Compartment Syndrome in the Foot and Leg

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KEYWORDS

• Exertional-related leg pain • Compartment syndrome
• Chronic exertional compartment syndrome • Sports injuries • Athletes

KEY POINTS

- Athletes with chronic exertional compartment syndrome require a comprehensive approach for proper diagnosis and treatment.
- Although chronic exertional compartment syndrome predominantly affects the lower leg, its affliction on the medial, central, and lateral compartments of the foot has also been reported.
- Intracompartmental muscle pressure readings are considered the gold standard for diagnosis, but newer noninvasive techniques are gaining popularity.
- Nonsurgical management involves anti-inflammatory medications, physical therapy modalities, and gait retraining.
- Surgical management can be achieved through an open, mini-open, or endoscopic approach to release the pathologic osteofascial compartment.

INTRODUCTION

Chronic exertional compartment syndrome (CECS) is a condition associated with strenuous exercise that can lead to debilitating pain only relieved by cessation of the causative activity.[1] The first published report of CECS was described by Mavor in 1956,[2] in which he reported bilateral anterior leg pain in a 24-year-old professional soccer player during exercise. The patient was treated successfully with a fasciotomy and fascia lata graft.[2]

The incidence of exercise-induced leg pain ranges from 14% to 27% in the literature.[3] Of these, 70% occur in the anterior leg compartment in endurance runners.[4,5] Literature regarding the relationship between CECS and gender is inconsistent. Although early reports suggested a higher male prevalence,[6,7] two more recent cohort

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studies have proposed a female predominance.[8,9] Because those cohort studies included military and/or athlete populations, the former was most likely due to selection bias. However, a more recent heterogeneous study found an increased incidence in males.[10] As such, the current knowledge of the incidence and risk factors of CECS is largely based on small or biased cohorts, such as elite athletes or servicemen.[9,11,12]

In an effort to quantify the incidence of CECS, a large retrospective cohort study of 8.3 million service members was studied by Waterman and colleagues[9] A total of 4100 diagnosed cases of CECS were identified, which correlated to an incidence rate of 0.49 cases per 1000 person-years. Of this cohort, increased risk was seen among those with increasing age, female sex, white race, junior enlisted rank, and those within the Army branch.[9] However, de Bruijn and colleagues[10] conducted a more recent retrospective cohort study that included a more diverse patient population in terms of age and activity levels. The authors compared patients evaluated for CECS and divided their cohort into CECS diagnoses based on elevated intracompartment pressures (n = 1411) and those with normal physical examination findings (n = 153). Overall, a younger age (median age of 25 years old), male gender, bilateral symptoms, and participation in endurance running or skating were all significant predictors of CECS.[10] More studies with larger patient populations and heterogenous data are required to accurately create a predictive model for CECS.

Although the incidence of lower leg CECS is widely reported, literature on its affliction in the foot is scarce. A little scientific literature exists on incidence and predominance in relation to age, sex, and sport. Of the case reports published, the majority were male, ranging from 16 to 40 years of age, and participated in an endurance sport, such as running.[13–20] The medial compartment comprises the majority of cases, followed by the central and lateral compartments.[13–21]

ANATOMY

The leg consists of four compartments that are divided by relatively inelastic crural fascia: anterior, lateral, superficial posterior, and deep posterior. The muscles of the anterior compartment include the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. The deep peroneal nerve pierces the intermuscular septum at the proximal fibula and travels within the anterior compartment of the leg. The lateral compartment includes the peroneus longus and brevis, which are accompanied by the superficial peroneal nerve and peroneal artery. The superficial peroneal nerve pierces the crural fascia on average 12.3 cm proximal to the ankle as it traverses distally, terminating in medial and intermediate dorsal cutaneous nerves 4.4 cm proximal to the ankle.[22] The posterior compartment is subdivided by the posterior intermuscular septum into superficial and deep compartments. The superficial compartment contains the gastrocnemius, soleus, and plantaris, while the deep compartment contains the popliteus, tibialis posterior, flexor hallucis longus, and flexor digitorum longus. The tibial nerve arises from the popliteal fossa and traverses distally where it gives off branches to innervate the superficial and deep compartment muscles. Some authors have postulated a “fifth” compartment of the posterior leg comprising solely the tibialis posterior muscle within its own osteofascial plane.[23] However, follow-up cadaveric studies were unable to corroborate such findings but suggest that aberrant anatomy, such as flexor digitorum longus hypertrophy can effectively compartmentalize the tibialis posterior tendon.[24] These findings corroborate the relatively high recurrence rates of CECS following posterior compartment fasciotomies. Therefore, a two-incision approach is often warranted.[23,24] Overall, approximately 60–75% of patients with CECS

experience pathology in more than one compartment.[8,25,26] The anterior compartment is the most commonly affected by CECS (42.5%), followed by the lateral (35.5%), and posterior (18.9%).[8]

In total, there are nine compartments in the foot: four interossei, lateral, medial, central superficial, central deep, and calcaneal compartments. The lateral compartment consists of the abductor digiti minimi and flexor digiti minimi. The medial compartment encompasses the abductor hallucis and flexor digiti minimi brevis. In a cadaveric study focusing on the central compartment by Manoli and Weber, dyed gelatin was injected in a controlled fashion.[27] The contents of each compartment were then assessed, revealing a new compartment deep in the superficial central compartment containing only the quadratus plantae muscle. This compartment was found to communicate with the deep posterior compartment of the leg through the retinaculum posterior to the medial malleolus, thus raising the possibility of concurrent foot and leg CECS.[27,28] Infusate was monitored in a separate cadaveric study by Guyton,[29] however, challenging Manoli and Weber's[27] theory in real-time computed tomography (CT) imaging. Guyton concluded that the barrier between the flexor digitorum longus and quadratus plantae becomes incompetent at pressures less than 10 mm Hg and therefore cannot be expected to generate isolated compartment syndrome.[29] Although foot CECS is rare and limited literature is available, the majority of case reports describe pathology in the medial compartment with unilateral symptoms.[13–16,18]

