

An unusual diagnosis for persistent leg pain in an adolescent girl

This condition should be in the differential diagnosis for any patient presenting with extremity pain—especially if the pain is out of proportion to the clinical findings.

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CASE

A 17-year-old African-American female presented to the emergency department (ED) complaining of right leg pain. She noted that the pain began approximately 2 weeks prior to evaluation and was originally felt in both calves. She said that the left leg pain resolved without difficulty after several days. However, the pain had continued in the right leg and was becoming increasingly severe. The patient described the pain as “achy” and “crampy.” She had tried OTC analgesics with minimal relief. She admitted that the pain was worse with ambulation and movement of her leg. It was a little better when she was not standing on it.

The patient denied any trauma or strenuous activity, although she did walk to catch the bus daily. She had no numbness or tingling in her extremities, chest pain, or

shortness of breath. She had no history of bleeding disorders and denied any significant medical history, although her family history was positive for sickle cell anemia. She had had an elective termination of pregnancy 7 months earlier, with no sequelae. She was currently taking birth control pills. Her last menstrual period was 1 month earlier and had been normal. Her social history was significant for tobacco use.

The physical examination revealed a slightly antalgic gait on initial observation. Mild edema to the pretibial surface was also noted, but no ecchymosis, erythema, or skin disruption to the pretibial surface was seen. There was no pallor to the lower extremity and no erythema to the calf area. The skin of the pretibial surface had a taut appearance and was mildly warm to palpation, but the differences from the other extremity were minimal. There was marked tenderness over the pretibial surface but only very mild tender-

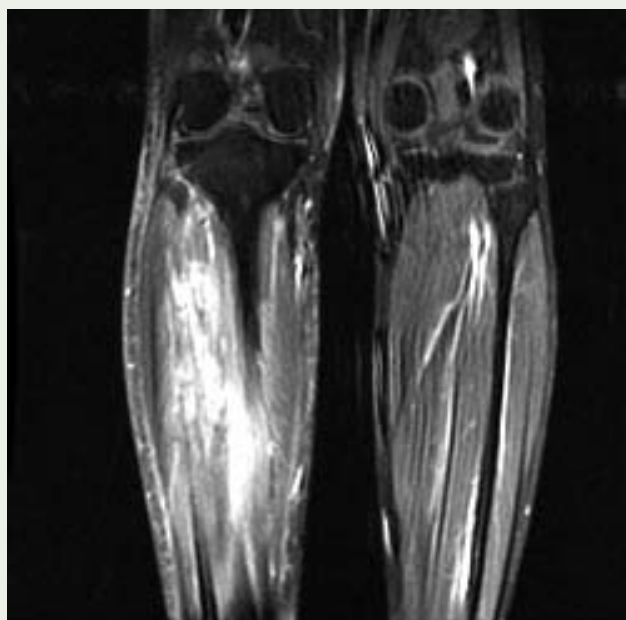


FIGURE 1. MRI views of the patient’s leg show increased signal within the bulk of the anterior tibialis, hallucis longus, and peroneus longus muscles and, more distally, the peroneus brevis muscle.

CASE REPORT | Compartment Syndrome

ness to palpation of the calf. There was no tenderness to palpation of the knee or ankle. Movement of the knee and ankle joints produced no pain. Deep tendon reflexes were 2+ throughout on both lower extremities, and strength was 5/5. Light touch sensation was intact throughout. The patient had full range of motion (ROM) in the leg and foot. Her peripheral pulses were bounding in both legs. The patient was examined by both the PA and the attending physician, who thought that there was some degree of tetany to the leg. The operating diagnosis was a simple muscle strain.

A complete metabolic panel was ordered, and electrolyte results were normal. The patient was given a muscle relaxant and ibuprofen in the ED, which provided her with a little relief. As the patient had no erythema and minimal tenderness to palpation of the calf, an ultrasound was not performed. There was no direct trauma, so plain films were not obtained. She was given crutches and made non-weight bearing. She was instructed to follow up with her primary care provider the next day and discharged with narcotic pain relievers.

Four days later, the patient returned to the ED complaining of worsening leg pain. She had seen her primary care provider, who had advised her to continue with the pain relievers. In the interim, her right leg had become increasing painful and the pain was now associated with dorsi and plantar flexion of the foot.

The examination revealed marked edema and erythema of the lower leg, with increased warmth. The patient was unable to bear weight and complained of paresthesia to the leg and foot. Although light touch sensation remained intact, the leg was exquisitely tender to even the slightest touch. Laboratory test results included a WBC count of 11,300/ μ L and an elevated creatine kinase level. Plain radiographs were obtained, and results were normal. An ultrasound was obtained to rule out deep vein thrombosis (DVT) and was negative. An orthopedist was then consulted and recommended MRI, which showed a fluid collection in the leg (see Figure 1, page 31). Compartment pressures were measured, and the readings were markedly elevated. Compartment syndrome (CS) was diagnosed, and the patient was taken to the operating room for a fasciotomy.

