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Chronic exertional compartment syndrome of the leg

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Abstract Chronic exertional compartment syndrome (CECS) is an underdiagnosed cause of chronic exertional leg pain. The syndrome most commonly occurs in young adult recreational runners, elite athletes, and military recruits. CECS is caused by increased intracompartmental pressure within a fascial space; however, the mechanism of why pain occurs is unknown. Symptoms are classically pain in the affected compartment at the same time, distance, or intensity of exercise. CECS is a clinical diagnosis; however, it is confirmed by intracompartmental pressure testing. Fasciotomy is the treatment of choice for athletes who would like to maintain the same level of activity. Athletes who have a release of the anterior and lateral compartments have a high success rate.

Keywords Compartment syndrome · Lower extremity · Fasciotomy · Chronic leg pain

Introduction

Chronic exertional compartment syndrome (CECS) is commonly overlooked as a cause of muscle pain. Typically, there is a 22-month delay in the diagnosis of the condition [13]. Studies of the etiology of chronic anterior leg pain indicate that CECS is the causative factor in 27% of cases [3, 4]. The delay in diagnosis combined with the relative frequency of occurrence underscores the importance that primary care providers should consider CECS as a possible diagnosis that may affect an individual's participation in sports or physical activity.

The earliest account of CECS is documented by Edward Wilson in 1912 who described his symptoms during an Antarctic expedition [3, 25]. French and Price were the first to correlate symptoms with documented increased intracompartment pressures in the British Medical Journal [2]. Mavor recorded the first surgical treatment of CECS in the Journal of Bone and Joint Surgery in 1956 when he described bilateral leg surgery on a soccer player who suffered leg pain from chronic exertion [22, 25, 31].

The pathophysiology of CECS is related to a marked increase in tissue pressure within the confinement of a closed fascial space during exercise. Muscle volume can increase up to 20% of its resting size during exercise. Increased muscle volume causes an increase in the internal pressure within the fascial compartment [7, 28]. There are several theories as to why pain occurs as a result of increased muscle volume and intra-compartmental pressure. The prevailing theory is that during progressive muscle activity rising intracompartmental pressures cause impaired muscle tissue perfusion [14, 30]. Deoxygenation of muscle results in increased cell permeability, causing a shift of fluid into the interstitial space. Compromise of the microcirculation leads to ischemia and ultimately results in pain. There have been no studies to show a causal relationship between increased intracompartmental pressures and ischemia. Additionally, there have been several theories refuting the ischemia theory. Using SPECT scanning Trease et al. [4, 30] demonstrated that there was no significant difference in the relative perfusion in patients diagnosed with CECS and those in the control group. This study led to theorization that pain is not from a lack of perfusion in the affected compartments, but rather from a disproportionate oxygen supply versus demand [27, 29].

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Amendola, et al. [1] made the conclusion that pain was not related to ischemia by proving that there are no consistent ischemic changes in patients with CECS after exercise, using nuclear medicine blood flow imaging. Other theories attribute pain to stimulation of sensory or intramuscular pressure fibers in the fascia from excessive stretch [3, 4, 17, 30]. There is a paucity of studies researching the cause of CECS, secondary to underdiagnosis.

Incidence

The incidence in the general population is unclear because of the difficulty in diagnosis and the delay in seeking medical attention. CECS should be suspected in any athlete who presents with chronic anterior leg pain which worsens with prolonged use and resolves upon cessation of activity. 95% of cases of CECS occur in the anterior and lateral compartments of the leg [5, 8, 14, 32]. In select groups of athletes, other potential locations of CECS include the superficial and deep posterior compartments of the lower leg, as well as, the compartments of the thigh and forearm [4, 12]. CECS occurs most commonly in young adult recreational and elite runners, military recruits, and athletes who participate in ball and puck sports [2, 8, 25]. There have been no demonstrated differences in the incidence in men and women [27, 28]. The median age of occurrence is 20 years old [28].