PATHOPHYSIOLOGY

The etiology of CECS is much debated in the literature, but a pathologically elevated muscle compartment pressure is found as the key event in its pathogenesis. During intense exercise, muscle volume can increase by up to 20% of its resting size, which in turn causes a rise in pressure in each osteofascial compartment.[7] Contributors to this rise in pressure, however, are often multifactorial. Fascial thickening or inelasticity, microtrauma to muscle, myopathies, and vascular compromise all likely play a role in this pathologic event.[30,31]

In a case-control study published by Turnipseed in 1995, patients with CECS were found to have thicker and stiffer fascia compared with age-matched controls (0.35 mm + 0.12 mm, 109 + 65 MN/mm; vs 0.22 mm + 0.06 mm, 60.3 + 22 MN/mm).[32] Partial fascial tears can also contribute to compartment noncompliance in a similar mechanical fashion (**Fig. 1**).[32]

During intense exercise, microtrauma afflicts the muscle tissue. This, in turn, affects the biochemical and mechanical characteristics of the inelastic fascial compartment. Inflammation caused by microtrauma increases fluid flow from the capillaries to the adjacent interstitial space.[5,31] This, in turn, increases volume and pressure within the osteofascial compartment, impairing revascularization and generating pain. In multiple studies, CECS patients were shown to endure greater deoxygenation of muscles during exercise and delayed reoxygenation after activity.[31,33] This double-crush phenomenon propagates patient-reported symptoms during and immediately following activity. In a retrospective case series of 197 patients, the role of obstructed venous outflow in causing the pathology of CECS was investigated.[34] When comparing resting and stress-CT angiography, transient venous outflow obstruction was appreciated, likely caused by hypertrophied muscles in the affected compartment.[34]

With numerous conflicting studies in the literature, the distinct pathophysiology of CECS is up for debate. In a case control study by Edmundsson and colleagues,[35] biopsies from tibialis anterior muscles were obtained at the time of decompression fasciotomy and at a one-year follow-up. These authors found that patients with CECS

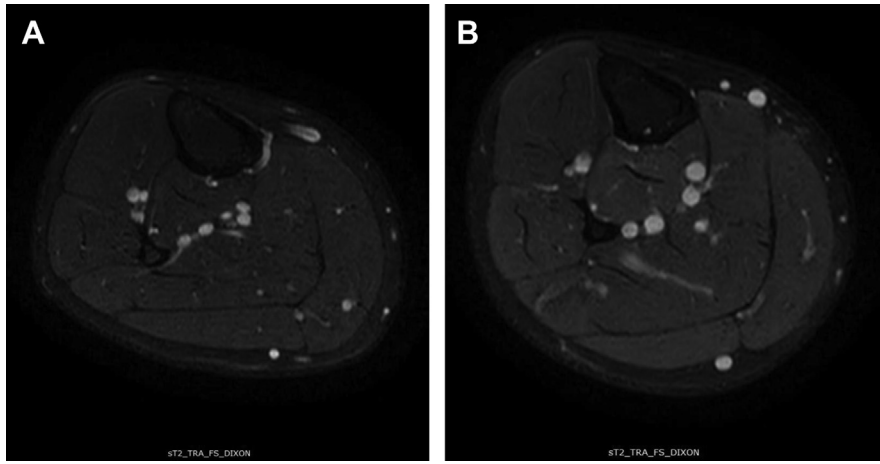

Fig. 1. Axial fat-saturated T2-weighted MRIs. (*A*) Pre-exercise imaging reveals a 1.8 cm diameter of anterior tibialis muscle belly extending 3 mm proud through an apparent fascial defect. (*B*) Post-exercise imaging has exacerbated the fascial herniation of the anterior tibialis muscle belly, which now stands 5 mm proud of fascial defect.

displayed lower capillary density (273 vs 378 capillaries/mm^2, $P = .008$), lower number of capillaries around muscle fibers (4.5 vs 5.7, $P = .004$), and a lower number of capillaries in relation to the muscle fiber area (1.1 vs 1.5, $P = .01$) compared with the control group. This study provides compelling evidence that a paucity of microcirculation is a pathogenic factor in the affected compartments of CECS.[35]

Many theories exist to explain the pain generated by the rise in intracompartmental pressures. The predominant theory is that progressive muscle hypertrophy inside a relatively inelastic osteofascial compartment comprises microcirculation, leading to ischemic pain.[36,37] Following deoxygenation of muscle tissue during exercise, there is increased cell permeability, leading to a shift of fluid in the interstitial space.[36] This results in increased intracompartmental pressures, reducing the microvascular circulation and resulting in ischemic pain. However, there are no studies to prove a causal relationship between intracompartmental pressures and ischemia. In fact, multiple studies have refuted this finding.[38,39] In a prospective blinded evaluation of 34 patients with suspected CECS, thallium-201 single-photon emission computed tomography (SPECT) was used for quantitative and qualitative assessments of perfusion between those compartments with and without CECS.[38] Overall, no compartment perfusion deficit was appreciated in those with CECS, suggesting that the pain stimulus in those patients is not related to ischemia. These nonischemic results are also concordant with the results of Amendola and colleagues[39] Utilizing MRI with methoxy isobutyl isonitrile uptake to assess perfusion to muscle in CECS patients, findings corroborated that the pathophysiology of CECS was in fact unrelated to ischemia.[39]

Other studies have proposed alternative theories for the etiology of pain in the setting of CECS.[36,40,41] The fascia itself is innervated by sensory nerves. It is proposed that in the setting of increased intracompartmental volume, sensory fibers stretch and stimulate pain.[40,41] Similar theories proposed by Humphries and colleagues[42] state that the rise in intramuscular pressure during exercise prompts a receptor-mediated

cascade for pain. Finally, metabolic by-products that result from intense activity might also act as a pain mediator in CECS.[38,42]

CLINICAL EXAMINATION

Obtaining a thorough history and physical examination is critical to making an accurate diagnosis of CECS. Because it is relatively rare and underdiagnosed, there can be as much as a 22-month delay in diagnosis of this condition.[36] Typically, patients will complain of pain throughout a particular compartment of the leg at the same time or distance of a particular triggering exercise.[36,43] This nonspecific pain will persist until the cessation of the strenuous activity. Patients will generally describe this sensation as cramping, aching, or burning that begins within 15–20 minutes of the offending activity.[44]

