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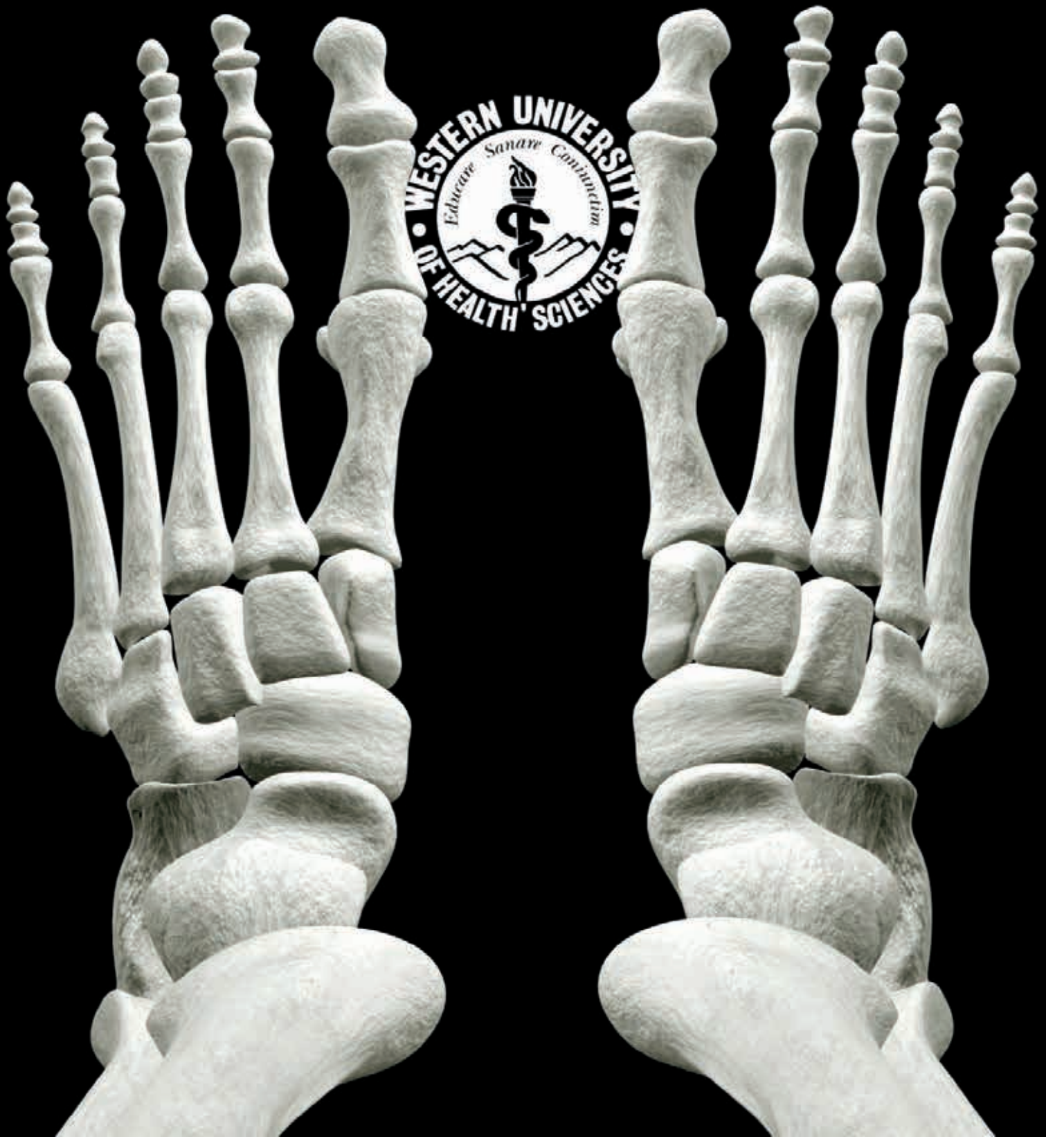
Journal of Lower Limb Medicine



Letter From the Staff

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Dear Reader,

Thank you for taking the time to delve into the third annual issue of *Extremitas*: *Journal of Lower Limb Medicine*. We are thrilled to be publishing another issue filled with case studies and review articles regarding our passion, the lower extremity, by our very own WesternU students. Our goal is to share the work of students from all disciplines and to spread the word of the importance and diversity of the lower limb, the facets of which shine through this year's edition. Assorted topics ranging from surgical procedures to sports injuries to diabetic complications are explored this year, and we hope to continue to challenge students from all professions to research topics they find interesting. We invite you to find a topic in this journal that sparks an interest and fuels you to explore further.

We would like to thank Western University of Health Sciences and the College of Podiatric Medicine, for this would not have been possible without their continued support. Please contribute your thoughts and feedback about this published work to the editors at ExtremitasJournal@westernu.edu. If you have a topic of interest regarding lower limb medicine, we invite you to submit your original articles, case studies, reviews, or any other formats that utilize evidence based practices to *Extremitas* for next year's issue. Be on the lookout for announcements regarding submission requirements and deadlines in the fall semester.

We appreciate your support and readership and look forward to another successful year sharing in WesternU's tradition of academic scholarship with you. Enjoy!

Sincerely,
The *Extremitas* Staff

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Os Trigonum Syndrome: Ballet and the Bone

Josephine Lyons, DPM 2017

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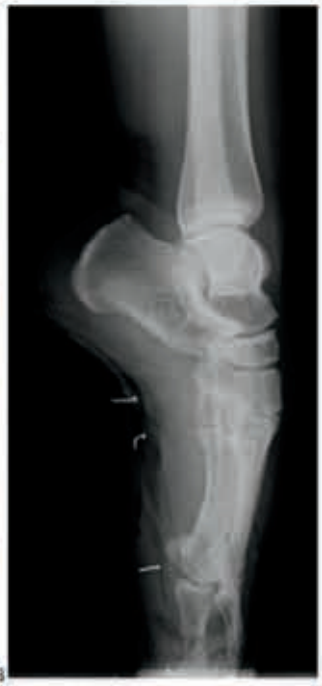

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Figure 1A. A ballet dancer demonstrating *en pointe* technique 1B A radiograph of *en pointe* (9)

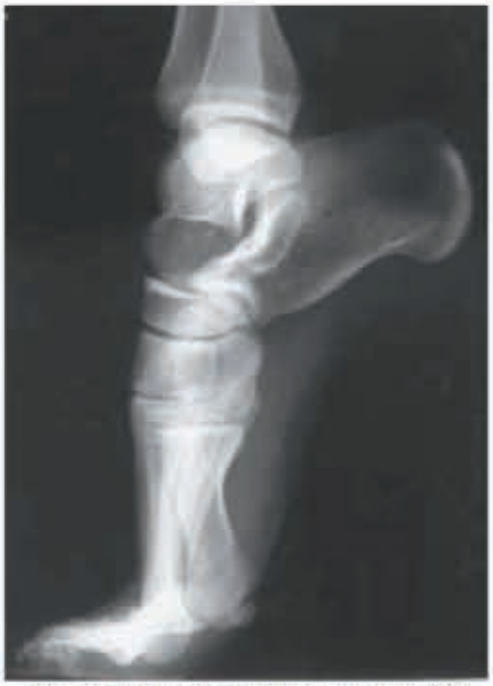

Figure 2: A radiograph of a dancer in *demi pointe* (10)

Introduction:

The os trigonum is an accessory bone at the posterolateral aspect of the talus. The bone stems from a secondary ossification center of the talus which fuses during early teen years.[1] However, a single traumatic event or repetitive micro-trauma to the area can cause this bone to be compressed against surrounding structures, resulting in irritation. While this is an uncommon phenomenon in the general populous, a significant portion of the dance community is affected, particularly ballet dancers *en pointe*.[2,3] Their frequent weight bearing on a hyper-plantarflexed ankle causes Os Trigonum Syndrome (OTS), an osseous cause of Posterior Ankle Impingement Syndrome (PAIS). This syndrome causes pain in the posterior ankle, often exacerbated by rapid or repetitive plantarflexion.[4]

A thorough physical exam along with MRI is the current primary evaluation technique, and definitive diagnoses may be made with an ultrasound-guided injection of a local anesthetic.[4,5] Treatment for OTS involves a trial of conservative interventions, such as NSAIDS, rest, and range of motion exercises, usually followed by surgical intervention for a more definitive end to symptoms. Surgical excision of the os trigonum is most often performed arthroscopically using the two portal technique described by van Dijk.[6] This procedure has shown decreased post-operative scarring, morbidity and wound complications, while improving patient recovery time, satisfaction and return to normal activity.[7] The ability to return to activity quickly with few complications is especially important for dancers, whose profession necessitates early return with full mobility and strength of the foot and ankle.

It should be noted that while Os Trigonum Syndrome can be debilitating, it is not universal, nor exclusive, to dancers. In fact, while many athletes who plantarflex frequently –such as footballers and gymnasts– have a distinct os trigonum bone, only those who traumatically hyper-plantarflex will develop OTS.[6,8] Special attention during developmental years, as well as proper care during all athletic training sessions can reduce symptoms and avoid OTS altogether.

Etiology:

Os Trigonum Syndrome (OTS) is one specific branch of a broader diagnosis known as Posterior Ankle Impingement Syndrome (PAIS). PAIS is characterized by inflammation and irritation to the posterior aspect of the ankle, and can be caused by any number of etiologies ranging from joint damage to soft tissue impairment.[2] Any individual who forcibly hyper-plantarflexes his foot is at risk of developing OTS. This could include dancers, football or soccer players, javelin throwers, gymnasts, figure skaters, downhill runners, or those involved in motor vehicle accidents who forcibly plantarflex.[1,4]

Ballet dancers are particularly at risk because their training includes standing *en pointe* where the dancer balances on her phalanges –primarily the hallux –as it rests on a block inside the pointe show (Figure 1).[9] The position of secondary risk is *demi pointe* wherein the dancer stands on the metatarsal heads and phalanges (Figure 2).[10] Both of these positions induce compression forces to the posterolateral ankle, where the os trigonum is located, commonly likened to a nut being crushed in a nutcracker.[1,8] In addition, dancers adopt a "turn out" in classical ballet. This involves externally rotating the entire lower limb from the hip joint, keeping the knee and ankle in alignment with the anterior femur. This is a highly desired position and many dancers often turn out improperly to achieve the stance. Improper turnout involves forcing a joint (particularly the hip) beyond its capable range of motion. This action is compensated at the knee and ankle joint, causing strain on those everted structures, and consequently, on the os trigonum.[5,6,8] Any *pointe* exercises, including *relevé, plié,* or *grande plié,* cause such repetitive stress to this area that injury is not only likely, but expected.

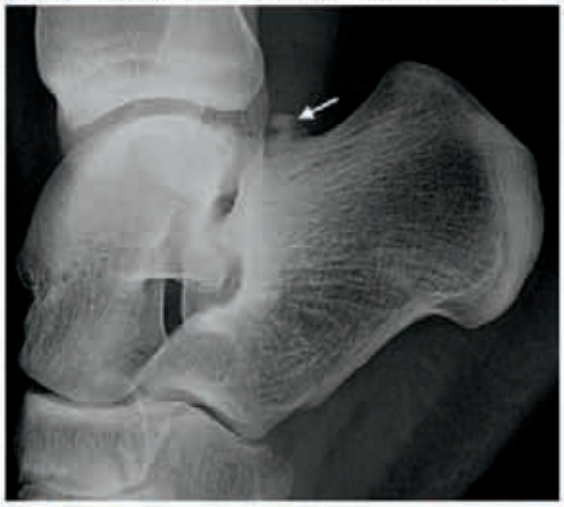

Figure 3: Radiograph showing os trigonum (arrow) (9)

Relevant Anatomy:

The os trigonum (originally described by Rosenmuller in 1804) is the second most common accessory ossicle, located at the posterolateral aspect of the ankle.[9,11] It is most often located superior to the calcaneus and distal to the tibial plafond (Figure 3).[9] The bone may be round, oval or triangular in shape, with

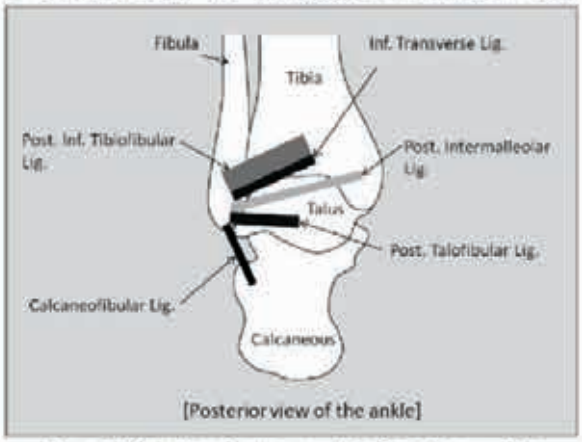

Figure 4: Relevant anatomy surrounding the os trigonum (4).

smooth margins, and may have an associated synchondrosis with the posterolateral talus.[1]

The bone begins as a secondary ossification site at the posterior aspect of the talus, appearing between ages 8-10 in females and 11-13 in males.[1,12] Usually, this site fuses to the talar body within, one year of appearance, but in 7% of the population, the site remains as a separate os.[1] 30% of all dancers have an os trigonum present, and account for 62% of all reported cases of OTS.[2,3]

The os trigonum is located lateral to the flexor hallucis longus (FHL), which runs between the medial and lateral tubercles of the posterior process of the talus.[1] Other nearby anatomical structures are demonstrated in (Figure 4).

Diagnosis:

Symptomatic Presentation:

The os trigonum is only a functional deficit if symptoms are present.[4,8] These symptoms can include pain in the retrocalcaneal space – described as a deep pain, swelling, tenderness, stiffness, and most often a "catching" feeling inside the posterior ankle during range of motion exercises.[12] The pain may be recurrent, and secondary synovitis can develop if symptoms are not addressed.[4,9] Dancers are reputed to have a high pain tolerance, and have an excess range of motion in ankle joints when compared to the general populous, so symptoms of OTS may go unreported for a significant amount of time.[5]

Other important indications of OTS include a history of trauma, ankle instability, or recurrent sprains, as these may indicate increased risk for developing inflammation in the posterior ankle. One important indication of OTS is reproducible pain, specifically worse when the foot is plantarflexed relative to the ankle, i.e. *en pointe* (A).

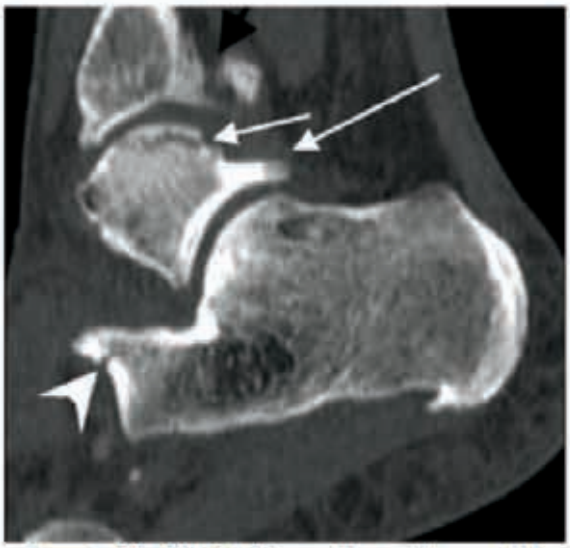

Figure 5: CT of Stieda's Process (long white arrow) (4)

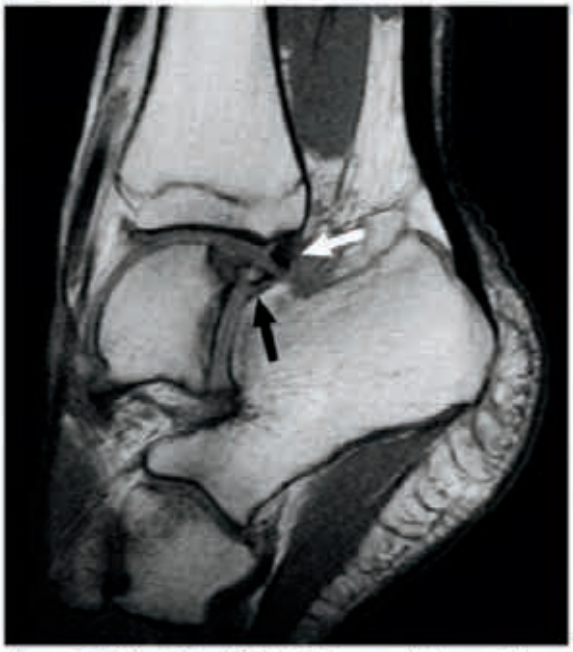

Figure 7: A T1-weighted MRI of a 16 year-old dancer with an os trigonum (white arrow) (1)

Differential Diagnoses:

The differentials for OTS include other osseous causes of PAIS, such as Stieda's process or a Shepherd fracture. A Stieda's process (Figure 5) is a posterolateral talar process that is contiguous with the talus.[4] When *en pointe*, this process can become compressed in the same manner as an os trigonum. If compressed with enough force, a break in the

Stieda's process, known as a "Shepherd's fracture" will result.[5] Other differentials for PAIS (listed in Figure 6) include osteophytes, loose bodies, pericapsular fat, retrocalcaneal bursitis, or tendonitis of the Achilles or flexor hallucis longus (FHL).[2,4,5,9]

Clinical Evaluation:

Clinical history and exam should be tailored to focus on the posterior ankle, the mechanism of injury, and reproducibility of symptoms. The history should focus on differentiating an overuse injury vs. a traumatic injury, as this will later determine treatment modalities. Physical exam includes forceful plantarflexion of a relaxed, non-weightbearing foot. This elicits pain in the posterior ankle.[1] The area may or may not be tender to palpation, and anterior drawer tests may elicit ankle instability.[12] Generally there is a loss in range of motion, a feeling of catching within the joint, and possible crepitus posterior to the malleolus.[9] The ankle should undergo a wide range of motion exercises, strength testing (particularly of the nearby FHL), and weightbearing and non-weightbearing exercises.

Imaging studies are warranted for full evaluation of possible OTS. Following Ottawa ankle rules, radiographs should be taken if the dancer is unable to bear weight and has tenderness to palpation in the area of concern.[4] The radiographs should be 3 views weight-bearing, with the lateral view providing the best visualization (Figure 3). If the patient is a dancer, then radiographs would be best taken while *en pointe* or *demi pointe*.

Following radiographs, an MRI (both T1 and STIR) is best to visualize the soft tissue structure surrounding the area (Figure 7).[4,5] If present, the os trigonum will be visualized as a sclerotic object, without central fatty

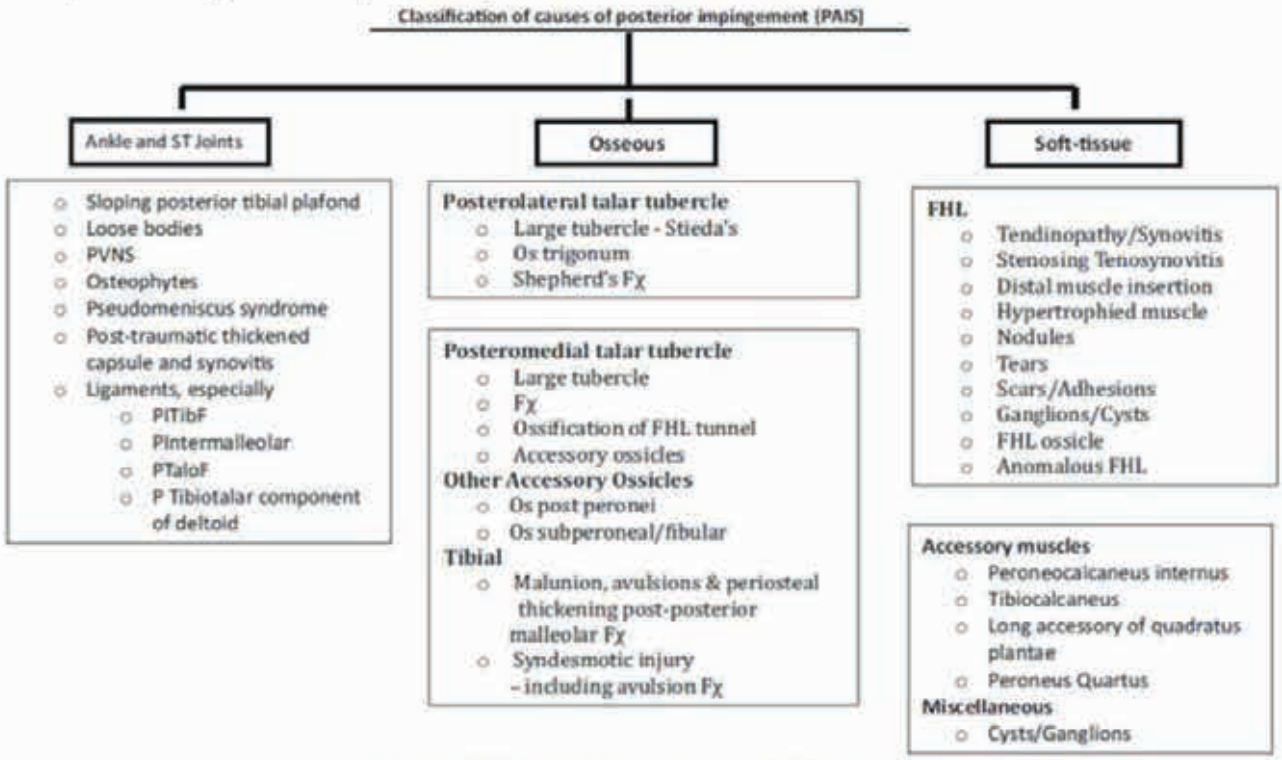

Figure 6: Differential diagnoses for PAIS (2)

marrow, differentiating it as a separate os, rather than an attached Stieda's. Other significant findings in MRI include increased fluid intensity suggestive of increased ankle instability, increased sclerosis within the posterior ankle suggestive of chronic stress to the area, and bone marrow edema which could indicate posterior impingement.[4] These symptoms alone are not definitive of OTS, but combined with history and physical exam, any of the above findings could indicate more long-standing and more complicated side effects of OTS. The most common concomitant finding of OTS is FHL tenosynovitis, present in 60% of all OTS cases.[1] This irritation of the tendon sheath will present on MRI as fluid around the FHL tendon which ends abruptly at the posterior talus, where the os trigonum lies.

