OS TRIGONUM SYNDROME

Gerard V. Yu, DPM
Amanda Meszaros, DPM
Theresa L. Schinke, DPM
Michael B. Canales, DPM

INTRODUCTION

The os trigonum is an anatomical variant of the posterior process of the talus where a secondary center of ossification located within the lateral tubercle fails to fuse with the main body of the talus. This secondary center generally appears between 8-11 years of age and fuses within 1-3 years of its appearance. Controversy exists as to the true nature of the os trigonum as a distinctly separate entity as opposed to a posttraumatic remnant of a fracture of the posterolateral process of the talus. Os trigonum syndrome is a symptomatic disruption or inflammatory response within the fibrous tissue, which unites the accessory ossicle with the posterior process of the talus. The origin and etiology of os trigonum syndrome has been the topic of much debate amongst both podiatric and orthopedic physicians. Outlining the somewhat complex derivation of os trigonum syndrome requires a thorough understanding of the anatomy of the posterior ankle.

The posterior process of the talus is made up of two tubercles, the medial tubercle and the lateral tubercle. Between these tubercles is a groove that allows the passage of the flexor hallucis longus tendon from the posterior ankle to enter the tarsal tunnel. The course of the flexor hallucis longus tendon is angled at this level, and its sheath is often thickened to accommodate the higher degree of friction encountered as it passes through the posterior ankle. The lateral tubercle of the posterior talus is generally larger, and it is here that the secondary ossification center associated with the development of os trigonum is found. Should this lateral tubercle present as an intact but abnormally elongated process or extension of the posterior talus, it is referred to as Steida's process. When existing as a separate entity, it is referred to as an os trigonum, trigonal process or accessory ossicle. The os trigonum articulates with the talus via a fibrous,

fibro-cartilaginous or cartilaginous union. The ossicle also has ligamentous attachments to the main body of the talus. The posterolateral process serves as an attachment point for the posterior talofibular ligament and the posterior talocalcaneal ligament. Aside from its articulation with the talus, the os trigonum communicates superiorly with the posterior capsule of the talo-crural joint, inferiorly with the posterior talocalcaneal ligament, medially with the flexor hallucis longus tendon sheath and laterally with the origin of the posterior talo-fibular ligament. The posterior process of the talus derives its blood supply via an anastamosis of the medial calcaneal artery and the communicating branch of the peroneal artery.

LITERATURE REVIEW

The os trigonum was first described as an accessory ossicle in 1804 by Rosenmuller who identified it based on its trigonal contour. In 1882, Shepherd revealed his theory that the os trigonum is not an accessory ossicle created by the failed fusion of a secondary ossification center. Shepherd believed that the os trigonum appears only as a result of a fracture of the posterolateral process of the talus.[1]

In contrast, Bardelen, in 1883, presented a theory that identified the os trigonum as a primitive or intermediate tarsal bone, retained in a segment of the population. He supported this theory on the basis that the ossicle forms from a distinctly separate cartilaginous body that does not fuse with the main cartilaginous body of the talus until the third fetal month.[2] Shepherd was challenged by O'Rahilly and Turner in 1953 who concluded that os trigonum is, indeed a non-unified remnant of the secondary ossification center or nucleus of the posterior talus.[1,3,4]

McDougall, in 1953 again challenged this view, supporting Shepherd's conclusions while speculating that the os trigonum is always secondary to fracture and that its smooth rounded edges are

natural changes due to erosion of the fracture edges over time. McDougall also proposed three mechanisms by which fracture of the posterolateral talar process may occur, resulting in the appearance of os trigonum: 1) impingement of the lateral tubercle between the calcaneus and tibia over time leads to stress fracture, 2) acute fracture of the lateral tubercle due to sudden, severe trauma with the foot plantarflexed, 3) avulsion fracture of the lateral tubercle by traction of the posterior talofibular ligament while stressed in dorsiflexion.[5] Recent literature supports the widely accepted view that the os trigonum is a true accessory ossicle which appears when the secondary ossification center of the talus fails to fuse by age 18 years.

PREVALENCE

The incidence of os trigonum in the general population ranges from 1.7-50%, 33-50% of which present bilaterally.[6] There does not appear to be an increased prevalence in women or men; the entity does not appear to be more common in certain age groups. Athletes who participate in sports that require a high degree of plantarflexory strain, for example, football, soccer, ballet and other forms of dance are more prone to both a fracture of the posterolateral process of the talus (Shepherd's fracture) and symptomatic os trigonum syndrome.