The most common symptoms include claudication (90%), muscle group tightness (60%), and paresthesias (25%) of the affected compartment.[32] Physical examination is vital to differentiate CECS from other common causes of exercise-induced leg pain. Specifically, the patient should be examined before and after completing an activity that reproduces his/her symptoms.[36] Palpating the affected compartment may elicit pain and/or paresthesias. A general firmness can also be appreciated in the involved musculature. Gait analysis should also be conducted, as overpronation is associated with anterior compartment CECS.[45] Runners with CECS often display increased vertical ground reaction forces, longer stride length, and reduced cadence.[46]

Lower leg CECS is predominantly a bilateral syndrome.[10,11,47] In a retrospective cohort study of 1411 patients with lower leg CECS, 74% of patients had bilateral pathology. Interestingly, these findings were compartment-dependent, where 53% of lateral CECS had bilateral syndrome compared with 72% and 78% in anterior and deep-posterior CECS, respectively.[10] Patients who experienced unilateral symptoms were more likely to have a history of previous lower leg trauma (14.4% vs .9%, $P < .01$) or vascular pathology (7.2% vs 3.6%, $P < .01$) compared with patients with bilateral symptoms.[10]

Knowledge of the anatomy of each compartment is paramount to properly diagnosing CECS (**Table 1**). If the anterior compartment is primarily affected, weakness in dorsiflexion and a drop-foot gait are often observed. Weakness in eversion and plantarflexion results in lateral and posterior compartment CECS, respectively. Pedal CECS, however, is often more difficult to deduce compartment involvement. The data available in the literature suggests that CECS of the foot does not share a similar clinical picture to that of the leg. This could be because of increased compartmental musculature impacts during the causative activity.[28] According to some reports, patients with foot CECS experience pain in the affected compartment within 10–90 minutes of strenuous exercise, which subsides after 10–20 minutes of rest.[15–17,20] However, other descriptions consist of aching or cramping pain within the affected compartment within 15 minutes of activity that persists and intensifies up to 24 hours later.[13,14,18] Physical examination findings include a tense, cyanosed, protuberant compartment. Passive range of motion of digits, especially the hallux in the medial compartment of CECS, reproduces pain.

DIAGNOSIS

The diagnosis of CECS is often difficult to deduce because there are multiple etiologies with similar presentations of exercise-related foot or leg pain. This could include medial tibial stress syndrome, stress fracture, nerve entrapment, popliteal artery entrapment syndrome, claudication, or plantar fascial tear.[1] Along with a

Table 1
Lower leg compartment anatomy, signs and symptoms, and incidence of the respective compartment

Compartments	Muscles	Neurovascular	Signs/ Symptoms	Incidences (Debrujin, 2018; Velasco, 2020; Davis, 2013)
Anterior	Tibialis anterior, extensor hallucis longus, extensor digitorum longus, fibularis tertius	Deep peroneal nerve, anterior tibial artery	Dorsiflexory weakness, drop-foot	42.5–43%
Lateral	Peroneus longus, peroneus brevis	Superficial peroneal nerve, peroneal artery	Eversion weakness	19–35.5%
Superficial Posterior	Gastrocnemius, soleus, plantaris	Tibial nerve	Plantarflexion weakness	18.9–34%
Deep Posterior	Popliteus, tibialis posterior, flexor hallucis longus, flexor digitorum longus	Tibial nerve, posterior tibial artery		

comprehensive history and physical examination, there are multiple invasive and noninvasive diagnostic tools that will assist in delineating between these similar conditions.

Currently, intracompartmental pressure measurement remains the gold standard for the diagnosis of CECS. To obtain such values, a needle or catheter is inserted into the symptomatic osteofascial compartment (Fig. 2). Either pre- or post-exercise, the patient is placed supine on an examination table where the projected area is prepped

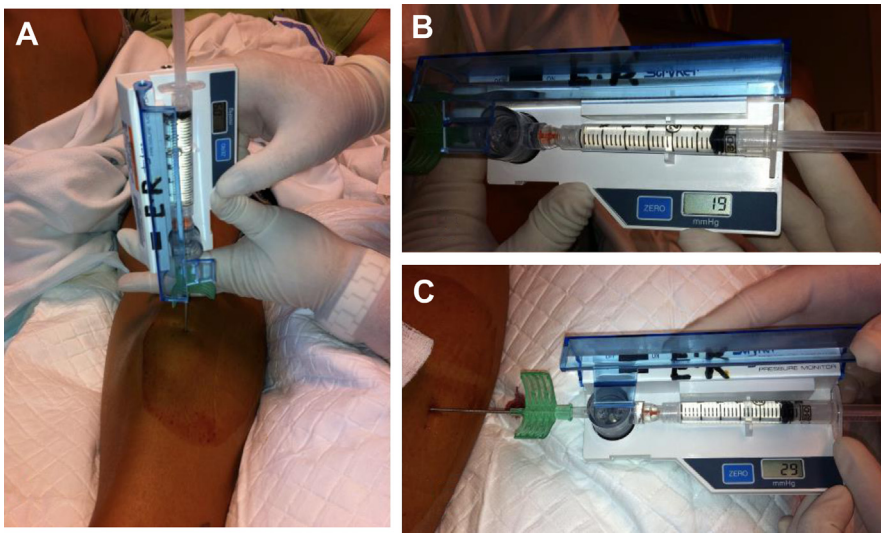

Fig. 2. (*A*) Indwelling slit catheter for diagnosis of CECS in the anterior compartment of the leg. (*B*) Pre-exercise measurement of 19 mm Hg. (*C*) 5 minutes post-exercise measurement of 29 mm Hg, confirming the diagnosis of CECS.

aseptically. Typically, 0.5–1 mL of lidocaine plain is raised as a skin weal along the portal site to anesthetize the skin.[26,48,49] One study concluded that the needle pain from intracompartmental pressure measurements had a median rating of 5 out of 10 with this technique, thus reducing the concern to avoid or minimize its utility in the diagnosis of CECS.[48]

The diagnostic criteria for CECS were first proposed in a retrospective study of 45 patients by Pedowitz and colleagues[7] based on intramuscular compartment pressures. When the history and physical examination are suggestive of CECS, one or more of the following objective criteria is diagnostic: (1) 15 mm Hg pre-exercise intracompartmental pressure measurement, (2) 30 mm Hg 1-minute post-exercise measurement, or (3) 20 mm Hg 5-minute post-exercise measurement.[7] It is important to note that this criterion was based solely on lower leg CECS and a majority of the patients in this cohort had anterior compartment pathology.

