Footdrop Without Significant Pain as Late Presentation of Acute Peroneal Compartment Syndrome in an Intercollegiate Football Player

David Marcu, MD, William H. Dunbar, MD, and Lee D. Kaplan, MD

Acute compartment syndrome (ACS) within an extremity is an orthopedic emergency that is usually associated with a high-energy fracture, crush injury, or vascular injury. ACS without fracture, resulting from a noncontact sports injury, is rare.

We present the case of a National Collegiate Athletic Association (NCAA) Division I football player with ACS caused by a peroneal muscle rupture. The condition was recognized only after significant pain and a marked functional deficit had developed. The authors have obtained the patient's written informed consent for print and electronic publication of his case report.

Case Report

An 18-year-old NCAA Division I football player was on a team road trip when he presented to our university's emergency department (ED) the evening before his game with a chief complaint of right footdrop. When participating in practice 3 days before presentation, he had planted his right leg to make a cutting move to the left and experienced a "cramp" of the proximal lateral aspect of the right leg. He did not roll his ankle or experience frank knee pain. He was able to finish the practice but continued to have pain along the outside of the leg, without any instability, catching, or locking. Cramping pain and swelling of the lateral leg worsened overnight, and he also experienced burning sensations over the dorsum of the right foot. The next day, he went to the training room, had his leg taped, and again participated in practice, which exacerbated the symptoms. The pain and burning sensations returned that night, and he did not practice the next day. This was not expressed to the training staff. That day, he had subjective weakness and continued pain, paresthesias, and swelling. He sought treatment at a local ED, where a Doppler ultrasound study of the right leg was negative for deep vein thrombosis. On the morning of the team's trip, he found it difficult to walk because of leg weakness, and he had a complete footdrop when the team bus arrived. We were contacted by the team's training staff, and the player was referred to the ED. He was seen by the orthopedic resident there approximately 76 hours after initial injury.

On examination, the very muscular young man was in no apparent distress. His right lower leg showed mild swelling laterally. The knee was ligamentously stable and without an effusion. There was no pain about the foot or ankle to palpation or range of motion. There was mild tenderness to palpation along the distal half of the anterolateral aspect of the leg, and the lateral compartment was taut but compressible. The patient described his pain as mild and as having improved over the previous 2 days. There was no increase in pain with passive motion at the ankle or toes, including passive inversion and plantarflexion at the ankle. Sensory examination showed decreased sensation along the dorsum of the foot and lateral leg but retained sensation

> "It is prudent to be aware of later ACS manifestations, including footdrop and other functional deficits, and to realize that patients with ACS can present without any significant pain on examination."

Dr. Marcu is a resident in Orthopedic Surgery, Department of Orthopedics and Rehabilitation, University of Wisconsin, Madison, Wisconsin.
Dr. Dunbar is a practicing orthopedic surgeon in Wisconsin.
Dr. Kaplan is Chief of UHealth Sports Medicine and Associate Professor in the Department of Orthopedic Surgery, University of Miami, Miller School of Medicine, Miami, Florida.

Address correspondence to: David Marcu, MD, University of Wisconsin, Department of Orthopedics & Rehabilitation, K4/732, 600 Highland Ave, Madison, WI 53792 (e-mail, dave.marcu@gmail.com)

Am J Orthop. 2009;38(5):241-244.

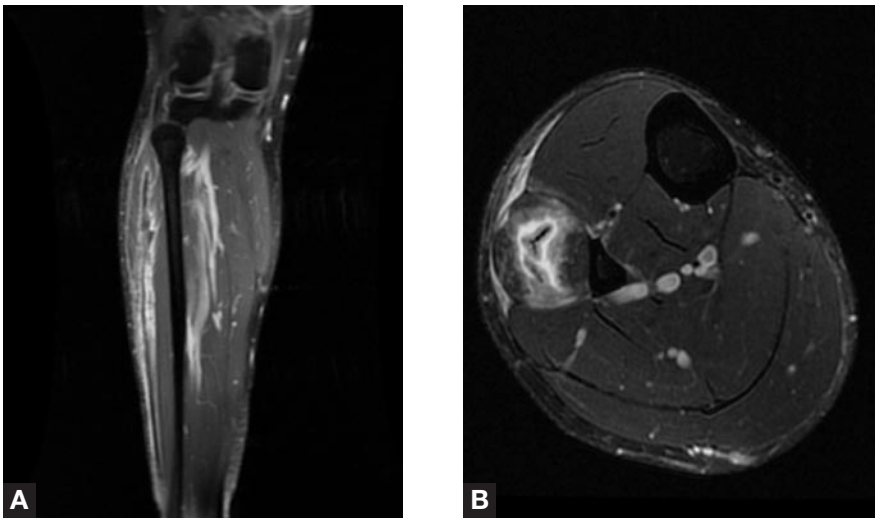

Figure 1. Coronal (A) and axial (B) T_2-weighted magnetic resonance imaging shows hematoma and edema in lateral compartment from ruptured peroneus longus muscle at its musculotendinous origin.