ETIOLOGY

The etiology of os trigonum syndrome is highly variable. It becomes symptomatic during strenuous activities and is generally dependent on the position of the ankle joint during activity. Most commonly, a symptomatic os trigonum may be attributed to repetitive microtrauma due to impingement of the ossicle between the calcaneus and the posterior-inferior aspect of the tibia. Plantarflexion and eversion of the ankle joint will recreate the symptoms that may be attributed to detachment of or inflammation within the fibrous junction between the ossicle and the posterior talus. Smaller ossicles often become symptomatic secondary to bony impingement and also to soft tissue compression as well.

Plantarflexing and everting the foot compresses the accessory bone between the flexor hallucis longus tendon and the posterior talofibular ligament, resulting not only in an os trigonum syndrome, but

FHL tendonitis as well. The FHL tendon passes through a bifurcation of the posterior talo-calcaneal ligament and the posterior tibio-talar ligament. Thickening of the FHL sheath in this area during contraction applies pressure directly on the ossicle, aggravating its syndesmosis. Primary FHL tendonitis may also be a contributing factor in symptomatic os trigonum; in the presence of inflammation, the tendon sheath tends to thicken and stenose over time, creating a medial compression force with flexion of the hallux.[7]

In a more acute setting, forced plantarflexion may result in fracture of the lateral tubercle or complete disruption of the fibrous bridge. Fallat examined this acute injury and its relationship to ankle sprains. He reported that less than 1% of ankle pain that is recalcitrant to conservative therapies following a sprain may be attributed to a symptomatic os trigonum, disrupted at the time of initial injury.[4] Excessive dorsiflexion, too, may create a symptomatic os trigonum by causing excessive traction on the posterior talofibular ligament which originates from the lateral tubercle. Traction causes the lateral tubercle to be forced inferiorly, creating a compression force against the calcaneus. This mechanism of injury is most common in the supinated, high-arched foot type. In contrast, a pronated or planus foot tends to disrupt the synchondrosis because of added traction on the origin of the posterior talocalcaneal ligament. As the talus adducts and plantarflexes in response to subtalar and midtarsal joint pronation, the talocalcaneal ligament is stretched, creating a compression force directly over the ossicle. Finally, direct trauma to the posterior ankle may stimulate an inflammatory response within the fibrous junction.[8]

CLASSIFICATION

Watson and Dobas proposed a classification scheme to categorize anatomical variants with predicted mechanisms of injury.[4,5] Other classification schemes have not been commonly recognized (Table 1).

CLINICAL FEATURES

In the clinical setting, the presentation of os trigonum syndrome is variable based on the mechanism of injury. A history of trauma may or may not be present. Most patients complain of deep,

aching pain in the posterior ankle that is aggravated with weight-bearing, especially on uneven terrain. The patient describes poststatic dyskinesia, intermittent swelling, pain and stiffness with plantarflexion of the ankle that may be acute or insidious in nature, dependent on the type of injury sustained.[4,5,7,9]

Physical exam reveals pain upon palpation of the posterior aspect of the ankle joint, just anterior to the Achilles tendon. In the acute setting, ecchymosis may be noted in this region. Crepitus upon STJ and ankle ROM may be indicative of a fractured lateral tubercle or loose body secondary to fracture. Pain in the posterolateral ankle is enhanced at end range of motion with active and passive ankle joint plantarflexion. Passive dorsiflexion of the hallux/FHL manual muscle testing also increases

Table 1

CLASSIFICATION SCHEME FOR OS TRIGONUM

I   Asymptomatic, normal appearance of the lateral tubercle without clinical consequence

II Steida's process- an enlarged tubercle that is prone to injury in plantarflexion

III Os trigonum- an accessory ossicle prone to irritation due to repetitive microtrauma

IV Os trigonum- a 'fused' accessory ossicle with a cartilaginous or synchondrotic bridge with the main body of the talus - tend to undergo acute fracture and result in post-traumatic arthritis

tenderness in the posterior ankle. Pain is augmented in activities requiring a greater degree of subtalar joint motion, and peroneal spasm may result if a large ossicle has partially obscured or fused the posterior subtalar joint facet. Chronic conditions may present with tarsal tunnel-like symptoms due to continued inflammation and traction along the course of the FHL tendon.[4]

