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# Acute Exertional Compartment Syndrome with Rhabdomyolysis: Case Report and Review of Literature

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Study Design A  
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Statistical Analysis C  
Data Interpretation D  
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**Conflict of interest:** None declared

**Patient:** Male, 17  
**Final Diagnosis:** Acute exertional compartment syndrome  
**Symptoms:** Foot drop • leg pain • paresthesia  
**Medication:** —  
**Clinical Procedure:** Fasciotomy  
**Specialty:** Orthopedics and Traumatology

**Objective:** Rare disease  
**Background:** Acute exertional compartment syndrome (AECS) is a rare cause of leg pain often associated with a delay in diagnosis and potentially leading to irreversible muscle and nerve damage.

**Case Report:** We present the case of a previously healthy, high-level athlete who presented with the acute onset of unilateral anterior leg pain and foot drop the day after a strenuous workout. He was diagnosed with compartment syndrome and rhabdomyolysis. His management included emergent fluid resuscitation, fasciotomies, debridement of necrotic muscle from his anterior compartment, and delayed primary closure. After six months of intensive outpatient physical therapy, including the use of blood flow restriction treatments, the patient returned to sports and received a NCAA Division I Football scholarship.

**Conclusions:** We describe the details of this patient's case and review the literature related to acute exertional compartment syndrome. The occurrence of acute compartment syndrome in the absence of trauma or fracture, though rare, can have devastating consequences following delays in treatment. AECS requires prompt diagnosis and surgical intervention to prevent these consequences. Diagnosis of atraumatic cases can be difficult, which is why awareness is equally as important as history and physical examination. While diagnosis is primarily clinical, it can be supported with direct intra-compartmental pressure measurements and maintaining a high index of suspicion in acute presentations of exertional limb pain.


**MeSH Keywords:** Anterior Compartment Syndrome • Athletic Injuries • Compartment Syndromes • Leg Injuries • Necrosis

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## Background

Acute exertional compartment syndrome (AECS) is characterized by a rise in pressure within a closed fascial space in the absence of a specific traumatic event [1–5]. This rise in pressure can override capillary perfusion pressure leading to a reduction in myocyte oxygenation and ultimately resulting in myonecrosis and neurologic damage [1,2,6]. In comparison to chronic exertional compartment syndrome, which is relatively benign and typically a self-limiting condition, AECS is a surgical emergency. Many clinicians are aware of the potential for acute compartment syndrome (ACS) in the setting of fractures, vascular injury, or significant non-fracture extremity trauma. However, there is limited awareness to the risk of ACS in a non-fracture, non-traumatic presentation. Due to its rarity and atypical presentation, the diagnosis is often delayed [1–7]. Without prompt treatment with fasciotomy, AECS can lead to muscle necrosis and neurovascular injury [1–7]. Therefore, it is critical for clinicians to maintain a high index of suspicion for AECS in patients with acute presentations of non-fracture, atraumatic limb pain.

## Case Report

A 17-year-old healthy African American male high school football player presented to the emergency department (ED) with the subacute onset of right leg pain and the inability to dorsiflex his foot. His past medical history was unremarkable. In the week preceding his presentation, he had attended an off-season football training camp. The day prior to his presentation, the patient developed relatively mild anterior leg pain after a strenuous workout but went to bed that evening thinking it was typical post-exercise muscle soreness. He woke the next morning with worsening pain in the anterior leg and the inability to dorsiflex his right foot. He denied any constitutional symptoms such as fevers, chills, sweats, or other musculoskeletal complaints.

On physical examination, he had a swollen and tense anterolateral leg, pain with passive dorsiflexion and plantarflexion stretch, inability to dorsiflex the right foot, decreased (4/5) great and lesser toe extension, and decreased sensation to touch over the deep peroneal nerve distribution in the first web space. He maintained normal sensation to the remainder of the foot and ankle. He denied any posterior leg pain and had 5/5 plantar flexion and toe flexion strength. Compartment measurements were performed using an Intra-Compartmental Pressure Monitor (Stryker, Kalamazoo, MI, USA). The anterior compartment pressure measured 54 mm Hg with all others less than 25 mm Hg. Labs demonstrated a creatinine kinase (CK) of 19,011 U/mL (reference range 24–170 U/mL), AST 293 U/L (reference range 5–40 U/L), and ALT 112 U/L (reference range

4–41 U/L). Radiographs of the leg and ankle were negative for any abnormalities. Subsequent tests during his admission for glucose-6-phosphate dehydrogenase, sickle cell disease, and hemoglobin electrophoresis were all negative. Serum electrolytes and electrocardiogram were normal.

