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Statement of Purpose

We discuss a case involving elevated compartment pressures discovered 5 days after open reduction internal fixation (ORIF) of a calcaneal fracture. Although surgical intervention was performed, the patient still went on to develop contractures. Our goal with this study is to make readers aware of the delayed development of compartment syndrome (CS) and the complications associated with comminuted calcaneal fractures.

Literature Review

Up to 10% of calcaneal fractures can be complicated by delayed onset CS with high or low impact injuries¹. Patients often present initially with severe pain and edema, which may cause the symptoms of CS to be overlooked. It is important to understand that CS may not be present initially, rather developing a few days post-injury. CS, first described by Richard VonVolkman in 1872, is a podiatric emergency and is associated with fractures 69% on the time¹. Pressures > 30 mmHg or within 10-30 mmHg of the diastolic blood pressure is considered diagnostic. Of the 9 compartments described by Manoli, the calcaneal compartment tends to have a slightly higher pressure in general than the other compartments². A high degree of suspicion should be exercised in patients with calcaneal fractures classified as Sanders type 3 or 4 since more comminution causes greater soft tissue damage and larger hematoma³. It is possible that further trauma to the tissue from manipulation and continued bleeding from cancellous bone of the calcaneus may cause elevated compartment pressures. Early detection is critical since patients with missed CS diagnoses have been shown to have significantly lower functional outcome than those without. Long term complications include claw toes, loss of function, pain, muscle atrophy, and contracture³. Volkmann's contracture results when prolonged ischemia causes myonecrosis, fibrosis, adhesions, and nerve injury lead to a variety of weakness and contracture depending on which muscles and nerves are affected⁴.

Figure 1A & 1B



1A: Lateral view radiograph reveals comminuted displaced calcaneal fracture. 1B: CT confirms intra-articular involvement Sanders 2C.

Case Study

A 35 year old male construction worker with no past medical history of fell off a scaffolding while at work. The patient noticed immediate pain and swelling and was unable to bear weight on his left foot. Upon presentation to our Emergency department, the patient was rating his pain as 10/10 to the left heel. His neurovascular status was intact and there were no signs of compartment syndrome at this time. Minimal ecchymosis was noted to plantar medial arch and the patient did not have any open wounds. Non-weight bearing radiographs revealed a comminuted calcaneal fracture with a critical angle of Gissane of 150 degrees and Bohler's angle of 4 degrees (Figure 1A). For surgical planning, a CT scan was obtained which revealed an intra-articular comminuted calcaneal fracture classified as Sanders 2C (Figure 1B). Within 24 hours, the patient was taken to the operative room for ORIF of his calcaneal fracture under general anesthesia. The patient was placed on the operating room in a supine position and a lateral extensile incision was made along the heel. Dissection was taken down to bone and the fracture fragments were visualized. Manual distraction with a Steinmann pin through the tubercle was performed to assist with reduction. The fragments were then fixated with an 8-hole calcaneal plate and eight 3.5 mm screws (Figure 2). The area was closed with 3.0 Vicryl and staples.

Figure 2



Lateral radiograph showing plate and screws for fixation of the fracture fragments with good alignment.

Five days later, the patient returned to the ER with swelling and uncontrollable pain, even after administration of IV pain medication (Figure 3A). A fracture blister was now noted to the medial ankle and pulses were not palpable. He also related to numbness and tingling in his toes with pain upon passive stretch. The patient was emergently taken back to the operating room where compartment pressures were measured (Figure 3B). Compartment pressures were 73 mmHg in the medial calcaneal compartment and 45 mmHg in the interosseous compartment so fasciotomies were performed (Figure 4A & 4B). Serousanguinous fluid was expressed, the area was irrigated, and drains were placed in the incision sites.

3 months later, the fracture blister persisted and became erythematous (Figure 5A). The patient was taken back to the OR for irrigation and debridement. Upon incision, hematoma was noted in the area so it was evacuated with manual compression (Figure 5B & 5C). A non-pulsating bleeding vessel was noted so this was ligated and hemostasis was confirmed by deflating the tourniquet. A skin plasty was finally performed to primarily close the wound.

Results

Approximately 11 months after ORIF, the patient's wounds were healed; however, he developed Volkmann's contractures with a cavovarus foot structure (Figure 6A & 6B). The deformity was created by peroneal weakness with overpowering of the tibialis posterior. In the midst of contracture, at 24 months post-operatively, the patient's deformity was asymptomatic and he was ambulating in orthotics for support. He returned to work and was able to perform his normal daily activities.

Figure 3A & 3B



Figure 3A: Edema noted to surgical foot compared to the non surgical foot.

Figure 3B: Intra-operative compartment measurements

Figure 4A & 4B

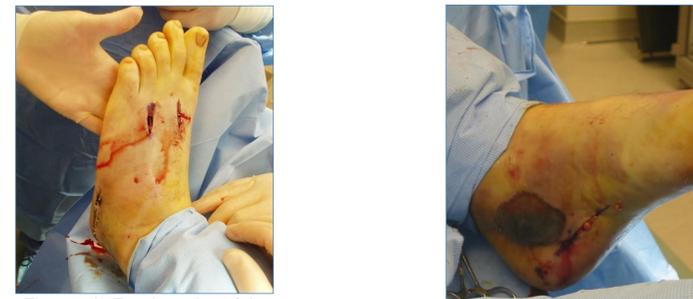


Figure 4A: Fasciotomies of the interosseous compartment.

Figure 4B: Plantar medial fasciotomy to decompress the calcaneal compartment.

Figure 5A & 5B



Figure 5A: Inflamed hemorrhagic fracture blister.

Figure 5B: Hematoma evacuation from blister area.

Figure 5C: Remaining defect after hematoma evacuation.

Discussion

In our patient, the combination of sudden onset of severe pain unrelieved by pain medication, numbness and tingling, and non-pitting edema led to a high index of suspicion for CS five days after ORIF. Presentations of delayed compartment syndrome such as this, may be attributed to severe edema or hematoma formation that develops after the injury. If a patient is taken to the OR shortly after the initial injury, hematoma and edema may not have had a chance to develop. Calcaneal fractures involving the sustentaculum tali, such as our patient's Sanders 2C fracture, have the potential to disrupt the medial calcaneal arteries and cause bleeding in the compartment⁵. What was thought of as a hemorrhagic fracture blister was actually a large, deep hematoma that may have put further pressure on the area. Also, fibrosis and scarring from the patient's previous calcaneal injury may have left the compartment with less potential space to accommodate edema⁵. Findings of pain, pallor, paresthesias, paralysis, pulselessness, and with pain out of proportion on passive stretch typically are delayed findings, and it was difficult to differentiate these symptoms in our patient due to his acute fracture status². It was not until 5 days post-operatively, when the typical late findings of CS presented, that we suspected CS. We applied the gold standard for diagnosis and treatment by performing intra-compartmental pressures with decompression fasciotomies; however, our patient still developed Volkmann's contracture post-operative. Thus, early detection is critical to patient outcomes and even with surgical treatment, one may still see long term sequela.

Figure 6A & 6B

Figure 6A: Evident varus deformity on radiograph.

Figure 6B: Volkmann contracture with varus deformity on the left foot.



References

References:
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