DIAGNOSIS

A wide variety of acute and chronic conditions have been described for the differential diagnosis of os trigonum syndrome. First, a Shepherd's fracture of the posterolateral process of the talus must be considered. Other common possibilities include a retrocalcaneal spur, FHL tendonitis and paratenonitis, posterior calcaneal fracture, medial/lateral/posterior malleolar fracture, osteoarthritis, Achilles tendonitis, retrocalcaneal bursitis, partial avulsion of the Achilles tendon, Haglund's deformity and lateral ankle joint or subtalar joint instability.[10,11] Less common causes of posterior ankle pain include peroneal tendon subluxation, Cedell's fracture of the posteromedial process of the talus, tarsal coalition, osteochondral lesion or flake fracture of the talar dome, tarsal tunnel syndrome, stenosing tenosynovitis of the FHL, pseudomeniscus syndrome of the STJ and periostitis within the groove for passage of the FHL tendon. In pediatric patients, Sever's disease, or calcaneal apophysitis should also be considered.[4,10-13]

The diagnosis of os trigonum syndrome is not uncommonly reached by employing a series of radiologic evaluations. Plain film radiographs prove an

Figure 1A. Lateral radiograph demonstrating an unusually long posterior lateral process of talus with symptoms mimicking os trigonum syndrome.

Figure 1B. Lateral radiograph of fracture of posterior lateral process with significant symptomatology necessitating surgical removal.

extremely valuable aid in diagnosis; the lateral and oblique views are particularly beneficial. Os trigonum is often an incidental finding on radiograph, appearing on average in 10% of the population. In symptomatic patients the accessory ossicle or elongated Steida's process is often visualized abutting the distal posterior edge of the tibia, raising the possibility of posterior ankle impingement in symptomatic patients.[14] However, there can be significant difficulty in distinguishing a true os trigonum from a fractured lateral tubercle on plain film (Figure 1). One would expect a true accessory ossicle to display a smooth, rounded contour while a fracture fragment would display a characteristic 'jagged' edge, however, it has been shown that over time fracture fragments may appear smooth as well due to erosive wear and tear.[7] Over time the eroding fracture fragments may represent a non-union, where characteristic sclerotic edges may be visualized. Chao suggests obtaining a 30-degree subtalar oblique view to differentiate between an os trigonum and an acute fracture.[6] Contralateral views are not exceptionally useful, although recommended, since the os trigonum as well as the fractured Steida's process may present unilaterally or bilaterally.

Bone scan is useful in diagnosing os trigonum syndrome by demonstrating increased uptake of the tracer in the posterolateral ankle. It can, in the experience of the senior author (GVY), be most valuable in distinguishing between an acute fracture of the posterolateral process and a normal, asymptomatic os trigonum. A focal positive scan may strongly suggest the diagnosis of an acute fracture of the lateral tubercle, showing a hot spot or increased uptake of the radiotracer at the site of the ossicle (Figure 2). A non-fractured free accessory ossicle that has been aggravated by impingement may also result in a positive scan due to the presence of inflammation or pseudoarthrosis of a prior fracture.[8,9,14] A positive scan strongly suggests the need for CT scan, MRI or tomography in certain clinical scenarios. This imaging technique will identify the extent of osseous involvement and aid in more clearly delineating the fracture lines from the body of the talus. CT scans, like plain film radiographs are not able to clearly distinguish between a true os trigonum and an old fracture that has recently become symptomatic.[4,14] Sopov and associates revealed uptake in the region of the os trigonum of 27 soldiers on 99mTc methylene-diphosphonate bone scan. Of the 27 soldiers displaying uptake in the os trigonum, 17 were asymptomatic. The conclusion reached from this study by its authors was that bone scintigraphy holds little value in determining painful os trigonum in active soldiers. Sopov suggested that perhaps the high-topped boots of the soldiers increased pressure and osteoblastic activity of the posterior talus during soldiers' routine exercise.[14] We believe that such scans when properly correlated with clinical

Figure 2A. Typical bone scan of patient with symptomatic os trigonum syndrome. This focal uptake is typical in patients with symptomatology attributed to the os trigonum itself and not involving pathology of the subtalar joint.

Figure 2B. Conventional bone scan showing extensive uptake throughout the subtalar joint in patient with os trigonum symptomatology but also decreased ROM of the STJ. An area of focal uptake representing the ossicle itself can be seen. Removal of this bone alone is likely to fail. A STJ arthrodesis with or without removal of the bone fragment was performed.

symptoms are an excellent diagnostic study; the senior author routinely employees this test to confirm that there is in fact an osseous problem of this specific geographic area.