Risk factors for development of CECS include anabolic steroid and creatine use, which increases muscle volume [5]. Eccentric exercise also increases the risk due to decreased fascial compliance [5]. Aberrant biomechanics in a runner such as a rear foot landing or overpronation can increase the risk of compartment syndrome secondary to differential weight loading and pressure on individual muscle groups in the lower leg [2].

Anatomy

A thorough understanding of lower extremity anatomy is imperative to diagnosing CECS and determining the compartments involved. The lower leg is divided into four compartments: anterior, lateral, superficial posterior, and deep posterior [13, 25, 26] (Fig. 1). The tibialis posterior muscle is contained within its own fascia, and is therefore referred to as the fifth compartment of the leg [2–5, 26].

The anterior compartment contains the deep peroneal nerve, tibialis anterior muscle, and long toe extensors. Increased pressure in this compartment can cause signs of loss of sensation to the first web space and weakness with dorsiflexion of the toes and ankle.

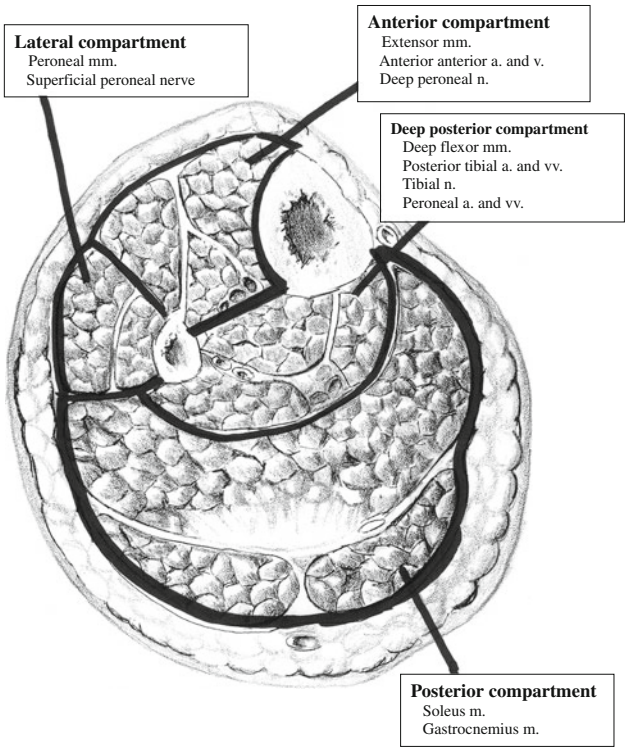

Fig. 1 Compartments of lower leg

The lateral compartment is comprised of the peroneus longus and brevis muscles, as well as the superficial peroneal nerve. Signs of compression of this compartment include weakness of foot eversion and loss of sensation to the dorsum of the foot.

Contained within the superficial posterior compartment are the gastrocnemius and soleus muscles and the distal segment of the sural nerve. Symptoms of compression would manifest as numbness of the lateral foot and distal calf.

The deep posterior compartment contains the tibialis posterior muscle, long toe flexors, the peroneal artery, and tibial nerve. Increased pressure would be demonstrated by weakness of plantar flexion and numbness on the plantar aspect of the foot.

Symptoms and signs

Obtaining the history of compartment syndrome is very important because the physical exam is often unrevealing. A classic history is development of pain in a compartment of the leg at the same time, distance, or intensity of exercise [8, 28]. The pain increases in intensity as the patient continues exercising. An athlete will discontinue the activity when the pain, muscle tightness, or muscle dysfunction becomes unbearable. Pain and tightness resolve after a rest period. Patients typically describe pain as

burning, aching, or pressure. A patient may also complain of being unable to achieve the same level of exercise the day after symptoms occur. Symptoms occur bilaterally 70–80% of the time [7, 25, 29]. Other symptoms include numbness and tingling in the dermatomal distribution of the nerve running through the compartment or weakness of the affected muscle. A classic presentation of compartment syndrome would be a runner who experiences burning in the leg and numbness on the dorsum of the foot 15 min into a run. The burning and numbness resolve within 30 min of rest.