The validity of these criteria has been questioned by follow-up studies in the literature. In one study by Roberts and colleagues,[50] with the exception of pre-exercise pressures, many of the intracompartmental pressure measurements overlapped with healthy controls for the lateral and deep-posterior compartments. This suggests that the Pedowitz criteria may only be reasonable for diagnosing anterior compartment CECS. Another review in 2014 studied the validity of the Pedowitz criteria for the anterior compartment specifically.[51] Overall, the majority of measurements pre-, during, and post-exercise in healthy individuals were within the diagnostic range for CECS, again questioning its efficacy even within the anterior compartment.[51]

In 2020, a prospective cohort study of 864 patients (n = 442 with CECS; n = 422 healthy controls) with exertional-related leg pain was studied to better delineate a relationship between compartment-specific intramuscular pressure readings and CECS.[52] Overall, the median 1-minute post-exercise values in patients with CECS were 33 mm Hg in the deep-posterior compartment, 35 mm Hg in the superficial-posterior compartment, 40 mm Hg in the lateral compartment, and 47 mm Hg in the anterior compartment, compared with 12 mm Hg, 12 mm Hg, 14 mm Hg, and 18 mm Hg in the control group, respectively. In concordance with this study, there is robust evidence to support lower intracompartmental pressure measurements in the posterior and lateral osteofascial compartments compared with the anterior osteofascial compartment.[50–52] As such, a physician should consider lowering threshold values for the Pedowitz criteria when a patient presents with posterior and/or lateral compartment symptoms.

In contrast to acute compartment syndrome, there is no definite value that governs CECS. In a meta-analysis of 32 studies in 2012, the average pre-exercise values ranged from 7.4 mm Hg to 50.8 mm Hg in CECS patients compared with 5.7–12 mm Hg in controls.[53] During exercise, mean pressure readings ranged from 42 mm Hg to 150 mm Hg in CECS compared with 28–141 mm Hg in controls; 1-minute post-exercise values ranged from 34 mm Hg to 55.4 mm Hg and 9–19 mm Hg in CECS and control patients, respectively. Although it appears that post-exercise measurement is the most reliable value for the diagnosis of CECS, no statistically significant conclusion could be reached.[53] In 2018, Zimmerman and colleagues[48] conducted a descriptive analysis of 501 service members with exercise-related leg pain who had intracompartmental pressure measured 1 minute post-exercise. Of those diagnosed with CECS, 68% had an intracompartmental pressure measurement of >35 mm Hg. However, pain scores had a negligible correlation with compartment pressures.[48] This ultimately challenges the validity of intracompartmental pressure measurement testing and has paved the path for implementation of newer noninvasive diagnostic tools.

More recently, new techniques have emerged to assess intracompartmental pressure readings on a more dynamic scale with anthropometric data. A cohort study of 40 men ($n = 20$ with CECS; $n = 20$ healthy controls) underwent intracompartmental pressure readings continuously before, during, and after exercise.[54] Pressure readings were conducted with an indwelling transducer-tipped catheter wire inserted into the tibialis anterior muscle 3 cm distal and lateral to the tibial tuberosity. Overall, CECS patients had higher pressure measurements immediately after standing than controls (35.5 mm Hg vs 23.8 mm Hg, respectively; $P = 0.006$). During exercise, the greatest difference in pressure readings corresponded with the maximal tolerable pain in both groups (114 mm Hg in CECS vs 68.7 mm Hg in controls; $P < .001$). This corresponded to an overall greater specificity (95%) but decreased sensitivity (63%) compared with the Pedowitz criteria.[54] The authors argue that continuous pressure monitoring is better tolerated because only one stick is required for data collection. However, it is important to note that this technique requires greater physical demand to produce valid waveforms. This should be taken into consideration based on the athlete's skill level.

The new techniques emerging for the diagnosis of CECS have focused on noninvasive tools to enhance patient comfort and lower the risks that accompany catheter readings, such as bleeding and infection.[1,26] Although risks remain minimal, noninvasive measures, such as MRI, near-infrared spectroscopy (NIRS), methoxyisobutyl isonitrile (MIBI) perfusion imaging, thallous chloride scintigraphy, and triple-phase bone scan technology may prove diagnostic capability in certain patient populations. Because the pathophysiology of elevated compartment pressures is characterized by intracompartment swelling, an MRI may demonstrate diffuse high signal intensity on T2-weighted images. In one study, T2-weighted signal intensity increased by 27.5% following exercise in the anterior compartment compared with baseline.[55] This effect was not apparent in the control group and disappeared in the CECS group post-fasciotomy. MRI is also useful to rule out other differential diagnoses, including fascial defects (see **Fig. 1**), medial tibial stress syndrome, or stress fractures that may mimic exertional-related lower extremity pain. However, this may be difficult to obtain immediately following exercise in certain institutions, thus reducing its validity as a diagnostic tool.[39]

NIRS can measure tissue deoxygenation of skeletal muscles that occurs with increased intramuscular pressure in CECS. As earlier discussed, CECS pain is often associated with ischemia, and therefore NIRS has proven to be a sensitive tool for its diagnosis.[36,37,56] In one diagnostic cohort study, the sensitivity of NIRS was 85% for diagnosing CECS, which is greater in comparison to intracompartmental pressure measurement in some literature.[57] This same study, however, reported rather low sensitivity but high specificity of MRI for the diagnosis of CECS (>10%) T2-weighted signal intensity–sensitivity (40%); specificity (100%).[57]