A negative scan even in the presence of radiographic confirmation of os trigonum should lead the physician to reconsider other differentials at this point, focusing on soft tissue pathology. However, even a negative scan cannot entirely rule out os trigonum syndrome as a possibility. Disruption of only the fibrous bridge without any osseous pathology has been reported to result in a negative scan.[8,9,14]

Additional testing, such as MRI will more clearly identify any soft tissue pathology. Inflammatory changes are perhaps the most important finding on MRI. The os trigonum is expected to display a marrow signal consistent with its corresponding tarsal bones. Marrow edema, visualized as an abnormally high signal, is indicative of inflammatory changes within the os trigonum (Figure 3). Signs of bony impaction, such as contusions or occult fractures may also help to confirm or refute the diagnosis (Figure 4).[14] High signal fluid within the FHL sheath and surrounding the ossicle or fracture fragment are indicative of soft tissue inflammation, further confirming the diagnosis of os trigonum syndrome (Figure 5).[10] Visualizing an ossicle of unusually low signal when compared with the surrounding tissues suggests an old fracture. Sclerosis at a site of non-union or pseudoarthrosis is visualized on MRI as an area of low signal. The additional finding of soft tissue edema in this case makes MRI a useful diagnostic tool in differentiating acute from long-standing injuries. An MRI also allows for evaluation of the STJ and ankle joints as well as adjacent muscle tendon complexes.

There are a number of other diagnostic tests that may be employed if there exists a high suspicion of os trigonum syndrome. Mann advocates the use of 0.75-1cc of lidocaine into the posterior ankle utilizing a lateral approach, injecting just posterior to the peroneal tendons. This diagnostic block is considered positive if the patient's symptoms are relieved with ROM of the ankle and FHL manual muscle testing. He suggests that a positive block be followed by an injection of corticosteroids into the same area as a first-line conservative therapy.[16] Fluoroscopy is often reserved for intraoperative guidance during
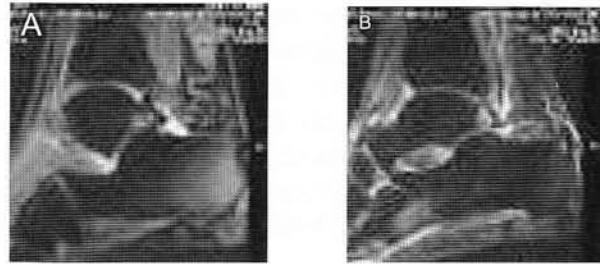

Figure 3A. MRI showing inflammatory changes in the posterior ankle. The area of high signal surrounding the ossicle is indicative of soft tissue inflammation, further confirming the diagnosis of os trigonum syndrome. The os trigonum should display a marrow signal consistent with its corresponding tarsal bones

Figure 3B. STIR image showing marrow edema, visualized as an abnormally high signal within the posterior talus. This is indicative of inflammatory changes within the os trigonum due to repetitive microtrauma.

Figure 4. MRI showing prior posterior talar facet fracture with the added presence of os trigonum and soft tissue inflammation. The presence of such fractures may refute the diagnosis of os trigonum syndrome, particularly in patients with STJ complaints as well. An isolated STJ fusion was performed with complete resolution of symptoms.

Figure 5. MRI demonstrating a fibrous union of the os trigonum to the posterior talar body. The presence of edema at the site raises the suspicion of acute injury.

excision of the os trigonum when conservative measures have failed. However, a fluoroscopically guided diagnostic block may confirm that the pathology exists purely within the synchondrotic or fibrous bridge connecting the os trigonum to the posterior talus.[17]

Stress lateral radiographs are most beneficial in ascertaining the exact etiology of the patient's complaints. Stress views in extreme dorsiflexion and plantarflexion may visually recreate the mechanism of injury, allowing the interpreter to confirm impingement of the ossicle and the mobility of or disruption of the bridge due to ligamentous traction. Once the diagnosis of os trigonum syndrome has been made, the physician must determine if any accompanying pathologies exist. For example, FHL tendonitis is a common secondary sequelae of os trigonum syndrome. A tenogram can aid in identifying defects and abnormalities within the FHL sheath, however, we believe the MRI is the preferred study of choice and when properly performed and interpreted provides unsurpassed detailed information as to the type and extent of pathology.

