CHRONIC EXERTIONAL COMPARTMENT SYNDROME OF THE LOWER EXTREMITY: A Review

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INTRODUCTION AND HISTORY

Chronic exertional compartment syndrome (CECS) is a condition in which patients experience pain with exercise that usually subsides with resting.¹ CECS is defined as increased pressure within a closed fibro-osseous space that can cause reduced blood flow and tissue perfusion within that space. These changes within a confined compartment can lead to ischemic pain and damage to the tissues of that compartment. However, pain is relieved relatively quickly with rest and there is typically no permanent damage to the tissues. CECS is often a recurrent condition that is associated with repetitive exertion. CECS is often seen among athletes with elevated exercise regiments that push their intra-muscular pressures within the affected compartment to become painful and tight, preventing them from further activity.²

CECS can present itself as being acute or chronic. Acute compartment syndromes, whether introduced by repetitive exertion or by way of trauma, require immediate attention to prevent irreversible damage to the tissues of the offending compartment. Patients that present with acute compartment syndromes will often display symptoms of severe pain that is exacerbated with passive stretch of the muscles and does not resolve with immediate resting. Development of parenthesis and pallor can be followed by the loss of pulse in the distal extremity. Acute compartment syndromes often occur following high-energy trauma with or without an open fracture. Exercise-induced acute compartment syndrome may not develop symptoms until 24-48 hours following exercise.²

Wilson first described exertional compartment syndrome in 1912 in an expedition to Antarctica.³ The symptoms of CECS were later described by Vogt in 1943 as "March Gangrene." It was not until 1962 that French and Price first documented elevated intracompartmental pressures in the lower extremities as the cause of chronic exertional compartment syndrome. Surgical treatment by way of a fasciotomy was first described by Mavor in 1956.²

ANATOMY

Chronic exertional compartment syndrome has been reported in the hand, forearm, thigh, gluteus, lower leg, and foot. The 3 most commonly affected areas of the body by CECS are the forearm, the thigh, and the lower leg.^{2,3} The lower leg is divided into four muscle compartments, which are the anterior compartment, the lateral or peroneal compartment, the superficial posterior compartment, and finally the deep posterior compartment (Figure 1). The anterior compartment consists of the anterior tibial muscle, extensor hallucis muscle, and the extensor digitorum muscle. The lateral or peroneal compartment contains the peroneal muscles. The superficial posterior compartment contains the soleus and gastrocnemius muscles. The deep posterior compartment consists of the posterior tibial muscle, the flexor digitorum longus muscle, and the flexor hallucis longus muscle (Figure 3).² There has been discussion within the literature that looks at the existence of a fifth compartment within the deep posterior compartment. The failure of surgical decompression with a fasciotomy within the deep posterior compartment suggests that the tibialis posterior muscle may exist within its own osseofascial compartment (Figure 2).³

FREQUENCY

The incidence of CECS in the population is largely unsubstantiated, however reports have estimated it at 14%-27%.⁵ CECS has a higher male predominance, although this was based largely on military records and athletic programs. More women have become involved in competitive sports and recreational activities, so now it is reported that the incidence among men and women appears to be similar. The most common reported anatomical area of occurrence is in the lower leg.³

PATHOPHYSIOLOGY

Pain caused by CECS is derived from the same process in acute compartment syndrome, which is compromise of the vascular supply leading to ischemia. Various mechanisms have been proposed as to the cause of this ischemia including arterial spasm, capillary obstruction, arteriovenous collapse, and venous outflow obstruction.3 Strenuous exercise can cause muscle fibers to swell to 20 times their resting size leading to a 20% increase in muscle volume and weight.¹ In agreement with Laplace's Law, stating that a capillary membrane subject to internal and external pressures reaches an equilibrium based on those forces, the blood flow through muscles are regulated by the arteriole based on the tension placed on the vascular wall.² The increase in intramuscular pressure in turn causes a decrease in arteriolar blood flow. When the blood flow is not sufficient to meet muscular tissue oxygenation from the decreased venous return and perfusion, the patient experiences pain with continued activity. Increased intracompartmental pressures during the relaxed phase of exercise have the greatest effect on muscle ischemia. Transient increases in compartmental pressure have been demonstrated with increased pressure in muscles at rest or postexercise.5

In a person without CECS these pressures normalize within five minutes after cessation of exercise. However, patients with CECS, have pressures that remain elevated for 30 minutess or longer in many cases.⁴ It is not clear as to why patients who experience CECS have increased total intramuscular pressures at rest and have higher pressures longer after exercise. Some have postulated that these patients have fascial defects over the anterior lateral lower extremity. In one study by Styf et al, they found that 39%-46% of patients have fascial defects or hernias that were 1-2 cm in size.⁶ It was found that these hernias occurred often at the exit of the superficial peroneal nerve and in turn the defect or the muscle bulge compressed the nerve. However, while these defects can be a contributing factor, these are not present in all patients as the study found. The study does show that another contributing factor of pain can be compression of nerves and occasionally Tinel's sign may be found at the herniated site.

