Chronic Exertional Compartment Syndrome in Athletes: A narrative Review

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Abstract

Chronic exertional compartment syndrome of the lower leg accounts for approximately 75% of sports-related chronic leg pain. Nevertheless, the exact and timely recognition in athletes might pose a great challenge to sports physicians. Among a variety of possible differential diagnoses such as tenosynovitis, stress fractures, periostalgia, or popliteal artery entrapment syndrome the physician has to be able to identify the correct entity as promptly as possible. Consequently, profound knowledge about exercise-associated pathologies of the musculoskeletal, nervous and vascular system, as well as the capability of interdisciplinary thinking are critical. Within the course of this narrative review the authors would like to equip the reader with a comprehensive overview about one of the most important entities of non-traumatic lower leg pain in sports medicine. Hence, chronic exertional compartment syndrome of the leg is going to be looked at from different perspectives such as epidemiology, sports at risk, potential explanations of pathophysiology, current and prospective diagnostic procedures, as well as the latest therapeutical options.

Zusammenfassung


Introduction

Exact and timely recognition of underlying pathology in competitive athletes suffering from exercise induced non-traumatic calf pain might be challenging for sports physicians. Among a variety of possible differential diagnoses such as tenosynovitis, stress fractures, periostalgia, popliteal artery entrapment or chronic exertional compartment syndrome (CECS) one has to be able to identify the correct entity as promptly as possible. Consequently, profound knowledge about exercise-associated diseases of the musculoskeletal, nervous and vascular system, as well as the ability to reason interdisciplinarily are crucial. Thus, the diagnosis of CECS often surmises a process of exclusion of other differential diagnoses (see table 1).
Within the course of this narrative review the authors would like to equip the reader with a comprehensive overview about one of the most common entities of non-traumatic lower leg pain in sports medicine. Hence, chronic exertional compartment syndrome is going to be explored from different perspectives such as epidemiology, sports at risk, potential explanations of pathophysiology, current and prospective diagnostic procedures, as well as the latest therapeutic options.

Epidemiology/Sports at Risk

In 1956, Mavor published a case-report of an “anterior tibial syndrome” in a 24-year old professional football player. In this case, the treatment strategy involved decompression of the fascia and transplantation of a fascia lata graft.[1] At present, chronic exertional compartment syndrome (CECS) of the lower leg accounts for up to 75% of sports-related chronic leg pain in endurance athletes.[2] It represents a overuse muscle injury affecting mostly well-conditioned young athletes (particularly runners) with a median age of 20 years.[3] According to a review by Turnipseed et al., 59% of athletes experienced bilateral symptoms (41% unilateral).[4] Therefore, specification of compartments revealed involvement of anterior compartment in 45%, deep posterior in 40%, superficial posterior in 10% and lateral compartment in 5% of cases.[5] Athletes that participate in endurance sports, particularly long distance running, soccer, and ballet or squash are primarily at risk of developing CECS.[6]

Pathophysiology

In 1948 Hughes et al. suggested fatigue of the anterior tibial muscles to be a potential etiology for pain and ischaemic necrosis in a case-series of football players.[7] It was described in this series that several risk factors such as eccentric exercise, poor running biomechanics, and inadequate weight loading combined with local fatigue may provoke overuse and swelling of specific compartments.[8,9,10] Currently, various contributors for the pathophysiology of CECS are up for debate:

*Inelastic fascia* – Due to rigid sheaths of fascia around the muscle, expansion of the muscle is limited and may cause elevated intracompartmental pressures. Experimental elasticity testing demonstrated that the