In order to simplify the complex process of recognizing and correctly diagnosing os trigonum syndrome, Martin presented a diagnostic schematic for the diagnosis and treatment of the disorder in the *Journal of Foot Surgery* in 1989. His plan begins with radiographic evaluation of a patient complaining of posterior triangle pain. If the plain films suggest a diagnosis of fracture and are consistent with the clinical presentations, then the patient should be treated with appropriate fracture care. Films non-specific for trauma should be evaluated for other possible differential diagnoses and conservative treatment initiated. If pain persists after conservative therapy, a bone scan should be ordered. A negative scan should lead to further evaluation with conservative and symptomatic treatment. A positive scan leads to a high suspicion of os trigonum syndrome and should be confirmed with CT scan; and MRI should also be considered as a further valuable diagnostic imaging study. A CT negative for occult fractures or gross osseous pathology should stimulate a more aggressive course of conservative therapy and re-evaluation for a possible inflamed ossicle. A positive CT scan would show a traumatic fracture or periostitis or stress fracture of an elongated Steida's process. In either case, after failure of a more aggressive course of conservative therapy, surgical excision of the ossicle or fracture fragment must be considered.[18]

TREATMENT

Os trigonum syndrome, acute fractures of the lateral tubercle and chronic conditions secondary to prior fracture or repetitive injury are all treated conservatively and symptomatically following initial diagnosis. All acute fractures and injuries are treated with 6 weeks non-weightbearing cast immobilization. Mann recommends casting in 10-15 degrees of equinus while most podiatric physicians believe neutral position is acceptable.[19] All chronic injuries should be treated initially with RICE therapy, NSAIDs, stretching exercises, ultrasound and rehabilitation. Iontophoresis with dexamethasone may also be considered as a conservative treatment modality.[6] Should these initial treatment modalities fail in either the acute or chronic setting, corticosteroid injections may prove beneficial. Corticosteroid injections should be reserved as a second line therapy because when utilized during the first 6-8 weeks of therapy they are implicated in the inhibition of potential bone healing.[14] Caution must be taken when injecting corticosteroids into this general area because weakness of the Achilles and flexor hallucis longus may result.[6] If symptoms persist, an additional 4 weeks of cast immobilization is recommended.[4,11,19,20] Paulos reports a 33% success rate with conservative therapy alone, and an additional 10% of patients improved with a second, more aggressive round of therapy.[20] Podiatric and orthopedic surgeons alike agree that surgical excision of the ossicle or fracture fragment should be considered following failure of 4-6 months of conservative treatment.

When indicated, surgical excision may be approached in three manners: 1) lateral incisional approach, 2) medial incisional approach, 3) arthroscopic excision. The lateral incisional approach is the most often employed method of excising a symptomatic ossicle (Figure 6A). A 4-6 cm lateral curvilinear incision is made posterior to the peroneal tendons and anterior to the tendoachilles, taking care to avoid the sural nerve; the sural nerve is more readily retracted forward of the surgical site minimizing potential injury during the surgical procedure (Figure 6B). Blunt dissection is carried down to the deep fascia and the posterior ankle joint capsule is incised to expose the talus. The ease of dissection at the posterolateral ankle offers superior access to the subtalar joint as well. The FHL tendon should be exposed and identified prior to excising the ossicle. This is
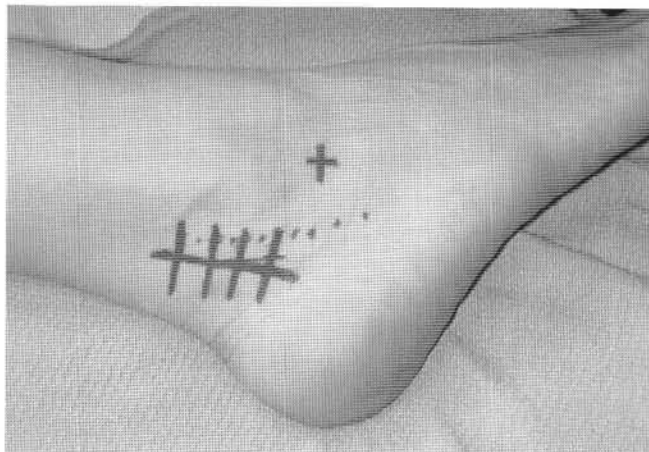
Figure 6A. Lateral incisional approach for removal of os trigonum. The incision is made midway, or preferable slightly posterior to the bisector of the distance between the tendoachilles and the peroneal tendons.
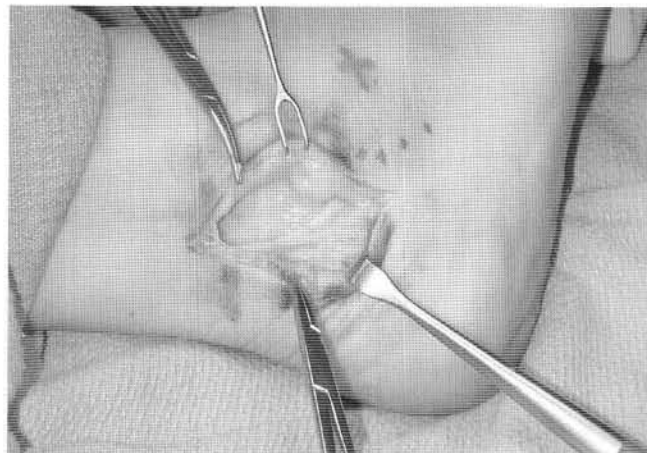