Definitive diagnosis of OTS is made by ultrasound-guided injection of a local anesthetic, to see if pain is relieved after injection. Ultrasound of an os affected by OTS is expected to show hyper-echoic nodular capsular thickening, in the lateral tubercle of the posterior talar process (near to the os trigonum location).[4] After identification, the area surrounding the os may be injected with local anesthetic. Typically, 1-1.5mL of 1% Lidocaine is injected into the posterolateral capsule. If desired, the Lidocaine can be mixed with 0.5mL of triamcinolone (40mg/mL) to reduce symptoms of inflammation.[6,9,13] After injection, the physician should perform the physical exam again. Although specific parameters such as range of motion are not expected to change, the patient is expected to have pain relief. If the injection completely relieves pain, then the diagnosis is presumed Os Trigonum Syndrome, and the patient is presumed to be a good surgical candidate if desired.[11] If there is residual pain after the injection, then it may be presumed FHL tendon involvement as well, this may necessitate release.

Treatment:

Conservative:

The conservative treatment options are those expected in the case of inflammation of an injured joint, but special care should be taken with duration and patient adherence. Conservative measures may include any combination of icing, stretching, ultrasound, massage, NSAIDS, range of motion restriction, training modifications, or acupuncture.[1,5,9,11] Corticosteroid injection should be avoided due to risk of nearby FHL tendon rupture, a career ending rupture for dancers.[9] Rest is extensively encouraged, but for many dancers this is either not feasible or an unappealing treatment course. Rather than expect compliance to an unrealistic conservative treatment regimen, the physician should attempt to adapt treatment plans to dancing. This can include training modifications such as a specialized brace, range of motion restriction during full routines, extensive stretching, and avoidance of hard dance surfaces, as the resistance of the ground may prove more than the dancer's foot can withstand. *Pointe* work should be reduced from the usual 45+ hours per week (for professional dancers) to half, with dancers marking their steps rather than a full-out performance.[6] Dancers should be encouraged that conservative therapy must be attempted for 2-4 months before surgery can be considered.

Surgical:

Surgical decompression of the posterior ankle is achieved by complete excision of the symptomatic os trigonum. The arthroscopic two-portal technique most common today was originally described by van Dijk, et al.[6] Modifications have since been employed based on the injection procedure. Specifically, if the injection of local anesthetic completely relieves symptoms, than only an excision of the os trigonum need be performed, and the lateral

approach is preferred. If the injection fails to relieve all symptoms, then it is presumed that the nearby FHL tendon is involved, and a medial approach is preferred to facilitate release of the FHL tendon sheath.[6,9] The lateral approach involves excision posterior to the peroneals. The medial approach is posterior to the medial malleolus.

A full surgical technique is described in Vila et al (Figure 8).[3] Briefly, the patient is put under general or spinal anesthesia and place prone with a thigh tourniquet and a bump to support the lower extremity and allow free motion at the ankle. A 4mm scope is used to identify the symptomatic os, and provide vantage for excision. A 3.5mm shaver and burr are used to resect any remaining exostoses. The FHL and surrounding neurovascular structures should be identified and preserved. If performing a tendon release, the sheath of the FHL is incised and the tendon freed. After excision and release is completed, range of motion should be performed intra-operatively to ensure no impediment remains. Nylon sutures are used to close scope portals.

The benefits of an arthroscopic excision are improved patient satisfaction, faster recovery and return to activity, and reduced post-op complications such as scarring, morbidity, and wound complications.[7] Complications of surgery include damage to nearby neurovascular structures, ligamentous damage or joint defects.[5] Post-operatively, van Dijk described 3 weeks of non-weightbearing followed by physical therapy (PT) for 4-6 weeks.[14] More recent recommendations are to place the patient in a splint for 5 days, and then begin progressive weightbearing as tolerated in a supportive brace, with PT. Dancers should be warned that while final results can be achieved by 9 weeks, a return to previous abilities may not be achieved for 6 months.[5,9,14]

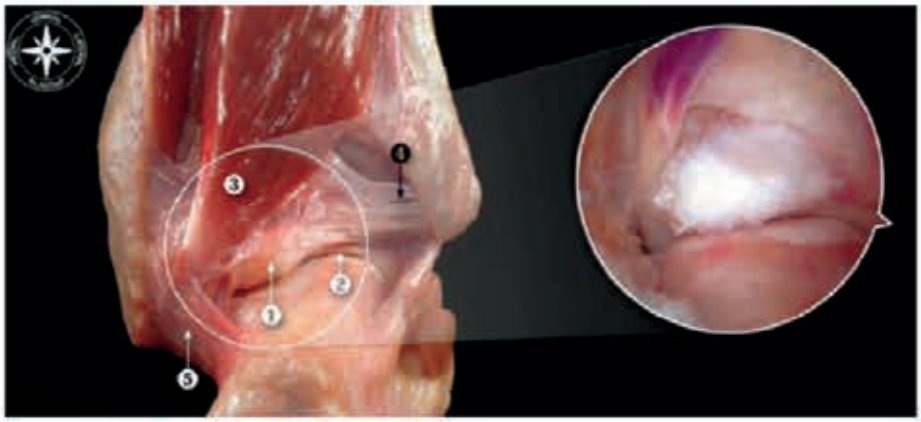

Fig. 8 Endoscopic view of the posterior region of the ankle compared with the anatomical view. 1. Postero-lateral process. 2. Posterior subtalar joint. 3. Flexor hallucis longus muscle. 4. Posterior talofibular ligament and intermalleolar ligament. 5. Flexor hallucis longus retinaculum.

Conclusion:

Os Trigonum Syndrome is a rare condition encountered by athletes, especially dancers, who hyper-plantarflex at the ankle joint, causing irritation and inflammation to the os and surrounding structures. Pain produced from activities such as *pointe* work, especially with improper turnout technique, can lead to complications in the posterior ankle. Ballet dancers are particularly vulnerable to non-fusion of the os trigonum if *en pointe* training begins during early teen years when the ossification site appears and unites with the talus.

Fortunately, accurate diagnosis is possible with thorough history and physical examination and adjunct imaging studies such as plain film radiographs and MRI. Definitive diagnosis is possible with local anesthetic injection to the area of irritation. The course of surgical planning (after conservative measures are exhausted) is dependent on this initial injection procedure. If pain can be completely relieved than excision of the os should relieve symptoms. If pain persists after injection, then the patient will likely need a release of the FHL tendon sheath in addition to os trigonum excision.

While many dancers develop an os trigonum due to early and strenuous *pointe* technique, it should be noted that only symptomatic cases require surgical intervention. Once symptoms arise, surgery is the definitive cure. However, symptoms can be reduced and serious injury avoided with proper attention given preventative care. Young dancers should be encouraged to avoid any undue stress of the joints before adequate turnout and strength requirements are achieved. Dancers of all ages should stretch thoroughly before and after dance training, to avoid shocking force to foot structures. If injury does occur, immediate steps should be taken to modify training. With these precautions any dancer, even in the presence of an os trigonum, can expect to have a long and fulfilling career.

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Case Study: Bilateral sequential lumbosacral plexopathy causing monophasic lower extremity weakness - a lesser-known manifestation of diabetic neuropathy

Michelle M. Happenny, COMP-Northwest 2017

Introduction

The term "diabetic neuropathy" encompasses a broader pathological spectrum than is commonly recognized. Presented here is a case study of an often-overlooked manifestation of
lower extremity neuropathy related to diabetes, as well as a summary of how to better assess patients' neurological complaints.

The Case

History of Present Illness

N. C. is an 80 year old African-American female with longstanding diabetes mellitus type II seen in Neurology clinic for electrodiagnostic studies. She complains of bilateral foot drop, leg muscle atrophy, and numbness.

In 2013, Mrs. C first noted numbness of the plantar aspect of the right foot. She developed similar symptoms in the left foot four or five months later. Since that time, the numbness progressed to involve both feet distal to the ankles. She also reports tingling in the fingers of both hands.

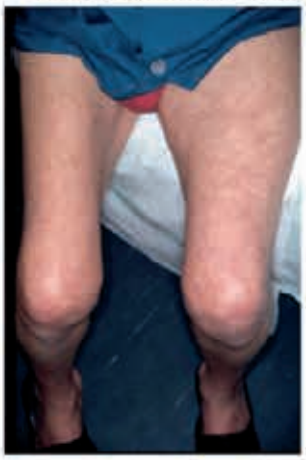


Figure 1: This patient suffered muscular atrophy due to diabetic plexopathy, albeit more prominently than our patient.[A]

In November 2014, a new set of symptoms developed. She first developed burning pain and swelling in her left foot, followed by weakness. Two months later, the right foot was similarly affected, with the events proceeding in the same fashion: pain, swelling, and then weakness. This newer pain resolved, but over the next few weeks, she developed bilateral foot drop as well as weakness in her thighs. Mrs. C then became non-ambulatory in the span of months.

Given this new set of symptoms, Mrs. C was referred to Physiatry for evaluation. A lumbosacral MRI was performed in November 2014 to evaluate the spinal cord and nerve root structures. This study revealed no evidence of herniated disc, severe spinal stenosis, nor foraminal encroachment to explain the symptoms. Repeat imaging in May 2015 revealed no significant or concerning interval change.

Interestingly, despite no medical intervention, the patient has regained strength during the latter part of 2015, after six months' plateau in her symptoms. As of December 2015, she was ambulatory with the assistance of a walker.

Further Medical History

In addition to her type II diabetes, Mrs. C's past medical history is significant for stage III chronic kidney disease, essential hypertension, hyperlipidemia, rheumatoid arthritis in remission, and anemia of chronic disease. She takes 81mg daily aspirin, 5mg daily lisinopril, and 300mg gabapentin three times a day. Up until two weeks prior to her electrodiagnostic testing, she had been on 5mg glipizide each morning, as well as 40mg daily lovastatin. These were discontinued due to side effects. Between 2004 and 2014 she had been on metformin (titrated up from 500mg BID to

1000mg BID over the years), but this was stopped due to renal impairment.

Mrs. C has had elevated hemoglobin A1c measurements on record dating from July 2001, when it was 6.7. Since that time the values have waxed and waned, ranging from 7.0 to 10.7 between April 2014 and September 2014. The most recent measurements from March, August, and September of 2015 were 6.5, 6.0, and 6.1, respectively.

Physical Examination

Vital Signs: Blood pressure 105/42, Pulse 80, Weight 109 lb.

General: Well groomed, thin-appearing female, appears stated age; appropriate affect

Neurologic: alert, fluent, following commands. Normal tone in bilateral lower extremities. Diffuse mild muscular atrophy of bilateral calves and feet. No fasciculation appreciated.

Strength: Deltoid, biceps, triceps, first dorsal interosseous, abductor pollicis brevis: 5/5 bilaterally. Iliopsoas, gluteus medius/minimus, hip adductors: 4/5 bilaterally.

Hamstring group and quadriceps: 4/5 bilaterally.

Tibialis anterior, extensor hallucis longus, flexor hallucis longus: 0/5 bilaterally.

Sensation: normal to light touch and vibration except: absent vibratory sense bilaterally at great toes, minimal at ankles, and normal at knees.

Reflexes: 2+/4 triceps, biceps, brachioradialis; 1+/4 patella bilaterally; 0/4 Achilles bilaterally.

Electrodiagnostic Studies

Nerve conduction studies (NCS) were performed of the right upper and lower extremities, evaluating sensory and motor function; electromyography (EMG) of the right lower extremity was also performed. A summary follows:

Sensory:

Median nerve: decreased velocity

Ulnar nerve: slightly decreased velocity, low amplitude

Radial nerve: slightly decreased velocity

Sural nerve: no response

Motor:

Median nerve (abductor pollicis brevis): decreased velocity

Ulnar nerve (abductor digiti minimi): within normal limits

Common peroneal nerve (tibialis anterior and extensor digitorum brevis): no response

Tibial nerve (abductor hallucis brevis): no response

EMG:

Tibialis anterior: 2+ fibrillations, 4+ positive sharp waves, no motor unit potentials recruited

Vastus lateralis: minimal insertional activity, 1+ positive sharp waves, normal motor unit potentials recruited

Discussion

Diabetic neuropathy is the most common complication of diabetes mellitus.[1] The risk of neuropathy is correlated with the amount of time one has suffered from diabetes: fewer than 10% of patients have clinically evident polyneuropathy at diagnosis, but prevalence rises to 50% after 25 years.[2]

Although distal axonal polyneuropathy is the most well-known form of diabetic neuropathy, also of note are autonomic neuropathy, diabetic ophthalmoplegia, acute mononeuropathies, a predilection toward compression neuropathies, and various polyradiculopathy syndromes.[2,3] Figure 2 provides a visual for several of these forms.

Diabetic Neuropathy: an "umbrella" term

The most familiar manifestation of diabetic neuropathy is also the most prevalent: distal, symmetric – "stocking and glove" – polyneuropathy, or DSPN.[4]

This form of neuropathy, caused by microvessel disease and subsequent ischemia of the nerve fascicular sheath, affects nerves in a length-dependent pattern.[2] It will first involve the longest nerves in the body and thus the patient will notice tingling, numbness, or burning/shock-like pain in their toes. With loss of protective sensation and proprioception, these individuals are at higher risk for falls, ulcers, and lower extremity amputations.[5] In this sense,

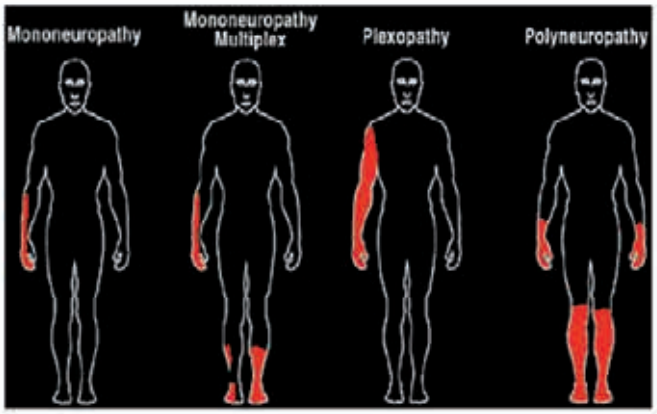
diabetic neuropathy is classically an assault on the lower extremity. As the disease process progresses, the rest of the feet and the legs will be involved, then fingers and arms. Most severely, the trunk could be affected, first along the midline and then extending laterally. On electrodiagnostic studies, one will find decreased amplitude of nerve potentials as well as mild to moderate slowing of conduction velocities, reflecting the length-dependent axonal degeneration of DSPN.[4,5]

Autonomic neuropathy is another manifestation of diabetes' impact on the nervous system. Patients will complain of or have findings consistent with impairment of sudomotor activity, such as difficulty with thermoregulation, dry eyes or mouth, pupillary changes, cardiac arrhythmias, postural hypotension, gastrointestinal manifestations such as gastroparesis, constipation, or diarrhea, and genitourinary effects like erectile dysfunction or incontinence.[4]

Cranial neuropathies in diabetes most commonly afflict cranial nerve III, leading to a "down and out" eye due to the spared actions of cranial nerves IV and VI, along with eyelid ptosis and a dilated pupil.[4] Next most frequently affected are cranial nerves IV and VI.[4] Involvement of cranial nerve VII and subsequent "facial droop" can occur in diabetes mellitus, but also from other etiologies, or idiopathically (as in Bell's palsy).[5]

Mononeuropathies, such as carpal or cubital tunnel syndromes, are by no means pathognomonic for diabetic neuropathy, although diabetic patients do show a tendency toward these sorts of "compression" neuropathies. The most common locations of compression and subsequent nerve damage are the wrist (median nerve), elbow (ulnar nerve), and fibular head (peroneal nerve).[4] As can be deduced from the name, these syndromes will involve a single nerve in a predictable distribution, or in the case of mononeuropathy multiplex, multiple peripheral nerves.[1]

Finally, the remainder of diabetic neuropathies include plexopathies and polyradiculopathies (also known as 'amyotrophy') and diabetic cachexia. The latter can be differentiated from amyotrophy by the presence of weight loss and wasting accompanied by pain, without specific weakness; however, polyradiculopathy syndromes in diabetes will involve pain along with weakness (unilateral or bilateral) of discrete muscle groups, as a subacute and monophasic episode.[5] There is typically a slow, variable recovery of function without any particular therapeutic intervention hastening the response.[2] The lumbosacral plexus is most frequently affected, but thoracic and cervical



plexopathies are not unheard of; however, the upper extremities are only rarely affected by this process.[2] Overall, according to *Harrison's Principles of Internal Medicine*, one-third of patients with diabetic neuropathy will suffer from such a syndrome.[4]

Figure 2: Depiction of several types of neuropathies.[B]

Pathophysiology

Hypotheses about the etiology of diabetic neuropathy range from ischemia of the vasa nervorum, to perivascular inflammation and damage to neighboring nerve fascicles, to building-block-level disruptions of myo-inositol transport or sorbitol metabolism.[2] Whatever the final verdict is at this point in time, experts agree that hyperglycemia as found in poorly controlled diabetes causes a metabolic derangement that results in nerve damage.[6]

Diabetic lumbosacral radiculoplexus neuropathy (DLRPN) and lumbosacral radiculoplexus neuropathy (LRPN) are thought to be immune-mediated phenomena given the

multifocal, "patchy" distribution of ischemic and vasculitic changes noted on nerve biopsies. The typical metabolic ischemia - endoneurial microvessel disease - of DSPN is remarkably absent in these samples. Furthermore, similar patient symptoms, clinical findings, and

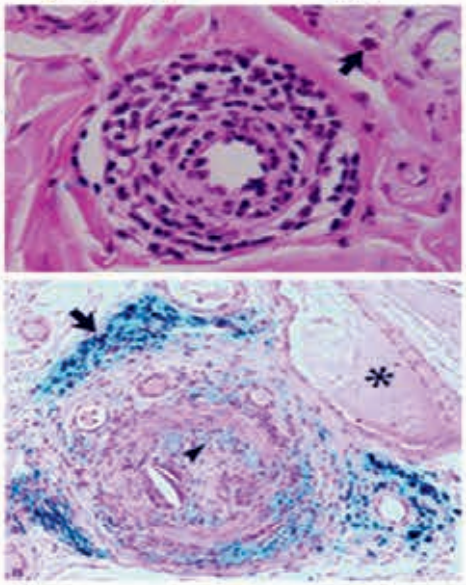


Figure 3: Epineural microscopic vasculitis in DLRPN.[1,C]

histological change in both DLRPN and LRPN, suggest that hyperglycemia – the key difference between these patient populations – plays a somewhat lesser role in the disease process.[7] Given Mrs. C's history of rheumatoid arthritis, an immunologic basis for DLRPN seems to fit, at least in the context of this particular case.

