[Orthopaedic Surgery]

Isolated Anterior Compartment Syndrome After a Bimalleolar-Equivalent Ankle Fracture in a Collegiate Football Player

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Compartment syndrome after an ankle fracture is an extremely rare and potentially devastating event. The authors report a case of an isolated anterior compartment syndrome in a college student athlete who suffered a bimalleolar ankle fracture dislocation. A review of the literature highlights the importance of vigilance when the sports medicine physician and the community orthopaedist are treating these seemingly basic orthopaedic injuries.

Keywords: Compartment syndrome, ankle fracture, bimalleolar, fasciotomy

Compartment syndrome is an extremely rare complication following isolated ankle fracture. It has been reported with both closed and open treatment of this injury.^{1,2,5,9-11,13,15,16} The majority of cases involve at least the deep posterior compartment. We report the delayed development of an isolated anterior compartment syndrome after operative treatment of a bimalleolar equivalent ankle fracture in a college athlete.

CASE REPORT

A 19-year-old college athlete weighing 135 kg (297 lb) with a medical history of hypertension injured his right ankle while playing touch football with friends. In the emergency department, he had a grossly deformed right ankle. Radiographic examination revealed a right ankle bimalleolar-equivalent fracture dislocation with obvious syndesmotic disruption (Figure 1). The patient underwent uneventful immediate closed reduction. The ankle was placed in a well-padded splint, iced, and elevated. Physical examination before and after reduction revealed normal dorsalis pedis and posterior tibial pulses, active flexion and extension of the toes, and normal sensation throughout. Pain was well controlled.

The following day (14 hours after injury), under general anesthesia, the patient underwent open reduction of the fibular fracture and placement of a syndesmotic screw through a standard lateral approach. Because of the patient's size, a 3.5-mm limited contact dynamic compression plate was used to stabilize the fibula; a 4.5-mm cortical screw was placed across 4

cortices to stabilize the syndesmosis (Figure 2). Tourniquet time was 90 minutes at 300 mmHg. Postoperative evaluation revealed normal sensation, good capillary refill, and a normal dorsalis pedis pulse. His pain was well controlled with occasional morphine sulfate.

Although initially cleared for discharge by the physical therapy team on postoperative day 1, the patient's pain waxed and waned. He required increasing doses of intravenous pain medication (hydromorphone), and discharge home was postponed for further observation. Physical examinations performed multiple times each day revealed indurated but soft leg compartments, no pain with passive flexion/extension of the toes, and normal sensory findings. The intermittent bouts of severe pain raised concern for compartment syndrome, but with the inconsistent subjective and objective findings, no intervention was deemed necessary.

In the overnight hours of postoperative day 5, the patient awoke in severe pain. Examination once again revealed an indurated but soft lower extremity, no pain with passive flexion or extension of toes, and normal sensory findings. Because of the severity of the patient's pain, bedside measurement of compartment pressures was performed. A handheld Stryker system (Kalamazoo, Michigan) revealed the following compartment pressures: anterior, 18 mmHg; lateral, 20 mmHg; superficial posterior, 15 mmHg; and deep posterior, 8 mmHg. The patient's blood pressure at this time was 127/55 mmHg.

On morning rounds of the same day, the patient's physical examination changed. He began to experience significant

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Figure 1. Anteroposterior (A) and lateral (B) radiographs demonstrating bimalleolar-equivalent ankle fracture dislocation.

Figure 2. Postoperative anteroposterior (A) and lateral (B) radiographs status post open reduction and internal fixation of the fibula with syndesmosis fixation.

pain with passive flexion of his toes, hyperesthesia over the anterior compartment, and a subtle decrease in sensation over the dorsum of his foot. It was also noted that his anterior compartment felt slightly firmer than it had during previous examinations.

The patient was taken emergently to the operating room 5 days after his initial surgery. After induction of general anesthesia, repeat compartment pressure measurements were taken using an arterial transducer system: anterior, 60 mmHg; lateral, 20 mmHg; superficial posterior, 50 mmHg; and deep posterior, 25 mmHg. Needle location was at the level from the fracture as it was for the earlier floor measurements. Blood pressure at the time was 140/67 mmHg. A standard 2-incision, 4-compartment fasciotomy revealed normal, nonbulging,

lateral, superficial, and deep posterior compartments. However, the distal two-thirds of the anterior compartment had dusky, noncontractile musculature. The proximal aspect of the tibialis anterior and extensor digitorum longus were pink, contractile, and bleeding. Serial irrigation and debridement of nonviable tissues were performed over the next 6 days, with gradual suture closure of the wounds. The viable proximal musculature and its distal tendons were salvaged in continuity. Final wound closure and split-thickness skin grafting of the distal lateral incision were performed on postoperative day 11/6. The patient was maintained in a neutral, removable posterior splint, and early range of motion was initiated.

FOLLOW-UP

Two years after his initial surgery, the patient is doing well. There was a postoperative, polymicrobial abscess within the anterior compartment early in his recovery that required incision and drainage and a course of intravenous antibiotics. He has a plantigrade foot, 5° of active dorsiflexion, and normal sensation. He is able to ambulate freely without use of assistive devices; he has no pain; and he plays flag football. Radiographic evaluation revealed a well-healed fracture.