Figure 6B. Identification of the sural nerve as it travels into the foot. Note bifurcation of the nerve. The nerve should be meticulously and carefully retracted and protected during the surgery to avoid postoperative entrapment and neuritic symptoms.
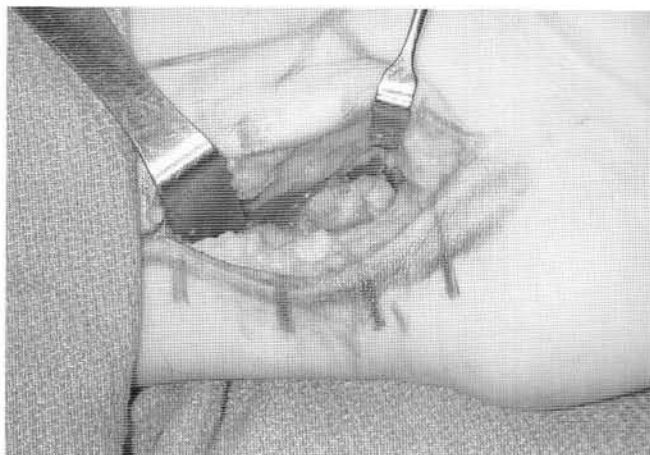

Figure 6C. Identification and isolation of the fracture fragment. Extirpation is facilitated by manipulating the bone with a single skin hook. Particular care should be taken to avoid injury to the FHL tendon which is just slightly medial and deep to the fragment.

Figure 7. Intraoperative x-rays confirming removal of the entire fracture fragment. The main body of the talus is checked carefully to ensure no spike or rough edges remain. A small hand rasp is effective in remodeling this area. Power instrumentation is not recommended due to potential damage to the surrounding soft tissue.

accomplished by manipulating the hallux under direct observation (Figure 6C). Following excision, the talus should be inspected for sharp or irregular projections and the FHL tendon inspected for any gross pathology (Figure 7). The FHL tunnel should be inspected for any loose bodies that may have displaced after fracture.

The lateral incision is a safe approach due to the lack of large neurovascular structures in the area and is technically easier to perform than a medial approach. A possible complication involves stiffness of the peroneal tendons due to interference in surgery, along with sural nerve neuritis, also secondary to trauma during surgery. Abramowitz et

al employed the lateral incisional approach to excise a symptomatic os trigonum in 41 patients. The study noted that sural nerve injury was the most common complication, with four patients developing complete sensory loss of the nerve.[21]

A medial incisional approach is justified in patients suffering from concomitant FHL tendonitis, common among ballet dancers. Here, care must be taken to avoid all structures of the medial neurovascular bundle and the flexor tendons (Figure 8A). The medial approach offers better exposure to the FHL tendon and its sheath, which may be divided if severe tendonitis and stenosis is present (Figure 8B). The medial approach may result in less
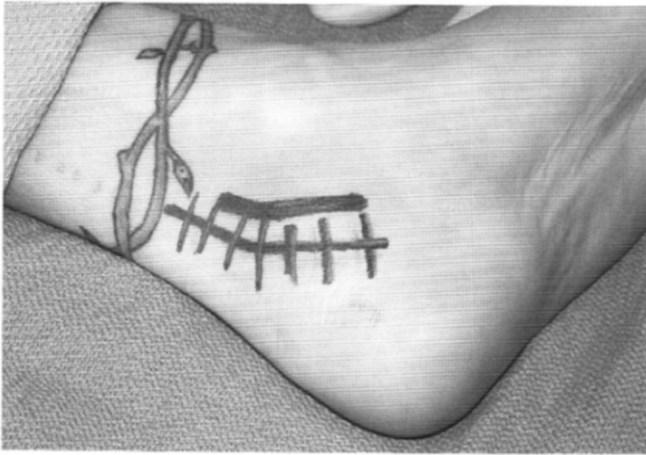
Figure 8A. Medial incisional approach for removal of os trigonum. Dissection is carried down through the subcutaneous tissues to the level of the flexor retinaculum (laciniate ligament) which is incised to visualize the muscle of the FHL tendon.
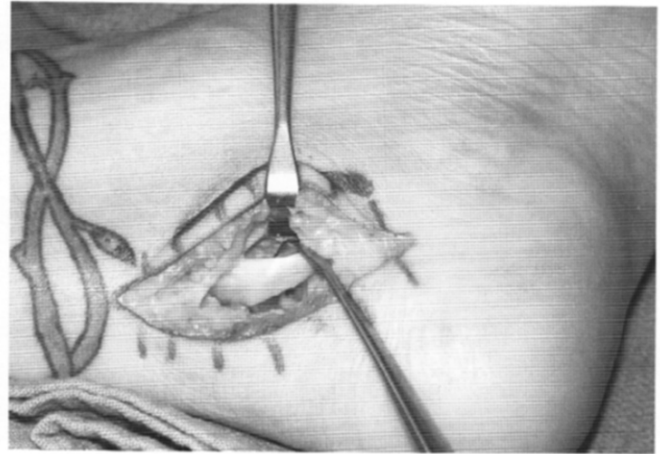