There are also studies that associate impaired glucose tolerance, or "pre-diabetes," with the development of sensory neuropathy. In one small prospective study of 107 patients with idiopathic neuropathy, 36 participants were later found to have impaired glucose tolerance based on American Diabetes Association criteria.[8] Unfortunately, more robust research has yet to be conducted, including whether it pertains to plexopathy as well. Effort is being put into evaluating the association between hyperglycemia, dyslipidemia, inflammatory factors, and activation of the renin-angiotensin-aldosterone system – components of the metabolic syndrome – and neuropathy.[5]

Regarding risk factors for this particular type of neuropathy, a specific HbA1c cut-off has not yet been determined. In the 2008 ADVANCE randomized controlled trial that looked at strict vs. typical glycemic control in 11,140 patients, there was no significant difference in "new or worsening neuropathy" between the control group and intervention group (7.3% vs. 6.5% HbA1c).[9] Perhaps the situation is more apt to arise in certain clinical scenarios: according to *Adams and Victor's Principles of Neurology*, diabetic multiple mononeuropathies and plexopathies "often emerge during periods of transition in the diabetic illness," such as when insulin is added to the regimen or adjusted in some way.[2]

Diagnosis

In evaluating any neurologic complaint, *Harrison's* advocates a useful method of "Seven Key Questions" for any clinician to utilize.[4] We will apply them to Mrs. C's case to assist with classifying this disorder.

1.	What systems are involved?
2.	What is the distribution of weakness?
3.	What is the nature of the sensory involvement?
4.	Is there upper motor neuron involvement?
5.	What is the temporal evolution?
6.	Is there evidence for a hereditary neuropathy?
7.	Are there any associated medical conditions?

Table 1: Seven key questions in evaluating the patient with neuropathic signs or symptoms.[4,D]

1. Mrs. C had both sensory and motor involvement.
2. Weakness was primarily distal, and asymmetric at the onset but becoming symmetric over time. Some objective proximal weakness was also noted on examination.
3. Per the HPI, Mrs. C suffered from burning pain, which we would consider a small fiber manifestation.

4. No upper motor signs (spastic paralysis or paresis, hyperactive reflexes, pathologic reflexes) were noted in Mrs. C's examination.[10]
5. The unilateral evolution of Mrs. C's symptoms would fall into the acute (0-4 weeks) category, with the bilateral involvement being better termed subacute (4-8 weeks). The term "sequential" best describes that the bilaterality was stepwise.
6. No family history of similar symptoms.
7. Mrs. C's longstanding diabetes provided a major clue to her situation, and her rheumatoid arthritis is another key diagnosis; prior workup for other autoimmune disorders or contributors like vitamin imbalances (i.e., B6 toxicity or B12 deficiency) were negative.

Now we can meaningfully describe the facts of the case. Mrs. C. suffered a subacute, distal > proximal, asymmetric, sensorimotor disorder with a history of type II diabetes mellitus. *Harrison's* system of pattern recognition, Table 2, can now help us with diagnosis. We see that Mrs. C best fits Pattern Four, allowing us to narrow the differential diagnosis.

Impression

In addition to length-dependent DSPN, Mrs. C's clinical picture supports a diagnosis of bilateral sequential diabetic lumbosacral plexopathy. The heavily affected nerve roots in Mrs. C's case include L5, S1, and S2, as evidenced by her physical examination as well as electrodiagnostic testing.

Pattern 1	**Symmetric proximal and distal weakness with sensory loss** - Inflammatory demyelinating polyneuropathy
Pattern 2	**Symmetric distal sensory loss with or without distal weakness** - Cryptogenic or idiopathic sensory polyneuropathy, diabetes mellitus and metabolic disorders, drugs, toxins, amyloidosis, Charcot-Marie-Tooth disease
Pattern 3	**Asymmetric distal weakness with sensory loss** - *Involving multiple nerves:* multifocal CIDP, vasculitis, cryoglobulinemia, amyloidosis, sarcoidosis, infectious causes, tumor infiltration - *Involving single nerves/regions:* the above plus compressive mononeuropathy, plexopathy, radiculopathy
Pattern 4	**Asymmetric proximal and distal weakness with sensory loss** - Polyradiculopathy or plexopathy due to diabetes mellitus, meningeal carcinomatosis or lymphomatosis, hereditary plexopathy, idiopathic
Pattern 5	**Asymmetric distal weakness without sensory loss** - *With upper motor neuron findings*: motor neuron disease - *Without upper motor neuron findings:* progressive muscular atrophy, multifocal acquired motor axonopathy, multifocal motor neuropathy
Pattern 6	**Symmetric sensory loss and distal areflexia with upper motor neuron findings** - Vitamin or nutritional deficiency (B12, E, copper), hereditary leukodystrophies
Pattern 7	**Symmetric weakness without sensory loss** - Proximal and distal weakness: spinal muscular atrophy - Distal weakness: hereditary motor neuropathy, atypical Charcot-Marie-Tooth disease
Pattern 8	**Asymmetric proprioceptive sensory loss without weakness** - Paraneoplastic syndrome, Sjögren's syndrome, chemotherapeutic agents, vitamin B6 toxicity, HIV-related sensory neuronopathy, idiopathic

Pattern 9	**Autonomic symptoms and signs** - Hereditary sensory and autonomic neuropathy, amyloidosis, diabetes mellitus, porphyria, HIV-related autonomic neuropathy, chemotherapeutic agents

Table 2: Patterns of neuropathic disorders.[4, D]

Treatment

The ultimate concern regarding treatment of diabetic neuropathy is primary or secondary prevention through optimal glycemic control, via dietary and exercise modifications along with the aid of pharmacologic agents.[5] A second major concern is pain management. Among the options available for use in diabetic neuropathy, pregabalin, gabapentin, venlafaxine, duloxetine, and amitriptyline are recommended by the American Academy of Neurology, American Association of Neuromuscular and Electrodiagnostic Medicine, and the American Academy of Physical Medicine and Rehabilitation.[11] If pain is refractory to these agents, tramadol, oxycodone, or morphine sulfate can be tried.[11] This coalition notes that diabetic neuropathic pain is generally a chronic condition and that there is no evidence that these medications are effective in the long term, despite trials that indicate short term use is beneficial.

Regarding diabetic amyotrophy, the Cochrane Database attempted to determine whether there was evidence for the use of immunotherapy as treatment. As of 2012, only one controlled trial of intravenous methylprednisolone for the treatment of diabetic amyotrophy was found, and the results had not yet been published.[12] However, a 2014 case described one patient's positive response to 8 years of serial treatments with intravenous immunoglobulin for relapsing DLRPN; the researchers found that previously elevated serum levels of tumor necrosis factor α normalized after completion of each course.[13]

According to *Adams and Victor's*, "Motor recovery [in plexopathy] is the rule although months and even years may elapse before it is complete."[2] As the mechanism of the onset is unknown, so is the reason for the resolution. It may be comforting to remind patients of the typical course of motor symptoms once the diagnosis is made.

Conclusion

The looming presence of diabetes mellitus in our population warrants increased familiarity with the subject of diabetic neuropathy. Indeed, as demonstrated by the lack of robust evidence, more data on plexopathy in particular needs to be obtained.

N. C.'s case provides an interesting study of pathology affecting the lower extremity, an illustration of the variety of diabetic neuropathy, and serves as an example of neurologic assessment that is user-friendly for budding clinicians. It also reminds us that no matter how unfamiliar a clinical situation, careful evaluation of the patient can provide the answer and thus guide appropriate treatment.

Acknowledgements

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Total Ankle Arthroplasty versus Ankle Arthrodesis: a Meta-Analysis.

Jordan Crafton, DPM 2017

Abstract

Background

The choice of total ankle replacement or ankle fusion has been a debated topic for end-stage arthritis of the ankle joint. For many years, ankle arthrodesis was the gold standard for patients with end-stage ankle arthritis. A systematic review and meta-analysis led by Haddad et al assessed studies at a chronological end-point of March 2005. Nearly 10 years have passed since then. The goal of this meta-analysis was to serve as a comparative update, assessing the literature from April 2005-July 2014.

Objective

This current meta-analysis reviews the outcomes of total ankle arthroplasty and ankle arthrodesis, for the chronological period of April 2005-July 2014.

Methods

Two comprehensive literature searches of PubMed were conducted. For one search, "total ankle arthroplasty" was used as the search term; for the second search, the search term "ankle arthrodesis" was used. Exclusion criteria were identical to those proposed by Haddad et al. While 326 articles were identified, 224 articles were excluded. This yielded 40 arthrodesis studies with 1599 ankles (1562 patients) and 62 arthroplasty studies with 9079 ankles (8268 patients).

Results

Though the percentage of excellent subjective outcomes decreased compared to the earlier meta-analysis, the percentage of good subjective outcomes, as well as combined good/excellent subjective outcomes, substantially increased in the ankle arthroplasty group. The percentage of poor subjective outcomes decreased as well, though the implant revision rate slightly increased.

Conclusion

In the past 10 years, patient satisfaction following ankle arthroplasty has improved. This trend may be explained by improvements in implant technology and physician training. As a caution, revision rates have slightly increased as well. Ankle arthrodesis outcomes have not significantly changed during this time period.

Introduction

The decision to use total ankle arthroplasty or ankle arthrodesis for debilitating ankle impairments has been a debated topic among foot and ankle surgeons for quite some time. The most common indications, according to Haddad et al., are post- traumatic injury, osteoarthritis, rheumatoid arthritis, and osteonecrosis among other causes[1]. The gold standard for these types of injuries has been ankle arthrodesis, but with recent advancements, total ankle arthroplasty has been implemented much more often than before. Haddad et al. also concluded, "…the intermediate outcome of total ankle arthroplasty appears to be similar to that of ankle arthrodesis…[1]" They also reported lower revision and amputation rates with arthroplasty compared to arthrodesis[1]. However, this article's research was performed from January 1, 1990-March 2005. This current meta-analysis investigates cases that have been reported in the literature from April 2005-July 2014 in regards to the outcomes of total ankle arthroplasty and ankle arthrodesis.

Methods and Patients

A comprehensive search of PubMed was performed for all relevant articles published between April 2005-July 2014 using key words "ankle arthroplasty" for one search and the other using "ankle arthrodesis." 326 articles were

included initially. Upon further review 224 articles were excluded based on certain criteria leaving 40 arthrodesis studies with 1599 ankles (1562 patients) and 62 arthroplasty studies with 9079 ankles (8268 patients). Exclusion criteria were:

1. Authors report on total ankle arthroplasty or tibiotalar ankle arthrodesis or other studies from each group that provides sufficient data.
2. Outcomes of interest at an average of 2 years or more after the operation
3. At least 10 patients in the treatment group.
4. Revision only studies are excluded.
5. All study designs are eligible including randomized controlled trials, prospective and retrospective non-randomized controlled trials and uncontrolled case series.

Using Microsoft Excel, data were compiled and calculated. The arthroplasty group included 2941 females and 2842 males with a mean age of 60.9 years, and the arthrodesis group included 432 females and 763 males with a mean age of 50.0 years. Some studies did not report data on gender. The mean follow-up time for arthroplasty was 51.3 months and the arthrodesis group was 54.2. Patients were evaluated using various scoring systems including AOFAS, VAS (pain), and evaluated using subjective outcomes of excellent, good, fair and poor, which were then pooled according to the following: very satisfied, good plus excellent/satisfied, moderately satisfied, and disappointed/somewhat disappointed. Good plus excellent was not separated in some studies so a combined good plus excellent category was created. 5 and 10-year survivorship data were recorded for arthroplasty. Revision rates, conversion to arthroplasty, amputation rates and the reasons behind each decision were also analyzed.

Results

Subjective Outcomes, AOFAS scores, VAS scores, and Survivorship

For subjective outcomes of excellent, good, fair, and poor, 1218 patients reported outcomes of 14.29%, 71.76%, 9.52% and 4.43% respectively, for arthroplasty with a combined good plus excellent outcome of 86.05%. In arthrodesis cases, 735 patients reported on excellent, good, fair, and poor of 22.02%, 50.45%, 17.41% and 10.12% for arthrodesis respectively with a combined good plus excellent outcome 71.4%. The mean AOFAS scores for arthroplasty and arthrodesis were 79.76 for 3393 patients reporting and 78.20 for 796 patients reporting respectively. VAS scores for arthroplasty and arthrodesis were 3.9 for 2451 patients reporting and 7.1 with 164 patients reporting respectively. Average 5 and 10 year survivorship data for arthroplasty patients was reported at 87.69% and 75.91%.

For completeness 3 yr. (4 studies), 4 yr. (5 studies), 6 yr. (5 studies), 8 yr. (3 studies), 14 yr. (1 study) and 15 year (1 study) survivorship were 87.9%, 92.1%, 85.4%, 89%, 45.6% and 73% on average respectively with limited data.

DEMOGRAPHICS		
	Total Ankle Arthroplasty	Ankle Arthrodesis
Female:Male	50.5% / 49.5%	37.2% / 62.8%
Mean Age	60.9 years	50.0 years
Mean Duration of Follow up	51.3 months	54.2 months

Revision and Amputation Rates

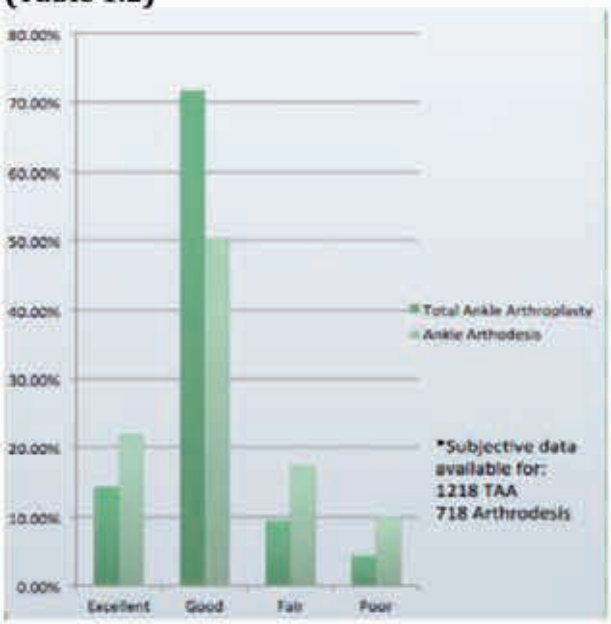
Revision rates were reported for arthroplasty at 13.61% with the most common causes being categories of aseptic loosening/subsidence/persistent pain/implant failure (27.77%), stiff ankle/impingement/bone fragments/pain/arthrofibrosis, bone overgrowth (10.16%), varus or valgus

deformity/instability/malalignment, deformity in general (10.16%), infection (9.3%), and polyethylene insert/bearing subluxation/implant failure (9.2%). The arthrodesis group had a revision rate of 10.26% with the most common causes being categories of non-union/unsatisfactory union/malalignment (32.24%), infection (21.05%), hardware malfunction (15.79%), and pain (10.53%). The amputation rate for arthroplasty was .07% and for arthrodesis it was 1.13%. (*see Table 1.1 and 1.2*)

(Table 1.1)

Table 1.1		
Revisions and Amputations		
Revisions		
Total Ankle Arthroplasty	**Revsions/Ankles**	**%**
Total Ankle Arthroplasty Revision Rate	1236/9079	13.61%
Conversion to Arthrodesis Rate	168/9079	1.91%
Amputation rate	6/9079	0.07%
Most Common Causes of Revision		
Aseptic loosening/subsidence/persistant pain/implant failure	368/1236	29.77%
stiff ankle/impingment/bone fragments/pain/arthrofibrosis/Bone overgrowth	105/1236	10.16%
varus or valgus deformity/instability/malalignment /deformity in general	105/1236	10.16%
PE insert/bearing sublux/implant failure	95/1236	9.20%
Conversion to Arthrodesis % of revisions	175/1236	16.94%
Amputation % of revisions	6/1236	3.43%
Arthrodesis	**Revsions/Ankles**	**%**
Total Revisions Tibiotalar Arthrodesis	152/1599	10.26%
Amputation rate	16/1599	1.13%
Most common Causes of Revision		
Non-union/unsatisfactory union/malalignment	49/152	32.24%
Infection	32/152	21.05%
Hardware malfunction	24/152	15.79%
Pain	16/152	10.53%

(Table 1.2)



Discussion

This systematic review explored how outcomes and patient satisfaction have changed since Haddad et al.[1] reported the outcomes for ankle arthroplasty and arthrodesis in 2007. They reported AOFAS scores for arthroplasty and arthrodesis at 78.2 and 75.6 respectively compared to this review where they were 79.76 and 78.20 respectively. Haddad et al. did not report VAS in their report and although some studies reported VAS score, the data was limited. Haddad et al. reported subjective outcomes for excellent, good, fair and poor at 38%, 30.5%, 5.5% and 24% respectively for ankle arthroplasty. Ankle arthrodesis was reported for excellent, good, fair and poor at 31%, 37%, 13%, and 13% respectively. Comparatively, this current review reported corresponding values for excellent, good, fair, and poor outcomes for arthroplasty at 14.29%, 71.76%, 9.52%, 4.43% respectively, and for arthrodesis the corresponding values were 22.02%, 50.45%, 17.41%, and 10.12% respectively. The 5 and 10 year survivorship for ankle arthroplasty in the study by Haddad et al. was 78% and 77% respectively. In this current study the survivorship was 87.69% and 75.91% respectively.

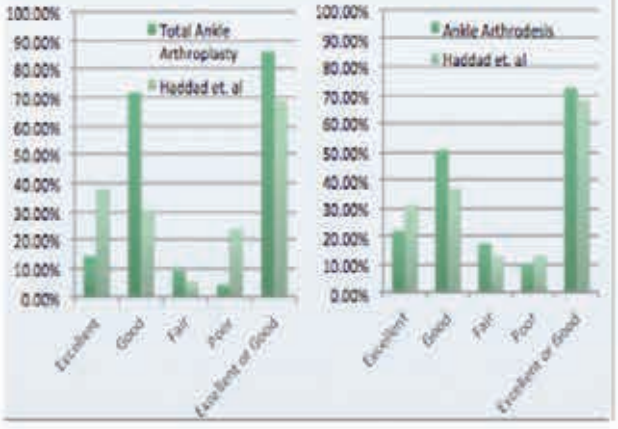
Revision rates for their study were 7% for arthroplasty, with the most common cause being loosing and/or subsidence (28%) and for arthrodesis the revision rate was 9% with nonunion being the most common cause (65%). Corresponding revision rates for this current review were 13.65% for arthroplasty, with the most common causes being the complication group consisting of aseptic loosening/subsidence/persistent pain/implant failure (29.77%) and for arthrodesis 10.26% with the most common causes being the complication group consisting of non-union/unsatisfactory union/malalignment (32.24%). Amputation rates for their study were 1% for arthroplasty and 5% for arthrodesis. This current study reports corresponding data of .07% and 1.13%.

(Table 2)

RESULTS: Side by Side Comparison with Previous Study

	Current Study	Haddad et al. study	Difference	Current Study	Haddad et al. study	Difference
	Total Ankle Arthroplasty	Total Ankle Arthroplasty		Ankle Arthrodesis	Ankle Arthrodesis	
AOFAS	79.70	78.20	+1.50	72.61	75.60	-2.99
VAS	3.9	N/A	N/A	7.1	N/A	N/A
Excellent	14.29%	34%	-33.71%	31.00%	31.00%	-9.0%
Good	71.76%	30.60%	+41.26%	50.48%	37.00%	+13.48%
Fair	8.82%	8.82%	+0.02%	17.41%	13.00%	+4.41%
Poor	4.63%	24%	-19.37%	10.13%	13.00%	-2.88%
Combined Excellent and Good	86.05%	68.50%	+17.55%	72.47%	68%	+4.47%
5-year survivorship	87.69%	78%	+9.69%	N/A	N/A	N/A
10-year survivorship	75.91%	77%	-1.09%	N/A	N/A	N/A
Revision	13.65%	7%	+6.65%	10.26%	9%	+1.26%
Amputation Rate	0.07%	1%	-.93%	1.13%	5%	-3.87%

RESULTS: Side by Side Comparison with Previous Study



5-year survivorship has increased ankle arthroplasty by about 10%, although 10 year survivorship decreased by 1.5%. (can you be a little more clear on what you're comparing here?) Most notably, the percent of good plus excellent outcomes for total ankle arthroplasty has improved from 68.5% to 85.9%; a 17% increase. That same data for arthrodesis has increased by 4.47% from 68% to 72.47%. Revision rates have barely increased- 5.7% for arthroplasty and 1.1% in arthrodesis. Amputation rates for both procedures have decreased. Arthroplasty has dropped by .03% and arthrodesis by 3.87%. AOFAS scores stated relatively the same although improvement of 1.56 points in arthroplasty and a decrease by 2.99 points in arthrodesis occurred (see table 2 below). The VAS pain score for arthroplasty was 4.2 points lower than for arthrodesis although data were very sparse on the arthrodesis side.

Arthroplasty is continuing to become utilized more often and provide better results despite the increase in revision rates. Survivorship and patient satisfaction are increasing for both procedures, but much more dramatically in ankle arthroplasty. The gold standard treatment for ankle arthritis has always been arthrodesis, but as this study has shown, patient satisfaction, AOFAS, VAS, and amputation rates in total ankle arthroplasty are superior to ankle arthrodesis. The complication rate from arthroplasty increased, but the risk is possibly outweighed by the other scores and outcomes reported.