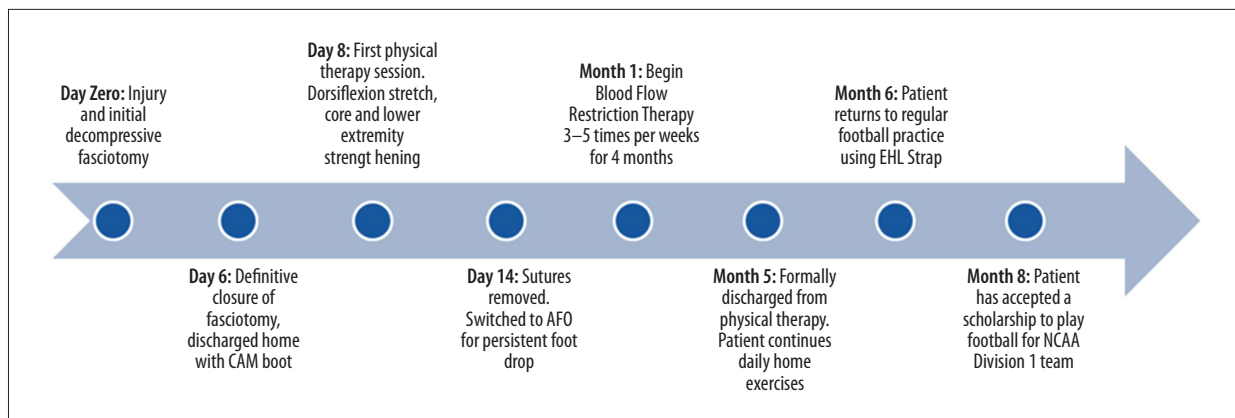
The patient was diagnosed with acute exertional compartment syndrome of the anterior compartment of the right leg with rhabdomyolysis.

## Treatment

The Internal Medicine Service was consulted for management of his rhabdomyolysis. The patient received intravenous fluid resuscitation with a total of 4 L of isotonic crystalloid (0.9% normal saline) during his time in the ED and was then placed on a maintenance fluid rate of 300 mL/hour. He was taken to the operating room for emergent anterior and lateral compartment fasciotomies six hours after his presentation to the ED and approximately 24 hours after the onset of his initial symptoms.

## Surgical technique

Once in the operating theater, general anesthesia was induced. The right leg was prepped and draped in standard fashion and a final timeout was performed. To avoid any additional ischemia, no tourniquet was utilized during the procedure. Due to his clinical presentation and isolated elevated pressures within the anterior compartment, only the anterior and lateral compartments were released. The deep and superficial posterior compartments were not entered. An extensive longitudinal incision was made midway between the anterior crest of the tibia and the fibula. The superficial peroneal nerve was identified as it pierced the fascia to become subcutaneous and was protected throughout the procedure. A transverse incision was made over the lateral inter-muscular septum and the anterior and lateral compartments were released in their entirety. The anterior compartment demonstrated significant tension with bulging of the muscles upon fascial release. The muscles within the proximal anterior compartment were dark brown to grey in color and minimally contractile compared to the lateral compartment muscles which were pink, healthy, and readily contractile to stimulation and electro-cautery. There was no evidence of any focal traumatic injury or hematoma formation. To avoid unnecessary debridement and allow for maximum potential muscle recovery, no muscle was debrided at the index procedure. The incision was left open and a negative pressure wound dressing (V.A.C., KCI Medical) was applied. The patient underwent two additional irrigation and debridement procedures at 48-hour intervals (post-operative day 2 and day 4) with serial debridement of necrotic muscle in the anterior compartment. Delayed primary closure was performed at the fourth operation, six days after his initial presentation. Overall, the



**Figure 1.** Recovery and rehabilitation timeline.

patient lost approximately 80% of his tibialis anterior muscle and 50% of his extensor hallucis longus (EHL) muscle belly. His extensor digitorum longus and peroneus tertius required only minimal debridement. The lateral compartment muscles remained healthy throughout his treatment course.

