

Clinical Syndrome and Diagnostic Techniques

There is no clear clinical picture that identifies this syndrome. The injury may be acute or chronic in nature. The acute injury is usually an inversion type ankle injury with the lateral lesion having a greater incident of antecedent trauma. A commonly associated injury includes damage to the lateral collateral ankle ligaments. The acute phase of this injury may not resolve for three to six weeks and symptoms may persist for several months. The stage I injury

presents a diagnostic dilemma due to lack of a defined clinical syndrome. Ankle joint motion is usually full without marked pain or crepitance. However, the patient will complain of deep aching pain greatest after activity that responds somewhat to ice and anti-inflammatory agents. A possible reason for the difficulty in detecting grade I lesions may be due to the lack of sensory nerve distribution within the cartilage and lack of injury to the collateral ligaments in a mild sprain.

It is possible to develop a chronically painful ankle as the result of an osteochondral dome fracture. The chronic pain begins once the acute phase subsides. Patients who may have sustained a grade II lesion will generally exhibit more pain in the acute episode. The likelihood of developing a chronically painful ankle increases with each injury. These individuals may exhibit swelling of the ankle with activity, possibly experience a clicking sensation. The patient usually sustains more damage to the collateral ankle ligaments than to the talus. The ankle joint range of motion may be limited due to traumatic synovitis.

Diagnosis of stage III and IV lesions is usually made in the face of acute trauma. The patient presents with a severely painful, edematous ankle with possible lateral ankle ligament disruption.

The neglected or undiagnosed injury is more likely to have a painful, crepitant, unstable ankle with readily identifiable radiographic signs of injury.

Standard radiographical views of the ankle will determine the stage and extent of the injury. A ten degree internal rotation of the standard anteroposterior ankle view will clearly visualize the lateral aspect of the talar dome for identification of the lesion. The lateral view will reveal the size of the lesion in the anteroposterior direction. This view assists the surgeon in deciding to make an anterior or posterior approach. Other radiographic techniques such as tomograms or CAT scans may prove useful.

Another procedure that has been quite beneficial in diagnosing stage I lesions and evaluating small intra-articular loose bodies is the use of air as a contrast media. Arthrograms and double contrast studies (air and radiopaque dye) may also be of some value in differentiating and identifying these lesions.

Treatment

Treatment of these injuries is either conservative or surgical. The goal of therapy should be relief of symptoms rather than healing of the lesion.

In a recent long term followup paper several conclusions were presented.

1. A delay in diagnosis and treatment produced poor results if the injury was stage III or IV.
2. The fracture type appears to be the most important variable in the prognosis.
3. In cases not responsive to conservative treatment, surgical procedures performed within one year of the onset of symptoms showed the best results.

Conservative therapy, in general, consists of immobilization, supportive therapy, physiotherapy, orthosis, casting for a period of 4 to 12 weeks.

Recommendations for stage I and II are cast immobilization for a period of 4-8 weeks. Stage III lesions should be treated with cast immobilization for 4 to 8 weeks. If symptoms persist surgical resection within one year of the onset of symptoms. Stage IV lesions should be treated by excision of the lesion with drilling of the subchondral bone followed by early protective movement to stimulate fibroblastic proliferation within the defect.

Occasionally, large fragments may be amenable to open reduction with rigid interfragmentary fixation. Surgical approaches are either lateral or medial. Often times the medial approach may involve an osteotomy of the medial malleolus followed by rigid fixation.

Os Trigonum and Posterior Talar Process Fracture

Acute or chronic posterior ankle pain in the running athlete can produce a diagnostic challenge. The normal and pathological anatomy of the posterior talus will be reviewed in relation to ankle function. Differentiation of the os trigonum, acute and chronic fracture of the posterolateral process will be discussed. The clinical syndrome, differential diagnosis, radiographic appearance, and treatment methods will be addressed.

The posterior margin of the talus is bounded by the medial and lateral posterior processes or tubercles. The lateral tubercle is the larger of the two. Together they form a fibro-osseous groove for the tendon of the flexor hallucis longus.

The medial process is the smaller of the two. It has attachments to the posterior portions of the deep fibers of the deltoid ligament. The lateral process carries the eponym of Steida's process and has attachments to the posterior talofibular ligament and posterior talocalcaneal ligament. Inferiorly it articulates with the posterior facet of the calcaneus and superiorly with the tibia.