Conclusion

Notable improvements can be made to this study. Scores such as AOFAS and VAS were reported mostly for the total patient base even with patients that were lost to follow-up or who had died before questioning. The patient number reported for those scores is likely lower but the average scores are still of worth mention. Additionally, one AOFAS score was included that was recorded at 12 months instead of 24.

Nonetheless, this study is worthwhile and clinically relevant due to the subjective outcome increase that patients reported with combined excellent/good results in the arthroplasty group as well as a substantial decrease in the amount of poor results in the same group. 5-year survivorship increases, and a decrease in the overall amputation rate compared to the Haddad et al study were impressive findings. Careful selection is needed by the clinician to determine which procedure is best for the patient on an individual basis but it appears that total ankle arthroplasty patient outcomes have improved. This could be due to improved training of surgeons, better technology of implants or rather, more likely, a combination of both. There is much skepticism surrounding surgeons performing ankle replacements due to the complexity of the foot and ankle joints, as well as the risk of patients needing a conversion to an arthrodesis or an amputation. From this study it appears that the risks are decreasing. Total ankle arthroplasty may be the answer to debilitating ankle arthritis that no other procedure has been able to adequately solve.

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Polymyalgia Rheumatica and Giant Cell Arteritis and their Effect on the Lower Extremity

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Introduction

Polymyalgia Rheumatica (PMR) is an inflammatory rheumatic condition that presents with aching and morning stiffness in muscles of the shoulders, hips, and neck, as well as edema and tenosynovitis in the hands and feet. Giant Cell Arteritis (GCA) can be associated with PMR and is a vasculitis of medium and large sized vessels. It has been observed by Warrington that patients with PMR and GCA may develop a lower extremity (LE) vasculitis that can increase the risk of LE claudication.[21]

Although LE involvement of vasculitis and peripheral arterial disease (PAD) is less frequently seen in patients with PMR and GCA compared to extra-cranial involvement and other presentations, studies suggest that these findings may cause significant morbidity, thus requiring immediate treatment. One study between a population of GCA patients and a control group found that 12% of the GCA patients had LE artery abnormalities such as stenosis and vasculitis compared to none in the control group. [20] Additionally, 6% of the GCA patients had occlusive changes in the posterior tibial artery and 3% had occlusive changes in the dorsalis pedis artery. Another study concluded that about 21% of patients with GCA had LE manifestations of vasculitis. [21]

Although these studies contained a small population of GCA patients, repeated demonstration of similar results suggest that physicians may be overlooking LE vasculitis and PAD in patients with GCA and PMR.

Epidemiology

Polymyalgia Rheumatica and Giant Cell Arteritis occur almost exclusively in adults over the age of 50. There is an increased prevalence with age, the highest incidence being in people aged 70-80.[1] Regionally, PMR and GCA has its highest incidence in Scandinavia and in people of Northern European descent.[5]

Patients diagnosed with GCA have a 50% chance of developing PMR[2,] while patients with PMR have a 5-30% chance of developing GCA.[1,3,4] Some patients with PMR and GCA have developed a lower extremity vasculitis leading to peripheral artery disease.

In a population-based study of 168 GCA patients, 46 patients presented with large-artery complications while only one patient (0.6%) had lower extremity stenotic disease attributed to GCA.[11] PMR patients were at increased risk of developing PAD compared with the referent cohort (hazard ratio 2.50; 95% CI 1.53–4.08).[21]

Pathogenesis

The etiology of PMR and GCA has been associated with sequence polymorphism in two different domains of the human leukocyte antigen (HLA)-DR4, more specifically HLA-DRB1*04 alleles: B1*0402 and B1*0403. GCA also has a similar distribution in the HLA-DRB1 alleles which map the antigen binding cleft for the HLA-DR4 molecules, indicating an important role in antigen binding and presentation in both PMR and GCA.[8] A mutation within the HLA domains causes an alteration to the cells that control the immune system, resulting in unregulated inflammation.

Multiple studies have shown that there are increases in pro-inflammatory cytokines in patients with PMR/GCA. PMR patients have been shown to have higher than normal levels of pro-inflammatory cytokine IL-6, which is believed to cause the constitutional symptoms of PMR.[9] The levels of Th17 have been shown to be increased, while Treg has been shown to be decreased in patients with PMR. Th17 cells produce IL-17, a pro-inflammatory cytokine, that is synergistic with tumor necrosis factor (TNF) and IL-1. TNF is known to cause apoptosis and IL-1 is a regulator of pro-

inflammatory cytokines. Treg is a suppressor cell that regulates the immune system, prevents the body from attacking self-antigens, and from developing autoimmune diseases.[10]

The lower extremity vasculitis that can be present in PMR and GCA is currently not well understood, but has been hypothesized to be related to dendritic cells located in the media-adventitia border of blood vessels. The dendritic cells become activated by toll like receptors within the vessels and mediate an immune response involving macrophages and pro-inflammatory cytokines that cause the vasculitis. The chronic inflammation caused by PMR/GCA is thought to be the cause of peripheral artery disease.[21]

Figure 1: Th1-cell-mediated and Th17-cell-mediated immunity.[11]

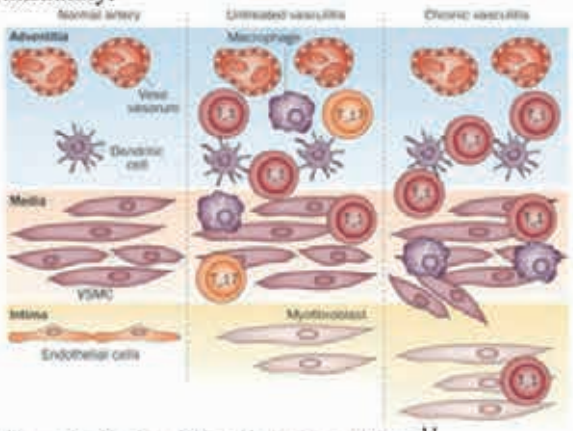
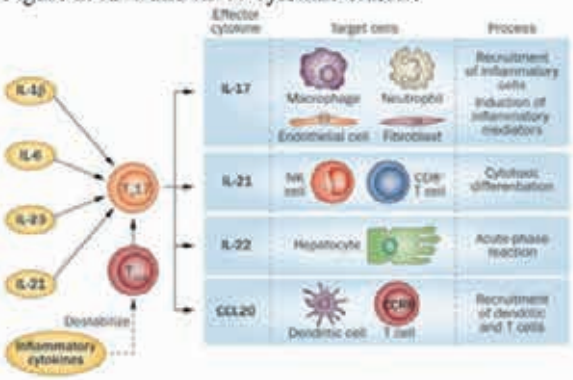


Figure 2: IL-6 and IL-17 cytokine cluster.[11]



Clinical Manifestations

PMR is commonly characterized by symmetric morning stiffness and aching at joints in the neck, shoulders, torso, and hips. Stiffness is present in all patients with PMR, which can last an hour or longer. This stiffness can become more severe following extended periods of inactivity. Pain is the second most common symptom of PMR affecting the same joints. PMR can also manifest as bursitis or synovitis at distal joints in the knees, wrists, hands, and feet. Although not as common, swelling can occur in patients afflicted with PMR at the distal joints as well. This swelling can present in the ankles and feet as well as the wrists and hands.[1]

Some of the less common symptoms associated with PMR include a decreased range of motion, weakness, fatigue, and a low-grade fever. Muscle tenderness is also a rare feature of PMR, which may lead to confusion due to the term "polymyalgia" in the name of the disease. Patients with PMR have a higher than normal tendency to develop LE claudication and PAD according to several studies.[20, 21] One such study using a population based cohort of 353 PMR patients found that despite adjusting for comorbidities such as hypertension, dyslipidemia, and diabetes mellitus, PMR patients were still at an increased risk of developing PAD and claudication compared to the referent cohort.[21]

GCA may present initially with systemic symptoms that include fever, fatigue, and weight loss. Another frequently noted symptom in GCA patients is a new onset headache, which has begun to persist acutely. These headaches tend to be located around the temples. Jaw claudication is seen in about half the patients with GCA which is noticed rapidly at the onset of chewing leading to severe pain. Temporary or permanent vision loss is a less commonly seen symptom but is one that needs to be monitored as it is considered among the most feared complications of GCA due to how sudden and painless the vision loss can occur. GCA can also involve the large vessels such as the aorta and its major branches, which can lead to dissection, aneurysm, stenosis, and occlusion.

Figure 3: European League Against Rheumatism and the American College of Rheumatology (EULAR/ACR) classification criteria for PMR.[6]

PMR classification criteria scoring algorithm—required criteria: age 50 years or older, bilateral shoulder aching and abnormal CRP and/or ESR*

	Points without US (0–6)	Points with US† (0–8)
Morning stiffness duration >45 min	2	2
Hip pain or limited range of motion	1	1
Absence of RF or ACPA	2	2
Absence of other joint involvement	1	1
At least one shoulder with subdeltoid bursitis and/or biceps tenosynovitis and/or glenohumeral synovitis (either posterior or axillary) and at least one hip with synovitis and/or trochanteric bursitis	Not applicable	1
Both shoulders with subdeltoid bursitis, biceps tenosynovitis or glenohumeral synovitis	Not applicable	1

*A score of 4 or more is categorised as PMR in the algorithm without US and a score of 5 or more is categorised as PMR in the algorithm with US.

LE involvement in patients with GCA may include stenotic disease, claudication, vasculitis, as well as a propensity for peripheral arterial disease. According to a study of GCA patients with LE vasculitis, it was found that 84.2% of the patients had symptoms of LE claudication an average of 3 months prior to the diagnosis of GCA.[20]

Diagnosis

There is a classification criteria developed by the European League Against Rheumatism and the American College of Rheumatology (EULAR/ACR). The criteria includes: being age 50 and older, morning stiffness, high level of CRP or ESR, hip pain or limited range of motion, absences of rheumatoid factor or anticitrullinated protein antibody, absence of other joint involvement and shoulder bursitis/synovitis. Each of the symptoms are given a numerical value (Figure 3) if the score reaches greater than 4 (or 5, depending on whether an ultrasound is used to identify underlying bursitis or inflammation) then a diagnosis of PMR is made.[6] A study was done to evaluate the sensitivity and specificity of the PMR classification criteria developed by the EULAR/ACR, showing 93% sensitivity and 82% specificity. With an ultrasound included to examine the shoulder and hips, there was an increase of specificity from 82% to 91%.[7] Some clinicians use the EULAR/ACR criteria along with a low dose glucocorticoid to see if the symptoms resolve quickly, which would help support the diagnosis of PMR.

The diagnosis of giant cell arteritis is based on a patient being age 50 and older with symptoms of new headaches, abrupt onset of visual disturbances, symptoms of polymyalgia rheumatica, jaw claudication, unexplained fever or anemia, high erythrocyte sedimentation rate (ESR) and/or high serum C-reactive protein.[21] If a patient presents with these symptoms and GCA is highly suspected, a temporal artery biopsy is done. An MRI/angiography, conventional angiography, ultrasound, or PET scan can be done prior to biopsy to contribute to the diagnosis of GCA and to determine the disease progression.

Treatment

Classically, glucocorticoid treatment for polymyalgia rheumatica has been the first line therapy using a low to moderate dose. The goal of treatment with glucocorticoids, such as prednisone or prednisolone, is relief of symptoms associated with polymyalgia rheumatica. Initial treatment consists of a low dose of a glucocorticoid followed by slow tapering until the patient no longer needs glucocorticoid therapy, which may take 1-2 years or occasionally longer. This dose can vary from 10-25 mg/day based on the severity of symptoms associated with polymyalgia rheumatica, with 15 mg/day being the most common initial dosage. Relapse of symptoms is

treated much the same way as the initial therapy. Depending on severity of the relapsing symptoms, patients should be re-started on anywhere from 7.5-25 mg/day of glucocorticoid therapy.[14]

Monitoring for side effects associated with sustained glucocorticoid treatment such as hypertension, glucose intolerance, weight gain, and osteoporosis, is common practice for those undergoing glucocorticoid treatment for polymyalgia rheumatica. Giant cell arteritis is commonly treated concomitantly with PMR, albeit at a slightly higher dose of glucocorticoid therapy in order to provide relief of vascular inflammation associated with GCA. Glucocorticoid-sparing treatment options are available, although they are vastly underutilized due largely in part to the inferiority of relief of symptoms as compared to low dose glucocorticoid treatment. These alternative treatment options include methotrexate, etanercept, infliximab, and tocilizumab.

Conclusion

While LE manifestations of GCA and PMR are relatively rare, it is important that these findings are not overlooked by physicians. Symptoms of PAD, claudication, and pitting edema in the LE should be routinely evaluated in patients who are afflicted with either PMR or GCA. Conversely, in patients presenting with claudication symptoms, physicians should elucidate whether there are any of the concerning symptoms of GCA or PMR that may be the underlying cause. Most of the recent studies on PMR and GCA relating to the LE have been limited in terms of population and length of study, there is a great need for more longitudinal and large-scale studies in this area.

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Continuous Flow Cryotherapy vs. Icing: A Literature Review of Pathophysiology & Postoperative Efficacy

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Introduction

Cryotherapy is a defined as any method of cooling used for therapeutic pain and edema relief[1,10]. Modalities of cryotherapy can range from a frozen bag of peas to continuous flow cold machines. Newer devices used by podiatric physicians include the cryo/cuff ankle device (Figure2), EBIce, and Iceman (Figure 2), which are basic systems composed of an ice water reservoir that maintains water at a given temperature attached to tubing that then filters the cold water to a cuff to be applied to the affected area[10].

Figure 1. Iceman[10]

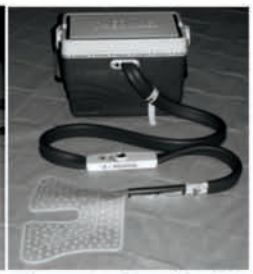

Figure 2. Cryo/Cuff Aircast [10]

With development of new machinery, physicians and surgeons must consider the utility of each modality and classify each on a risk vs. benefit spectrum. In the realm of podiatric medicine, cryotherapy has been utilized in the post-operative period to decrease pain and edema. The following is a review of literature to evaluate the mechanism of action by which cryotherapy is expected to achieve therapeutic results. The review will also focus on current podiatric and orthopedic literature comparing traditional icing protocol to the continuous flow cold therapy machinery.

Current Research on Cryotherapy as a Pain Cycle Mediator

Nadler and Kruse, published sports medicine physicians, attribute the decrease in pain attenuation from cold treatment to theory of cold-induced neuropraxia. They describe that pain sensation begins at nocioceptors in the skin and surrounding tissue structures. Nocioceptors are free nerve endings of afferent neurons that detect signals of tissue damage. They then relay these messages to the CNS as pain (Figure 3)[7]. There are 5 main types of nocioceptor channels: thermal, chemical, mechanical, polymodal, and silent[8]. Each channel is a variation of a transient receptor potential (TRP) channel. These channels can detect substances such as arachidonic acid, substance P, globulin, histamine and other substances released during cell lysis and tissue damage[7, 8]. When a channel is stimulated beyond a given threshold, an action potential is initiated and then transferred to the spinal chord and brain[7].

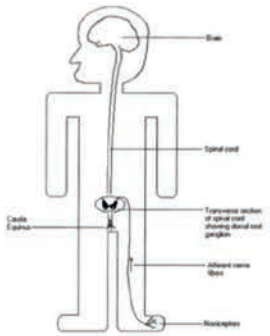

Figure 3. Pain Perception Pathway[7]

Cryotherapy as described by Nadler and Kruse, intervenes in the pain pathway by decreasing the temperature to the area of tissue damage. The decrease in temperature increases the activation threshold of tissue nocioceptors and decreases conduction velocity of pain nerve signals[8]. Consequently, it is difficult to initiate a pain relaying action potential, and even when one is initiated it is relayed to the CNS at a much-delayed speed. It must be noted that cold also decreases the amount of substances that are released from damaged tissue, thus decreasing the amount of stimuli present for nocioceptor activation[13]. Pain suppression is thus a result of the overall depression of nocioceptor signal activation.

Conversely, McDonald and Gutherie attribute pain relief from cyrotherapy to the gateway control theory. Authors state that the sensation of cold overwhelms and blocks the

inflow of pain stimuli to the cortex. This theory relates that non-painful input, in this case thermal input, causes inhibitory cells to block transmission cells from relaying nocioceptor signals to the CNS[10]. The theory is based on the fact that pain is a complex process with much cross talk between pathways, allowing it the capacity to be regulated and inhibited with substances such as GABA and substance P[10]. The authors also describe another regulatory pathway in which cold therapy intervenes in the pain-spasm-pain cycle[10]. Colder temperature decreases nerve conduction velocity at the neuromuscular junction. This causes fewer muscle spasms in affected areas, thus leaving the damaged muscles in a relaxed state associated with less pain[10]. By discussing both theories of pain signaling, authors relate that the mechanism is highly complex with multiple pathways leading a sensation of pain.

Research Comparing Cryotherapy vs Standard Icing in Pain Management

Scheffler et al. compared the effectiveness of standard icing protocols versus continuous cold flow machinery in regard to pain suppression. In this study authors compared 25 patients who underwent identical bilateral foot surgeries and postoperatively designated the right foot to undergo cryotherapy using the cryo/cuff apparatus and the left to undergo an ice cap and elevation. The left foot in this study served as a control. The study methods ensured that both the cryo/cuff and the standard ice were use for the same amounts of time and at the same intervals. Authors compared the right versus left foot in regards to pain via a visual analog scale (Figure 4). Participants were asked to mark their level of pain on the spectrum in four postoperative visits.

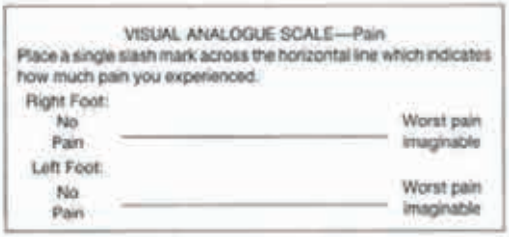

Figure 4. Visual Scale Analog used by Scheffler et al.[13]

Authors found that 80% of the participants had less pain in the right foot at their first postoperative visit. They concluded, based on their survey that the use of the cryotherapeutic device offered subjective pain relief in patients.

According to a prospective, multicenter, randomized trail by E.P. Su et al. the only statistically significant difference in the post-operative course of the two groups following total knee arthoplasty was in the amount of narcotics used. The study examined 280 patients who were post-operatively randomized to either a cryopneumatic device or ice w/ static compression following a total knee arthroplasty. The found that the cyropneumatic therapy group used an average of 509 mg of morphine while the control group used an average of 680 mg. Authors then concluded that due to the lower amount of morphine used by the cyrotherapy group, the device offers a valuable benefit over standard icing protocol in terms of pain management[4]. This direct comparison of the two ice modalities shows the superiority of cryotherapeutic devices over standard icing protocol.

Cryotherapy as a Mediatory of Edema

The imbalance between intravascular and interstitial hydrostatic and osmotic pressures causes an increased pressure in capillaries (figure 5) leading to leakage into the interstitial space[2].

Figure 5. Edema Pathophysiology

According to Fahad Attar et. al, an increase in intravascular hydrostatic pressure causes an axonal reflex in post-capillary venules. This innate reflex causes vasoconstriction of pre-capillary arterioles, which then prevents the increase in intravascular hydrostatic pressure that could lead to capillary leak.[2] This is especially relevant while the limb is in a

dependent position. The normal microcirculation of the foot is such that even an increase of blood pressure, from placing the foot in a dependent position (an increase of approximately 25 mmHg), stimulates stretch receptors in post-capillary venules to induce the local sympathetic axon reflex.

The local sympathetic axon reflex is disturbed and cannot be initiated by increased blood pressure post-operatively[2]. As a result, the body's innate mechanism of edema prevention is no longer intact. Additionally, substances released by the damaged tissues are vasodilatory in nature, leading to more extensive dilation of capillaries[2]. Authors determined the extent of the loss of the vasoconstrictive reflex determined to last greater than 72 hours post-operatively. The authors examined the microcirculation of the foot in patients who underwent elective foot surgery using laser doppler flowmeter. They stimulated increased intravascular pressure by placing patients in dependent positions and reporting change in microvascular flow. The study found that the normal vasoconstriction was not reached until 72 hours postoperatively.