Figure 8B. The FHL tendon is visualized and will be retracted anteriorly along with the neurovascular bundle to expose the os trigonum. Intraoperative c-arm fluoroscopy is highly recommended to facilitate identification of the fragment prior to surgical extirpation.
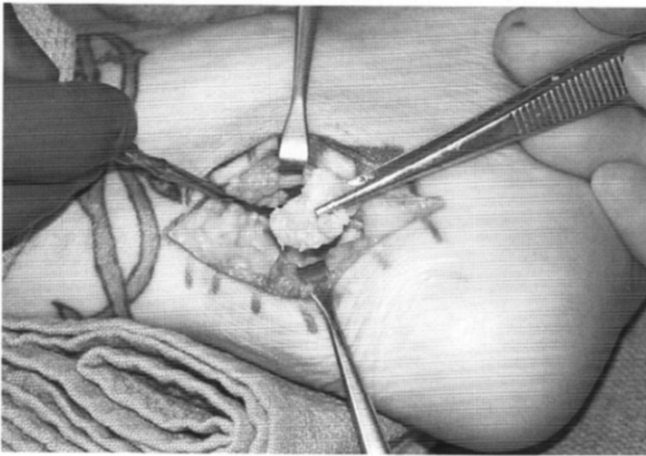

Figure 8C. Appearance of the bone fragment following successful removal. Meticulous dissection technique ensures minimal post-operative edema and fibrosis as well as minimizes potential damage to the surrounding neurovascular structures and tendons.

postoperative stiffness and a quicker return to activity, however, it is technically more difficult to perform and the risk of neurovascular damage is much greater (Figure 8C).[10,16,22]

Lastly, arthroscopic excision of a symptomatic os trigonum is an option supported by both podiatric and orthopedic surgeons to promote a quick return to activity. A 30° 2.7mm arthroscope provides the best possible access to the joint. Three portals are created to accomplish excision: 1) anterolateral portal- anterior and distal to the tip of the lateral malleolus, 2) posterolateral portal in the soft tissue lateral to the Achilles at the level of the tip of the lateral malleolus, 3) accessory anterolateral portal- distal and superior to the anterolateral portal. The camera is inserted into the anterolateral portal while the instrumentation is positioned through the posterolateral portal. Inflow enters through the accessory portal to increase joint distention and improve visualization.[1,11,19] Following identification, the ossicle is outlined and shelled out with a banana knife, removed via a grasper and the area is smoothed with a shaver. Arthroscopy, though difficult to achieve spatial orientation and somewhat technically difficult to perform, may offer a faster return to activity for serious athletes and dancers. Minimal incisions result in a lower occurrence of skin necrosis, incisional neuromas and scarring. Overall, the arthroscopic approach has been shown to significantly decrease surgical morbidity offering full recovery at 2-3 months.[1,11] Unfortunately, we have not had occasion to perform resection of an os trigonum with this technique. We have routinely employed either a posterolateral or posteromedial incisional approach for this procedure.

The course of postoperative care is similar for all three approaches. Generally a compressive dressing is applied immediately postoperatively to control swelling, followed by a short leg cast 5-7 days later. Most physicians limit immobilization to 2 weeks NWB before initiating a stretching and strengthening regime. Others promote even earlier (immediate-1 week) postoperative weight-bearing and aggressive ROM exercises to promote a return to full function.[4,7] Aggressive physical therapy will prevent excessive fibrosis and accumulation of scar tissue that may later limit motion around

the ankle and subtalar joints.[14] We recommend a short period of several weeks of absolute immobilization to allow healing the tissues and to avoid wound dehiscence.