DISCUSSION

Thick layers of tissue called fascia separate groups of muscles in the arms and legs from each other. Inside each layer of fascia is a confined space, called a *compartment*, that includes muscle tissue, nerves, and blood vessels. Fascia do not expand, so any swelling in a compartment will lead to increasing pressure in that compartment, which will compress the muscles, blood vessels, and nerves. If this pressure is high enough, blood flow to the compartment will be blocked, and CS can develop.

“Compartment syndrome occurs when perfusion pressure falls below tissue pressure in a closed compartment space.”

The most common area for an anatomic compartment to occur is in the lower extremities. The lower leg is divided into four compartments: anterior, lateral, superficial posterior, and deep posterior.¹ CS usually results from infection or trauma such as a crush injury or fracture. In cases where there is no obvious trauma to the affected area, an apparent atraumatic CS can occur. The most common presenting symptom is pain.

Compartment syndrome occurs when perfusion pressure falls below tissue pressure in a closed compartment space. An integral mechanism of this disorder is the elevation of the pressures in the compartment. The elevation will occur when tissue injury results in swelling and bleeding and subsequently increases the pressure inside the muscle compartment. The inflammatory response associated with trauma drives fluid into the interstitial compartment from the intravascular channels. There is a reduction in venous outflow secondary to pressure on the venules and lymphatic drainage of the compartment. At the same time, inflow of arterial blood continues, further increasing intracompartmental pressure.

Normal compartmental pressure is maintained by the interstitial fascia that maintains an adequate difference between

TEACHING POINTS

- Two types of compartment syndrome (CS) are generally recognized: acute compartment syndrome (ACS) and chronic exertional compartment syndrome (CECS). ACS is usually associated with trauma to the area or with external forces. CECS typically occurs in athletes who have repetitive loading leading to microtrauma of physical activity.
- The classic and pathognomonic Ps of CS include *pain, pulselessness, pallor, paraesthesia, and paralysis*, though these are not seen in every case.
- The definitive diagnostic test is measurement of compartment pressures. Several techniques are used, but the most common involves inserting a wick catheter into the affected compartment and connecting it to a pressure monitor.
- Definitive treatment for ACS is immediate fasciotomy; but treatment of CECS may begin conservatively, with behavior modification and physical therapy. Conservative treatment is not always successful, however, and fasciotomy may eventually be needed to completely relieve symptoms.
- CS must be part of the differential diagnosis for any patient presenting with extremity pain. It should be high on the list if the patient has known trauma, but further investigation is warranted in anyone with pain out of proportion to the clinical findings.

arterial and venous pressure at the capillary level. Normal tissue pressure is 0 to less than 10 mm Hg.² The fascia have no stretch fibers; therefore, any stressors such as bleeding into the compartment or swelling can exponentially increase the pressure within the compartment. The increased pressure compromises capillary blood flow. Increased pressures can also compromise venous and lymphatic drainage in the affected area. When pressures rise higher than 20 mm Hg, capillary blood flow is at risk. At 30 to 40 mm Hg, nerve and muscle necrosis become possible.² Furthermore, at this level hypoxia begins within the tissues of the compartment secondary to the decreased flow of the capillaries.

Two types of CS are generally recognized: acute compartment syndrome (ACS) and chronic exertional compartment syndrome (CECS). The increased pressure in ACS is usually associated with trauma to the area, such as a fracture or blunt injury. It can also be associated with external forces such as a cast or burn eschar. This most commonly occurs in the anterior compartment of the leg. Another potential cause of ACS is reperfusion after an ischemic event. With correction of an arterial injury, reperfusion and edema may extend beyond the original injury, leading to an increase in surrounding pressures.

CECS typically occurs in athletes who have repetitive loading leading to microtrauma of physical activity. The anterior and deep posterior compartments are the most commonly involved. With the chronic form, there is usually pain and crampy muscle tightness with minimal clinical findings. The pain is also usually limited to the involved compartment. The typical patient is a runner who has pain only with exercise. The pain will usually resolve with cessation of the activity, though the intensity will increase with each exacerbation.

The classic and pathognomonic Ps of CS include *pain*, *pulselessness*, *pallor*, *paraesthesia*, and *paralysis*, though these are not seen in every case. The patient with ACS will have a primary complaint of pain. Often the patient will appear to be at a level of discomfort not supported by the examination. The patient may also report paresthesia in the cutaneous nerves. However, the other Ps may not manifest until late in the course of the disorder.