Similar to NIRS, thallium chloride scintigraphy can demonstrate reversible ischemia in affected compartments during exercise through its SPECT scanning.[58] Takebayashi and colleagues[59] performed a quantitative analysis of post-exercise thallium chloride SPECT of the lower leg. The mean values of mean percentage uptake were 75% in the anterior compartment, 69% in the lateral compartment, 72% in the superficial-posterior compartment, and 68% in the deep-posterior compartment. However, there were discrepancies between clinical and SPECT diagnoses in 33% of these patients, thus questioning its accuracy as a diagnostic tool.[59]

MIBI perfusion involves intravenously injecting technetium-99 m to monitor its uptake in peripheral muscles. Muscle hypoxia demonstrates an inverse relationship with MIBI uptake. In some reports, a decreased concentration of MIBI is seen in the affected compartments of CECS.[60] Similarly, CECS may also become evident in

dynamic bone scanning. Imaging will reveal decreased radionuclide concentration in the area of increased compartment pressure, with increased soft tissue concentration at the superior and inferior poles of the pathologic compartment. Further research is required to improve the sensitivity and specificity of noninvasive diagnostic testing of CECS.[61]

TREATMENT

There are both conservative and surgical interventions for the treatment of CECS. Conservative options include activity modification and limiting activity to a level that elicits minimal symptoms, physical therapy for stretching and strengthening of the involved muscles, massage with soft tissue release, and anti-inflammatories or orthotics for cases with excessive pronation.[62] There are also other manipulation and physical therapy techniques, such as ultrasound, myofascial release, and strain-counterstrain.[63]

Surgical intervention involving fasciotomies or fasciectomies of the affected compartment should be considered if symptoms are refractory to conservative care after a few months or if patients are experiencing extreme pressure elevation.[64,65] Only involved compartments that were measured with increased intracompartmental pressures are released. Anterior and lateral fasciotomies have a greater success rate of 80%, while deep and superficial posterior fasciotomies have lower success rates of 50%.[66,67] The decreased success of the deep posterior compartment is attributed to more complex anatomy, poor visualization, and inaccessible small muscular subdivisions.[68]

There have been multiple described incision techniques, including single, double, and triple incision fasciotomies, as well as endoscopic techniques. Regardless of the technique, all fascia hernias must be addressed with the fascial incision to adequately relieve the intracompartmental pressure. The advantages of an open fasciotomy include full visualization as well as the ability to excise a strip of the fascia during the release to decrease scarring and reoccurrence.[69] Endoscopic fasciotomies or small incision fasciotomies involve 1–3 small incisions where a subcutaneous plane overlying the fascia is made between the small incisions and the fasciotomy may be made with endoscopic guidance to confirm release of the fascia.[70,71]

TECHNIQUE

Anterior and Lateral

There is a high rate of coexistence of CECS in the anterior and lateral compartment, and both compartments may be addressed from the same incisions to gain access to both compartments.

Mark out a point approximately 11–12 cm above the tip of the fibula to mark the location of the superficial peroneal nerve. The incision is based midway between the anterior aspect of the fibula and the tibia crest (**Fig. 3**). This is an optimal position to allow both access to the lateral and anterior compartments to be released. The incision is generally between 4 cm and 6 cm. The dissection toward the anterior compartment of the leg allows visualization of the superficial peroneal nerve before incising the compartments. Full thickness skin flaps are raised bluntly and the superficial peroneal nerve is identified. The superficial peroneal nerve is then released and retracted out of the way. Variations of the nerve path must be appreciated: it may exit directly through the lateral compartment fascia posterior to the septum, through both the anterior and lateral compartments by traversing a fibrous tunnel within the intermuscular septum, or immediately anterior to the intermuscular septum from the anterior compartment alone.

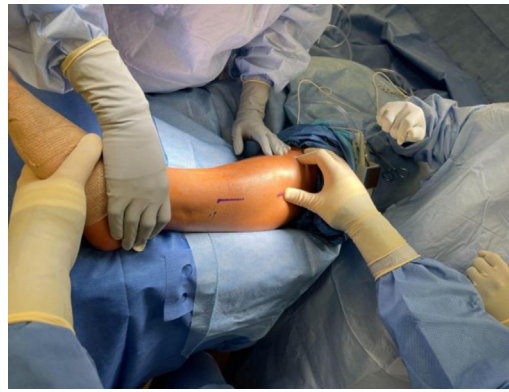

Fig. 3. Incisions for fasciotomy release of the anterior and lateral compartments are marked out. A 4–6 cm incision is made at the midline between the tibial crest and the anterior edge of the fibula 11–12 cm proximal to the distal fibula. An accessory portal is marked out at a level midway between the initial incision and Gerdy's tubercle.

The fascia is identified and a subcutaneous plane is made proximally and distally utilizing blunt instrumentation (Fig. 4). The fascia is then cut and released distally to the level of the extensor retinaculum with a curved scissor with the curve pointing anteriorly to avoid any damage to the superficial peroneal nerve.

An accessory incision is made proximally and dissected down to the level of the fascia. A subcutaneous plane is made distally to connect the two incisions as well as proximally to the level of the tibial tuberosity. The fascia is then incised from distal to proximal utilizing a metzenbaum scissors, hook blade, or a scalpel depending on surgeon's preference to the level of where the accessory incision is. The fascia is then further released from the accessory incision proximally to the level of Gerdy's tubercle. Through the same incisions retracted posteriorly, the lateral compartment may be released in the same fashion just 1 cm behind the anterior intermuscular septum right over the level of the peroneal tendon from the level of the distal fibula

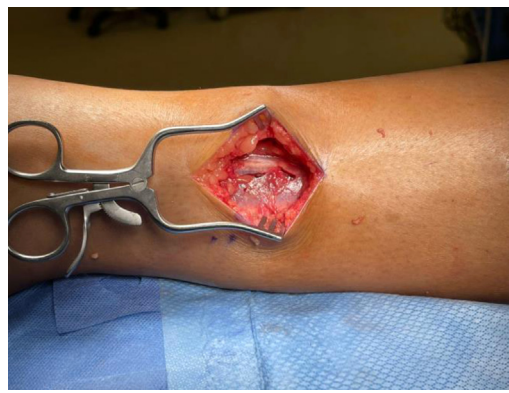

Fig. 4. Dissection is carried down to the level of the fascia where the fascia is incised and subfascial planes are created to perform the fasciotomy with care to identify and retract the superficial peroneal nerve.