The physical exam can be used to differentiate CECS from other causes of chronic exertional leg pain [17]. An athlete should be examined after completing the exercise that reproduces symptoms. Physical exam finding of CECS are pain on palpation of the muscles involved, pain with passive stretching of the muscle, and firmness of the involved compartments. Muscle herniation through defects in fascia can be palpated in 40–60% of patients with the syndrome [8, 10, 26]. The herniated muscle is normally palpated over the anterior tibia. Fascial defects often occur at the sight of the peroneal nerve's exit from the lateral compartment. Physical exam may also include an assessment of gait or gait analysis because pronation is a common physical finding [13]. Because the syndrome primarily affects the venous system, arterial pulses are normally intact. The neurological exam may reveal weakness or numbness of the affected muscle. Physical findings that suggest the compartment involved are

1. Weakness of dorsiflexion–anterior compartment
2. Weakness of eversion–lateral compartment
3. Weakness on plantar flexion–posterior compartment

Differential diagnosis

The differential diagnoses of chronic exertional leg pain include Medial Tibial Stress Syndrome (MTSS), stress fracture, Fascial Defects, Nerve Entrapment Syndromes, Popiteal Artery Entrapment Syndrome, and claudication.

MTSS also known as shin splints is a periostitis of the postero-medial border of the tibia. A patient will complain of pain exacerbated by activity and partially relieved with rest. On physical exam the athlete will have diffuse tenderness of the postero-medial border of the tibia [12].

Stress fracture presents as pain over the tibia and fibula with daily activity and relieved by rest. On physical exam a patient will have point tenderness over the tibia or fibula, exacerbated by percussion [4].

Fascial defects are asymptomatic. Defects occur most commonly over the anterior and lateral compartment. However, symptoms can result from muscle herniation through fascia resulting in nerve compression. Patients typically complain of pain radiating to the dorsum of the foot. Physical exam may reveal muscle bulging.

Peroneal nerve entrapment can be diagnosed clinically by applying pressure over the point the superficial peroneal nerve exits the deep fascia. Patient will have lateral leg pain elicited by resistance to dorsiflexion and eversion of the ankle and a positive Tinel's sign [7].

Popliteal artery entrapment syndrome (PAES) is a rare condition in which the artery is compressed in the compartment following chronic exercise. The patient will report exertional calf pain. The physical exam may reveal diminished dorsalis pedis pulse with passive plantar flexion or active dorsiflexion [12]. Patients with a positive exam should undergo Doppler imaging to confirm the diagnosis.

Claudication is a result of poor arterial supply. Patients will have pain in the leg with a certain amount of activity and with leg elevation. Pain is relieved by rest and dependent leg positioning. Physical exam may reveal decreased arterial pulses and capillary refill.

Diagnosis

Because definitive testing for CECS is invasive, evaluating patients for other causes of chronic exertional leg pain is important. However, if the history and symptoms are convincing for CECS, testing for CECS should be pursued. Initial testing in the workup of chronic exertional leg pain includes a bone scan and EMG. A bone scan can help differentiate stress fracture from MTSS. A stress fracture on bone scan shows intense uptake in the affected area of the bone. A bone scan revealing diffuse uptake of the radioactive isotope is diagnostic of MTSS [1, 7, 10]. An EMG can assess for peroneal nerve entrapment, a common cause of anterior exertional leg pain. PAES can be evaluated by Doppler ultrasound at rest, with passive plantar flexion, and with active dorsiflexion. It is important to recognize that a patient can have a concurrent cause of chronic anterior leg pain, such as shin splints. If a patient fails to respond to adequate treatment for a diagnosed cause of chronic exertional leg pain, testing for CECS should follow.

