Compartment Syndrome Following Recurrent Ankle Inversion Injury

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Compartment syndrome is an elevation of the interstitial pressure in a closed osseofascial compartment that results in microvascular compromise. The condition may be further classified as either acute or chronic. Common causes of acute compartment syndrome include fractures, severe blunt soft tissue trauma, arterial injury, limb compression, and burns. Chronic compartment syndrome is caused by increased muscle volume most commonly occurring during exercise.

The diagnosis of an acute compartment syndrome is made by clinical signs and can be further confirmed by compartment pressure measurements. Measurements of compartment pressure are used for the evaluation of a compartment in which the diagnosis of compartment syndrome cannot be made or ruled out with certainty, as in a patient with altered mental status or in chronic exertional compartment syndrome. Signs and symptoms of acute compartment syndrome include pain out of proportion, increased use of analgesics, pain with passive muscle stretching, paresis, paresthesia, and diminished pulses. Pulses usually remain intact due to arterial pulses remaining higher than the pressure of the compartment compressing the artery. The following case report describes the clinical presentation and treatment of compartment syndrome following an inversion injury of the ankle.

**Case Report**

A 17-year-old male varsity baseball player suffered a minor right ankle inversion sprain while playing baseball. He was treated at that time with normal protocol of rest, ice, compression, and elevation. The patient was able to play in a baseball game the following day. After hitting the ball, he was rounding first base and suffered another inversion injury to his ankle. He was unable to continue playing and had to leave the field one hour later. He was taken to the local emergency department three hours after his injury. Radiographs of his ankle were normal, and he was diagnosed with a grade I ankle sprain. The patient went home but developed continually increasing pain in the right lateral aspect of his lower leg. Early in the morning the next day, the patient woke his father because of increasing pain. His father gave him some pain medicine, and the patient was able to fall asleep. He awoke the next day with increasing pain when he moved his foot. He was seen the next day in the office for evaluation of his “ankle sprain.”

On physical exam, he was a healthy male in extreme discomfort. He was unable to bear weight on his right leg. There was no difference in calf musculature size, but he had minimal swelling around his right lateral malleolus. His ankle exam was negative for instability and deltoid ligament tenderness, and had no deformity of his fibula or translation of the fibular head. He had an extremely tense anterolateral compartment of his tibia from his fibular head to lateral malleolus, and this area was extremely tender to touch. He was able to dorsiflex his ankle, invert his foot and extend his toes, but was unable to extend his big toe or evert his foot. He had significant pain referred to the lateral and anterior compartments on passive stretch of the big toe and ankle. No sensation in the region on his first dorsal web space and decreased sensation to light touch on the dorsal aspect of his foot was noted. The patient had full sensation on the plantar aspect of his foot. He also displayed a 1+ pulse of his dorsalis pedis artery on his right foot with a 2+ pulse of his right posterior tibial artery and his left foot pulses. The posterior musculature of the leg was soft and non-tender. The patient was diagnosed with compartment syndrome of the anterior and lateral compartments.

The patient was taken emergently to the operating room within one and a half hours from presentation, and under general anesthesia anterior and lateral compartment fasciotomies were performed.

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planned. Blood work was obtained intraoperatively, and all values were normal except his aspartate aminotransferase (AST) (57 U/L), alanine aminotransferase (ALT) (54 U/L), and creatine kinase (CK) (1383 U/L). A 10" (20 cm) incision was made along the anterolateral tibia from the region of the tibial plateau down to the distal lateral malleolus. Upon encountering the fascia of the lateral compartment, the lateral compartment was bulging and tense and had a violaceous color from a hematoma that was present (Figure 1). The anterior compartment appeared slightly tense, but showed no discoloration.

The lateral compartment was incised along its entire length, making certain to protect the superficial peroneal nerve that appeared 10 cm proximal to the lateral malleolus (Figure 2); it was noted to bulge 4 cm through the fasciotomy. The musculature of the lateral compartment initially had a dusky appearance. A few minutes following the release of the lateral compartment, the muscle coloration improved to a more normal appearance. The anterior compartment was then released. The musculature of the anterior compartment did not have the dusky appearance. The contractility and consistency of the muscles in the anterior and lateral compartment were tested and were normal. The wound was inspected for venous or arterial bleeding, but none was encountered. The wound was irrigated, left open, and dressed with wet-to-dry dressing supported with a burn dressing and a loosely applied elastic bandage. In the recovery room the patient’s pain had resolved, the numbness on the dorsum of his foot improved, and his right dorsalis pedis pulse returned to normal. He also had improved function of his extensor hallucis longus, but there was still some noted weakness.