His rhabdomyolysis improved over the course of the hospitalization through intensive IV fluid resuscitation with normal saline administered at a rate of 300 mL/hour. This rate of infusion was maintained until the serum CK was below 3,000 U/mL at which point fluid maintenance was discontinued on the fifth hospital day. Urine output remained adequate throughout his hospital course and there was no laboratory evidence of acute kidney injury. His BUN and creatinine at presentation were 11.7 mg/dL and 1.3 mg/dL, respectively. Within 24 hours of hospitalization, his creatinine improved to less than 1.0 mg/dL (reference range 0.67–1.17 mg/dL).

## Rehabilitation

The patient was discharged home in a Controlled Ankle Motion (CAM) boot so that he could begin early weight bearing and range of motion exercises. Outpatient physical therapy began two days after discharge. Initial therapy focused on dorsiflexion stretching to prevent the development of equinus contracture, and core and lower extremity strengthening. His sutures were removed at two weeks and he was converted to an Ankle Foot Orthosis (AFO) to maintain neutral dorsiflexion due to his persistent foot drop. The patient's rehabilitation process included 3–5 sessions of formal physical therapy per week for four months. These sessions included the routine use of blood flow restriction training at the Center for the Intrepid at San Antonio Military Medical Center. Blood flow restriction training is a form of exercise that utilizes brief and intermittent periods of partially restricted venous blood flow during low load resistance training to improve muscular strength and endurance. These loads are typically 20–30% of a patient's single repetition maximum [8]. Over the course of four months, the

patient gradually regained tibialis anterior function with dorsiflexion strength improving from 0/5 to 4/5 at his most recent follow-up. Upon discharge from therapy, he continued to lack EHL strength (0/5) for which he preferred the use of a toe extension strap to maintain great toe interphalangeal joint extension. He declined any surgical procedures to correct his toe drop (tendon transfer versus interphalangeal joint fusion). He maintained normal lesser toe extension. Six months after his initial presentation, he had 15 degrees of active ankle dorsiflexion and near normal sensation in the deep peroneal nerve distribution. His only complaint upon discharge was the inability to actively extend his great toe. He returned to play football at a very high level and accepted a scholarship offer to play Division 1 Football in college. Refer to Figure 1 for a visual timeline of our patient's rehabilitation.

## Discussion

The differential diagnosis for atraumatic leg pain in an athlete include medial tibial stress syndrome (MTS), chronic exertional compartment syndrome, tibial stress fractures, tendon pathology (including tendonitis, tendinosis, and rupture), nerve entrapment, and popliteal artery entrapment. While AECS is rare, it may lead to severe and devastating consequences in a matter of hours if left untreated [1–7]. The ability for a clinician to promptly diagnose and initiate treatment for AECS is crucial for the prevention of such consequences. We admit there was a delay in diagnosis with our patient due to both a delay in presentation as well as the failure to immediately recognize the compartment syndrome.

AECS was first described in 1943 by P.R. Vogt and since that time approximately 40 cases of true exertional ACS have been reported [1,7,10–19]. ACS has been reported after heavy exercise, military training, a variety of endurance sports, and even after brief exercise [11–19]. In 2004, Hope et al. compiled a review of 38 non-fracture ACS and compared these to 113 cases

of ACS with fracture [2]. They found that patients with non-fracture ACS were likely to experience up to eight hours in delay for fasciotomy when compared to fracture-related ACS.

In 2015, Livingston et al. reviewed 39 cases of pediatric non-fracture ACS and found that the presence of neurological deficits occurred 33% of the time [1] as compared to 20% in patients with tibia fracture-related ACS as reported by Shore et al. [20]. In one study, 50% of the non-fracture ACS group demonstrated motor weakness at initial presentation, and 63% presented with paresthesia [1]. Livingston et al. demonstrated that the incidence of myonecrosis in non-fracture ACS (54%) was significantly higher than fracture-related ACS (4%) [1,20]. Non-fracture ACS patients also had a lower rate of return to full activity (56%) compared to fracture-related ACS [1]. Risk factors associated with non-fracture ACS include vascular abnormalities, thrombi, blood dyscrasias, anticoagulation use, infection, soft tissue injury, illicit drug use, and metabolic conditions predisposing to myonecrosis [1,5–7,17,21,22].