Yet another, but less accepted theory exists called the mechanical damage theory. This theory states that myofibril damage and release of protein-bound ions results from prolonged exercise. This frequently effects runners and occurs in the anterior compartment. The release of protein-bound ions damage muscle and increases osmotic pressure so that swelling and local tenderness occur.⁷ Even though these explanations for the cause of pain in CECS seem plausible, no single theory has

been overwhelmingly accepted. It would seem that the pain caused by CECS is either ischemic or compression of a nerve, and needs further study. It is likely that a combination of anatomic restrictions contributes to the incidence and severity of CECS.

EVALUATION

As with any medical problem, chronic exercised induced lower leg and foot pain begins with a thorough history. Typically these patients are runners or involved in a sport that requires a lot of running such as soccer, basketball, and football. Almost all patients report that the pain adversely affects their performance and deny any trauma. Often the symptoms start as a dull ache that usually starts within 30 minutes of starting exercise. Patients then state that they develop tightness or fullness accompanied by burning, cramping, numbness, and aching as the exercise is continued. More often than not these symptoms may cause the patient to have to discontinue the activity and the symptoms may persist even after stopping the activity. Rest usually relieves the pain and the pain will not persist into the following day unless the activity or exercise is continued the next day. The pain is normally predictable as far as time after starting activity is concerned and after reaching a certain intensity level.² The pain is typically well localized to the entire affected compartment or nerve distributions. Approximately 80%-95% of patients present with bilateral symptoms.8

Anterior and lateral compartments present with pain over the anterior lateral aspect of the leg and may radiate to the ankle or foot dorsally. The anterior compartment is involved in 40-60% of patients and the lateral in 12-35% of patients. Deep posterior pain and tibialis posterior pain is located at the posteromedal border of the tibia and may radiate to the medial aspect of the foot. The deep posterior compartment is involved in 32-60% of patients. A minority of patients will report pain superficially and have a 2-20% involvement.^{8,10} Other symptoms the patient may relate are weakness in the affected extremity during activity that is usually described as a loss of control of the effected extremity. The patient may note bumps or herniations over the affected compartment. The patient usually denies any temperature changes or color changes of the affected extremity.

Unfortunately, physical examination of the resting patient is normally asymptomatic. A possible fascial hernia may be evident and typically occurs in 40-60% of patients, but can also occur in patients without CECS.⁸ Having the patient exercise prior to the examination is ideal for proper diagnosis. Involved compartments on physical examination may appear tense and palpation and

passive stretch may elicit pain. Neurological examination may reveal weakness and parasthesias to the distal innervations of deep and superficial peroneal nerves as well as the tibialis posterior. If the lateral compartment is affected, the patient may exhibit weakness upon inversion with loss of sensation on the anterior-lateral part of the leg and the dorsum of the foot. The anterior compartment will show weakness on dorsiflexion and loss of sensation in the first webspace. If the deep posterior compartment is affected, the patient may exhibit weakness in foot muscles and the medial ach.15 Patient should exhibit normal pedal pulses and should typically rule out any pain due to vascular abnormalities. Again if the physician has any inclination that the patient is exhibiting symptoms due to CECS, the physical examination must include the patent having exercised prior to the examination.

DIFFERENTIAL DIAGNOSIS

Patients with exercise induced lower leg pain, the differential diagnosis includes: CECS, tibial or fibular stress fracture, medial tibial stress syndrome (MTSS), fascial defects, nerve entrapment syndromes, vascular claudicating, and lower back problems (Table 1).⁸ In a study by Clanton et al, of 150 patients with exercise induced lower leg pain, they found that 33% had CECS, 25% had stress fractures, 14% had muscle strains, 13% had MTSS, and 10% had entrapments. In the same study looking only at anterior induced pain found in 98 patients that 42% had periostitis, 27% CECS, 13% with superficial peroneal compression. 5% with fascial defects and no CECS, and 13% with miscellaneous diagnoses.⁹