The wound was closed 36 hours later by placing three far-near-near-far sutures in the midregion for approximation, followed by placement of vertical mattress stitches for closure of the remainder of the incision (Figure 3).

Upon discharge the following day, the strength of his extensor hallucis longus was continuing to improve, as was sensation of his first dorsal web space. The patient had an uneventful postoperative recovery. At his first postoperative visit, stress radiographs of his ankle and fibular head were normal. His sensation and strength of his foot and ankle progressively recovered over the next couple of weeks. His lab values of AST (20 U/L), ALT (24 U/L), and CK (89 U/L) at two weeks had normalized. He was able to return to playing baseball two months after his compartment releases.

**DISCUSSION**

Acute compartment syndrome of the lower extremity is frequently associated with high-energy trauma, and commonly associated with fractures. Less commonly,
Compartment syndrome can occur in patients with muscular tears, musculotendinous injuries, or syndesmotic tears resulting in hemorrhage. In this patient, the compartment syndrome was caused by a double insult of inversion injuries of the ankle on successive days. We believe that the original injury contributed to the development of compartment syndrome after the second injury, because there was less potential space in the compartment to accommodate the swelling. The diagnosis of compartment syndrome was based on the clinical presentation of increasing pain out of proportion to the initial injury, pain on passive stretch referred to the compartment in compromise, and decreased sensation on the dorsal surface of the foot and loss of sensation to the first dorsal web space. We did not perform compartment measurements in this case due to the certainty of the diagnosis and the urgency of the situation. Even though no bleeding vessels were seen during the surgery, this could still be an etiology for the compartment syndrome supported by evidence of the hematoma in the lateral compartment.

Compartment syndrome from noncontact injuries to the ankle has been identified by other case reports in the literature. Moyer et al reported three cases of lateral compartment syndrome that involved young athletes who were initially diagnosed with injuries other than compartment syndrome. These patients presented later with compartment syndrome and required fasciotomies. Ancillo et al, Davis et al, Gabisan et al, and Mendelson et al reported cases of compartment syndrome involving ruptures of the peroneus longus muscle in noncontact injuries among athletes. Strachey and Jones reported on a rupture of the medial head of the gastrocnemius that resulted in compartment syndrome of the anterior, lateral, and superficial posterior compartments. In all these cases, the patients were found to have tears within the muscles of the lower leg causing the increase in pressure in the specific compartments. The musculotendinous junction is a frequent site of injury and can lead to sufficient hemorrhage and edema causing ischemic compromise within the compartment. In all of these cases, injuries within the compartment lead to bleeding and edema causing compartment syndrome.

Treatment for compartment syndrome requires emergent and immediate decompression. Delay in treatment can cause complications such as rhabdomyolysis, Volkmann’s contractures, renal failure, shock, hyperkalemia, and even death. The release of the compartments of the lower leg can be accomplished by either open or percutaneous fasciotomy. We chose an open fasciotomy due to the severity and length of time of the patient’s symptoms, and the fact that the entire length of the compartments was involved. It has been reported that the intact skin between the percutaneous fasciotomies can act as a constricting band once reperfusion is initiated causing a second compartment syndrome. The longer incisions in open fasciotomy have been shown to significantly decrease compartment pressures when compared to percutaneous fasciotomies.

Compartment syndrome of the lower leg must be considered in the differential diagnosis of any physician who encounters patients that present with this type of injury. With the athlete wanting to return to sports soon after injury, a second injury in an already compromised area may be problematic. The athlete, athletic trainers, coaching staff, and physicians must be familiar with and aware of this troublesome injury pattern. Symptoms that should make any physician suspicious of compartment syndrome include extreme pain out of proportion, pain with passive muscle stretching, paresis, paresthesia, and diminished pulses. In patients who are unresponsive, uncooperative, unreliable, or in patients with equivocal findings, compartment measurements can be helpful in confirming the diagnosis of compartment syndrome. Once the diagnosis is made, immediate preparations need to be made for compartment decompression.

REFERENCES