In 2016, Livingston et al. reported the largest case series of exertional ACS describing seven cases of AECS in athletes with a mean age of 17 [7]. The average time to decompressive fasciotomy in the AECS group compared to a fracture-ACS group was nearly five times longer (97 hours versus 19 hours, respectively, with a wide range from 19 hours to 336 hours) [7]. Much of the delay was attributed to delayed presentation and incorrect initial diagnosis. Patients who were diagnosed within 24 hours had no evidence of long-term complications. For reference, our patient presented to the ED approximately 18 hours after symptom onset and underwent surgical decompression within 24 hours. Each of the seven cases in the Livingston et al. series had anterior and lateral compartment releases and only one required a four-compartment release.

After a thorough review of the literature and all information available related to our patient, we feel he most likely developed isolated rhabdomyolysis in the anterior compartment of his right leg. This local muscle injury resulted in focal edema increasing intra-compartmental pressure eventually leading to compartment syndrome. Rhabdomyolysis is the process of cell death leading to spillage of intracellular contents [5,6,23]. Further consequences of rhabdomyolysis can include electrolyte imbalances and cardiac rhythm abnormalities. As a result, close monitoring of kidney function, urinalysis, electrolytes, and electrocardiogram changes is warranted while CK remains elevated. There have been two reported outbreaks of rhabdomyolysis at NCAA Division 1 Football programs in recent years. Thirteen players at the University of Iowa were treated for rhabdomyolysis in 2011 during off-season workouts. There were no reports of compartment syndrome in any of these players [24]. Bhalla and Dick-Perez reported the case of a young athlete developing rhabdomyolysis and bilateral

thigh compartment syndrome. Unfortunately, this patient developed renal failure requiring dialysis [25].

ACS is a clinical diagnosis often supplemented using intra-compartmental pressure (ICP) measurements [1–7,20,26–29]. Clinically, the classic presentation is unrelenting pain that is not relieved by rest. Pain is often said to be out of proportion to examination. These symptoms are distinctly different from those of MTS, chronic exertional compartment syndrome, and stress fracture, which tend to improve after a brief period of rest [1,6,7,16]. Although our patient presented with pain, it was relatively mild (5/10 pain score and he was capable of unassisted ambulation) when compared to other acute compartment syndromes encountered by the senior author. His chief complaint upon presentation was the inability to dorsiflex his foot with pain as a secondary issue. Clinicians may be familiar with the six P's of compartment syndrome: pain, pallor, pulseless, paralysis, paresthesia, and poikilothermia. The most common presenting symptoms are pain and paresthesia [1,15,16,20]. Pulselessness, paralysis, pallor, and poikilothermia are often late findings [1,7,30]. Patients typically have pain to passive stretch of the affected muscle(s), and the extremity may be swollen, or the compartments feel tense to palpation [7,27]. In exertional ACS, pain and swelling may be the only presenting symptoms [7]. Ulmer et al. found a low sensitivity (13% to 19%) but high specificity (97%) for clinical diagnosis of ACS suggesting the importance of maintaining a low threshold for further work-up [29]. In the setting of acute fracture, ICP measurements are at their peak when measured within 5 cm of the fracture site [28]. In non-fracture ACS, the clinician must identify the compartment suspected of increased ICP and maintain a low threshold for evaluating multiple compartments [2,29]. When measuring ICP, a delta pressure <30 mm Hg between diastolic blood pressure and ICP should be considered abnormal [1]. Intervention for AECS follows the same principles as fracture-related ACS with emergent fasciotomy of all involved compartments [1–27].

## Conclusions

The occurrence of ACS in the absence of trauma or fracture, though rare, can have devastating consequences following delays in treatment. Similar to fracture-related ACS, AECS requires prompt diagnosis and surgical intervention to prevent these consequences. Diagnosis of atraumatic cases can be difficult, which is why awareness is equally as important as history and physical examination. While diagnosis is primarily clinical, it can be supported with direct ICP measurements and maintaining a high index of suspicion in acute presentations of exertional limb pain.

## Conflict of interest

None.

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