<table>
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<tr>
<th>Vascular</th>
<th>Musculoskeletal</th>
<th>Nervous</th>
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<tr>
<td><strong>External vascular compression:</strong></td>
<td><strong>Overuse syndromes:</strong></td>
<td><strong>Nerve entrapment:</strong></td>
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<tr>
<td>Adductor- canal syndrome</td>
<td>Tenosynovitis, tendinopathies (e.g. achilles tendon), stress fractures, medial tibial stress syndrome</td>
<td>Neuropathy of saphenous, tibial or peroneal nerve</td>
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<td>Popliteal entrapment syndrome</td>
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<td>Cystic adventitial disease</td>
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<td><strong>Vessel wall processes:</strong></td>
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<td><strong>Neurogenic claudication:</strong></td>
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<tr>
<td>Atherosclerotic stenosis of femoral or popliteal artery</td>
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<td>S1-Radiculopathy</td>
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<td><strong>Endoluminal processes:</strong></td>
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<tr>
<td>Thromboembolic events in veins or arteries</td>
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fascia of the anterior compartment is stiffer than in the posterior compartment.[11]

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**Increase of muscle volume** – Various authors cite a study by Lundvall et al. describing an increase of muscle volume during exercise of roughly 20%.[13] However, this number is based on investigations in a 6 minutes bicycle ergometer test, and is therefore not comparable to biomechanical stresses generated through other endurance based activities. Furthermore, the ultimate adaptation of muscle volume seems to be load-dependent and is subject to significant interindividual variation.[14] The prevailing theory suggests that during progressive muscle activity, elevated interstitial pressure within the affected compartment outperforms capillary pressure and causes impaired muscle tissue perfusion. This leads to deoxygenation and triggers increased cell permeability with accompanying fluid shift into the interstitial space what itself compromises microcirculation even more.[15]

**Venous congestion** – Birtle et al. demonstrated that external venous occlusion of the anterior tibial compartment muscles in healthy limbs induced similar changes in terms of pain, fatigue, and increase in muscle thickness to those seen in patients suffering from CECS.[16] The fact that numerous veins follow a rather tortuous path through the different non-interconnected collagen layers of the muscular fascia may also be of pathophysiological significance when it comes to compression.[17] Nevertheless, only 3% of patients who experience CECS seem to be affected by venous insufficiency.[4]

**Microcirculation capacity** – In 2010 Edmundsson et al. presented some compelling evidence for low muscle capillary supply as a potential pathogenic factor in CECS.[18] The authors analyzed biopsies from tibialis
anterior muscles obtained during decompression fasciotomy and after a follow-up of one year. CECS patients displayed lower capillary density (273 vs. 378 capillaries/mm$^2$, p-value = 0.008), lower number of capillaries around muscle fibers (5.4 vs. 5.7, p-value = 0.004), and lower number of capillaries in relation to the muscle fiber area (1.1 vs. 1.5, p-value = 0.01) compared with healthy controls. At the one year follow-up there was no increase in capillary network. Thus, the authors concluded, «patients with CECS seem to have reduced microcirculation capacity».

**Diagnostics**

Numerous options of differential diagnoses (see table 1) and clinical resemblances might impede the course of a concise diagnostic procedure. Furthermore, physical examination is often unrevealing and suspicion of CECS is very much dependent on a detailed survey of patients’ history. In this respect, most common symptoms include claudication (90%) and muscle group tightness (60%).[4] Correspondingly, one has to be alert when patients describe exercise induced pain as aching, dull, sharp or pressure.[20] Paresthesias are often associated with pathology in posterior compartment (25%).[4] CECS might also be localized in the medial foot area and potentially misinterpreted as plantar fasciitis due to foot-arch pain in the running athlete.[21]

In 40-46% of symptomatic patients clinical examination reveals fascial defects near the intramuscular septum of the anterior and lateral compartment at the exit of the superficial peroneal nerve. In contrast, only 5% of asymptomatic individuals exhibit fascial defects.[20,22] Arterial pulses are normally intact, even in flexed knees. Occasionally, neurological findings such as paresthesia, numbness or nerve palsy with foot drop may occur.[20] With respect to advanced diagnostic procedures several options are available.