Paresthesia, pulselessness, pallor, and paralysis often develop when damage to the surrounding vasculature and muscles has been significant, with necrosis not far behind. Pain is increased with stretching of the muscle group involved by passive ROM and extension of the area. Palpation of the involved area will usually reveal a tense, firm muscle group. Clinicians should note that in patients with CECS, the examination may not elicit pain—unless the patient has recently exercised, exacerbating the condition. If the patient presents after exercise, the leg may be edematous with tense muscles. If the condition has progressed enough, muscle weakness and paresthesia to light touch may be present. The patient may also be hypotensive.

Plain films of the involved limb are typically normal, as are ultrasound findings—although ultrasound of the involved limb will exclude DVT from the differential. MRI may

reveal fluid collection but may be nonpredictive early in the course of CS—although in the case of our patient, it did help to define the diagnosis.

A common mistake is to diagnose shin splints or a stress fracture instead of CS. Stress fractures result from repetitive microtrauma, and plain films may appear normal early in the condition. A bone scan is usually diagnostic in this case. The bone pain in stress fractures is usually well-localized and exacerbated by activity. Shin splints are symptomatic in the middle and distal thirds of the tibia. Pain can range from dull achiness to sudden, severe and sharp pain. As with stress fractures and CECS, the pain of shin splints may be alleviated with rest and exacerbated by activity. Unlike with ACS, however, both passive and active ROM is painless in shin splints and stress fractures. Also, a patient with a stress fracture or shin splints will not have associated neurologic complaints.

The definitive diagnostic test is measurement of compartment pressures. Several techniques are used, but the most common involves inserting a needle or wick catheter into the affected compartment and connecting it to a pressure monitor. The values that determine the diagnosis are different for acute and chronic compartment syndromes. Pedowitz and colleagues determined that resting pressures higher than 10 to 15 mm Hg and 5-minute post-exercise pressures higher than 15 to 20 mm Hg are diagnostic of CECS.³ For ACS, many surgeons use pressures higher than 30 mm Hg as an indication for immediate intervention. Another diagnostic measurement is compartment pressures within 10 to 30 mm Hg of diastolic

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BP, known as the *delta pressure*. If the diagnosis is in question, serial measurements may be obtained. Consecutively elevated pressures are also an indication of an ACS.

Definitive treatment for ACS is fasciotomy. The procedure should be performed as soon as possible after diagnosis. A longitudinal incision is made in the affected compartment. Decompression should be followed by debridement of any dead tissue. The surgical incision is left open until pressures have decreased sufficiently for re-approximation of the wound edges. Delayed closure will usually follow 2 to 3 days later. The most common complication of the procedure is wound infection.

Treatment of CECS may begin conservatively, with behavior modification and physical therapy. Shoe design and training techniques and modalities must be addressed. Orthotics are used for limb misalignment or muscle imbalance. Conservative treatment is not always successful, however, and fre-

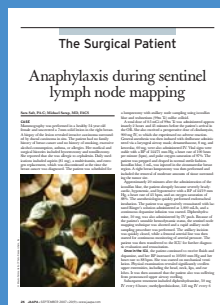
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CASE REPORT

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quent reevaluation is necessary. Fasciotomy may eventually be needed to completely relieve symptoms. Physical therapy is necessary after surgery in both forms of CS syndrome.

If left untreated, CS can have dire, long-reaching consequences. These include foot drop (with untreated anterior compartment pathology), acute renal failure secondary to rhabdomyolysis, and tissue necrosis in the affected compartment. Volkmann's contracture can also develop with long-term ischemia to the forearm due to increased pressures.² Contracture deformities of the hand, arm, and wrist will occur in varying degrees of severity according to the duration of the ischemia. This condition is commonly associated with forearm fracture and resulting vascular compromise.

Our patient had an atypical presentation. Her CS was not acute because her symptoms had slowly progressed over 2 weeks. Yet her only exercise consisted of walking to a local bus stop, so the diagnosis of CECS did not apply either. ACS has been associated with nontraumatic causes such as hypothyroidism, viral myositis, and bleeding disorders including sickle cell anemia. Our patient did not suffer from any of these disorders either.

In our patient, necrosis of the involved tissue as well as foot drop eventually developed. After surgery, a localized infection at the fasciotomy site developed, and the patient had to be taken back to the operating room for wound debridement. Eventually she was discharged to home for physical therapy and long-term follow-up.

In conclusion, CS must be part of the differential diagnosis in any patient presenting with extremity pain. It should be high on the list of suspected conditions if the patient has known trauma, but further investigation is warranted in anyone with pain out of proportion to the clinical findings. Taut, painful compartment areas; inability to bear weight; and particularly pain should all increase the index of suspicion. Failure to diagnose CS can result in long-term or permanent disability for the patient. **JAAPA**

Pamela Young practices in the emergency department at Albany Medical Center, Albany, New York. The author has indicated no relationships to disclose relating to the content of this article.

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