The gold standard of diagnosing CECS is the measurement of intracompartmental pressures. The Strkyer catheter is the device most widely used for intracompartmental pressure testing. The Strkyer catheter is a hand-held needle device that includes a pressure scale. The needle is placed into one of the four compartments. Normal saline is injected into the compartment and the pressure of the compartment is taken. After entering the compartment one can verify proper placement of the needle by externally compressing the compartment being measured and observing a pressure increase on the device. Compartment

pressures are taken prior to exercise and 5 min after the patient has exercised. A patient should perform the offending exercise until severe symptoms occur. They are then rested for 5 min and compartment pressures are taken. A resting pressure $\geq$15 mmHg and 5 min post-exercise pressures greater $\geq$20 mmHg are diagnostic of compartment syndrome [7, 10, 17, 21, 26, 29]. Joint position of the ankle and knee should be standardized during the test because it can affect intracompartment pressures.

There are evolving tests that may be used in the future to assess CECS such as MRI, infrared spectroscopy, and Thallium 201. MRI has been shown to be more sensitive post-exercise [6]. An MRI suggestive of CECS will demonstrate an increase in T2-weighted signal intensity within the involved muscle. Increased T2 signal has correlated well with increased intracompartment pressures [5, 24, 28]. A decrease in T1-weighted imaging may be seen if the patient has fibrosis and muscle atrophy [5]. MRI suggestive of CECS may also demonstrate muscular edema, muscular swelling, fascial thickening, and fatty infiltration of the muscle [6]. Fatty infiltration is associated with chronic ischemia. MRI is sensitive at diagnosing CECS, but is not specific [6].

Infrared spectroscopy is another promising test. This test measures oxygenated and deoxygenated blood in the muscles. Measurements are taken pre and post-exercise. Results that indicate CECS are delayed return to the level of oxygen at baseline when the muscle is measure post-exercise and an increased ratio of deoxygenated to oxygenated muscle post-exercise [5]. Infrared spectroscopy is very sensitive [5].

Thallium 201 with SPECT scan measures reversible ischemia. Images are taken prior to exercise. The patient exercises to 85% of their target heart rate or to the level that causes symptoms. Thallium is injected and serial SPECT images are taken. Pre and post-images are compared looking for reversible ischemia suggestive of CECS. Hayes et al. [15] conducted a study showing reversible ischemia in three patients with CECS. However, Trease conducted a blinded control study, which compared patients with and without a diagnosis of CECS. Trease found no difference in SPECT scan readings in patient's with and without increased intracompartmental pressures, which caused him to theorize that CECS is not caused by ischemia and SPECT scanning had no role in the diagnosis of CECS [30].

Treatment

The only definitive treatment of CECS is fasciotomy [20]. However, there are also conservative methods, which have been used to treat CECS. All other methods of therapy are based on expert opinion or reports in case studies. No randomized control trials have been conducted to evaluate the effectiveness of conservative treatment. As with most conservative treatment methods, these should be started early in the disease course. Because there is an average 2 year delay in the diagnosis of CECS, conservative methods are often implemented late with low success rates. The conservative treatment methods, which have been noted in case studies to have the most success, consist of discontinuing the activity that elicits symptoms or decreasing the intensity of training [7, 8, 21]. For example, a runner can become a cyclist because cycling has a lower risk of compartment syndrome. Athletes can also be instructed to go into a resting period. After the resting period the athlete can slowly increase exercise. While the athlete is increasing exercise they should ice daily and use NSAIDs [13, 26, 27]. Arch support orthotics should be designed to correct pronation during running for patients who have *pes plano* valgus [8, 12]. Other documented conservative treatment methods include avoidance of running on hard surfaces, changing footwear, and changing the biomechanical techniques of running [11, 12, 17]. Deep tissue massage, ultrasound, and stretching before exercise are modalities, which may prolong the time before symptom onset [8, 26, 31]. An osteopathic physician can treat patients by using manipulation techniques of myofascial release and strain–counterstrain [5].

If athletes are not obtaining relief from conservative measures or desire to continue competition at the same level, a compartment fasciotomy is the definitive treatment. Only involved compartments are released. Anterior and lateral fasciotomies have the best outcome with a greater than 80% success rate [10, 11, 17]. Deep posterior fasciotomies have a lower success rate of 50% [5, 10, 23]. Decreased success in the release of the deep posterior compartment has been attributed to more complex anatomy, poor visualization, and inaccessible small muscular subdivisions [19]. Release of the deep posterior compartment may be used to improve symptoms.