in the first dorsal web space and the medial leg and plantar aspect of the foot. Motor examination showed 0/5 strength of the peroneals, tibialis anterior, extensor hallucis longus, and extensor digitorum communis. There was 5/5 strength of the flexor hallucis longus, the flexor digitorum, and the gastroc-soleus complex. There were palpable dorsalis pedis and posterior tibial pulses.

Radiographs showed no abnormalities of the knee, tibia, fibula, or ankle. Magnetic resonance imaging (MRI) of the right leg was obtained to differentiate between peroneal nerve entrapment and compartment syndrome. T_2-weighted MRI showed high signal in the lateral compartment with rim-enhancing fluid collections in the proximal peroneus longus (Figure 1), consistent with partial-thickness tearing of the peroneal muscles versus hemorrhagic necrosis of the proximal portion of these muscles. There was also enhancement of the fascia between the lateral and anterior compartments but no evidence of high signal within the anterior compartment itself. A compartment monitor (Stryker, Kalamazoo, Mich) demonstrated a lateral compartment pressure of 108 mm Hg (rechecked at 90 mm Hg), anterior compartment pressure of 25 mm Hg, superficial posterior pressure of 22 mm Hg, and deep posterior pressure of 19 mm Hg. Blood pressure was 147/85 mm Hg.

The patient was taken to the operating room, where fasciotomies of the lateral and anterior compartments were performed. Before incision, compartment pressures were rechecked: lateral, 96 mm Hg; anterior, 26 mm Hg; superficial posterior, 22 mm Hg. A lateral fasciotomy was then performed through a lateral skin incision. The peroneal musculature appeared dark and swollen but contracted in reaction to electrocautery (Figure 2). The fascia separating the anterior and lateral compartments was identified and incised. The anterior musculature was red and healthy-looking. Another lateral incision was made, in line with the first but more proximally. The lateral fascia was incised at that level as well, and the 2 fascial incisions were connected subcutaneously. The more proximal peroneal musculature also appeared dark and swollen but was reactive; the anterior musculature at that level again appeared healthy. We identified a piece of tissue resembling peroneal musculotendinous junction; this tissue was free proximally and confluent with peroneus longus distally. We then explored the peroneal nerve to ensure that it was intact and that there were no particular sites of entrapment. We identified the common peroneal nerve just posterior to the fibular neck, as well as its bifurcation into the superficial and deep branches (Figure 2). A loculated hematoma found in this region was evacuated. We traced the deep branch into the anterior compartment and saw it was not impeded, and we traced the superficial branch distally through the zone of musculotendinous rupture and freed it from a surrounding hematoma and adherent musculature. After the fasciotomies were performed and the

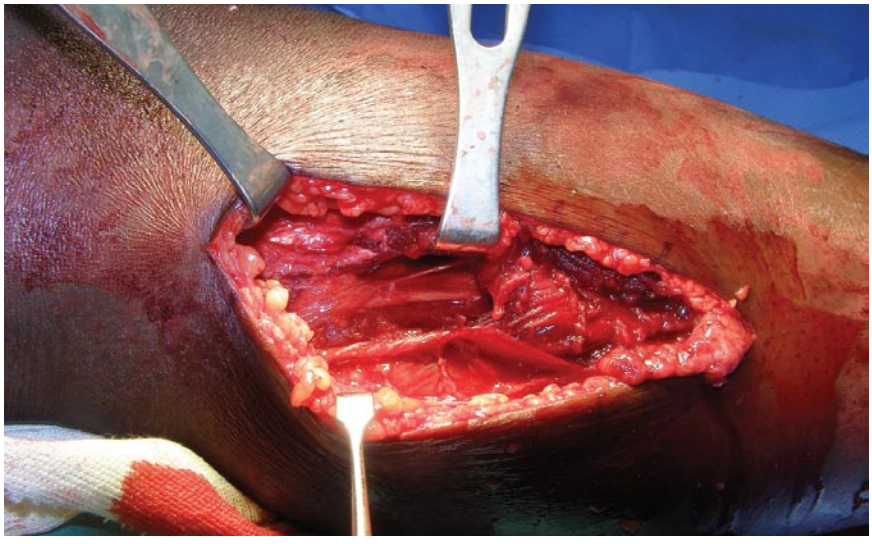

Figure 2. Intraoperative photograph shows bifurcation of common peroneal nerve.

nerve explored, compartment pressures (lateral, 15 mm Hg; anterior, 22 mm Hg; superficial posterior, 19 mm Hg; deep posterior, 22 mm Hg) confirmed compartment release. The wounds were copiously irrigated, and the skin was primarily closed without any significant tension using vicryl and nylon sutures (Figure 3). The patient's incisions were dressed, and his leg was placed into the padded brace to prevent equinus.