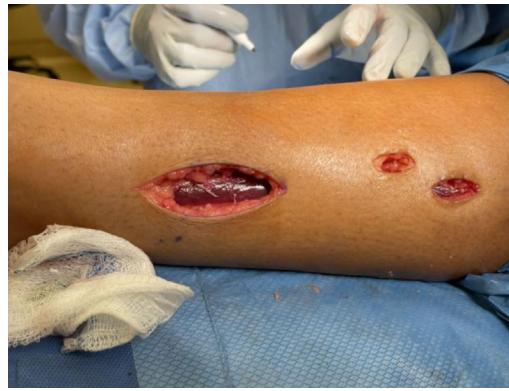

Fig. 5. Fasciotomies were completed with exposed muscle belly to confirm complete release to the anterior and lateral compartments.

to the fibular head with an additional accessory portal made more posteriorly if necessary (Fig. 5).

After release of the anterior and lateral compartments, the wounds are packed with moist gauze and the tourniquet should be released for visualization and control of any significant bleeding. This also allows for direct visualization to prevent damage of the saphenous vein and posterior tibial bundle when performing the superficial and deep posterior compartment releases.

Superficial Posterior

An incision is made at the middle one-third of the leg, 1 cm behind the posterior medial border of the tibia, approximately 6 cm in length (Fig. 6). Dissect down to the level of fascia over the gastrocnemius tendon, clearing subcutaneous tissue with blunt finger sweep dissection. The saphenous nerve and vein are visualized just medial and posterior to the medial border of the tibia overlying the deep posterior compartment of the leg and should be retracted. The fascia is then incised and released proximally to the level of the knee joint and distally to the level of the Achilles tendon, freeing up the soleus and the gastrocnemius muscles (Fig. 7).

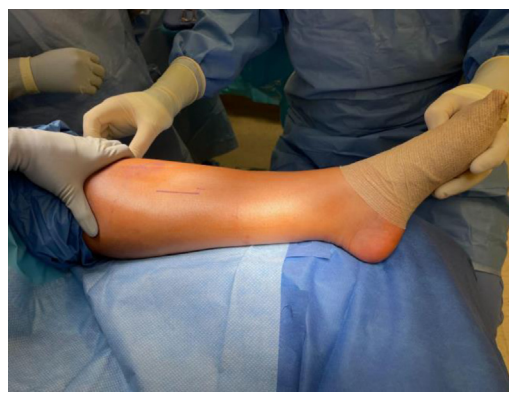

Fig. 6. The superficial and deep posterior compartments are accessed through an incision made at the middle one-third of the leg just behind the posterior medial edge of the tibia.

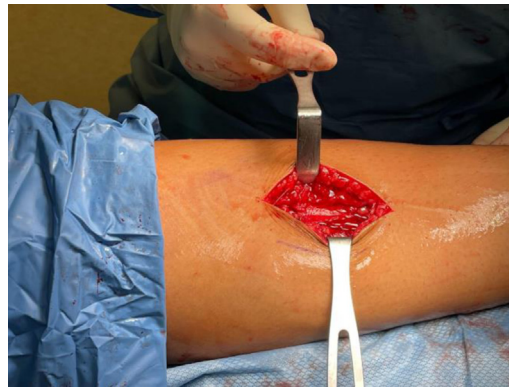

Fig. 7. Dissection is carried down to the level of the fascia overlying the gastrocnemius and soleus muscles with care to identify and retract the saphenous nerve and vein.

Deep Posterior

The deep compartment is exposed next after the superficial posterior compartment. The soleus must be released and elevated posteriorly off the posterior edge of the tibia. This exposes the deep compartment. The flexor digitorum longus can be visualized first, and the fascia can be incised and opened up. The most distal aspect can be elevated with periosteal elevators or blunt finger dissection to the level of the posterior medial malleolus. Proximally, the fascia is opened up with scissors or a periosteal elevator. When performing a deep posterior compartment release, attention must be given to adequate decompression of the tibialis posterior.[23]

Endoscopic techniques for all compartments except the deep posterior compartment are performed similarly with 2–3 stab incisions along the compartments, freeing up the subcutaneous plane utilizing blunt dissection with a periosteal elevator and releasing the fascia with a hook blade or scalpel, all with direct visualization of the fascia being released with a scope through an accessory portal.

After adequate fascial release in the affected compartments, the subcutaneous layers are closed, followed by skin closure. Simple skin incisions are closed for endoscopic procedures. A compressive dressing is applied postoperatively.

Surgical treatment of CECS has demonstrated success in returning to activity with generalized relief of symptoms. Fasciotomies of the anterior compartment of the leg have the highest success rates exceeding 85% in the literature, while deep posterior compartment fasciotomies elicit a lower success rate of approximately 70%.[72]

Postoperative course

Although an optimal post-operative rehabilitation program has not been established, there are general guidelines for returning to activity that may be monitored to cater to a patient's specific activity level. Patients may typically bear weight as tolerated and begin active and passive range of motion immediately following surgery, but crutches are often dispensed for comfort the first few days. Jazwari and colleagues[73] published a preferred protocol outlined in **Table 2**. More active walking, light jogging, and cycling are initiated when the wounds are healed at 2 weeks, and patients typically resume running training at approximately 6 weeks. Full rehabilitation and return to activities usually takes 3 months, but may be longer for patients who underwent deep posterior compartment fasciotomies.[72,74,75] Recovery and return to activity may also be assisted with physical therapy, which can be initiated 1–2 weeks after surgery.