The os trigonum is one of the 21 documented accessory bones known to occur in the foot. This is believed to be a secondary ossification center of the talus that fails to unite or forms a fibro-cartilagenous bar or is separated from the talus prior to ossification. In approximately 7% of the population it persists as a free ossicle and is termed a true os trigonum.

Clinical Syndrome and Differential Diagnosis

Patients who complain of posterior ankle pain may have a history of inversion ankle injury; prolonged downhill running, stadium step training, or participation in an activity that causes forced plantarflexion of the foot on the leg. These individuals very often have pain with and after activity, retromalleolar edema and ecchymosis and point tenderness with pain upon palpation anterior to the tendo Achilles. The patient may complain of pain with active and passive range of motion of the ankle and flexor hallucis longus. Usually their condition will be aggravated with jarring motions, stair climbing, and ambulation on uneven surfaces

There are many conditions about the ankle that can produce acute and chronic pain including: lateral ankle instability, tendinitis, or partial rupture of the tendo Achillis, Achilles tendinosis or bursitis, osteochondral dome fracture, peroneal subluxation, lateral ankle impingement syndrome, malleolar fracture, and entrapment neuropathy.

Classification

There are four types of posterolateral process of the talus visible on a lateral radiograph. This classification assists in simplifying and distinguishing these disorders.

Classification of Posterior Tubercle

Type 1. Normal lateral process; anatomical norm associated with few clinical symptoms.

Type 2. Enlarged posterior lateral process (Steida's process). This enlarged process may fracture as the result of a compression mechanism between the calcaneus and posterior tibial malleolus or during inversion ankle trauma.

Type 3. True os trigonum. May become irritated as the result of an acute injury or chronic microtrauma.

Type 4. Os trigonum attached via a cartilagenous band to the talus, which may be disrupted with an acute plantarflexion injury.

Note: In the event of a fractured Steida's process which goes on to nonunion, the free ossicle may become mobile. Mobility may cause remodeling of the fracture margins so that they take on a smooth appearance. This may become radiographically indistinguishable from a true os trigonum.

Diagnosis

Posterior ankle pain may be associated with any number of clinically different disorders. Evidence of a free os trigonum on a lateral radiograph is by no means diagnostic of a fracture.

The posterior ankle can be readily visualized using a standard lateral ankle projection. Sharp jagged cortical margins are a suggestive of a posterior process fracture. Smooth cortical margins are normal for a true os trigonum or long standing Shepard's fracture.

Occasionally the need arises to perform dorsal and plantar flexion stress radiography or fluoroscopy to observe the freely mobile ossicle.

Other modalities such as tomography or CAT scan may be of some assistance for a greater appreciation of the exact size and configuration of cortical margins. Triphasic bone scans may also be employed as a diagnostic aid to determine a site of symptomatology in recalcitrant cases of posterior ankle pain.

Treatment

Acute cases of fracture of the posterior process or acutely symptomatic os trigonum are initially treated by 4 to 6 weeks of below knee casting.

In chronic symptomatic cases of os trigonum unresponsive to casting, a short course of injectable steroid may be indicated.

Should conservative measures appear inadequate surgical excision is recommended. This has proven to be quite predictably successful with minimal complications. Patients are usually able to resume full activity in 4 to 6 weeks.

Lateral Talar Process Fracture

Acute or chronic persistent pain about the lateral aspect of the ankle may be due to overt or occult fractures of the lateral process of the talus. Lateral process fractures constitute approximately 25% of all talar fracture types. The injury is very often misdiagnosed at the time of initial trauma. Neglected fractures of the lateral process result in degenerative joint disease of the lateral ankle and underlying subtalar joint.

Anatomy, Mechanism of Injury, Classification

Recall that the talus is almost wholely articular. The lateral process is the lateralmost aspect of the body of the talus and is covered with hyaline cartilage. The articular surface is triangular in outline with its base dorsal and apex distal corresponding to the medial articular surface of the fibula. The underlying surface of the lateral process is intimately associated with the posterior facet of the talus and forms the superior portion of the posterior subtalar joint. The lateral process is partially restrained by the capsular ligaments of the lateral ankle and subtalar joint. Fractures of the lateral process are bi-intra-articular fractures.

