Exertional Compartment Syndrome

Here's an update on this often-misdiagnosed overuse condition.

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Introduction

Exertional compartment syndrome can come in two forms—acute or chronic (AECS and CECS, respectively). Chronic exertional compartment syndrome (CECS) has become an issue that has plagued many active individuals, particularly those engaged in repetitive strain (e.g., runners). The symptoms described by individuals have been consistent: pain, cramping, muscle weakness, numbness, and tingling.[9,11] However, the time required for these symptoms vary between individuals.

CECS has been classified as an overuse injury, the result of "repetitive microtrauma [resulting in] formation of scar tissue and tissue degeneration[8]" One of the first reports to discuss CECS was from 1950.[4,5] Since then, CECS has become a much more widely understood and researched area of study.

Diagnosis of CECS has developed from simply clinical to actual diagnostic testing, with the gold standard now being intra-compartment pressure testing.[7] With the continued prevalence of these ailments, it becomes increasingly important to understand what can be done to alleviate the symptoms among active individuals worldwide.

Anatomy

With CECS, understanding anatomical relationships becomes increasingly essential. The musculature is separated into compartments, with each compartment lined by a fascial layer. Each of these compartments can contain multiple muscles, vasculature, and nerve supply. While exertional compartment syndrome can occur anywhere in the body, the most common location is the lower leg.

Within the lower leg, there are four different compartments: anterior, superficial posterior, deep posterior, and lateral. To be more specific, the anterior compartment is the one most commonly affected by this ailment.[2,4,5,10] Muscles contained in this compartment are the tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus tertius). These muscles act to dorsiflex the ankle and extend the toes. Neurovasculature involved includes the deep peroneal nerve as well as the anterior tibial artery and vein. The next most common compartment affected is the deep posterior, which includes the posterior tibial neurovascular bundle, as well as the posterior tibial, flexor digitorum longus, and flexor hallucis longus.

The symptoms of CECS will be representative of which compartment is affected, so understanding the anatomy becomes fundamental to making a clinical diagnosis.

Pathophysiology

CECS results from repetitive loading/impact or exertional activities, while its counterpart, AECS, is typically a result from a severe injury (i.e., fracture, or crush injury).[1] The first known article to discuss CECE pathophysiology occurred in 1975 by Dr. Reneman.[10] Despite the plethora of studies since then, the exact pathophysiology behind CECS is still unknown. However, the most highly hypothesized cause is due to an overall increase in intrafascial pressure.

Normally with exercise, metabolic demands to tissue are increased and the fascial layer will allow stretching of the fascia to accommodate the swelling musculature. However, in the case of CECS, the fascial layer surrounding the muscles is less pliable, essentially forming a physiologic barrier. This eventually leads to a pressure increase in a limited space, creating more restriction. With repetitive loading, musculature continues to swell and it creates a positive feedback loop of pressure increase with continued exercise. The overall increased pressure of the compartments leads to compression of compartmental contents that are more pliable—nerves, arteries and veins—leading to the classical symptoms of CECS. By decreasing blood supply, the muscle can undergo ischemic changes, as the metabolic de-

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mands to the tissue are not being met.[9,10] Symptoms will continue to be experienced until the time when the pressure is alleviated and the neurovasculature can once again meet the physiologic demands.

While the above describes the more common theory behind CECS, it is by no means the only thought. There have been multiple studies describing that the pain of CECS stems from the stimulation of varying receptors due to excessive fascial stretch.[12]

There may never be complete agreement regarding the underlying cause of CECS, but it continues to be well accepted that rest or cessation of activity results in symptomatic improvement.

Diagnosis

Ultimately, the test for chronic exertional compartment syndrome may be one of exclusion. After ruling out many of the more common diagnostic issues such as shin splints, stress fractures, and overuse syndrome by normal diagnostic measures, CECS should be considered as a possible diagnosis. The whole standard of diagnosing CECS is the measurement of intracompartmental pressures.[1,12] The first measurements were done by Francine Pierce, and later by Reneman. They used a simple needle and manometer, but this could not measure the pressure during exercise. The Stryker catheter is a device most used for intracompartmental pressures. A needle was placed in one of the compartments and normal saline was injected. The pressure was then taken prior to exercising after symptoms occur. Normal pressure is 0 to 8 mmHg. The capillary flow is compromised at 25-38 mmHg, and pain easily occurs at this level. Ischemia occurs when tissue pressures approach the patient's diastolic pressure.[1,12,13]

There are evolving tests to assess CESC. An MRI has been shown to be more sensitive post-exercise, with an increase of T-2 weighted signal intensity. Infrared spectroscopy measures oxygenated and de-oxygenated blood in the muscle. Finally, thallium 201 with a SPECT scan can measure reversible ischemia.[12]