The patient was discharged the next day into the care of his team medical staff. He initially required an ankle–foot orthosis and had poor control of the foot. Follow-up examinations revealed remarkable progress. Twelve weeks after surgery, he had 5/5 strength of the dorsiflexors and 4+/5 strength of the peroneals. Sensation throughout the superficial peroneal nerve dermatome continued to improve. The incisions healed well. The patient was working with the training staff on rehabilitation of right lower extremity strength. He continued to have occasional burning paresthesias, and his dorsiflexors and everters fatigued during repetitive activity. The right leg musculature was atrophied in comparison with the left but was improving. The patient was scheduled to participate in a modified winter conditioning and lifting program and to continue to undergo focused rehabilitation as directed by the team's medical staff. The possibility of returning to football was to be determined.

"We...determined that releasing the compartments would give this young athlete the best chance to recover from his functional deficits, whereas the nonoperative option had a decidedly grim prognosis."

Discussion

Acute peroneal compartment syndrome from peroneal rupture, which has seldom been reported,[1-5] is usually associated with an ankle inversion injury.[1,2,4,5] Some of the case reports are similar to ours in that the diagnosis of compartment syndrome was initially missed and, on later diagnosis, footdrop was present.[2] However, pain was a primary symptom in each of the published reports, and there was pain with passive stretch on physical examination. In our young athlete's case, there was significant pain on initial presentation to his training staff, over the next 2 days, and on presentation to a local ED. But the pain leveled off and then subsided somewhat, and it was functional weakness that then led the patient to seek medical attention again. Our examination did not elicit any significant pain on passive stretch. As we still suspected compartment syndrome, we sought more information (MRI scan, compartment pressures) before taking him to the operating room for the fasciotomies.

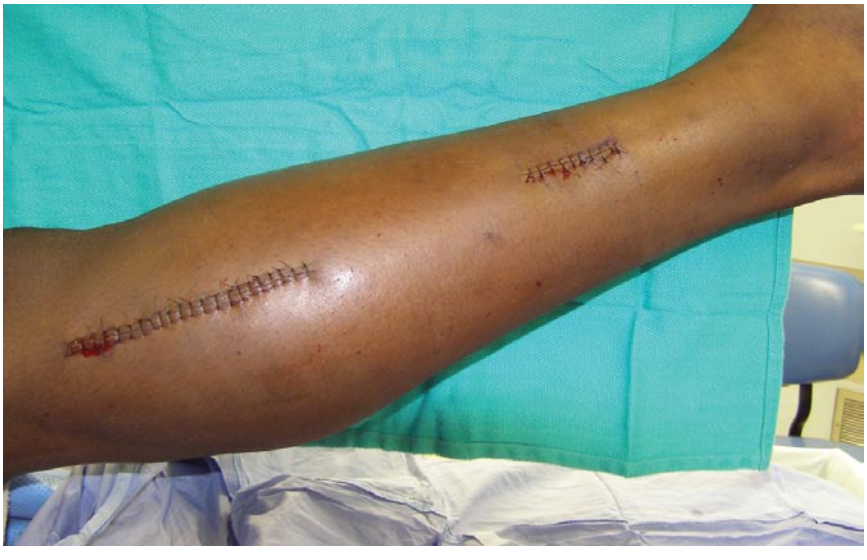

Figure 3. Intraoperative photograph shows primary skin closure of 2-incision fasciotomy.

This athlete ruptured his peroneus longus by eccentrically loading the lateral compartment musculature of the right leg while changing direction from right to left during practice. This resulted in an expanding hematoma, later evident on MRI. The patient had significant pain and paresthesias for approximately 48 hours after injury, but his presenting sign at our ED was paralysis, making this case unique. It is commonly recognized that pain is a relatively early sign of ACS, and paralysis of the musculature within the affected compartment is a relatively late manifestation. Paralysis can result from either muscle necrosis or damage to the innervation of those muscles. Evidence has shown that muscle necrosis occurs with intracompartmental pressure of more than 30 mm Hg for more than 8 hours.[6] Other data demonstrate that higher pressures are needed to arrest nerve conduction. A canine model showed that pressures of 40 mm Hg for 14 hours did not arrest nerve conduction but that conduction stopped with 50 mm Hg in less than 7 hours.[6] Our patient presented approximately 76 hours from time of muscle rupture, with lateral compartment pressures between 90 and 108 mm Hg, making it likely that both the peroneal musculature and superficial peroneal

nervous tissue took sufficient insult to be nonfunctional. The final pathology confirmed that there was some degree of myonecrosis of the lateral compartment. A nerve biopsy was not performed.