COMPLICATIONS

Complications of surgical excision include infection, persistent pain and posttraumatic arthritis following a fracture. More specifically, postsurgical adhesions and fibrosis may limit ROM and be a source of chronic pain. Chronic tendonitis may result due to trauma to the flexor hallucis longus tendon during surgery or chronic inflammation of the tendon sheath due to traction against improperly debrided bony edges.

Post surgical neuritis or other nerve disorders such as neuropraxia and neurontmesis have been encountered in a number of patients who underwent surgery elsewhere. On careful examination of these patients, the surgical incision is often directly overlying the neurovascular bundle and direct insult and injury have been sustained. Complex regional pain syndrome or reflex sympathetic dystrophy syndrome remain real entities which can result in a chronic pain syndrome in these patients. Any postoperative nerve disorder can be frustrating, challenging and discouraging to both the patient and surgeon. Some patients will become candidates for chronic pain management programs.

Traumatic techniques of resection can also result in injury and damage to the subtalar joint primarily and less frequently the ankle joint. As a result, significant degenerative arthritis develops and, if fails to respond to conservative treatment modalities, is likely to require joint arthrodesis.

The prognosis for os trigonum syndrome and acute and chronic fractures of the posterolateral talar process is excellent. Approximately 43% of all patients diagnosed with os trigonum syndrome will experience complete resolution of their symptoms with conservative treatment modalities alone. The remainder of patients experience relief through surgical excision with an extremely low rate of complications and chronic intractable pain. The average time to full recovery for an arthroscopic excision is 3 months, compared to 3-12 months reported for an open excisional procedure. With aggressive physical therapy and rehabilitation,

highly competitive athletes and dancers may attain full recovery within 6-8 weeks.[7,22] The prolonged recovery and convalescence reported for open techniques of resection have not been the experience of the senior author (GVY), although we have seen several patients who have suffered. It is our belief that this occurs as a result of traumatic technique with injury to the cutaneous nerves on either the medial or lateral aspect with or without concomitant injury to the deeper soft tissues resulting in an excessive amount of postoperative scar tissue and adhesions.

DISCUSSION

The diagnosis and management of an os trigonum syndrome or related entity involving the posterior process of the talus can be difficult and challenging. Often considerable time has transpired since the original inciting event or injury. Frequently misdiagnosis has led to extensive conservative treatment, and in some cases, surgical intervention, of an all together different clinical entity with no improvement in the original symptomatology. Patients, not uncommonly, are very frustrated, anxious and at times even hostile over their current condition, especially when it precludes them from participating in sports activities. We have seen several patients who have, unfortunately, experienced this outcome.

A careful detailed history and physical examination should raise the index of suspicion for this clinical entity. Conventional radiographs will either confirm or refute the clinical suspicions. In either scenario, further diagnostic workup or confirmation can be done with specialized studies including a conventional bone scan, CT scan or MRI . A series of diagnostic and therapeutic blocks with long acting local anesthetics, with or without short acting corticosteroid, accurately delivered to the posterior ankle joint area, should readily confirm the diagnosis.

Conservative treatment should be aggressive and includes a combination of immobilization followed by physical therapy. Various pharmacologic agents may prove beneficial including systemic and locally injected corticosteroids combined with long acting local anesthetics and NSAIDs. The earlier the correct diagnosis is established and the sooner conservative treatment instituted, the more likely it

is to be successful. Radiographic healing or failure to heal does not necessarily correlate with clinical outcomes; treatment must not be based upon the radiographic presentation of this entity. When patients fail to respond to conservative treatment, surgical exploration with excision is necessary.

Careful, meticulous and complete excision of the fragment of bone must be achieved to ensure a successful clinical outcome and resolution of symptoms. In some cases, exploration of the FHL tendon itself with debridement and or repair is necessary. Rarely will joint fusion be required as a definitive treatment.

The senior author believes that the single most important requisite for successful outcome with this type of surgery is a thorough working knowledge of the anatomy of the ankle and subtalar joints, including the periarticular structures. An awareness of the various nerve structures, especially the sural and posterior tibial nerves will ensure a low incidence of nerve injury. Meticulous, detailed, precise minimally traumatic technique then paves the way to a successful excision of the pathologic bone without damage to surrounding structures. Hemostasis is critical during and following the surgery. A tourniquet is routinely employed during this procedure but is released prior to closure to ensure maximum hemostasis.

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