Table 2
A postoperative rehabilitation program following compartment fasciotomies to return to sport

	Goals	Modalities	Weight-Bearing	ROM/Stretching	Exercises
Phase 1 (Day 1–14 post- operation)	Pain management, wound healing, edema control	Ice, elevation, compression	Crutches and partial weight bearing, consider CAM boot	Unrestricted non-weight bearing dorsiflexion/ plantarflexion	Unresisted ankle plantarflexion/ dorsiflexion
Phase 2 (Weeks 2–4 post-operation)	Normal ankle ROM	Ice, compression stockings	Wean crutches, progress to full weight bearing as tolerated	Begin alphabet/ankle rotation exercises, dorsiflexion towel stretches	Progression- light Theraband dorsiflexion/ plantarflexion/inversion/ eversion, pain free leg and calf press
Phase 3(Weeks 4–6 post-operation)	Improved ankle strength, normal gait pattern maintained for 1 mile	Ice, compression stockings, scar massage	Weight bearing as tolerated	Full and unrestricted	Increase theraband resistance, mini squats to real squats, single heel raise, pain-free cardio (forward and backward treadmill), elliptical, pool running
Phase 4 (Weeks 6–12 post-operation)	45 min low impact cardio, resistance weight training at 90% normal	Ice, compression, scar massage	Unrestricted	Unrestricted	Progression of weight machines, sit ups and push ups, pain free cardio treadmill, stairmaster
Phase 5 (Weeks 12–16 post-operation)	Running normal pace, return to sports	None	Unrestricted	Unrestricted	Running progression, agility drills, plyometrics

This may be adjusted given patient's ability and activity level.

Chronic exertional compartment syndrome of the foot. Unlike CECS of the leg, whose incidence and treatment is very well documented, CECS of the foot remains underdiagnosed and is only reported in the literature on an anecdotal basis. The medial compartment is most commonly affected either bilaterally or unilaterally.[13,17] The incidence of CECS of the foot remains unclear, however, as most of the compartment syndrome of the foot that is described is an acute compartment syndrome from trauma.

The clinical signs and symptoms of CECS of the foot are more ambiguous, diverse and with a lack of consistency of its counterpart in the leg. CECS of the foot has a similar presentation consisting of pain, cramping, paresthesias, or tightness on activity that is relieved by a period of rest or cessation of activity.[14] There may also be noticeable swelling on the abductor hallucis muscle as the medial compartment of the foot is most commonly affected, or a taut compartment to the affected compartment. Foot CECS may go undiagnosed because it may look like other conditions like a Lisfranc injury, tarsal tunnel syndrome, or plantar fasciitis. Just as in the leg, the most objective investigation is a dynamic intracompartmental pressure study, which can be supplemented with clinical symptoms or advanced imaging such as MRI, demonstrating relative hypertrophy to a compartment (**Fig. 8**).

When conservative care of rest, anti-inflammatories, physical therapy or orthotics fails, surgical fasciotomy is the most effective management of CECS of the foot. Jowett and colleagues[21] published a series of five patients and seven feet with confirmed CECS of the medial compartment, surgical release of the superficial and deep fascia demonstrated significant symptom relief in all but one patient. Fasciotomies over the affected compartments can be performed either with a single incision, or endoscopically with serial stab incisions along the compartment. Endoscopic fasciotomy of the most commonly affected compartment, the medial compartment consists of three stab incisions made along the abductor hallucis muscle at equal distances (**Fig. 9**). The surgery is performed proximal to distal. The soft tissues are mobilized along the line of the abductor hallucis muscle and a fascial plane is created to connect all of the stab incisions. Next, utilizing a blunt probe to identify a layer, the cannula is inserted through the stab incision placed proximal to distal. The fascia is identified and, utilizing a small blade for the endoscopic plantar fascial release system, the fascia is perforated and cut. The muscle belly will be visualized with the scope to extrude from the fascia, clearly being under significant tension (**Fig. 10**). Once the fascia was cut all the way to the central incision, the cannula was then repositioned from

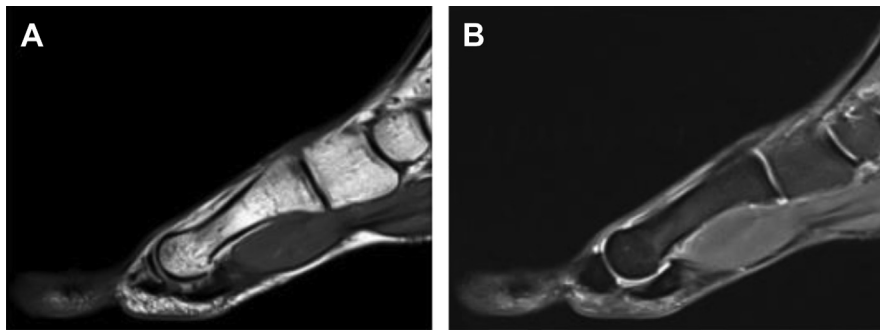

Fig. 8. Sagittal T1 (*A*) and T2 (*B*) weighted images on MRI demonstrating hypertrophy within the medial compartment to the abductor hallucis muscle belly.

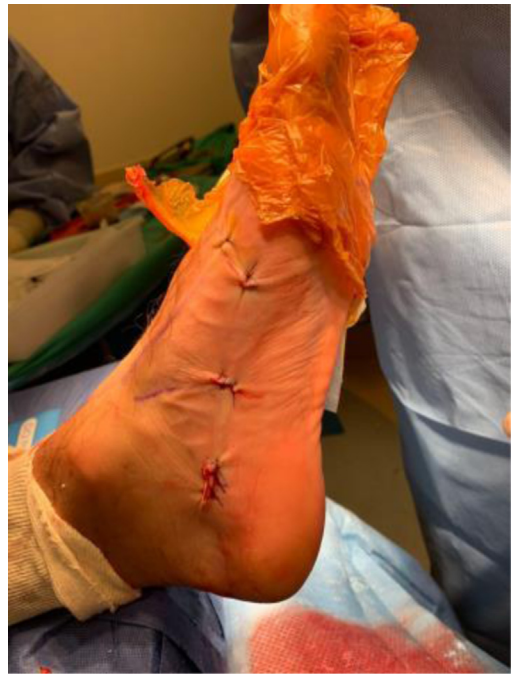

Fig. 9. Three stab incisions are made along the abductor hallucis muscle at equal distances.

the central incision to the distal incision, again being careful to be sure to be above the fascial layer and not penetrate deep. The hook blade is utilized to cut through the fascia with the muscle herniating out once the fascia is cut.