Since vasodilation causes the imbalance of hydrostatic pressure post-operatively leading to edema, icing is used to induce the vasoconstriction that is lacking. A study published by Knoblock K et al., showed that using a cryo/cuff ankle device changed the parameters of microcirculation in the ankle within the first 10 minutes of use. Authors looked at the difference in microcirculation at different tissue depths, superficially at 2 mm and deeper tissue at 8 mm, and found that following 10 minutes of use of the cyro/cuff aircast device, microvascular flow was reduced by 40% in the superficial tissue and 60% in the deeper tissue. Authors thus proved that cold therapy brings about the vasoconstriction that is naturally lacking in the post-operative period. In addition to this mechanism, it is also stated by Scheffler et al. that colder temperatures lead to slowing local cellular metabolism.[13] The decrease in cellular metabolism then decreases the oxygen requirement of local tissue which lead to fewer hypoxic injury and tissue destruction[13]. When discussed together these studies show that cyrotherapy is able to provide the vasoconstriction that is naturally unavailable in the post-operative period.

TABLE 1. **Measurement of standard deviation in relation to baseline circumference**

Baseline Circumference (cm.)[a]	L 21.7 cm. L	R 21.7 cm. R	N[b]
1st p.o.[c] evaluation	+0.041	−0.196	25
2nd p.o. evaluation	+0.283	−0.018	22
3rd p.o. evaluation	+0.179	−0.088	8
4th p.o. evaluation	+0.46	+0.18	5
Standard deviation + or − baseline	+0.167	−0.032	

[a] Baseline circumference determined preoperatively, represented by baseline mean circumference.
[b] $N = 25$.
[c] p.o., postoperative.

Figure 6. Left vs. Right Foot Circumference[13]

Research Comparing Cryotherapy vs. Standard Icing in Edema Control

The question still remains, does the cryotherapy device offer any benefit compared to standard icing protocols? Once again, Scheffler et al. in their prospective randomized trial evaluated this question by studying patients with bilateral foot surgery and following post-operative care of the right foot with a cryotherapeutic device and the left foot with an ice cap as a control. The primary outcomes of the study included circumference of the right and left midfoot to evaluate for edematous changes and a subjective survey of patients to determine their comfort preference. Authors found a standard deviation of .032 below circumference mean in the group using the device and also demonstrated that patients subjectively favored the cryotherapeutic device over standard applications of ice. The authors conclude then that the cryo/cuff aircase device decreases edema to a greater extent than standard icing.

Cryotherapy Complications

The detriments of the cryotherapy device use include frost bite, peroneal neuropathy, and Raynauds, among others. The vasoconstrictive effects of these newer instruments can become so extensive that it leads to hypoxia from extensive restriction of blood flow and oxygen supply. Additionally, this can cause impairment

of nerve condition, specifically peroneal neuropathy.[5] Other potential adverse reactions include Raynauds' phenomenon with cold induced vasospasm of peripheral vessels, or even Hunting Reaction[2, 13]. Hunting reaction, as described by Wilke and Weiner, refers to when change of temperature of the skin surface causes cold stress leading to oscillation between vasoconstriction and dilation of blood vessels.[1] The sequential dilation and then constriction leads to accumulation of edema, thereby worsening the healing process[1]. The final adverse reaction that must be acknowledged is the obvious frostbite attributed to freezing of tissue with either intra or extracellular crystal formation[1].

Two cases of frostbite published by Brown and Hahn display the true severity of extended and unsafe cryotherapy use. The first case was that of a 16 year old female with bilateral hallux valgus surgery who was discharged with ankle cryo/cuff. In the following days, the patient experienced bilateral foot pain and required compartment releases due to elevated plantar compartment pressures (figure 7).

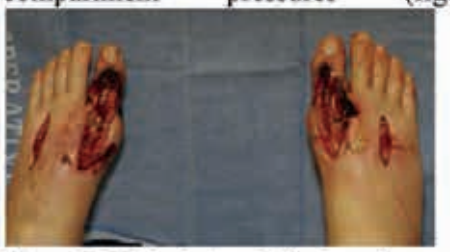

Figure 7. Clinical photo following aircast use[12]

Serial debridements following the procedure showed extensive necrotic tissue. The patient required bilateral split thickness grafts to cover damaged area and the patient went on to walk with a slight limp.The second case was very similar, a 19 year old female who also required a fasciotomy after cryotherapy use following hallux valgus and hammertoe correction. In both cases the author emphasizes the severity of the adverse effects of the use of the device.

To follow, Wilke and Weiner published a larger survey of 110 podiatrists and local hospitals to look at the incidence of major injuries. The authors found only 5 incidences of serious cold-related injuries resulting in a complication rate of 0.00225%. The five major categories that pre-disposed these patients to the adverse effects include: increased humidity at the wound site, age, social habits, pharmacological intake and other medical comorbidities. Pharmacological intake includes patients on beta-blockers, which prevent peripheral vasodilation. Medical comorbidities specifically include patients with known PAD, Raynaud's, diabetes, sickle cell or a history of cold injuries. We must also consider social habits such as caffeine which lead to peripheral vasoconstriction. Authors concluded that these are the patients in which cryotherapy devices must be avoided, but otherwise established that other patients showed a very low complication rate.

Conclusion

The focus of this article has been pathophysiology of using cryotherapy in general and then a comparison of continuous flow cold cryotherapy device to standard icing protocol. The review of literature has demonstrated that cyrotherapy devices have shown an improvement in patient pain and edema with very low complication rates. The complications, if present, are so severe that special care must be taken in giving instructions of limited use of cryotherapuetic devices and also patient selection to avoid those at elevated risk. High risk patients include those with known peripheral arterial disease, sickle cell patients, and those with a history of cold injury. Few practitioners have studied the comparison of standard icing protocol versus newer cyrotherapeutic devices, however there is a greater need for further understanding and research. Conduction of randomized clinical trials to determine the true efficacy of ice and cryotherapy devices in specific patient populations and given procedures would prove useful for future patient care.

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Management of Ankle Fractures Associated With Trauma to the Syndesmosis

Benjamin Kamel, DPM 2017

Introduction

Over the years, there has been a general consensus in the treatment of ankle fractures - the standard of care for ankle fractures dictates that anatomic reduction and restoration of the distal tibiofibular relationship needs to be achieved to obtain predictable clinical results. [1,2] However, when there is syndesmotic damage, there remains controversy involving a management strategy that reduces complications and maximizes outcomes. Considerations when dealing with the management of syndesmotic repair are: duration of fixation to allow healing of the syndesmosis, proper anatomic level of screw placement, biomechanical effects of the ankle joint after screw placement, and the number of screws. Some reasons why surgeons avoid syndesmotic screw fixation include fusion and the need for an additional operation for screw removal. [3] The current standard of care recommends the treatment of tibiofibular diastasis, a Maisonneuve fracture, and ankle instability is a syndesmotic screw fixation.[3].

A study by Leeds and Ehrlich found a correlation between the stability of the syndesmosis and restoring proper tibiofibular relationship and correct anatomical reduction [4] It has been recommended by Boden et al that a fibular fracture above the "critical transition zone," or 3 to 4.5 cm proximal to the ankle joint, required fixation in order to decrease the incidence of post-traumatic osteoarthritis (OA) of the ankle. [5] Research on evaluation, treatment, and long-term sequelae of distal tibiofibular syndesmosis injury is also rare. This injury is difficult to evaluate and diagnose and has a longer recovery time than other ankle sprains.[18] Therefore, it is important for those clinicians evaluating, treating, and rehabilitating ankle and lower leg injuries to fully understand the anatomy, biomechanics, and mechanisms of injuries involving the tibiofibular syndesmosis

Anatomy

The inferior tibiofibular joint is defined as a syndesmotic articulation between the convex surface of the distal fibula and the concave distal tibia. [15] The distal fibula is firmly attached at the fibular notch of the tibia by several syndesmotic ligaments.[15]

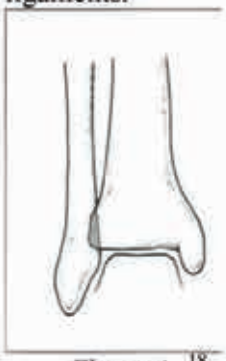

Figure 1.[18]

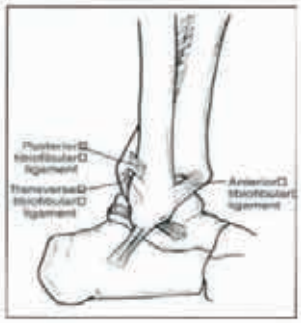

Figure 2.[18]

The stability of this articulation is integral in allowing for proper function of the ankle and lower extremity. The distal tibiofibular syndesmosis is held together by 5 structures: the Anterior-inferior tibiofibular ligament (AiTFL), Posterior-inferior tibiofibular ligament (PiTFL), Deep transverse ligament, interosseous ligaments, and the interosseous membrane.

Mechanism of Injury

The incidence of syndesmotic injury can occur with or without any associated ankle fracture. External rotation seems to bring the most opportunity for syndesmosis injury, despite other positions of the ankle, such as dorsiflexion, plantar flexion,

supination, or pronation. When the ankle is in the neutral position, external rotation appears to cause injury to the tibiofibular ligaments only, without damaging other structures.[16]

External rotation injures the structures of the syndesmosis by widening the mortise.[17] Normally, the talus is positioned between the medial and lateral malleoli and is unable to rotate substantially. However, with a great enough force to the forefoot, the talus is forced to rotate laterally, thereby pushing the fibula externally away from the tibia. Reports have shown that there is an increase in incidence of syndesmotic injury with patients who have recurrent ankle sprains in the past.[3]

Clinical Diagnosis

There are few classification systems to date for ankle fractures: Lauge-Hansen, Danis-Weber, Muller, and Edwards & Delee classifications. (Charts on next page)

The squeeze test can identify the presence of a syndesmotic injury.[3] The squeeze test can be performed by using both palms to gently press both medially and laterally on the heel. Positive squeeze test is indicated when ankle pain is elicited when the tibia and fibula are squeezed at the midpoint of the calf. The most common presentation of syndesmotic injury is in the presence of an associated ankle fracture.[3] In a study by Rasmussen et al, using 18 cadaver specimens, evaluated the role of the tibiofibular ligaments in ankle stability and the mechanisms that may cause their rupture.[14] The authors reported that mortise integrity was only minimally influenced after isolated incision of the anterior tibiofibular ligament. However, external rotation was greatly increased by incising both the anterior tibiofibular ligament and the anterior aspect of the deltoid ligament or the posterior talofibular ligament. Therefore, rupture of the distal tibiofibular structures may occur only with external rotation trauma. Anterior tibiofibular ligament injury in isolation must be rare, and complete rupture of the distal tibiofibular structures is probably combined with injury to the anterior aspect of the deltoid ligament, the posterior talofibular ligament, or both.

The Cotton test may be used to assess syndesmosis instability. The cotton test is a manual stress test that is used to identify the degree of lateral translation of the talus within the ankle mortise.[3] The cotton test is performed by stabilizing the proximal portion of the tibia while shifting the talus laterally. (Figure 3) A positive test is when there is an increased motion relative to the uninjured side and is indicative of a sprain of the distal tibiofibular syndesmosis.

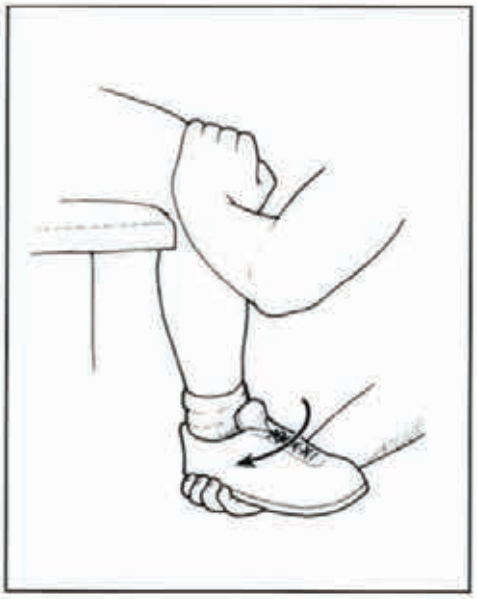

Figure 3.[6] The Cotton

Lauge-Hansen Classification				
Supination – Adduction	**Pronation – Abduction**	**Pronation – Dorsiflexion**	**Supination – External Rotation (SER)**	**Pronation – External Rotation (PER)**
I – transverse fx of the lateral malleolus	I – Rupture of deltoid ligament/medial malleolar fx	I – Fx of medial malleolus	I – Rupture of ant inferior tibio-fibular ligament	I – Rupture of deltoid ligament/medial malleolar fx
II – vertical fx of the medial malleolus	II – Rupture of ant inferior tibio-fibular ligament	II – Large anterior lip fx of tibia	II – Spiral oblique fx of lateral malleolus (extending anterior inferior to posterior superior.)	II –Rupture of anterior inferior tibia-fibular ligament, intra-osseous ligament, intra-osseous membrane
	III – Bending fx of fibula 1cm proximal to plafond	III – Fracture of superior lateral malleolus	III – Rupture of posterior inferior tibia-fibular ligament	III –Spiral fracture above syndesmosis (High fibular fracture)
		IV – Fracture of third malleolus (posterior tibia)	IV – Deltoid rupture/fracture of medial malleolus	IV – Rupture of posterior inferior tibia-fibular ligament

Table 1. [15] Lauge-Hansen Classification

Danis-Weber Classification- Lateral Malleolar Fracture	
Type A	Fracture below the level of the tibial plafond
Type B	Fracture at the level of the tibial plafond
Type C	Fracture above the level of the tibial plafond

Table 2. [15] Danis-Weber Classification

Muller Classification- Medial Malleolar Fracture	
Type A	Avulsion of tip of medial malleolus
Type B	Avulsion at the level of the ankle joint
Type C	Oblique fracture
Type D	Vertical Orientation

Table 3. [15] Muller Classification

Edwards & Delee- Chronic Tibiofibular Diastasis	
Type I	Straight lateral subluxation of the fibula with medial clear space on x-ray
Type II	Lateral fibular subluxation with plastic or angular deformity
Type III	Posterior rotatory subluxation of distal fibula behind talus with Posterior inferior tibia-fibular ligament intact
Type IV	Complete Ankle Diastasis with talus dislocated superiorly, wedged between the tibia and fibula

Table 4. [15] Edwards & Delee

Radiographic Assessment

The most reliable parameter for detecting the widening of the syndesmosis is determined by visualizing the width of the medial clear space on the AP view. (Figure 4)

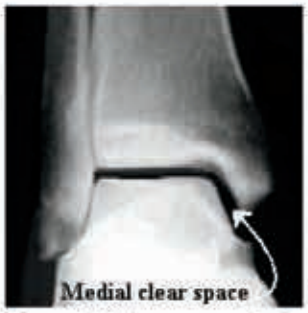

Figure 4. [9]

Normal clear space is 6mm or less and the normal tibiofibular overlap is greater than 6 mm or 42% of the fibula width. [7] Assessment of syndesmotic disruption on stress lateral radiograph has a higher correlation with anatomical separation than that on a stress mortise radiograph. [8]

Conservative vs. Surgical Management

Stable, undisplaced fractures of the ankle, significant comorbidities, or poor local skin or significant vascular problems are typical reasons why a surgeon might choose to treat an ankle fracture conservatively. Conservative treatments of ankle fractures include: rest, ice compression, elevation, short leg splint, or a walking boot with crutch walking.

However, if the history and clinical exam suggest a syndesmotic injury, routine radiographs should be performed and analyzed. If there is separation of the syndesmosis without a clear fracture the patient is then taken to the OR before there is too much swelling. If a significant amount of swelling has developed, it is best to wait 5-10 days until the swelling resolves.

Screw Placement

According to the AO principles of internal fixation, syndesmotic fixation is to be placed 2-3 cm proximal to the ankle joint. If the fixation is below the 2-3 cm mark proximal to the ankle joint the surgeon runs the risk of causing synostosis or calcification as well as chronic ankle pain, due to possible complications such as the screw breaking within the tibiofibular joint. [9] The screw(s) that are placed in the syndesmosis are described as positional screws as opposed to lag screws and are not to be used to compress or to approximate the syndesmosis. Positional screws are used in order to achieve stability between the tibia and fibula to hold the syndesmosis in position until the syndesmotic ligament healing occurs.

The type of screw, its placement location, and angulation are instrumental in the success of syndesmotic injury repair. Improper repair of the syndesmosis can lead to irritation or failure of the hardware, development of arthritis in the syndesmosis, and failure of the syndesmosis to heal properly. The AO manual endorses that a 4.5 mm or 3.5 mm cortical screws, placement of screws 2-3 cm proximal to and parallel to the ankle joint, and an oblique screw aiming 25° from posterolaterally to anteriomedially (Figure 5) as recommendations for optimal syndesmotic repair. [9]

Figure 5. [5]

A report by Mulligan and Hopkinson's syndesmotic repair using one screw engaging four cortices is less stable than the use of two screws inserted through three cortices. The explanation is that if a screw were engaged between four cortices as opposed to three cortices, then the patient would run the risk of having abnormal external rotation of the fibula during ankle

dorsiflexion. [8] If movement were to occur, the screw will loosen in the lateral cortex of the tibia rather than break. Screw engagement in the far cortex may risk screw breakage. It is important to inform patients of this common complication and that it does not imply that there was a surgical error.

The risk of stiffness is decreased by dorsiflexing the foot maximally as there is reassurance that the widest area of the talus is within the ankle mortise. [10] A study by Olerud showed that ankle joint dorsiflexion decreased by an average of 0.1 degrees for every one-degree increase in plantarflexion when the screw was being inserted. [10] It is important to maintain optimum range of motion for patients undergoing ankle surgery as it may lead to significant arthritis later on in life.

Bioabsorbable Screws

The disadvantages of bioabsorbable implants include both mechanical and biologic. Metallic screws maintain a much higher tensile and torsional strength than bioabsorbable implants. [12] Metallic screws, popularized by the Champy and AO systems, comprise a mixture of iron (62.5%), chromium (17.6%), nickel (14.5%), molybdenum (2.8%), and smaller amounts of other metals. [19] Stainless steel is strong and extremely rigid, making it difficult to bend and more susceptible to surface damage and resultant corrosion after adaptation. This is where the bioabsorbable implants are lacking in strength. The other disadvantage to using certain polyesters as bioabsorbable screws is the potential for a local inflammatory response leading to an undesirable sterile abscess. [13] Sterile abscesses aren't as common as infected abscesses, but is important to know that the treatment is for excision of the foreign body.

The advantages of absorbable screws include minimal osteolysis, radiolucency, elimination of subsequent procedure for removal, and minimized disruption of the ankle biomechanics. [13]

Post-operative Management

Aggressive rehabilitation with early range of motion and weight-bearing exercises allows patients to regain functional activity at the quickest rate. Traditional modalities such as rest, ice, compression and elevation to decrease swelling and inflammation should be utilized early and often in post-operative management. Mobility and strength-training exercises promote the natural healing process of the syndesmosis and associated ligaments, and thus accelerate return to previous levels of function. Patient getting started with a physical therapist as soon as possible can help with making sure that the patient maintains proper post-operative care. The postoperative rehabilitation protocol lasts from 2 to 6 months and includes progressive steps toward full recovery. Any acute signs of infection, hardware pain or arthritis warrant a patient to return to the OR for hardware removal.

Screw Removal

Controversy continues to exist between whether or not to remove the screws prior to full activity or ambulation. In general, syndesmotic screws are removed between 6-12 weeks, but there has been interest in delaying removal up to 4 months to avoid any syndesmotic widening. A study by Needleman et al concluded that prolonged duration of the screw within the syndesmosis has led to changes in ligament length/tension relationship and restricted ankle rotation. [11] They inserted the screws using AO technique and concluded from their findings of restricted ankle rotation and changes in normal ligament length/tension relationship that the screw should be removed prior to full activity.

Conclusion

Any ankle fracture warrants evaluation of syndesmotic injury. Surgically the ultimate goal is to restore proper tibiofibular relationship and correct anatomical

reduction while also decreasing the incidence of any post-operative complications such as post-traumatic osteoarthritis. There continues to be multiple controversies regarding screw fixation for unstable syndesmotic injuries. When it comes to the type of screws to use in trans-syndesmotic screw fixation for syndesmotic injury, although there is decreased tensile strength than the traditional metallic screw, the absorbable implant has been shown to exhibit a fair amount of advantages and fewer disadvantages. Patients who are looking to eliminate a subsequent procedure for removal or would like to maintain ankle biomechanics might look to choose bioabsorbable screws as their preferred fixation. On the other hand, if patients want to minimize their risk factors for post-operative complications and want to be reassured of a stronger fixation, then traditional screws would be the more appealing option. However, given that there are multiple advantages to the use of absorbable screws in trans-syndesmotic screw fixation, there is still no conclusive answer to which is best, therefore more research needs to be done to confirm the best type of screw to use during fixation.