**Intracompartmental Pressure Measurement** – The measurement of intracompartmental pressures represents the gold standard of diagnosing CECS. The normal resting intracompartmental pressures displays values of approximately 5-10 mmHg.[23] Generally applicable reference values are provided by a study of Pedowitz et al. which investigated 159 patients (mean age 26.8 years) within a period of 9 years (1978–1987).[20] In this context, pressure analysis was performed by direct puncturing of the compartment with a needle attached to a fluid pressure monitor device (e.g. Stryker Corporation, Kalamazoo, MI, USA). As a result, parameters such as pre-exercise pressure ≥15 mmHg, 1-minute post-exercise pressure ≥30 mmHg, and 5-minute post-exercise pressure ≥20 mmHg had been identified to be diagnostic for CECS. Therefore, the consideration of these criteria should result in less than a 5% incidence of false positive results. Furthermore, elevation of 10mmHg in a single compartment or failure to return to normal compartment pressures within 25 minutes after exercise were also considered pathological.[20] Nevertheless, there is a controversial discussion about the validity of these diagnostic criteria.[24] There are major implications such as modified knee or ankle joint position which might contribute to significant intraindividual differences in the course of pressure measurement.[25]

**Magnetic Resonance Tomography** – MRT is sensitive (especially post-exercise) for the diagnosis of CECS, but not specific. Furthermore, MRI demonstrates T2-weighted intensity in involved edematous muscles. Also indirect signs such as fascial thickening or fatty muscle-infiltration due to chronic ischemia can be
Infrared Spectroscopy – In 2005 Van den Brand et al. conducted a prospective cohort study (level of evidence, 2) in 50 patients validating that the sensitivity of near-infrared spectroscopy of 85% is comparable to MRI and intracompartmental pressure measurement, whereas associated specificity at a given sensitivity appeared to be lower with magnetic resonance imaging.[27]

B-Mode Ultrasonography – With regard to future diagnostic perspectives, the authors like to draw the attention to a potential non-invasive indirect method of intracompartmental pressure measurement. In 2007 Thalhammer et al. introduced a noninvasive technique to determine central venous pressure (CVP) using high-resolution compression sonography which allows reliable indirect assessment of CVP without intravenous catheterization.[28] Subsequently, several authors adapted this principle in order to determine the intracompartmental pressure in an animal model and a cadaver leg model.[29,30]

Therefore, a pressure sensing transducer was combined with an ultrasound transducer in order to obtain B-mode cross section views of the affected compartment (e.g. tibial anterior compartment). By comparing the compartment depth without and with a defined compression pressure the authors were able to determine a value of relative elasticity (in %). Sellei et al. performed these non-invasive measurements in human individuals with acute compartment syndrome and was able to demonstrate a high level of correlation with invasive intracompartmental needle measurement.[31] As a result, relative elasticity less than 10.5% of the anterior tibial compartment revealed a sensitivity of 95.8% and a specificity of 87.5% to an appropriate diagnosis of acute compartment syndrome.[31] In this respect, non-invasive ultrasound based compartment measurement might also be a valuable option for clinical application in sports medicine.

**Therapy**

Athletes suffering from CECS are at elevated risk of developing a non-reversible condition such as acute exertional compartment syndrome. Unfortunately, the likelihood of success of conservative treatment strategies such as eccentric training, stretching, and adaptation of running technique is limited. Occasionally, clinical examination and gait analysis reveal biomechanical alterations such as pes planovalgus. Therefore, foot orthotics with arch support to correct over-pronation while running may provide some relief to symptomatic patients. Patients with concomitant venous insufficiency might benefit from utilization of compression stockings.[4] Within a case series of 10 patients Breen et al. proposed a gait re-training over 6 weeks in order to alleviate symptoms by achieving a midfoot strike pattern.[32] As a matter of fact, to our knowledge, there are no randomized control trials which evaluate the effectiveness of non-surgical approaches. Nevertheless, conservative treatment should be continued for at least 3 to 6 months before considering surgical interventions such as fasciotomy.[12]