The patient also had functional loss of tibialis anterior, extensor hallucis longus, and extensor digitorum communis, which implies injury either to the anterior compartment musculature or to the common or deep peroneal nerves. Intraoperative findings showed healthy-appearing anterior musculature, and anterior compartment pressures were not significantly elevated, implying that it was the nerve supplying the anterior compartment that was affected. This can be explained by the anatomy of the peroneal nerves.

The common peroneal nerve courses through the proximal aspect of the lateral compartment, near the origin of the peroneus longus muscle, before its bifurcation sends the deep branch through the proximal lateral compartment and into the anterior compartment. Lateral compartment pressure elevation resulting from hematoma from rupture of the peroneus longus origin can affect the common peroneal nerve, even when there is no direct elevation of anterior compartment pressure.

Gabisan and Gentile[4] reported a case of acute peroneal compartment syndrome from an inversion ankle injury in which there was decreased sensation in the first web space and anterior compartment pressure of 60 mm Hg. They deduced that the altered sensation and weakness of dorsiflexion were caused by a combination of somewhat elevated anterior pressure and compression of the deep peroneal nerve within the lateral compartment. During surgery, we identified a loculated hematoma around the bifurcation of the common peroneal nerve and around the proximal deep and superficial peroneal nerves in the lateral compartment.

Some have argued against surgical release in cases of delayed presentation of ACS—that late fasciotomy risks postoperative infection, septicemia, and a worse prognosis for limb salvage by exposing a bed of already necrotic muscle to the environment.[7,8] We considered this before surgery but determined that releasing the compartments would give this young athlete the best chance to recover from his functional deficits, whereas the nonoperative option had a decidedly grim prognosis. Careful aseptic technique, copious irrigation, and perioperative antibiotic prophylaxis allowed our patient to heal without infectious complication, and he has already shown remarkable functional recovery.

Because of myonecrosis and damage to peripheral nerves, ACS can have devastating functional outcomes. Being mindful of the possibility of compartment syndrome in a patient who presents with pain and paresthesias after an athletic injury can help reduce time to recognition and surgical treatment of this progressive condition. It is also prudent to be aware of later ACS manifestations, including footdrop and other functional deficits, and to realize that patients with ACS can present without any significant pain on examination. It is even more important to be vigilant in the evaluation of high-level athletes, such as this NCAA Division I football player, as they are more at risk for significant soft-tissue injury and may be accustomed to playing through pain. Awareness and caution on the part of trainers, team medical staff, and orthopedic consultants can help limit morbidity and aid in recovery from these injuries.

Authors' Disclosure Statement and Acknowledgments

The authors report no actual or potential conflict of interest in relation to this article.

We appreciate the work of our patient's medical and training staff in his continuing care and rehabilitation, and we are grateful for the follow-up information that was provided.

References

1. Davies JA. Peroneal compartment syndrome secondary to rupture of the peroneus longus. A case report. *J Bone Joint Surg Am*. 1979;61(5):783-784.
2. Ebenezer S, Dust W. Missed acute isolated peroneal compartment syndrome. *Can J Emerg Med Care*. 2002;4(5):355-358.
3. Edwards PW. Peroneal compartment syndrome: report of a case. *J Bone Joint Surg Br*. 1969;51(1):123-125.
4. Gabisan GG, Gentile DR. Acute peroneal compartment syndrome following ankle inversion injury: a case report. *Am J Sports Med*. 2004;32(4):1059-1061.
5. Goodman MJ. Isolated lateral-compartment syndrome. Report of a case. *J Bone Joint Surg Am*. 1980;62(5):834.
6. Azar FM. Compartment syndrome. In: Canale ST, ed. *Campbell's Operative Orthopaedics*. 10th ed. St. Louis, MO: Mosby; 2003:2449-2457.
7. Finkelstein JA, Hunter GA, Hu RW. Lower limb compartment syndrome: course after delayed fasciotomy. *J Trauma*. 1996;40(3):342-344.
8. Williams P, Shenolikar A, Roberts RC, Davies RM. Acute non-traumatic compartment syndrome related to soft tissue injury. *Injury*. 1996;27(7):507-508.

This paper will be judged for the Resident Writer's Award.