DISCUSSION

Young Szalay and Roberts¹⁵ reported compartment syndrome following a Bosworth-type³ fracture dislocation, in which the distal end of the proximal fibular fracture becomes trapped behind the distal tibia. The patient developed increasing pain and paresthesia involving the dorsum of her foot within hours of surgery. Compartment pressures were measured and were normal in the lateral and deep compartments but markedly elevated in the anterior compartment. Fasciotomy was performed 6 hours after the onset of pain. Despite intervention, the patient developed contracture of the extensor hallucis longus, requiring further surgical intervention. Seyahi et al¹³ reported isolated anterior compartment syndrome that evolved 3.5 days following open reduction and internal fixation of a lateral malleolar fracture. The patient developed severe pain, pain with passive range of motion, anterior compartment swelling, dorsiflexion weakness, and sensory loss about the dorsum of the foot. Similar to this case, the result was anterior musculature necrosis requiring serial debridements and a stiff but plantigrade and functional ankle. Despite these more convincing presentations of compartment syndrome, isolated anterior compartment syndrome is a rare complication following an ankle fracture that may produce inconclusive physical examination results, including pain-free passive toe motion.

Upon further review of the literature, several reports of ankle fractures resulted in compartment syndrome involving multiple compartments or compartments other than the anterior compartment. Hawkins and Bays⁵ described acute compartment syndrome following cast treatment of a minimally displaced fracture of the medial malleolus. Despite emergency above-knee amputation, the patient developed

fatal rhabdomyolysis. Ashworth and Patel¹ described a young man with an unstable bimalleolar fracture dislocation treated initially with closed reduction and bivalved cast. Twenty hours after reduction, the patient developed altered sensation over the sole of his foot and pain with passive dorsiflexion of the great toe. Fasciotomy revealed a “very edematous” posterior compartment. Three months after surgery, neuromuscular examination results were normal. Joseph et al¹⁰ reported 2 cases of compartment syndrome following ankle fracture. All compartments were involved in both patients. Prompt recognition and early fasciotomy ensured that both patients regained full functional capacity without neuromuscular deficit. Zachariah et al¹⁶ reported a case of isolated lateral compartment syndrome 48 hours after open reduction and internal fixation of a Weber C fracture dislocation. Six weeks after 4-compartment fasciotomy, the patient had persistent hypesthesia over the dorsum of his foot and loss of terminal extension. Horne⁹ presented a case of late sequelae of what was probably a missed, isolated, deep compartment syndrome after an ankle fracture. Finally, Beekman and Watson² reported a case of delayed diagnosis of anterior, lateral, and deep posterior compartment syndrome following closed reduction of a Bosworth fracture-dislocation. This patient regained an active lifestyle but had sensory and motor deficits about the foot and ankle.

The present patient is an excellent example of the sometimes-reticent nature of compartment syndrome—in particular, isolated compartment syndrome. While this was a sports injury, his large size resulted in a high-energy external rotation injury to the syndesmosis and anterior compartment. His size made the physical examination difficult, as did the particular compartment involved. Passive flexion of the toes in a neutrally splinted ankle did not initially cause pain, and passive flexion of the ankle is not possible in the postoperative ankle fracture. Because the posterior compartments were not involved, passive extension was not painful, giving the examining physicians a false sense of security.

Luk and Pun¹¹ reported a case of missed compartment syndrome in a patient who also had dense tourniquet palsy after prolonged surgery for an ankle fracture. The patient had a tourniquet inflated to 450 mmHg in place for 3 hours 15 minutes, with 25 minutes of deflation after 2 hours. Postoperatively, the patient had significant motor and sensory loss, which was thought to be due to the tourniquet. The latter likely contributed to a missed compartment syndrome. This patient's tourniquet was in place for only 90 minutes and at 300 mmHg. He was neurovascularly intact postoperatively, and we do not believe that the tourniquet contributed to his isolated compartment syndrome. It is possible, however, that a significantly traumatized anterior compartment was more vulnerable to this duration of ischemia and further injury ensued.

This patient had familial hypertension, possibly exacerbated by his obesity. The hypertension, though mild, may have had

Figure 3. Diastolic blood pressure (DBP) vs time, demonstrating mild hypertension and a precipitous drop in diastolic blood pressure on November 12, 2008, coinciding with a change in the patient's physical examination findings and the decision to perform fasciotomy.

a protective effect on the involved musculature and deep peroneal nerve in the first 4 days following his initial surgery (Figure 3) while contributing to the vacillating clinical picture. Indeed, studies by Heckman et al⁶ and Heppenstall et al^{7,8} have documented that hypertension may exert a protective effect on underlying muscle in the presence of a developing compartment syndrome by supporting a normal pressure gradient (ΔP = diastolic pressure – compartment pressure). This is particularly true for traumatized muscle.⁸ Hypotension, however, may have a deleterious effect on a potential compartment syndrome.¹⁴

It is unclear when the compartment syndrome commenced and why the proximal musculature was spared. Delayed presentation of compartment syndrome has been reported as late as 5 days.^{4,16} It is possible that the compartment syndrome was still evolving, perhaps protected by mild hypertension.

This may have been a focal compartment syndrome, where the entire compartment was not involved. In support of this hypothesis is Matava et al's description of a nonuniform pressure gradient within a compartment in a canine model¹² (in contrast to a commonly held belief that pressure equilibrates throughout an enclosed compartment). Matava et al theorized that the relatively compliant proximal muscle tissue, with its loose fascial boundaries, allows for this “isolated” area of muscular compromise. A focal, isolated compartment

syndrome would be more difficult to diagnose. This highlights the inexact, sometimes subtle clinical picture of compartment syndrome.

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