DIAGNOSTIC STUDIES

In terms of radiographic diagnostic studies for CECS, plain film radiographs are always negative but can help rule out stress fractures and patients with MTSS. Evaluation of CECS has also been studied in terms of bone scanning. Like plain film radiographs, bone scans can provide diagnosis in terms of stress fractures and MTSS but not for CECS conclusively. However, a study by Takebayashi et al, using thallium-201 SPECT bone scan for CECS, found that the imaging can provide precise localization of an ischemic compartment.¹¹ As stated in their study accurate localization of CECS by clinical examination is limited to only 35% of cases.¹¹ However, another study by Oturai examining the thallium-201 SPECT bone scan for CECS found, contrary to Takebayashi's study, that a 50% sensitivity and 63% specificity showed that the SPECT bone scan was not useful for diagnosing CECS.12

Evaluation of CECS by magnetic resonance imaging (MRI) has become a more promising and realistic method of diagnosing CECS. Verliesdonk et al found that there was a significant increase in T2-weighted signal intensity in the anterior compartment of patients with proposed CECS and was found to be decreased after fasciotomy.13 MRI may prove to be a useful and noninvasive method for diagnosis of CECS, but will require further testing and long-term results. Another noninvasive method that has been proposed for diagnosis of CECS is near infrared spectroscopy (NIRS). Previous studies have shown that patients with reports of CECS show greater deoxygenation than those without.14 NIRS has shown that it is possible to measure hemoglobin saturation of deep tissues in a noninvasive manor. Oxygen saturation reflects in most part the venous saturation and then in parts the level of local ischemia. In a study examining NIRS for CECS found that this modality can confidently distinguish healthy from diseased legs and supported NIRS as a noninvasive, painless alternative in diagnosis of CECS.¹⁵

Resting and post exercise measurements of intracompartmental pressures have shown to be the best method of diagnosing CECS.² These tests are normally done at a larger, more well equipped outpatient facility and will require the physician to send a suspected CECS patient to this facility. A variety of methods for measuring pressures have been described including wick catheter and constant infusion catheters. There is no agreement on whether or not any method for obtaining pressures is better than another one.³ To further complicate matters, measuring pressures does have a learning curve and is difficult to obtain. Moreover, obtaining intracompartmental pressures is often impractical but again has been shown to be the best method for diagnosis with both pre and post exercise.

Nevertheless, Pedowitz et al, have developed a pressure criteria that, according to the authors, is convenient and easy to conduct. The diagnosis is based on having 1 of 3 of the following: A pre-exercise pressure of ≥15 mm Hg, and/or a 1 minute post-exercise pressure of ≥30 mm Hg, or a 5 minute post-exercise pressure of ≥20 mm Hg.¹⁰ Although a diagnosis can be made if one of these criteria are met, the more criteria met the more confidence in the proper diagnosis of CECS. Position of the ankle and knee can also affect pressures and it is recommended that pressures be taken with the knee at 10-30 degrees of flexion and the foot at 20 degrees of flexion. One study compared the diagnostic value of intracompartmental pressures, MRI, and NIRS for diagnosis of CECS and found that the sensitivity of NIRS was clinically equivalent to that of intracompartmental pressures of 85%. This study also provided an estimate of MRI sensitivity of 77%.16