OUTCOMES/COMPLICATIONS

There have been many published studies of fasciotomies following CECS with varying results, as most studies showing the effectiveness of fasciotomy for CECS are of smaller cohort sizes. Maffuli and colleagues[76] found that in a study of 18 patients with CECS post-fasciotomy, significant improvement was seen in Short Form 36-item Health Survey and a return to preinjury or higher level of sport after minimal incision fasciotomy. Another study of 7 patients with CECS of the leg by Ballus and colleagues[77] with an ultrasound-guided fasciotomy demonstrated a decrease in pain in all patients, with all but 1 patient returning to presymptomatic exercise levels. Drexler and colleagues[78] performed a retrospective case series study of 95 legs with CECS demonstrating significant long-term improvement in postoperative activity level and quality of life after fasciotomy. Packer and colleagues[79] compared patients with CECS who were treated with conservative treatment and those who underwent operative treatment, with the operative group having higher satisfaction rates.

Common complications from fasciotomies for CECS include infection, nerve damage or entrapment, vascular injury, hemorrhage, deep vein thrombosis, chronic regional pain syndrome, or lymphocele.[80] Complication rates of fasciotomy are found to be around 11–13%.[81] In addition, a patient, particularly an active athlete, may have a recurrence of compartment syndrome after the fasciotomy, with

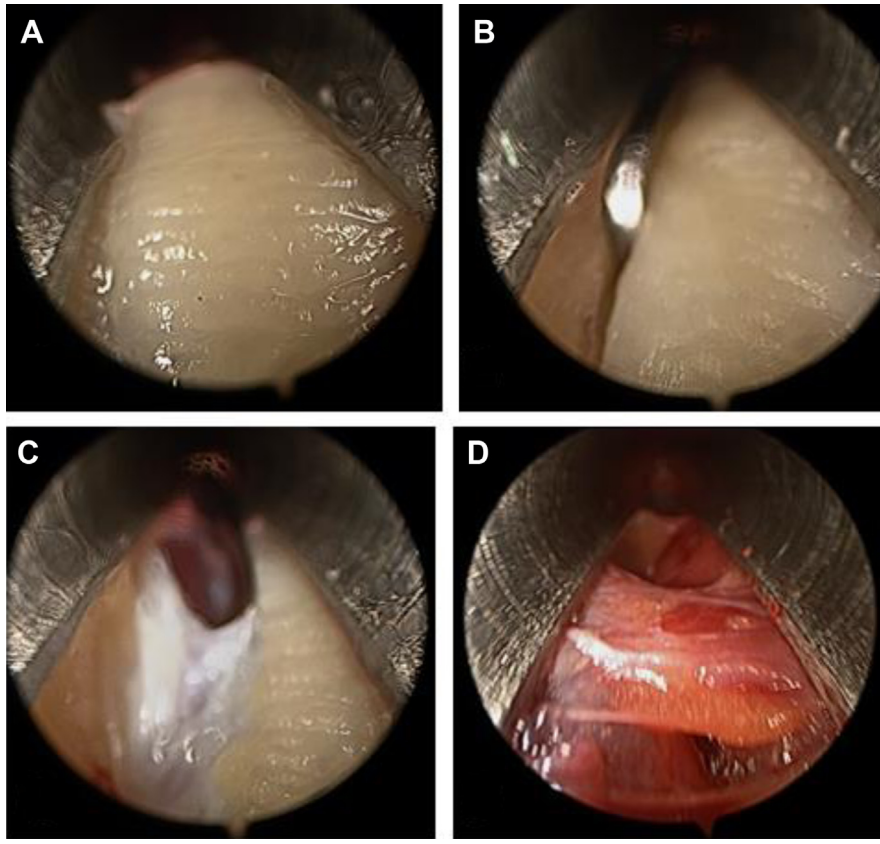

Fig. 10. (*A*) The cannula is inserted through the stab incisions along the fascial plane and the fascia is identified. (*B*) Utilizing a small hook blade from the endoscopic plantar fascial release system, the perforated. (*C*) The fascia is cut along the course of the medial compartment. (*D*) The released fascia and extruded muscle belly is identified and confirmed with the scope.

recurrence rates in the literature ranging from 6% to 11%.[82] Causes of recurrence are incomplete release of the fascia, poor rehabilitation after fasciotomy, or excessive scarring.[4,65] Although the endoscopic and smaller incision techniques have gained more favor for minimal scarring and tissue disruption as well as quicker return to activity given the smaller wound sizes, this technique has been shown to have increased complication rates, nerve damage, and symptom recurrence due to limited visualization.[64] In some instances, patients can develop an acute compartment syndrome on top of CECS.[83]

SUMMARY

CECS continues to be studied as its etiology is unclear. The evolution of alternative diagnostic options may provide more insight into the pathophysiology of the syndrome, which may provide more availability for a greater diversity of treatment options. Proper diagnosis through clinical and compartmental pressure evaluation is imperative. Conservative treatment methods may be effective early in the course of the condition, but the only definitive treatment is a surgical fasciotomy of the affected compartment. The literature demonstrates excellent success with the myriad of fasciotomy techniques that return patients and athletes back to normal, pain-free activity.

CLINICS CARE POINTS

- Chronic exertion compartment syndrome most commonly afflicts the leg, particularly the anterior compartment.
- Although chronic exertion compartment syndrome can occur with any sport, it is more common with participation in endurance running or skating.
- The most common symptoms include claudication, muscle group tightness, and paresthesias in the affected compartment.
- The gold standard for diagnosis of chronic exertion compartment syndrome is intracompartmental pressure measurement pre- and post-exercise.
- Conservative treatment for chronic exertion compartment syndrome include physical therapy, modification of activity, and NSAIDs.
- Surgical intervention involve open or endoscopic fasciotomies to release the affected compartment(s).
- Recurrence from surgical release is rare, and is most often the result of incomplete release, poor rehabilitation, and/or excessive scarring.

DISCLOSURE

The authors have nothing to disclose.

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