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Applications of the Angiosome Concept in the Lower Extremity

Caitlyn Lee, DPM 2017

Introduction

The angiosome concept, first presented by Dr. Ian Taylor in 1987, has had many clinical implications in treating the lower extremity in the case of critical limb ischemia and wound healing, as well as surgical planning. An angiosome is described as a three dimensional block of tissue fed by a main artery, referred to as a source artery.[1] This three dimensional theory stresses the connection of tissues between the skin and bone, distinguishing it from the previous concept of vasculature running within a single two dimensional tissue layer. Angiosomes communicate with each other at their borders through choke arteries which supply blood to neighboring angiosomes in the event of a disease process, trauma, or other damage to the source artery supplying that angiosome.[1] Plastic surgeon, Christopher Attinger, expanded on the angiosome concept by using handheld Doppler to map the entire vascular tree and predict the direction of flow in any given angiosome.[2] This method aids surgeons in incision planning, surgical wound healing assessment, flap harvest, and revascularization.

Angiosome Mapping

There are six angiosomes identified in the foot and ankle that are fed by the three main arteries to the foot: the posterior tibial artery, anterior tibial artery, and the peroneal artery. The posterior tibial artery gives rise to three angiosomes: the medial calcaneal branches, the medial plantar artery, and the lateral plantar artery, represented by the color purple, pink and blue, respectively, in Figure 1. The anterior tibial artery feeds the anterior ankle and then becomes the dorsalis pedis artery, yellow in Figure 1. The peroneal artery gives rise to two angiosomes: the anterior perforating branch and the lateral calcaneal branch, orange and red in

Figure 1.

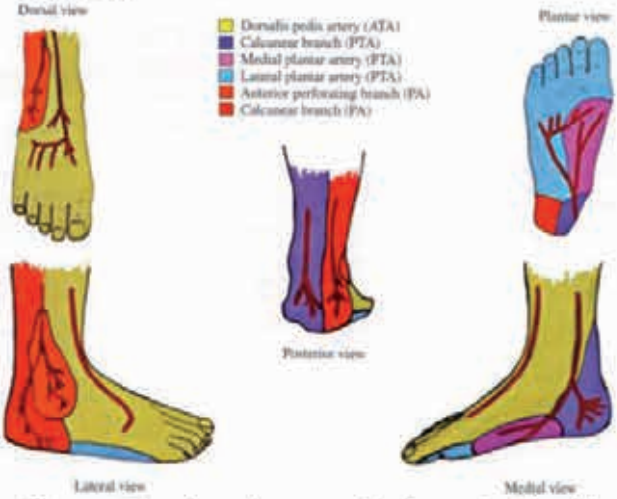

Figure 1. The six angiosomes of the foot and ankle with their source arteries.[1]

Once a source artery is identified, its quality and direction of flow can be determined with a Doppler.[2] Normal circulation is characterized by triphasic flow while biphasic indicates mildly compromised flow and monophasic indicates arterial compromise.[3] The direction is determined by putting finger pressure proximal and distal to the Doppler and evaluating the sound which results. With this exam, the provider can determine the source arteries that supply an angiosome and subsequently map the arterial tree, giving a better understanding of the vascular anatomy of the patient. Figure 2 illustrates this technique using the first dorsal metatarsal artery as an example. The artery is first identified using the Doppler, and then the dorsalis pedis is occluded using finger pressure at the proximal first interspace. Retrograde flow from the web space is indicated if the Doppler continues to pick up a signal.

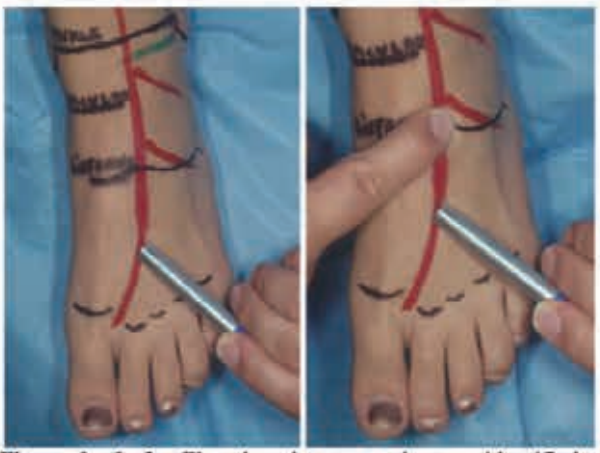

Figure 2. Left: First dorsal metatarsal artery identified with Doppler. Right: Occlusion of dorsalis pedis to determine if there is retrograde blood flow to the first web space, confirmed by picking up signal on Doppler.[2]

Surgical Implications

Incisions

When planning a surgical incision, there are four factors that need to be considered. The incision must provide: adequate exposure to the surgical site, adequate blood supply on both sides of the incision to optimize healing, preservation of motor and sensory nerves, and the incision should parallel a joint to avoid scar contracture and subsequent joint immobility.[2] Angiosome mapping techniques are useful in preoperative incision planning by allowing surgeons to identify the location and quality of source arteries to a given angiosome. In a vascularly intact patient, the best incision, in terms of wound healing, is the border between two adjacent angiosomes due to the maximal blood flow.[2] When an incision at the borders of angiosomes fail to provide adequate exposure, interruptions within angiosomes must be made and are resupplied by choke vessels which become patent within 4-10 days. In vascularly compromised patients, vascular mapping becomes increasingly important when planning on incision because blood supply in these patients is less likely to be reinstated by collaterals if the main arteries supplying that region of the foot are incised. When working near an occluded vessel, it is best to make the incision closer to the occlusion as to preserve as much of the collateral vasculature from the adjacent angiosome as possible. If the surgical site is surrounded by vascular compromise, direct revascularization by a vascular surgeon must be considered before proceeding.[2] Using the angiosome principle to plan surgical incisions ultimately optimizes the chances of successful wound healing post-operatively by ensuring adequate vascular supply.

Flaps

The angiosome principle and Doppler examination are also useful when planning both local and pedicled flaps in the foot and ankle which retains its own blood supply. Identifying a perforator artery and ensuring antegrade flow at the base of a local flap help the pedicle thrive and minimize the risk of failure. This principle is even more important when planning a pedicled flap, as it relies on a strong, patent artery for survival.[2] A Doppler is important in this instance to not only map out the vasculature of the dominant pedicle, but also to determine the patency, direction and quality of flow in the vessel. Regardless of the vascular status of the patient or the type of flap used, it is critical to have antegrade flow to the base of flaps which can be determined using Attinger's Doppler approach and the angiosome concept.

Amputation

The same principles for surgical incisions and flaps also apply to performing forefoot and midfoot amputations.[2] The optimal site for incision and closure of amputations in the foot are at the angiosome borders to ensure maximal blood flow and healing.[2] In vascularly compromised patients who have failed revascularization using bypass and require amputations in the foot, it is important to map out arterial-arterial connections as much as possible. If the choke vessels that an angiosome relies on for blood supply are ligated or damaged, the closure and flap are at high risk for necrosis. In many cases the dorsal circulation depends on the plantar circulation, or vice versa, such as the deep plantar artery which communicates dorsally and plantarly. It is vital that these communicating branches are not interrupted. In the setting of a transmetatarsal amputation or Lisfranc amputation, the lesser

metatarsals are removed laterally and the first metatarsal is removed medially to preserve the dorsal and plantar flow. Similar to flaps, it is important for both ends of the primary closure to have antegrade flow to ensure proper healing and blood flow to the reconstructed foot.

Implications in Diabetes

Diabetics have a higher propensity for developing peripheral vascular disease (PVD) due to the increased glucose levels which cause endothelial damage, inflammation and hypercoagubility to blood vessels. PVD in diabetics is both more severe and more diffuse than non-diabetics.[4] Critical limb ischemia (CLI) is the most severe form of peripheral vascular disease and is characterized as decreased blood flow to the foot, with rest pain in the lower extremity, ulceration and/or gangrene of the leg.[4] Diabetic peripheral neuropathy along with ischemic changes seen in CLI predisposes the patient to develop minor traumatic wounds which give rise to larger ischemic foot wounds.[4] For this reason, diabetics are 8-24 times more likely to have a lower extremity amputation compared to non-diabetics.[5] Even with the standard treatment of bypass and endovascular intervention for amputation prevention, there is a 15% failure rate.[2] This is attributed to failing to achieve local revascularization of the source artery to the ischemic area despite major vessel patency more proximally.[2] This supports the angiosome model which suggests more success in reconstituting the flow in the artery that directly supplies the ischemic area. Revascularization using the angiosome model has been shown by Attinger's study to aid surgeons in selecting vessels and forming preoperative strategies to help diabetic patients with CLI to avoid major amputations and promote healing of ischemic wounds.

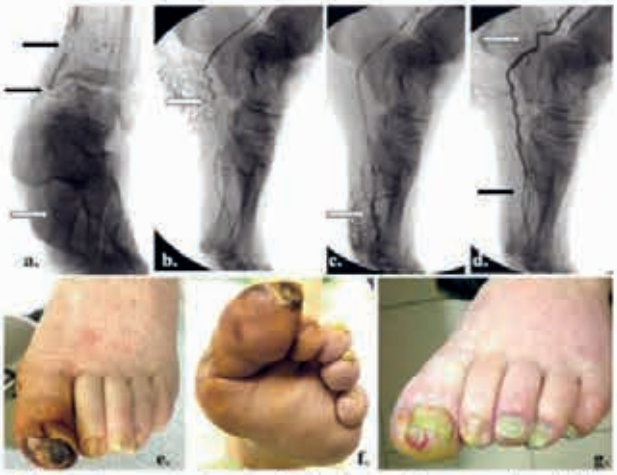

Figure 3. a-d: Revascularization of the posterior tibial and medial plantar arteries and the resultant reperfusion. e-g: Clinical results before vascular intervention and at 14 weeks post selective artery revascularization.[7]

Direct vs. Indirect Revascularization

Since the angiosome concept was first presented, there has been some debate between the efficacy of directly revascularizing the angiosome compared to indirectly revascularizing it. In direct revascularization the angiosome containing the ischemic region is targeted, where as indirect revascularization is bypass targeted at regions unrelated to the ischemic area.[3] Serra et al. conducted a study where diabetic patients with foot ulcers and impending amputations were treated using an angiosome targeted surgical technique.[4] This study revealed a significant increase in postoperative transcutaneous oxygen tension (TcPO2) and over 90% survival without major amputation compared to traditional procedures. In addition, Biancari and Juvonen conducted a meta-analysis which revealed a significantly lower risk of unhealed wounds, major amputations and limb salvage rates of 86.2% and 84.9% at one and two years using direct angiosome-targeted revascularization compared to 77.8% and 70.1% using indirect revascularization techniques, such as bypass, for ischemic foot wounds.[3] Neville et al. compared bypass to the artery directly feeding the ischemic angiosome and bypass indirectly feeding the angiosome and its impact on wound healing and limb salvage.[3] Their study revealed 91% complete wound healing using direct

revascularization whereas the indirect revascularization group had a 62% healing rate. Conversely, there is still discussion on the effects of direct angiosome-targeted revascularization. Spillerova et al. conducted a retrospective study which questioned the feasibility of an angiosome-targeted endovascular treatment since ischemic wounds are not usually localized to just one angiosome.[6] Although the theory of direct revascularization is accepted and supported by Biancari's meta-analysis, a clear definition of the angiosome approach when faced with ischemia covering two or more angiosomes needs to be developed and studied further. As angiosome-targeted approaches to surgery are utilized more, there will be further data to help support or refute the validity and feasibility of the angiosome concept in the future.

Conclusion

The angiosome concept has many clinical implications for the lower extremity from surgical planning to revascularization approaches, especially for diabetics with critical limb ischemia and neuropathy with ischemic foot wounds. Using a Doppler to map a detailed vascular tree within an angiosome helps diagram patients' individual anatomies. This helps predict the success and healing potential for the procedures that they will undergo. Although this theory and approach to revascularization is relatively new and requires further study and development, the current research supports the efficacy of using an angiosome-targeted approach in a variety of lower extremity procedures.

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An Introduction to Skating Biomechanics, Pathology, and Treatment

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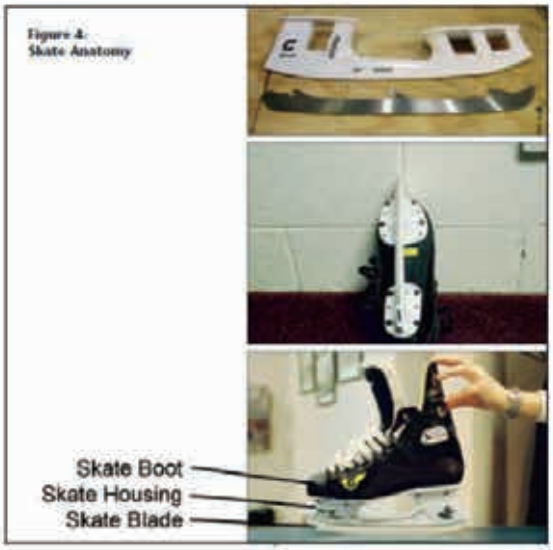
Introduction

Ice skating has come to be enjoyed by millions of people worldwide and has evolved into multiple disciplines and sports (hockey, figure skating, speed skating), each with their unique appeal and *je ne sais quoi,* but the biomechanics between the various disciplines remains largely the same. Physicians should know how to recognize, understand, and treat injuries resulting from ice skating. The purpose of this article is to summarize the biomechanics, pathology, and therapies related to skating.

Like the gait cycle, the skating cycle incorporates biphasic movements consisting of single and double support phases.[1] The principles of ice skating begin with the skate itself. Standard skate blades are attached to the boot under the 2nd metatarsal, and extends posteriorly under the heel and anteriorly beneath the 2nd toe.[1] All skate boots have a heel raise (~5-9°) and wedges which vary and can be adjusted.[1] The blade itself has characteristics that can be adjusted to suit an individual's preferences. A sharper blade gives better push-off and the "blade rocker" (i.e., the radius of the curvature of the blade) is anecdotally said to allow for increased agility at a greater rocker radius, but decreases stability while the opposite is true for a lesser rocker radius.[2] The adjustable nature of skate blades and the blade-boot attachment allows for accommodations to be made for the biomechanics of each individual skater.[1] Modifications ranging from metatarsal pads and orthotics to blade balancing and more complex manufacturer-performed skate adjustments can be made to further accommodate skaters.[3]



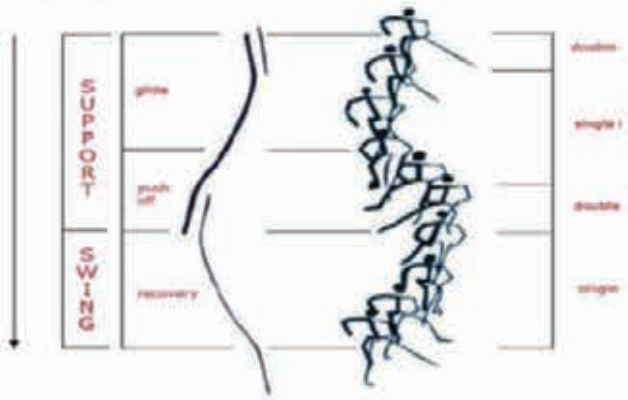
In this image from Humble, different aspects of a hockey skate and the parts of the skate are shown.

Skating injuries, due to the unique biomechanics and footwear, are a fairly common pathology that podiatrists should be prepared to treat. Approximately 11.1% of hockey injuries involve the lower leg, ankle, or foot.[4] In addition, it is worth noting that more than half of the injured players are aged 9 to 14 or 15 to 18,[4] so this should be taken into consideration when determining treatment. Many of these sports are highly competitive and demanding, so some patients may want to return to activity quickly. Patient education about the necessity of compliance and risk of reinjury is important in this setting.

Skating Cycle and Biomechanics

In order to discuss the biomechanics of the skating cycle, it is important to clarify the different phases occurring throughout the cycle. The skating cycle can be viewed as two different phases: swing phase and support phase. The swing phase occurs after the push-off (analogous to toe-off during the gait cycle)

and is entirely single-support as the foot is brought forward. The support phase, composed of the glide phase and push-off, begins following the swing phase with the skater in a period of double-support until push-off, which begins as the contralateral limb touches down.[2] During this period, the contralateral limb is acting as a balance, allowing the skater to complete propulsion.[1] At the propulsive point in the cycle, beginning approximately halfway through single-support glide phase, force is generated through the hip by abduction and extension, through the knee by extension, and at the ankle by plantar flexion and abduction.[3,2] The contralateral limb touches the ice as push-off occurs to begin the cycle anew.[2] This cycle maintains the center of gravity medial to the weight-bearing leg, resulting in a curvilinear pathway for the center of gravity which differs from the linear sinusoidal pathway observed in the gait cycle.[1]



MacLean illustrating the skating cycle. Notice the overall cycle is broken down into support and swing phase, as well as periods of double support and single support. Notice the path taken by the skater meanders as the skater progresses through the skating cycle.

Since ice is a low friction surface, generating enough force to accelerate on it requires the foot to be in an abducted position in order to push into the ice with the side of the blade at an angle tangential to the direction of motion.[1] This is accomplished by abduction at the subtalar joint and external hip rotation.[3] The level of abduction and external hip rotation needed depends on the particular stride pattern. During the first few steps of skating, the skater is said to be in an "acceleration stride pattern". These strides are quick and do not have a significant glide phase, allowing for continual positive acceleration. The foot will be in an abducted position and the hip will be more externally rotated. These acceleration strides usually last for the first three steps.[1] Technique changes as the skater's velocity increases, movement at the hip transitions primarily from extension (acceleration stride pattern) to abduction (steady-state stride pattern), facilitating a gliding motion. The "steady-state stride pattern" usually begins by the fourth stride, as the skater has now generated enough force to overcome the force of friction of the ice in order to glide. Gliding imparts negative acceleration due to friction, meaning that the skater slows between strides and regenerates speed with each step. The position of the foot is less abducted and the hip is less externally rotated in the "steady-state stride pattern" relative to the "acceleration strides."[1] Hip abduction and hip abduction velocity are greater in steady-state strides compared to acceleration strides.[5] The knee displays less flexion during acceleration strides than steady-state strides, likely due to plantar flexion being the primary contributor to ground reactive forces in acceleration and thus force generation.[5] During steady-state strides the knee extensor muscles are more active and allow for greater joint extension velocity, and thus greater skating velocities.[5]

In skilled skaters, hip abduction was greater at push-off than in less skilled skaters.[5] Less skilled skaters were found to have increased abduction during glide compared to skilled skaters.[5] This suggests that less skilled skaters, when compared to skilled skaters, have a higher proportion of the force generated contributing to side-to-side motion as opposed to forward movement. In addition, skilled skaters have greater plantar force in the forefoot, while less skilled skaters have greater plantar forces in the mid-foot and heel.[5] The skilled skaters demonstrate greater hip extension, abduction, and external rotation, which corroborates the hypothesis that greater magnitudes of range of motion are needed to achieve greater velocities.[5]

Unfortunately, there is limited data on the movement of the joints within the foot and ankle during the skating cycle. However, it is known that excessive pronation and supination of the foot can affect optimal interface angle (~45°) during the gliding phase[2] and is implicated in some overuse injuries.[3] Over-pronators (everted rearfoot position) during support phase often present with medial ankle instability, deltoid ligament stress, and tibialis posterior muscle strains. Rearfoot inversion during glide phase can cause excessive stress on the peroneal muscles, which are necessary for maintaining balance through compensatory eccentric contraction.[3] Additionally, the blade rocker creates a contact point on the ice that can be as small as one inch, causing greater need for strenuous eccentric muscular contraction to maintain balance, and contributing to overuse injuries of the peroneal muscles and tibialis posterior.[1]

Injuries and Therapies

Due to the unique characteristics of the biomechanics and footwear involved in skating, there are several injuries that may be sustained.

Due to forceful hip abduction, extension, and external rotation, there are often times muscular strain injuries to the hip flexors and adductors,[1] which are in charge of bringing the leg back to the glide phase from a fully extended, abducted and externally rotated position seen at the end of the propulsive phase of the skate cycle. A treatment involving rest, ice, gradual off-ice training and slow return to on-ice activity should be advised. A physical therapy referral should be considered depending on severity and patient goals. Although the adductor longus is the most common groin injury among skaters, stretching all muscles of the hip and thigh is critical for injury prevention.[3]

Skaters may experience exacerbations of Haglund's deformity and a concurrent bursitis by wearing a skate boot that is too loose.[2] Treatments involving skate-fitting, molding, or custom made boots are often necessary to prevent exacerbations of these pathologies, while doughnut-shaped pads may also take the pressure of the area of pathology.[3]

Hallux abductovalgus and tailor's bunions are also common in skaters, possibly attributable to the tight and stiff nature of the skates, but there is little research showing that hallux abductovalgus is caused specifically by skating. It is known that most skates offer no intrinsic support for the wide array of foot types they accommodate.[3] Without intrinsic support for the foot, biomechanical abnormalities and functional adaptation may present, which has been determined to be the most common etiology for hallux abductovalgus deformity.[3] Treatment of these pathologies involves use of custom orthotics and skate molding, and possible surgical management depending on severity and patient goals.[3]

Plantar fasciitis, often associated with figure skating,[3] can also be managed with orthotics[7] and a normal conservative course involving stretching of the posterior leg muscles[6] along with the plantar fascia-specifically,[8] icing, non-steroidal anti-inflammatory drugs,[9] taping,[10] night splints,[11] physical therapy and rest.