Different types of myofascial release have been described in the literature. There is open fasciotomy and subcutaneous fasciotomy. The advantage of open fasciotomy is full visualization of the compartment [20, 22] (Fig. 2). Some open fasciotomies include excision of strips of fascia to decrease scarring and reoccurrence. Subcutaneous fasciotomy involves one to two small incisions [3, 7]. There have been several case studies involving endoscopically assisted subcutaneous fasciotomy [18, 20, 21, 23, 31]. There are reports of increased complication rate and symptom reoccurrence with the subcutaneous method [21, 23, 27]. Unsuccessful subcutaneous fasciotomy is attributed to incomplete fascial release, nerve injury, and unrecognized fascial defect or muscle herniation [21].

Post-operatively a compressive dressing is applied for 2 to 3 days. Patients are encouraged to do range of motion

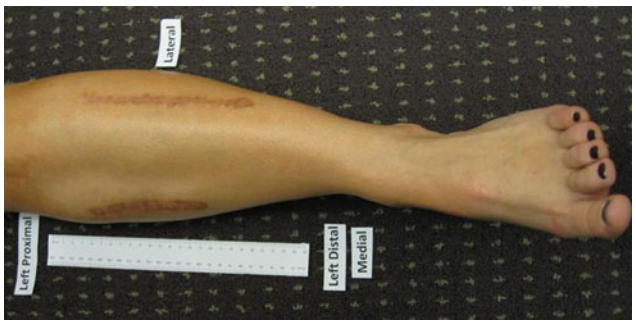

Fig. 2 Picture of the left leg following open fasciotomy

exercise and weight-bearing exercises immediately after surgery to prevent adhesions and scaring [22, 23, 31, 32]. Cycling and swimming can be started as soon as incisions are healed [8, 26]. Physical therapy can be started 1 to 2 weeks after surgery. The athlete can return to full activity in 6–8 weeks if they are symptom free and have recovered full strength and flexibility [10, 13, 17, 26].

Complications

The complication rate of fasciotomy is 11–13% [16, 26]. Complications include hemorrhage, infection, nerve damage, DVT, vascular injury, skin breakdown, altered sensation over the fasciotomy site, lymphocele, and nerve entrapment [9, 16, 27]. A case of complex regional pain syndrome was reported as a result of partial injury to a nerve [16].

An athlete can have reoccurrence of compartment syndrome after having a fasciotomy. The rate of reoccurrence is 6–11% [12]. Reoccurrence can result from incomplete release of the fascia, poor rehabilitation, or overactive scarring [7, 28, 31]. Patient non-adherence to instructions can also be a cause of failed fasciotomy. Reoccurrence rates in patients who have had open fasciotomy (2%) have been reported to be lower than those who had closed fasciotomy (11%) [2, 3, 12]. Most patients, who fail treatment initially, have resolution of symptoms with a second more extensive fasciotomy [2].

Patients can develop acute compartment syndrome on top of CECS [2, 5, 14]. Athletes should be educated on symptoms of acute compartment syndrome so that they seek immediate treatment [14]. Leg ischemia greater than 12 h causes irreversible damage to nerves and muscles.

Summary

The underlying cause of CECS continues to be the source of debate. The initial theory of the cause of pain from CECS was ischemia. However, modalities such as SPECT scanning have suggested that ischemia may not be the underlying cause. The evolution of alternative diagnostic options may provide more insight into the pathophysiology of the syndrome. As insights are gained into the etiology of compartment syndrome pain, a greater diversity of treatment options for CECS will be available. Conservative treatment methods may be effective early in the course of the condition; however, there is often a delay in both seeking treatment and making a diagnosis. The only definitive treatment currently is fasciotomy. Improved awareness of CECS by both providers and athletes may result in earlier intervention and a higher success rate of conservative therapy.

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