The proposed mechanism of injury involves forced dorsiflexion and inversion of the rearfoot resulting in tangential,

compression and shearing forces on the lateral process. Three stages of this fracture have been described consisting of fissure (stage I), fracture with displacement of the lateral process (stage II), fracture with subtalar joint dislocation (stage III). The fractures are often comminuted and displaced. In addition to standard ankle views, a medial oblique view offers clear visualization of the lateral ankle joint.

Clinical Syndrome

The acute traumatic injury presents with symptoms very similar to a severe lateral ankle injury. There will be moderate to severe pain and swelling. The development of ecchymosis may be delayed compared to a lateral ligamentous disruption. Point tenderness will be just distal to the fibular malleolus at the lateral talar process. The patient will have pain on range of motion of the subtalar and ankle joints.

Old cases of chronic persistent pain require a high degree of clinical suspicion. Standard radiographs may not reveal evidence of fracture and more sophisticated techniques such as CAT scan, tomogram, or even bone scan may be required.

Treatment

In the acute case where the fracture is well aligned, a below knee non weight-bearing cast for 6-8 weeks should be used. If the fracture is severely comminuted or there are large fragments, open reduction with internal fixation or excision of small fragments should be performed.

The chronically painful injury with exostosis formation may respond well to an arthroplasty of the lateral ankle joint and subtalar joint followed by casting for 4 to 6 weeks.

Impingement Syndromes

The presence of fibro-osseous prominences about the ankle create a limitation of motion, pain, and decreased performance by the running athlete.

The formation of exostosis about the ankle joint complex is the result of repeated microtrauma. The athlete that requires forced dorsiflexion and plantarflexion commonly develops marginal exostosis of the anterior and posterior aspect of the distal articular surface of the tibia. Runners and other athletes that have a history of ankle ligament instability or previous ligamentous injury develop moments of rotary instability resulting in marginal exostosis of the medial and lateral talar neck. In addition many runners develop degenerative changes in the ankle from biomechanical abnormalities. The degenerative changes and inflammation result in chronic synovitis, limitation of motion, and referred pain.

A variety of impingement syndromes exist and have been described in both the podiatric and orthopedic literature. They are usually designated for their radiographic appearance and referred pain. The most common are: anterior impingement syndrome, anterolateral corner compression syndrome, marginal exostosis, and osteophytosis.

Patients commonly present with diffuse pain about the ankle that has had an insidious onset and gotten progressively worse over time. Patients often complain that the pain is increased after activity, and may even require restriction of some activities. Occasionally they have a history of inversion ankle and foot injuries and relate some component of ankle instability albeit psychological. Other factors that contribute to the exostosis formation and exacerbation of symptoms include heel valgus or varus, pathological rearfoot pronation, and gastrocnemius equinus.

Standard radiographs of the ankle and rearfoot delineate the majority of these syndromes well. In cases of anterior ankle impingement charger views (stress dorsiflexion) will help determine if the osseous formation is blocking motion. Lateral ankle joint pain (fibulotalar articulation) may be secondary to old injury of either soft tissue or bone. Occasionally additional radiographic studies such as CAT scan or tomograms may be warranted.

Treatment

Treatment is conservative initially consisting of oral anti-inflammatory agents, ice massage, orthosis, and wedging of the running shoe. A course of injectable steroid in combination with other conservative methods of treatments and reduction of activity should decrease the inflammatory response and symptoms.

Indications for surgery include elimination of pain and deformity and improvement of function. In general arthroplasty procedures are followed by aggressive physical therapy.

Sinus Tarsi Syndrome

Occasionally runners will present with persistent pain about the lateral aspect of the rearfoot that is referred to the area of the sinus tarsi. The symptoms are insidious in onset and may be extremely persistent lasting for months. This condition is aggravated walking or running on uneven surfaces and may have some component of rearfoot instability. This syndrome is frequently described as occurring after inversion injuries of the foot and ankle. Another unrecognized etiology is excessive rearfoot pronation. As the talus adducts during pronation it slides off the sustentaculum of the calcaneus allowing the lateral process of the talus to jam into the lateral aspect of the sinus tarsi. The result is chronic inflammation of the synovium and capsule of the subtalar joint.

Clinically there is sharp pain to palpation at the sinus tarsi that increases with inversion of the calcaneus and adduction of the forefoot upon the rearfoot. Adolescents with tarsal coalition will present with similar symptoms but will have radiographic changes.