Extensor tendonitis, also known as "lace bite" or "skate bite," is a frequent complaint, often times self-diagnosed, occurring when the pressure from the upper laces along the top of the skate causes a tenosynovial reaction in the tibialis anterior and extensor tendons.[12] This problem may be resolve as the skate boot becomes worn in, but may also be treated with rest, redistributing the pressure of the laces so that there is less localized stress on the extensors, using insert-cushioning, or custom skate molding to accommodate the extensors.

Maintaining balance on a thin edge is a cause of overuse injuries for the frontal plane stabilizers, especially in over-pronators. This may lead to "overuse injuries" such as peroneal tendonitis, posterior tibial tendonitis, and Achilles tendonitis. Many times these injuries can be treated with custom orthotics which will help stabilize the frontal plane as well as provide arch support in the sagittal plane.[2] Heel lifts, custom skate molding, blade re-positioning, and localized padding can help with many of these injuries that require minor

adjustments to biomechanics and associated pressures involved. Each of these injuries requires close monitoring and slow return to skating after off-ice activities can be completed without pain. Due to the competitive nature of athletics, patient education is crucial to prevent premature return to activity and risk of reinjury.[3]

Although ankle fractures and stress fractures are common in many skating athletics such as figure skating and hockey, the injuries are most often procured during off-ice training[2] or obtained from high-impact collisions and jumping rather than the intrinsic nature of skating biomechanics. Nevertheless, it has been reported that skates are occasionally caught in a rut, which can lead to forceful external rotation of the leg and a high ankle sprain (distal tibiofibular syndesmosis).[13] The treatment should follow the standard course for high ankle sprains of athletes. Proper imaging includes a stress radiographs of the ankle syndesmosis to rule out a diastasis, and conservative care involves compression, ice and elevation to reduce swelling and quicken return to play.[12] If there is evidence of mortise widening or instability, surgery involving open fixation of the syndesmosis should be considered. Return to activity should only resume once off-ice training can be completed without pain or risk of reinjury.

Conclusion

Clinicians should be aware of common skating ailments of the lower extremity and the proper way to treat such injuries in order to allow athletes and weekend warriors to return to activity as quickly and safely as possible. Patient education on proper warm-ups and stretching, as well as off-ice training and rest, is important for the prevention of future injuries.[3] In addition, coaches and skaters should be educated on the importance of patient compliance.

There is limited data available pertaining to the biomechanics during skating, and more research needs to be done to further understand the nature of certain injuries in order to help return skaters to the ice safely and comfortably. To date, the link between skating biomechanics and hallux valgus, noted in up to 57% of skaters,[14] has yet to be elucidated.[3] Skate fitting and uncorrected over-pronation have been implicated,[14] but just how much the nature of the skate cycle itself contributes to these pathologic forces has not been established. The authors are interested in whether the skate cycle exacerbates hallux valgus formation in ill-fitting skates and if subtalar joint motion in the skate can also play a part. There is not enough data on subtalar joint motion in skates as of yet to determine if the motion is significant enough to become pathological. With increasing technological advancements, biomeasurements may provide more data in the future.

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Subungual Osteochondroma vs Subungual Exostosis: Evaluating the Utility of Histologic Studies

Riddhi Shah, DPM 2017. George Alvarado, DPM 2017

Introductions

Subungual osteochondroma is the most common bone tumor in the body and the most common benign bone tumor of the lower extremity, occurring primarily in the long bones but can also involve the calcaneus or talus.[1] This is in contrast to subungual exostosis which commonly arises on the distal phalanx of the hallux.[2] Subungual osteochondroma was described by Virchow in 1891 as originating from cartilaginous growth plates, and in 1847 Dupuytren described a bony outgrowth of the hallux that was later termed a "Dupuytren exostosis".[1,3] Subungual osteochondroma has been used interchangeably with subungual exostosis or has been thought to be a variation of the aforementioned. Lee et al concluded that subungual osteochondroma and subungual exostosis are distinct pathologies with different radiographic and histological features. The major histological difference between subungual osteochondroma and subungual exostosis is the composition of the cartilaginous cap as seen in Figure 1 and Figure 2.

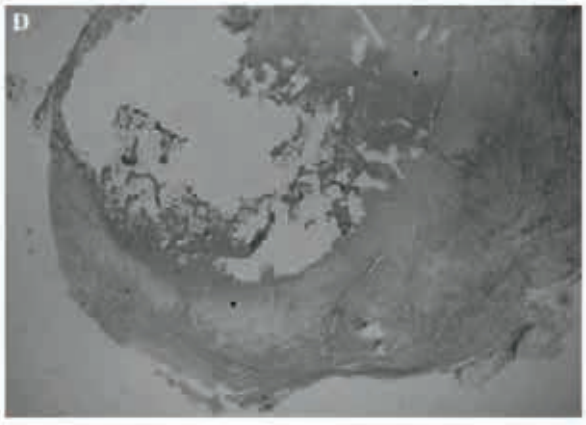

Figure 1: Histology of the subungual exostosis, revealing a mature trabecular bone pattern covered by fibrocartilaginous tissue.[3]

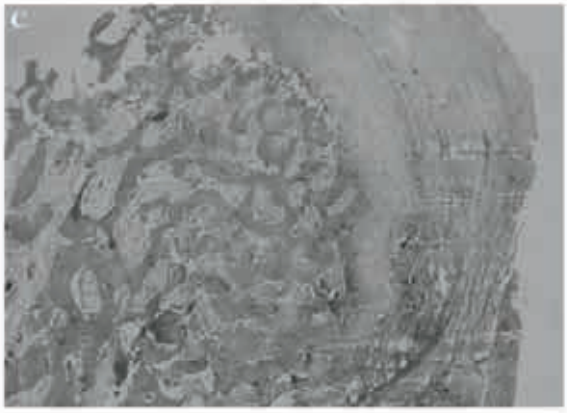

Figure 2: Hematoxylin-Eosin staining of osteochondroma revealing trabecular bone covered with hyaline cartilage.[3]

Consulting radiology, pathology, and oncology will help with making the correct diagnosis. Clinically, both subungual exostosis and subungual osteochondroma will usually be painless, but mechanical irritation due to shoe gear or certain activities will elicit pain prompting a visit to primary care or podiatry. Clinical signs and symptoms include subungual pain or discomfort, overlying nail plate elevation or deformity, erythema, throbbing sensation of the hallux, paresthesia, and vascular complications. Physical examination for both subungual pathologies reveals a firm fixed nodule with or without a hyperkeratotic surface.[1,4] Subungual osteochondroma and subungual exostosis are similar clinically and require the same surgery, however they exhibit different pathologies.

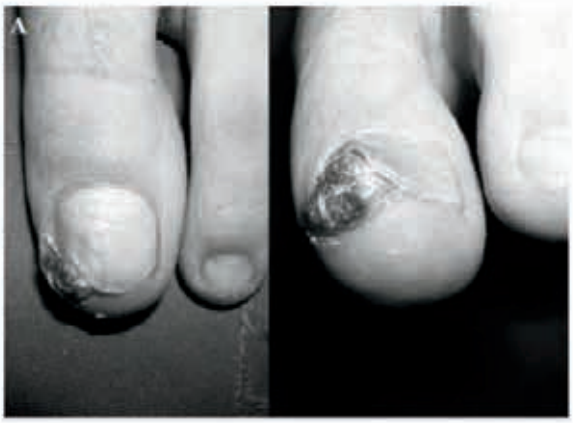

Figure 3: Subungual exostosis with a hard stony consistency that displaced the nail plate upward.[3]

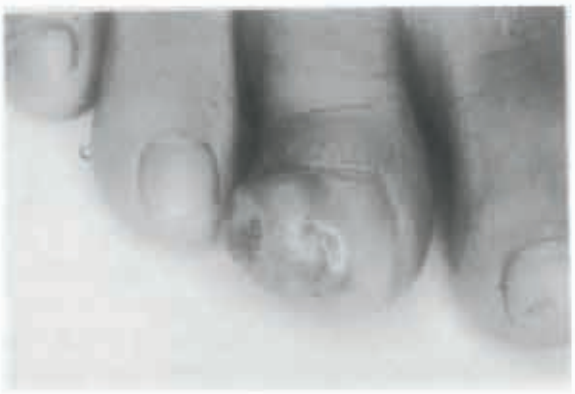

Figure 4: Firm, shiny, erythematous subungual osteochondroma.[6]

Symptoms

Subungual osteochondroma is a common benign skeletal neoplasm that is more common in toes than in fingers. It is more common in young adults and demonstrates a male to female ratio of 2:1.[5] Clinically, subungual osteochondromas present as a painless growth that is localized to the metaphysis of a long bone as seen in figure 4; it can cause local irritation, pain, and paresthesia if it impedes on a neurovascular bundle or adjacent soft tissue.[1]

Subungual exostosis is an uncommon, benign bony tumor that is frequently seen in the hallux, mainly seen in an older population of 20-40 years of age with a more female to male ratio of 2:1.1.[5] Though the pathogenesis is not clear, it can be due to an inflammatory or reactive growth process of the fibrous and cartilaginous tissue to make the fibrocartilaginous cap. On clinical exam, subungual exostosis presents as a fixed, firm, tender nodule that is usually less than 0.5cm in diameter and continuous with the distal phalanx as seen in figure 3.[4]

Subungual osteochondroma and subungual exostosis share similar traits that make it difficult to distinguish between the two. Both grow progressively and can cause pain and nail deformities, and can also compress both the nail plate and distal phalanx.[6]

Studies

Radiographs

Plain radiologic views, anteroposterior or dorsoplantar and lateral of the foot, aid in differentiating subungual exostosis and subungual osteochondroma for diagnosis. The subungual exostosis protrudes from the dorsal or dorsomedial aspect of the distal phalanx and extends distally away from the epiphysis as seen in figure 5.[2,3] The base of the exostosis can be pedunculated or sessile while the tip is either flat, cupped, or dome-shaped.[3] Bone trabeculae can be observed at the base if the subungual mass has matured. However, no periosteal reaction and destructive changes to the phalanx are seen.[5] Since subungual exostosis is composed of a fibrocartilaginous cap, the cap appears radiolucent.[3]

On the other hand, subungual osteochondroma presents as trabecular bone growth from the juxta-epiphyseal aspect of the distal phalanx.[5] The main radiologic feature of subungual osteochondroma is medullary bone that is adjacent to the exostosis stalk and the cortical bone which is seen in figure 6.[1,7] Additionally, subungual osteochondroma has an endochondral ossification, and can can grow slowly, then experience an accelerated growth through puberty and adolescence until the closure of the physeal plates.[5] Similar to

subungual exostosis, the base of a subungual osteochondroma can be pedunculated, sessile, or circumscribed. Its hyaline cartilage cap is radiolucent, unless it is calcified.[3] It is common to misdiagnose subungual osteochondroma for subungual exostosis due to their similar radiographic features.[5]

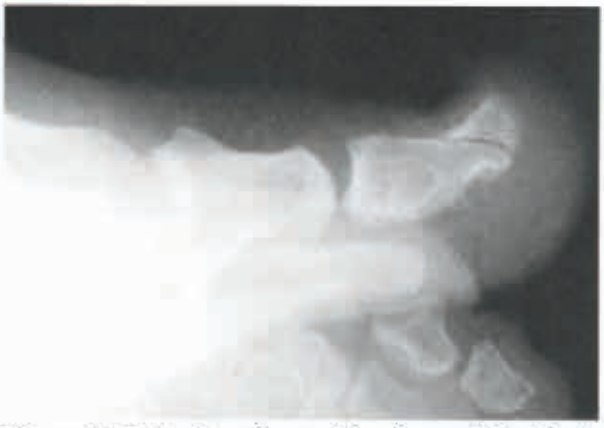

Figure 5: Lateral radiographic view of the foot showing subungual exostosis.[2]

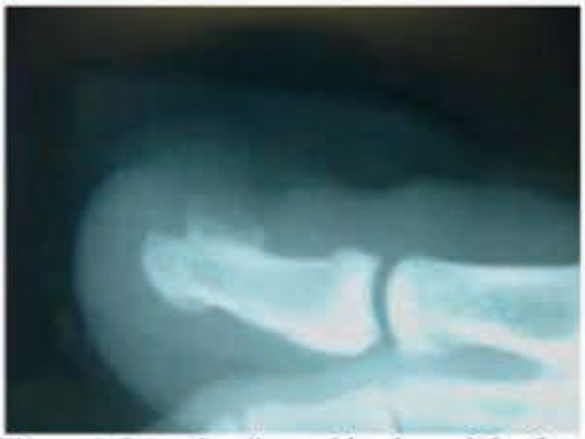

Figure 6: Lateral radiographic view of the foot showing subungual osteochondroma.[7]

Histology

Though plain radiography helps to differentiate subungual osteochondroma from subungual exostosis, the mainstay choice for diagnosis is histology. The main histologic difference between both is the cartilaginous cap. Subungual exostosis has a trabecular bone base or stalk with a fibrocartilaginous cap.[3,8,9] The subungual mass is composed of cancellous bone with osteoid trabeculae that has calcified areas. Beneath the fibrocartilaginous cap, which surrounds the physis, it has trabecular bone made by both proliferating fibroblasts and endochondral ossification. If the subungual mass is immature, it will have a thick cartilage cap; however, the mature mass has a thin cap of cartilage which has been replaced by trabecular bone.[3] Conversely subungual osteochondroma is primarily composed of hyaline cartilage.[3,8,9] Collagen fibers are observed to be either normal or slightly fibrotic in the papillary dermis. Islets of hyaline immature cartilage with chondrocytes are seen beneath the dermis.[3]

Surgery

Although subungual exostosis and osteochondroma are distinct pathologies, their surgical approach is the same. Classification systems of subungual exostosis related to clinical presentations such as size, shape, location, and protrusion/non-protrusion of the lesion can be helpful in directing the surgical approach listed in table 1.[10]

Table 1. Surgical Approaches to Subungual Exostoses Type[10]

Type	Surgical Approach
Type I (mild deformity)	Distal exostectomy ("fish-mouth"–type incision)
Type II (exostosis distal to the nail plate)	Distal exostectomy (semi-elliptical distal wedge) ± partial matrixectomy ("cold-steel" matrixectomy)
Type III (exostosis under the nail plate)	Nail unit resection (Kaplan's matrixectomy) ± dorsal exostectomy Total matrixectomy (Zaddick's matrixectomy) ± dorsal exostectomy
Type IV (medial or lateral condyle)	Cold-steel matrixectomy (Frost's matrixectomy) ± exostosis resection

In general the surgical steps for both subungual osteochondroma and subungual exostosis include:

1) Partial or total removal of the nail plate.

2) Incision and dissection down to normal bone base, removing the lesion with its cartilaginous cap and without overlying nail bed.

3) Burr to smooth the normal bone at what was the lesion base.[11]

4) Suture the nail bed and fix nail plate.[12]

Complications

Onycholysis and onychodystrophy are the main complications of surgical treatment of both subungual osteochondroma and subungual exostosis. DaCambra et al used a pedicled VAC dressing as postoperative wound management for subungual exostosis hoping for a favorable wound closure and preventing onychodystrophy. The dressings were changed every 2-3 days for 12 days and this decreased the morbidity and contributed to a quicker recovery time.[8] Basar et al found that nail bed damage due to onychodystrophy could not be avoided in the presence of a protruding lesion. However, in non-protruding lesions, a fish mouth incision of the nail bed was associated with less iatrogenic damage, and prevented nail deformity that could prolong recovery time.[12]

Recurrences

Complete excision of both subungual osteochondroma and subungual exostosis is necessary to prevent recurrences. Recurrence rate of subungual osteochondromas is low and reported to be <1.8%.[7] Recurrence of subungual osteochondroma is likely due to nonaggressive curettage of bone and can likely lead to cells of resected perichondrium or cartilage cap being left in non-resected bone or nail bed.[7] Additionally, repetitive trauma to the phalanx post resection can cause recurrence.[7] Dumontier et al states it is safer to excise at the base of cortex until spongy bone is observed to ensure that there is complete surgical removal, which may require removing part of the nail.[5] In the study performed by Basar et al, the fish mouth incision has no recurrences.[12]

Conclusion

Although often mistaken for the same pathology, subungual osteochondroma and subungual exostosis are histologically and epidemiologically distinct. A review of the current literature does not support the idea of two distinct surgical approaches, but rather an emphasis on preventing iatrogenic injury to the nail bed and aggressive curettage through a common surgicial approach in order to decrease the incidence of complications and recurrences respectively. This questions the use of obtaining histologic studies to determine the diagnosis. Is it necessary to make the patient go through the additional hassle and time of obtaining histological studies when in the end the same surgical procedure will be used? Besides being able to explain to the patient the exact pathological impression of their subungual lesion, there is no reason to obtain histological studies other than confirming the diagnosis.

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Chronic Exertional Compartment Syndrome: A Review of Treatment Techniques

Ana Emirzian, DPM 2019

Introduction

Chronic Exertional Compartment Syndrome (CECS) is characterized as pain induced in the leg due to exercise or other exertional activities that subsides soon after the exertion has ended.[6] Unlike its acute form, CECS is not caused to trauma and its pathophysiology is not completely understood. This article will present the current theories on the pathophysiology and etiology of CECS, while focusing on the methods of some of the treatment options available.

Pathophysiology

There are many presumed etiologies of CECS. One suggested mechanism is ischemia of the muscles involved due to intracompartmental pressure as the cause of the syndrome.[6] Persistent muscle contraction leads to an increase in intracompartmental pressure that usually subsides within 5 minutes, however in CECS patients, the pressure remains elevated even after the muscles are not exerting any force.[6] The muscles of the lower leg are contained within four compartments: lateral, anterior, deep posterior and superficial posterior; each surrounded by a layer of fascia. A study by Gershuni et al. found that during exercise, the volume of the compartments increased due to muscle contraction, resulting in an increase in the intramuscular pressure.[4] In patients with tight fascia, the pressure build-up exceeded 30 mm Hg, the threshold for perfusion of the muscles. Such a high pressure within the fascial compartments can lead to a decrease in blood flow to the muscle belly, resulting in painful ischemia.

Epidemiology

Chronic Exertional Compartment Syndrome is commonly experienced during repetitive loading or exertional activities found in about 14% percent of patients who complain of lower leg pain.[6] Runners often experience pain within the first five minutes of running, or after a certain distance, depending on the runner; the pain does not subside until the exercise has stopped.[6] A study by Davis et al. researched the characteristics of the patients who were diagnosed with CECS.[2] Of the 226 patients enrolled, 153 of them were diagnosed with the condition, with an age range from 13-69 years and a mean age of 24. Of those diagnosed 60.1% were female and 92.2% of the patients were reported to participate in sports, running being the most prominent form of exercise. These statistics should be considered when physicians encounter patients experiencing lower leg pain, to consider CECS as a differential diagnosis.

Treatment

The current standard of conservative treatment includes stretching, altering gait, providing orthotics, changing footwear or discontinuing the activity that results in pain.[8] Conservative approaches should be considered during the early stages of diagnosis, however if symptoms persist patients can opt for surgery.[8] Fasciotomy is the definitive treatment option and can be performed in an open surgical method or endoscopically.[6]

A case presented by Knight et al. provided a detailed account of a bilateral fasciotomy performed with an endoscope.[5] The patient was first sterilized and a tourniquet was inflated to to 250mm Hg. An incision was then made distally, centered on the superficial peroneal nerve. Another 4-6mm incision was made 12cm proximal to the tip of the lateral malleolus and 5cm lateral to the crest of the tibia, after which cuts were made down to the subcutaneous tissues. While the leg was kept at supine position a 30 degree arthroscope was placed to allow visualization of the spectrum suture passing device. The No.1 Polydioxanone suture was then placed and the skin retracted to

provide adequate view of the underlying fascia and protection of the superficial peroneal nerve.