When surgical treatment is considered, a major challenge for sports physicians is the identification of an appropriate time slot within the course of the season. Patients who underwent compartment release within 12 months of symptoms had improved outcomes in terms of postoperative satisfaction compared to individuals who had to wait longer for surgical release.[33] Thus, 81% of patients who underwent operation experienced satisfying results, whereas success rate of non-operative treatment appeared to be
about 41% within a mean follow-up of almost 6 years.[34] Surgical fasciotomy may be performed either open (with crural incisions up to 10 cm in length), or endoscopically assisted. Regardless of technique, when a fascial herniation is present, it must be included in the release to attain successful results. Endoscopically assisted fasciotomy represents an alternative technique that has been affirmed to be as safe and effective as an open fasciotomy.[35] A crucial advantage of this technique is the ability to visualize superficial peroneal nerve and saphenous vein while cutting the fascia with Smellie or extra-long Metzenbaum scissors. In order to identify potential bleeders the application of a tourniquet is not recommended during the time of surgery.[35] There is an ongoing clinical debate regarding the need to release all four compartments in every case or target the affected compartment only.[36] The authors' preference would focus on the release of affected compartments. In the course of post-fasciotomy follow-up total intramuscular pressure might remain more elevated than in normal individuals.[37] Risk of severe operation-associated complications such as injury of superficial peroneal nerve accounts for 11.5–13%. Almost 6% of patients require a second fasciotomy.[38] The surgical outcome depends on the compartment involved. Therefore, success rate in anterior or lateral compartment release accounts for 81%, whereas surgical intervention in deep posterior compartment is only successful in 50% of cases.[38] Recurrence rates using the open approach seem to be about 2%, while closed/endoscopic techniques display numbers up to 11%. [39] In this regard, optimal rehabilitation is key in CECS. Therefore, Blackman et al. [40] proposed a “return-to-play” protocol presented in Table 2.

Table 2: Return to play protocol after fasciotomy (adapted from Blackman et al.[40])

<table>
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<tr>
<th>Time after surgery</th>
<th>Permitted activity</th>
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<tr>
<td>Immediately</td>
<td>Mobilisation exercises of knee and ankle in order to minimize tissue adhesion within the operated area.</td>
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<tr>
<td>3–5 days</td>
<td>Initially, limited weight bearing with the aid of crutches, then full weight bearing as tolerated according to pain.</td>
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<td>10–14 days</td>
<td>As soon as wound healing is completed muscle dynamic strength training, cycling, and swimming could be re-established.</td>
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<tr>
<td>4–6 weeks</td>
<td>Gradual return to low-intense jogging. Return-to-sports training if one compartment has been released.</td>
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<tr>
<td>8 weeks</td>
<td>Return-to-sports training if multiple compartments or both legs were subject to fasciotomy.</td>
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<tr>
<td>12 weeks</td>
<td>90% pain-free</td>
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<tr>
<td>90% pain-free</td>
<td>Return-to-performance and full sports participation allowed if 90% of sports associated stresses are pain free.</td>
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Conclusion

The diagnosis of exercise-induced lower leg pain due to chronic exertional compartment syndrome represents an intersection of disciplines such as vascular medicine, orthopedic surgery and neurology. In the course of the clinical examination, it is essential to discriminate various findings and prime the correct diagnostic path. If conservative therapy options are not successful within 3-6 months, surgical fasciotomy is required. Indeed, surgical outcome depends on the compartment involved.

Practical implications

- Although, CECS accounts for up to 75% of sports-related chronic leg pain in endurance athletes, it
often surmises a process of exclusion of other differential diagnoses.

- Currently, various contributors for the pathophysiology of CECS are up for debate such as inelastic fascia, venous congestion, increase of muscle volume, or decrease in microcirculatory capacity.
- In terms of diagnostics, the measurement of intracompartmental pressures represents the gold standard of diagnosing CECS. Nevertheless, B-mode sonography might have to potential of a fast available, cost-effective, and non-invasive diagnostic technique in the future.
- Surgical fasciotomy may be performed either open or endoscopically assisted.

Competing interests
None declared.

Contributorship
This narrative review was planned, reported and written by Dr. Gaehwiler. Prof. Thalhammer, Prof. Hirschmueller, Dr. Isaak, Mr. Tiernan, and Dr. Grumann revised the manuscript and gave very valuable comments.

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