A distinguishing feature of this syndrome is the cessation of pain with the infiltration of a small amount of local anesthetic into the sinus tarsi. The patient may experience relief for several weeks following the initial injection. Standard radiographs will reveal normal joint contours with no soft tissue changes.

Subtalar inversion stress views are usually negative. Subtalar arthrography may reveal the formation of micro recesses of the anterior joint capsule that may account for some of the symptomatology but is not diagnostic. Electromyograms of the peroneal musculature and histological studies of the sinus tarsi have revealed only nonspecific changes.

Treatment

A variety of modalities are utilized in conservative therapy such as whirlpool, ultrasound, strapping, oral anti-inflammatory medication, and below knee casting. More aggressive treatment can be accomplished through a series of short acting steroid and local anesthetic injections. Biomechanical control of pathologic motion is equally as important.

Cases resistant to conservative therapy may resolve by evacuating the entire contents of the sinus tarsi including the talocalcaneal interosseous ligament. A more radical approach would be subtalar joint fusion.

Calcaneal Avulsion Fracture and Anterior Process Fracture

The generation of an avulsion fracture from calcaneus is usually the result of a severe forced inversion injury of the foot. The fracture is the result of the extensor digitorum brevis muscle belly stretching beyond its physiologic limits. The fracture fragment is located at the distal medial aspect of the superior surface of the calcaneal body. The bifurcate ligament and interosseous ligament are in close relation to the origin of extensor digitorum brevis and must be considered possible etiological agents.

Physical exam of the acutely injured foot will reveal marked swelling about the rearfoot and ankle with loss of the normal foot contours. Many patients believe they have suffered a serious fracture of their foot because of the rapid onset of edema. Hematoma formation is disseminated throughout the superficial fascia with ecchymosis extending from the digits to the lateral malleolus. The point of maximum tenderness usually indicates the site of the avulsion fracture. The patient will have moderate pain with the active extension of their toes against resistance. Passive motion of the ankle and subtalar joints will elicit much less pain than expected. Standard foot and ankle radiographs will reveal a small cortical fragment at the level of the calcaneocuboid joint. This must be differentiated from an anterior process fracture of the calcaneus.

Treatment is usually conservative consisting of ice, rest, compression bandaging, and elevation for 48 to 72 hours. Extremely severe injuries may require evacuation of the hematoma followed by the infiltration of some local anesthetic and soluble steroid to decrease the symptoms. It is rare to perform primary excision of the fracture fragment with repair of the muscle belly.

The patient's weight-bearing status will be determined by the severity of their symptoms when ambulatory. A below knee walking cast is applied after the initial compression cast is removed.

Patients most often return to full activity in 5 or 6 weeks post injury. Should the patient experience persistent pain from the fracture site, excision of the fragment may be necessary.

Fractures of the anterior process of the calcaneus have received significantly less attention in the literature than other fractures of the calcaneus. Its incidence is not readily known but it is believed to be more common than reported in podiatric and orthopedic literature. This may be secondary to the lack of anatomical significance associated with the anterior process.

Anatomically the dorsal distal portion of the calcaneus is continuous with the saddle shaped articular surface that articulates with the cuboid and occasionally the navicular. AT this level the anterior process is the primary attachment of the bifurcate ligament.

There are two postulated mechanisms of injury. The first is an avulsion fracture that results from a severe inversion injury. While the foot is in a plantarflexed and inverted position it undergoes a violent plantarflexory inversion-type force changing the tension in the bifurcate ligament and fracturing the anterior process. The second and less common is a compression fracture of the anterior process which is generated by a sudden dorsiflexory eversion force.

The clinical appearance is quite similar to that of the calcaneal cortex avulsion fracture. There is ecchymosis, edema, and joint tenderness at the level of the calcaneocuboid joint. Extreme pain will be produced with adduction and inversion of the forefoot as well as with dorsiflexion and abduction. The patient does not usually tolerate weightbearing.

This fracture may not be readily demonstrable on conventional foot and ankle radiographs. A lateral oblique view is highly recommended. The fracture must be distinguished from the topographical ossicle—os calcaneus secondarius.