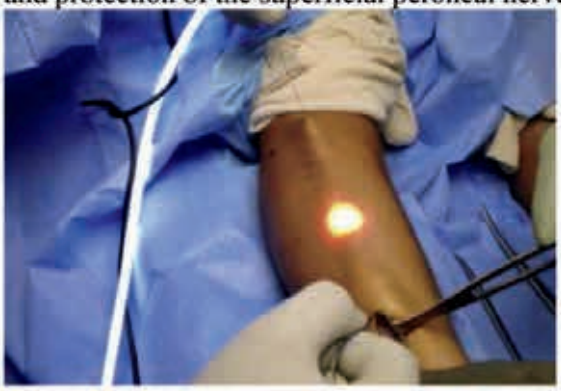

Figure 1. Using the suture to retract the skin and to allow visualization of the fascia.[5]

Next, incisions were made to the anterior and lateral compartments. Incisions ran longitudinal on the leg. After the fascia was incised the tourniquets were released and the skin was closed using No. 2.0 vicryl. Patients had minimal soft tissue damages and were allowed to return to full activity within 6 weeks of the surgery.

A study by Sebik and Dogan found success in relieving pain in all patients that underwent the surgery via the endoscopic method.[7] Of the six patients involved in the study, all had early rehabilitation and minimization of soft tissue damage, allowing full recovery within six weeks.

Fasciotomies can also be done the open surgical method. Bresnahan and Hennrikus provide a detailed account of the surgery, based on a case performed on a high school soccer player.[1] Similar to the endoscopic fasciotomy, and prior to anesthesia the pressure in the anterior compartment of the patient's leg was measured, indicating an above normal resting pressure value (figure 2). Once this was established a tourniquet was set and a 2.5 inch incision was made above the defected fascia, on the anterolateral aspect of. Blunt dissection of the fascia followed; similar to endoscopic procedure, the superficial peroneal nerve was made sure to be identified and gently dissected. The fascia was opened in both the lateral and anterior compartment spanning the leg proximally and distally from the incision point. Once the fascia was released the tissue was closed with vicryl, resulting in a decrease in compartmental pressure.

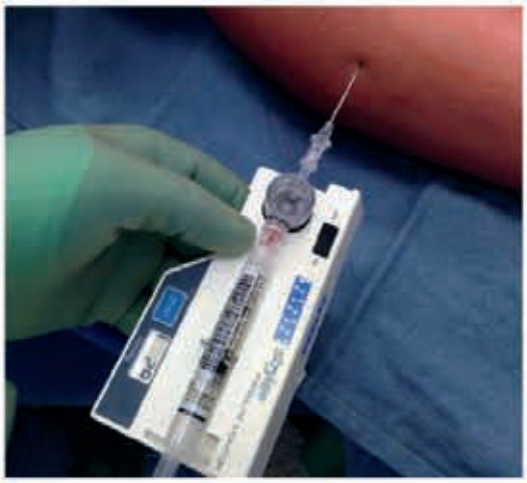

Figure 2. Measuring intracompartmental pressure using a Stryker Pressure Monitor System wick catheter.[1]

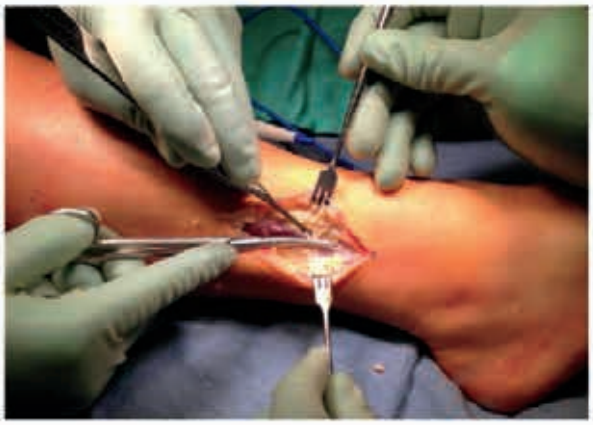

Figure 3. Releasing the lateral compartment fascia.[1]

Both the open and endoscopic methods of fasciotomies described by Bresnahan and Hennrikus and Knight et al. respectively, have proven to be successful in majority of cases, allowing patients to resume activity without pain or discomfort.[1,5] This prompts the question: why would a patient or physician would choose one method over the other? A study performed by Wittstein et. al sought to analyze the results of 14 cases of endoscopic procedures in 9 patients, as opposed to the open method, and found success in avoiding any neurovascular injuries in all patients.[9] In addition, 8 of the 9 patients were able to return to normal

preoperative activities after proper post operative healing. Endoscopy improves on the open method by reducing the risk of an inadequate release, complications involving wound healing, soft tissue disruption or neurovascular injury.[5] The major difference lies in the optimal visualization of the compartments of the leg and the superficial peroneal nerve (figure 1) to ensure the proper release of the compartment, leading to less soft tissue damage.[5] However, more studies should be done to properly compare the surgical methods.

Although fasciotomies are proven to be effective in treating CECS, a study by Diebal et al. study found a non-invasive approach to helping relieve pain for runners.[3] The study examined the affects of forefoot running on 10 patients in line for surgery to improve pain. Just 6 weeks after the intervention the patients exhibited decreased compartmental pressure. Patients reported significant decrease in pain, with an increase in running distance (figure 4). Patients were instructed to avoid initial hindfoot strike. Hindfoot strike led to a dorsiflexed ankle when contacting the ground, increasing the contraction of the tibialis anterior muscle. In addition to this, during toe off, the plantarflexed ankle led to increased contraction of the gastroc/soleous complex. An increase in contraction forces caused increase in compartmental pressure. During forefoot running the knee is not extended fully which decreases the activity of the tibialis anterior. The hamstrings are more active during lift off which relieves the calf muscles, causing less contraction. The decreased activity of these muscles allows for decreased pressure in the compartments of the leg, and improvement of painful symptoms.

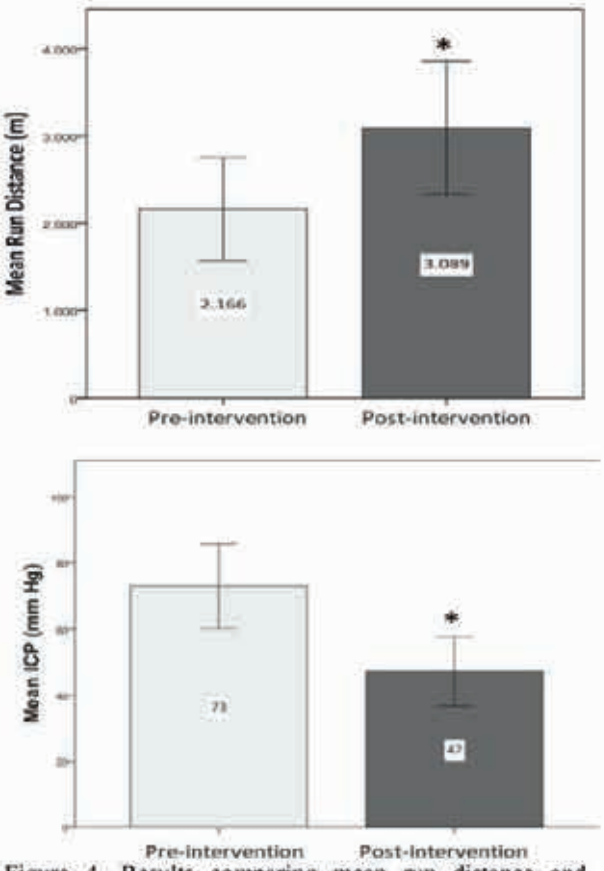

Figure 4. Results comparing mean run distance and pressure, before and after the intervention.[3]

Conclusion

The symptoms associated with Chronic Exertional Compartment syndrome can have significant lifestyle effects for patients. CECS is more commonly found in patients under the age of 40, with symptoms appearing during exertional activities.[6] The syndrome exhibits increase pressure in the compartments of the leg leading to ischemia and pain. Conservative treatment can be used during the early stages; however open or endoscopic surgical techniques are the definitive treatment for relieving the pain.

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Achilles Tendon Repair: A review of recent literature for the treatment of Achilles tendon rupture

Eduardo Hernandez, DPM 2017; Daniel Kim, DPM 2017; Alexander Lister, DPM 2017

Introduction

The Achilles tendon, the strongest tendon in the human body, is the connection point between the triceps surae muscle and the calcaneus.[1] The function of the Achilles tendon is to facilitate heel lift during the propulsive phase of gait. If this tendon is ruptured, ambulation will be challenging.

An Achilles tendon rupture is an injury that typically plagues athletes - from weekend warriors to professional athletes. Historically, the Achilles tendon has been repaired surgically, but in recent years there have been numerous emerging articles that state non-surgical treatment with early weight bearing has resulted in no difference in re-rupture rates as compared to surgical treatment.[2] The purpose of this article is to present some of the latest evidence for surgical vs. conservative treatment of Achilles tendon ruptures.

Anatomy

The Achilles tendon (tendo calcaneus Fig.1 and Fig. 2) is formed from the distal aponeurosis of gastrocnemius and soleus muscles. The tendon inserts into the middle ⅓ of the posterior calcaneus. The Achilles tendon is lined by the paratenon, a structure comprised of thin sliding membranes which function as a sheath for the tendon to move freely within the surrounding tissue.[3]

The Achilles tendon has three sources of blood supply: proximally, the musculotendinous junction, distally, at the insertion point at the calcaneus, and the paratenon, spanning the entire length of the tendon itself.[4] The vessels that surround the paratenon provide the most important supply to the tendon.[5] The two main vessels responsible for this blood supply of the Achilles tendon are the posterior tibialis artery and peroneal artery.[6]

Nerves supplying the gastroc-soleus complex and collateral branches from cutaneous nerves are the major nerves innervating the Achilles tendon.[7]

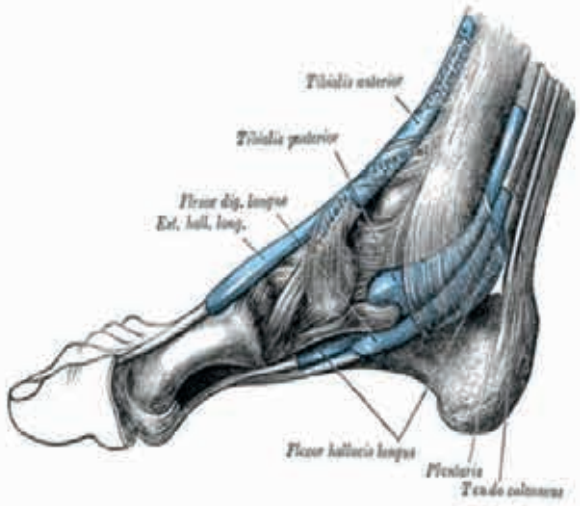

Fig 1. Anatomy of Achilles tendon and surrounding structures, medial. Henry Vandyke Carter - Henry Grey (1918 *Anatomy of the Human Body*) Gray's Anatomy 20th ed. 10 January 2016. (in public domain at Bartleby.com: Gray's Anatomy, Plate 1242).

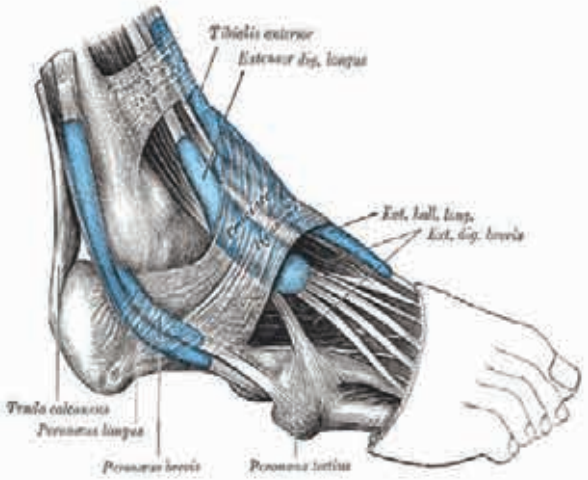

Fig 2. Anatomy of Achilles tendon and surrounding structures, lateral. Henry Vandyke Carter - Henry Grey (1918 *Anatomy of the Human Body*) Gray's Anatomy 20th ed. 10 January 2016. (in public domain at Bartleby.com: Gray's Anatomy, Plate 438).

Pathophysiology

Achilles tendon injuries and ruptures may occur in either an acute or chronic setting. Metabolic diseases, flouroquinolone use, and increased duration/frequency level of activities are just a few predisposing factors associated with Achilles tendon ruptures.[7]

One of the most common metabolic diseases that have been implicated includes diabetes, due to deposition of advanced glycated end products that deteriorate tendons. Other metabolic diseases associated with tendon injuries include gout, hypercholesterolemia, and other more rare metabolic illnesses.[8]

The FDA has issued a black box warning for the use of fluoroquinolones in association with Achilles tendon ruptures. Specifically, those over the age of sixty, have received a kidney transplant, a lung transplant, or have used anabolic steroids are at a greater risk for Achilles tendon ruptures while consuming fluoroquinolones. The mechanism by which this rupture occurs remains unclear.[9] Proposed mechanisms range from decreased fibroblast proliferation to inhibition of tenocyte migration required for tendon repair and response to injury.[9]

As cited in Maffulli et al, Hattrup and Johnson found that in the majority of cases, the etiologic basis for the rupture appears to be a combination of intra-tendon degeneration and mechanical stress. In other words, Achilles tendon ruptures don't necessarily occur by traumatic injury to a healthy tendon but instead are caused by eccentric contraction on a pathologic asymptomatic tendon.[10] Moreover, the watershed area has been associated with Achilles tendon ruptures.[5]

Watershed zones/areas have been shown to have the greatest propensity to rupture. Specifically in relation to the Achilles tendon, the watershed area is an area of decreased vasculature found two to six centimeters above the insertion.[5] It is an area where the supply from different vascular territories anastomose. They are more prone to degeneration because nutrients and oxygen delivered by blood supply to the tendons must reach these zones via small capillaries making them vascularly impoverished. It has been hypothesized that in areas of impoverished vascularity, cells depend on the ability of tissue fluids to diffuse through the tendon for their survival.[11] The term "watershed zone" is not unique to the Achilles tendon. Watershed zones have been found to be the most common site of rupture in other tendons, including the posterior tibial and supraspinatus.[6]

According to Chen et al., the watershed zone has surgical implications simply because the tissues around tendons act as conduits for arterial supply to the tendon. Due to the hypovascularity at the midsection of the Achilles tendon, it is less likely to heal after surgery. Chen et al. performed an angiographic study on twenty cadaveric lower limbs in which they found a drastically reduced number of blood vessels by anatomic dissection, radiographically, and histologically between two to seven centimeters proximal to the insertion of the Achilles tendon.[6]

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Treatment

Recent Studies of Surgical Therapy for Achilles tendon Rupture

Since the 1990s, there have been numerous publications arguing for operative repair of an Achilles treatment ruptures. The main argument for surgical treatment is to surgically reinforce the Achilles to reduce the chances of re-rupture. Surgical intervention allows the physician to take measure to ensure the ruptured Achilles will not re-rupture. In 2001, The Journal of Bone and Joint Surgery published a prospective randomized study, by Moller *et. al*, comparing surgical treatment to conservative treatment.[5] Of the 53 treated conservatively, 21 patients re-ruptured.[5] Of the 59 treated surgically, one patient re-ruptured.[5] The study used Fisher's test to analyze the data, a p-value of 0.0013 was obtained, showing that these results were statistically significant.[5] In conclusion, they determined that surgical treatment along with early functional treatment is the optimal therapy for Achilles tendon ruptures.

In a more recent review, Wilkins and Bisson reviewed and compared seven level 1 trials of

operative and nonoperative treatment for Achilles rupture repairs through meta-analysis.[12] The Coleman methodology score for these seven articles were within a range of 78-97.[12] The results showed surgical treatment yielded 3.6% re-rupture rate while conservative treatment yielded 8.8% re-rupture rate.[12] When this data was pooled and meta-analyzed, it was found to be statistically significant.[12]

In 2015, another systematic review of meta-analyses comparing surgical to conservative treatment for Achilles tendon rupture was done by Erickson *et. al*. In their review of 9 different articles comparing types of treatment, they discovered seven of the nine articles showed surgical treatment decreased rupture rates of Achilles tendon repairs.[13] Lastly, Erickson *et. al.* discovered that those who did have a lower re-rupture rate were able to return to work sooner.[13]

The Achilles tendon is very important when it comes to propulsion; it is a major connection between the foot and the rest of the body. Past and recent studies have shown surgical intervention is an effective treatment for Achilles tendon ruptures.

Recent Studies of Conservative Therapy of Achilles tendon Rupture

The evidence for the advantages of conservative care for Achilles tendon ruptures have become more prevalent over the years. Conservative treatment includes non-invasive procedures and because of the daunting stigma behind 'surgery', patients are more responsive towards this method. Moreover, by avoiding surgery, operating room complications, infection, and contamination during the hospital visit are completely avoided. But more importantly, studies show that the rate of re-rupture of the Achilles tendon when comparing surgical to conservative treatment is nearly the same. In one study looking at 24 patients who were treated operatively vs. 23 patients who were treated conservatively, when it came to re-rupture, pain, and biomechanics there was no statistically significant difference.[14] This finding coincided with a study done in 2012 utilizing the Egger and Begg test and statistical analysis which showed that as long as functional rehabilitation with early range of motion (ROM) was employed, re-rupture rates were equal in surgical and non-surgical patients (risk difference 1.7%, p=0.45). Conservative treatments also had fewer complications than surgical treatments.[15]

The general outline for conservative treatment includes serial casting. This includes initially placing the patient's foot in about twenty degrees of plantarflexion and then placing a cast over the foot, supported with an ankle boot with a rocker bottom sole and heel lifts.

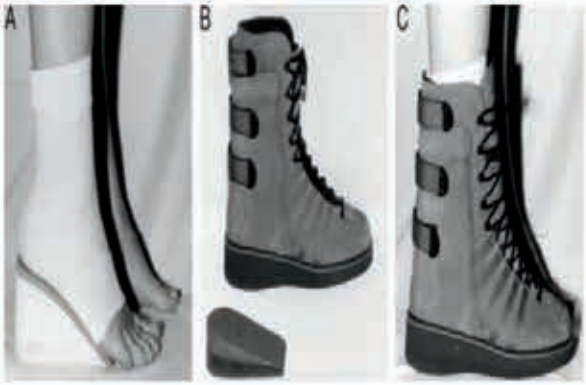

Figure 3. Casting Method and application of Ankle Brace. Adapted from "Nonoperative treatment of of Acute Rupture of the Achilles Tendon," by Weber, Martin, 2003, The American Journal of Sports Medicine, 31(66).

The thickness of the heel lifts and initial amount of plantarflexion may vary slightly amongst physicians. The cast is removed after seven days and the integrity of the Achilles tendon is measured via the Thompson test and palpation during physical exam. The Thompson test is a diagnostic test to evaluate for a complete rupture of the Achilles tendon. With the patient's leg completely extended and the foot hanging off the table, squeeze the calf to see if the foot plantar flexes. If plantarflexion does not occur, you have a complete Achilles rupture.

After examination, the foot is once again placed in a plantarflexed position of twenty degrees and the cast is changed every 10-14 days for hygienic purposes. During this time the patient is instructed to use the boot all day and only instructed to remove it during the evening

when in bed; as the cast alone if WB cannot hold the foot in the wanted position. Lastly at six weeks, the cast is removed and the heel lifts are gradually decreased in the boot depending on how the patient is doing, and ideally completely removed at eight weeks but still instructed to wear the ankle boot for an additional four weeks for support. Throughout the treatment course, Physical Therapy is administered two to three days post injury with isometric strengthening exercises and a stationary bike. At 6 weeks ROM exercises are implemented for the ankle, and at eight weeks calf muscle strengthening is added to the Physical Therapy regimen.

Lastly, one important thing to consider during conservative treatment of an Achilles tendon rupture is dynamic rehabilitation, early ROM, and whether it is crucial for obtaining similar results to those who receive surgical intervention. The idea of casting and holding the foot in a plantarflexed position is to expedite the healing of the rupture. However, regaining strength and trying to get back to a 'normal biomechanical' foot requires extensive dynamic exercises and strengthening. In a blinded, randomized, controlled, parallel superiority trial; comparing 29 patients in a weight bearing groups vs. 27 in the control group (no early dynamic rehabilitation) demonstrated that the weight-bearing group at twelve months had a better quality of life.[2] With that in mind, the evidence shows that having physical therapy that includes dynamic rehabilitation, stretching, strength and ROM exercises early on and continuously through therapy is the best method thus far in decreasing healing time and getting back to normal function.

Conclusion

Previous research showed surgical treatment for an Achilles tendon rupture as the best treatment to prevent re-rupture. Through more recent literature, we have discovered that conservative treatment is just as viable of an option as surgical treatment when it comes to this injury. Additional in-depth, longitudinal studies with meta-analyses are necessary to further understand the result of surgical treatment and conservative treatment for an Achilles tendon rupture.

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