Treatment

The best conservative treatment for CECS is to stop the activity that has created the problem. Many athletes, however, are unwilling to stop their activity, and other conservative measures can be explored if the pain is relieved with cessation of activity. The runner can try running on a softer surface, and can also make better footgear and orthotics. Physical therapy techniques may be effective as well in reducing the time and the volume of exercise activity. IV saline, analgesics, anti-inflammatories, hyperbaric oxygen, and massage have also been tried with some success. Osteopathic manipulation may also be considered.[2,9,12,13]

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The only definitive treatment for CECS is fasciotomies. In 1956, Mavor recorded the first surgery for CECS in a soccer player with the condition. A fasciectomy is also utilized for the recurrence of the condition by itself or in combination with an extended fasciotomy. The surgical release of the fascia has a reported 90% success rate with a complication rate of 3-11%. Recurrence rates are 3-12% in the literature. The newest method of treatment for CECS is to change the first stride pattern of runners. Eighty seven percent of CECS patients have the condition from the sport they participate in, with 69% of these cases involving runners.[9,13,14]

Running technique can also affect gait biomechanics. A forefoot striker gait pattern leads to decreased reaction forces, stride length, and ground contact time. It also influences the anterior compartment pressures, which are decreased in a forefoot striker.[14]

Case Reports

CASE #1:

A 27-year-old male presented to the office with a history of chronic shin pain following fun runs, with a pain that radiated from a 3 to a 5 out of 10. His pain was described as a chronic dull ache to the left anterior shin area. The patient's past medical history was essentially unremarkable with the exception of his history of being a cross country coach. Examination was negative with the exception of a swelling noted on the anterior aspect of the leg. An MRI was ordered, which showed a suspected fascial tear presenting as a mass. The patient was instructed to use compressive therapy and was placed in orthotics. He also tried PRP injections, and was eventually sent to the University Health Science Center for compartment pressures with suspected chronic exertional compartment syndrome.

CASE #2:

The patient presented with progressive bilateral lower leg pain that had been present over the previous month or two. He stated that conservative care failed to give him relief and the pain was exacerbated by running or any type of heavy activity. The pain was described as a sharp ache with prolonged activity at the level of a 3 that can rate up to a 5 out of 10. He denied any history of trauma. The patient's past medical history was essentially unremarkable. A musculoskeletal exam revealed pain in the lower leg anteriorly as well as posteriorly, especially with activity. The patient was diagnosed as having chronic exertional compartment syndrome and was treated with a heel lift, physical therapy, and placement in orthotics. The patient states that the physical therapy and orthotics did help the condition, but the best course of action was decreasing his intensity of exercise, which lowered the patient's pain back to a subclinical level.

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CASE #3:

The patient presented with a history of his feet falling asleep when running more than 3 miles. The patient related a history of diffuse numbness from the lower leg that radiated into his feet, with the pain most noted with any type of running activity. The patient had been averaging approximately 15-20 miles running per week, and the pain had increased over the last 2-3 months. The patient tried only conservative care, including shoe therapy, arch supports, sock changes, re-lacing shoes, and stretching; however, the pain persisted. The patient's past history was unremarkable. The physical exam was unremarkable with the exception of a mild midfoot exostosis, bilaterally. The patient does have mild limb-length discrepancy, which was treated with a heel lift. The patient's diagnosis was chronic exertional compartment syndrome. He was treated with anti-inflammatories and instructed to try a different type of gait pattern. The patient's heel to toe gait pattern was changed into a forefoot striker. After two-year follow-up, pain had resolved and he even recently ran a half marathon without any issues.

Discussion

It has been over a 100 years since Dr. Edward Wilson discussed CECS for the first time when his patient had pain in his lower leg during a return trip from the South Pole. The cause of this syndrome still remains elusive. Although the most common location seems to be the lower leg, it can also be found in the foot, thigh, upper extremity, and back muscles. Since this diagnosis is commonly overlooked, the literature states that a 22-month delay in correct diagnosis can occur.[2,14]

The exact pathophysiology is unknown. It has been hypothesized that CECS results from microtrauma from overtraining, ischemia from an increase in blood volume during exercise, or decreased elasticity within a compartment with nerve entrapment. The importance of intracompartmental pressure measurement to confirm the diagnosis is still debatable, but it can lead to a definitive diagnosis.[8,14]

Treating the condition with a fasciotomy is still considered the best treatment for CECS. However, if possible, conservative care should be tried prior to surgery. These conservative care measures may include decreasing the athlete's activity, cross-training, or the many other conservative measures discussed earlier. Recently, a change in foot stride patterns has shown to be promising for correction of this problem. Running distances have been shown to increase over 300% with the forefoot striker running technique, and may have a value as the future preferred conservative treatment of this issue. The increasing awareness of CECS by both doctors and athletes may result in earlier intervention and a higher success rate of conservative and surgical care for this condition.[12,14] **PM**

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