Table 1

DIFFERENTIAL DIAGNOSIS FOR PATIENTS WITH CHRONIC EXERTIONAL COMPARTMENT SYNDROME

DIAGNOSIS	SYMPTOMS	PHYSICAL EXAMINATION	DIAGNOSTIC STUDIES
Chronic Exertional Compartment Syndrome	Pain starts within first 30 minutes of exercise and can radiate to ankle/foot. Pain ceases when activity is stopped. Bilateral in 80-95%. Daily activities usually not provocative.	Typically benign. Fascial herniations in 40-60%. Reproduction of symptomswith exercise.	Elevated intracompartment tissue pressures. MRI may show increase T2-weighted signal intensity.
Medial Tibial Stress Syndrome	Pain along posteromedial aspect of mid and distal tibia. Usually exacerbated by activity and only partially relieved by rest. Daily activities may exacerbate pain.	Point tenderness at rest over posteromedial tibial periosteum and soleus fascia.	Bone scintigraphy may reveal linear uptake along posteromedial tibia. Compartment pressures to rule out deep posterior compartment syndrome.
Stress Fracture	Localized area of pain over tibia or fibula initially relieved by rest. Usually occurs after change in training routine. Daily activities may exacerbate pain.	Point tenderness over tibia or fibula. Often exacerbated with percussion.	Plain films usually negative initially. "Dreaded black line," cortical thickening or callous can be seen after 2-4 weeks. Bone scintigraphy can show localized uptake within one week. MRI may aid in diagnosis.
Fascial Defects	Often asymptomatic. Can be painful if associated with superficial peroneal nerve compression or muscle ischemia at defect. Often associated with CECS.	Defect can be visible in thin patients. Herniated muscle belly may be tender to palpation.	Intracompartmental tissue pressures should be tested if associated with symptoms of CECS. MRI may be helpful to rule out other potential etiologies.
Nerve Entrapment Syndromes	Parasthesias and burning pain along involved nerve distribution. Can be associated with weakness.	Weakness and atrophy of muscles innervated by involved nerve. Positive Tinel's sign at site of entrapment.	EMG and nerve conduction studies can localize area of entrapment.
Radiculopathy	Radiating pain at rest that follows a specific dermatone. Can be associated with weakness, parasthesias.	Localized weakness and diminished reflexes in musculature innervated by involved root. Decreased sensation. Positive long tract signs.	MRI of lumbar spine.
Vascular Claudication	Rare cause of leg pain in healthy adults. Pain with activities and leg activation. Pain relieved with leg in dependent position.	Diminished pulses and capillary refill. Loss of hair and pallor in advanced changes.	Ankle/brachial index. Arterial Doppler studies. Angiogram is the gold standard.

TREATMENT

There are few nonoperative treatments of CECS, with only cessation of activities that is certain to fully alleviate pain. This is mostly unacceptable to athletes especially patients that are competitive. The physician may suggest an alternative for the recreational athlete such as cycling as a substitute for running, to maintain fitness and may lower the risks associated with CECS.³ Another modality available is massage therapy. For patients with mild symptoms or those who will not undergo a surgical option, massage therapy can offer some relief. Blackmen et al found that patients who underwent massage treatment for a period of 5 weeks with stretching had an increase in the amount of exercise that could be tolerated but had no decrease in the amount of intracompartmental pressures pressures.¹⁷ Physical therapy is also another modality present. Passive range of motion exercises combined with a regimented physical therapist has been reported to be successful for short periods of time. However, these two modalities remain largely with regard to successful treatment.

The mainstay of treatment of patients who are unwilling to modify their exercise programs is surgical intervention in the form of subcutaneous fasciotomy of the involved compartment.² Multiple techniques have been described. Fasciotomes for the anterior and lateral compartment are performed by a single anterolateral incision. Care must be taken to avoid and decompress the superficial peroneal nerve and skin incisions can be primarily closed. Release of the posterior compartment performed through a single incision or double incision has been described.³ Care must be taken to avoid the saphenous vein and nerve.

In patients with symptoms in the superficial posterior compartment, release of the lateral head of the gastrocnemius is indicated. Endoscopically assisted fasciotomy is a documented alternative to the open method and has also had good results. The surgeon has the advantage of access to the entire length of the compartment and excellent visualization of the superficial peroneal nerve and branches.² Results of these releases have noted a high level of pain relief and satisfaction among patents.¹⁸ Most studies cite a 90% relief of pain regardless of the technique used.³ Studies have also found that patents that underwent anterior and lateral releases as compared with deep posterior compartment release faired much better in the long-term and this was mostly attributed to inadequate release of tibialis posterior muscle.

Complications ranging from 4.5-13% have been reported for surgical release. Possible complications include hemorrhage, hematoma formation, infection, nerve entrapment, swelling, vascular injury, lymphocele, and deep vein thrombosis.² Recurrence rates after fasciotomy vary from 3-12% and have been attributed to inadequate release of the involved compartment and post surgical fibrosis leading to reducing space needed for muscular expansion.³

SUMMARY

Chronic exertional compartment syndrome is an uncommonly encountered problem but presents as a difficult dilemma to both diagnose and manage. There needs to be proper understanding of the compartments of the foot and lower leg. Patients that present with symptoms of CECS need an accurate diagnosis that begins with a proper history and physical that is backed with both post- and pre-exercise intracompartmental pressures. In patients with recurrent CECS, fasciotomy is the treatment of choice to allow for full return to activities. Close attention should be paid to insure a full release of the fascial defects and to avoid neurovascular damage.







Figure 2.



Figure 3.

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