The treatment of this fracture will vary depending upon the size of the fracture fragment and the amount of displacement. Incomplete fractures respond well to compression bandaging for 4 to 6 weeks, with partial weightbearing

as tolerated. Complete well aligned fractures should be casted for five to eight weeks or until radiographic consolidation occurs. If the fracture fragment is large enough one should consider open reduction and internal fixation. In the event of nonunion of the fracture site or prolonged disability, it should be treated similarly to an osteochondral fracture of the talus with excision of the fragment, remodeling of the joint surface, and the drilling of subchondral bone. If degenerative changes are present an arthroplasty should be performed. Fusion is rarely indicated.

Miscellaneous

It is possible that congenital syndromes such as osteochondritis dessicans, synovial chondromatosis or normal occurring topographical ossicles may simulate one of the aforementioned syndromes. The presence of repeated microtrauma or even acute trauma may present the physician with a most challenging diagnostic delemma.

Summary

There are a great number of conditions that may affect the osseous structure of the ankle and rearfoot. The results of repeated microtrauma may have significantly long lasting debilitating effects on the runner that often require patience from both the physician and the patient to effect improvement. Early recognition of osseous pathology by the physician may decrease the treatment course and restore the runner to pre-injury activity level.

References

Bauer M, Jonsson K, Linden B: Osteochondritis dissecans of the ankle (a 20 year follow-up study). *J Bone Joint Surg* 69B:93-96, 1987.

Berndt AL, Harty M: Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg* 41A:988-1022, 1959.

Boberg J, Kalish SR, Banks AS: Ankle conditions. In McGlamry ED (ed): *Comprehensive Textbook of Foot Surgery,* vol 1. Baltimore, Williams & Wilkins, 1987, pp 508-518.

Brodsky AE, Khalil MA: Talar compression syndrome. *Am J Sports Med* 14:472-476, 1986.

Cain TD: Posterior talar process fracture and os trigonum syndrome. In McGlamry ED (ed): *Doctors Hospital Surgical Seminar Syllabus, 1986.* Tucker GA, Doctors Hospital Podiatry Institute, 1986, pp 130-133.

Canale ST, Belding RH: Osteochondral lesions of the talus. *J Bone Joint Surg* 62A:97-102, 1980.

Cavalierre RG: Talar fractures. In McGlamry ED (ed): *Comprehensive Textbook of Foot Surgery,* vol 2. Baltimore, Williams & Wilkins, 1987, pp 873-933.

Cavalierre RG, Ruch JA: Talar fracture. In McGlamry ED (ed): *Doctors Hospital Surgical Seminar Syllabus, 1985.* Tucker GA, Doctors Hospital Podiatry Institute, 1985, pp 206-209.

Fjeldborg O: Fracture of the lateral process of the talus. Supination-dorsal flexion fracture. *Acta Orthop Scand* 39:407-412, 1968.

Flick AB, Gould N: Osteochondritis dissecans of the talus. *Foot Ankle* 5:165-185, 1985.

Hamilton WC: Injuries to the ankle and foot. *Clinics Podiatric Medicine and Surgery* 3:169-197, 1986.

Hawkins LG: Fracture of the lateral process of the talus. A review of thirteen cases. *J Bone Joint Surg* 47A:1170-1175, 1965.

Hontas MJ, Haddad RS, Schlesinger LC: Conditions of the talus in the runner. *Am J Sports Med* 14:486-490, 1986.

Kelikian H: *Disorders of the Ankle.* Philadelphia, WB Saunders Co, 1985, pp 676-755.

Nicholas JA: Ankle injuries in athletes. *Orthop Clin North Am* 5:153-175, 1974.

Pettine KA, Murrey BF: Osteochondral fractures of the talus (a long term follow-up). *J Bone Joint Surg* 69B:89-92, 1987.

Resnick D, Niwayama G: *Diagnosis of bone and joint disorders.* Philadelphia, WB Saunders, 1981, pp 2260-2266, 2713-2714.

Sarrafian S: Anatomy of the *Foot and Ankle.* Philadelphia, JB Lippincott, 1983, pp 88-96.

Tachdjian MO: *The Childs Foot.* Philadelphia, WB Saunders Co, 1985, pp 616-637.

Yu G, Boberg J, Mahan K: Miscellaneous ankle conditions following sprained ankle. In McGlamry ED (ed): *Doctors Hospital Surgical Seminar Syllabus.* Tucker GA, Doctors Hospital Podiatry Institute, 1984, pp 52-61.