Cracking Under Pressure

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Paul's Case

Paul is a 35-year-old male who was brought to the Emergency Department (ED) by ambulance following a rollerblading fall. The patient states that he lost control while rollerblading along a local river path and travelling down a short embankment. He states that he sped into a grassy area at the bottom of the hill. He fell to the ground, and his left ankle was forcefully externally rotated. He was unable to stand and could not bear weight on his ankle. Paul sustained no other injuries and is otherwise healthy. He takes no prescription medications and has no allergies. Emergency Medical Services (EMS) arrived on the scene shortly after the fall and rapidly transported the patient to the hospital. He received intravenous opioid analgesia (morphine 10 mg IV) during transport.

On examination in the ED, the patient is in mild distress, complaining of pain to the injured limb. Edema and soft tissue swelling of the left lower leg are noted. Pedal pulses are present and the distal neurological exam is normal. There are no breaks to the skin integrity. Plain radiographs of Paul's left leg reveal a comminuted fracture of the tibia and a mid-shaft fracture of the fibula (Figure 1). Paul is fitted with a plaster splint for immobilization of the fracture.

Two hours following the fall, Paul is complaining of increased pain in his right leg. He states that the pain is quickly becoming intolerable. Is the analgesia wearing off or is something else going on?

Read on for more on Paul.

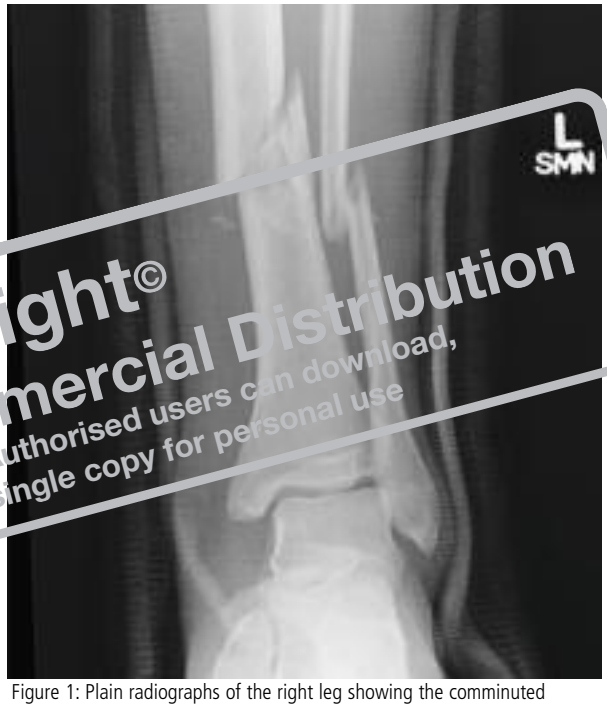

Figure 1: Plain radiographs of the right leg showing the comminuted fracture of the distal tibia and fibula.

Questions and Answers

1. *Is the fracture causing all of this pain?*

In all cases presenting with musculoskeletal trauma, physicians must be vigilant for compartment syndrome (CS). CS can occur anywhere in the body where fascia, connective tissue, or bone rigidly confine organs and vessels in an area of fixed volume. This syndrome manifests itself clinically when pressures rise high enough within the specific body compartment to cause ischemic damage leading to organ/tissue dysfunction. Common areas prone

to the development of CS include the abdomen, mediastinum/thorax, skull, and the extremities (particularly the forearm and lower leg).

The most widely accepted explanation for the pathophysiology of CS is the arterio-venous pressure (AVP) gradient theory, which can be explained as follows: 1) initially, there is a rise in the compartment's interstitial pressure, which is directly associated with bleeding or edema from the primary injury; 2) as the compartment pressure rises, venous drainage decreases and so does the AVP gradient; 3) capillary blood-flow to the surrounding tissues decreases and ischemia begins; and 4) finally, anaerobic metabolites accumulate and tissue death occurs, promoting more edema and further increases in compartment pressure. The final step closes the loop on this vicious cycle (Figure 2) and the clinical manifestations of CS become apparent.

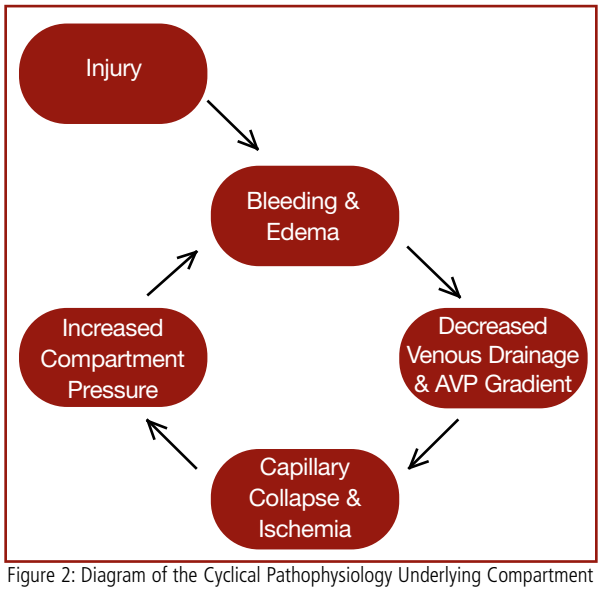

Figure 2: Diagram of the Cyclical Pathophysiology Underlying Compartment Syndrome. AVP = Arterial Venous Pressure Gradient.

2. How is CS diagnosed in an extremity?

A high index of suspicion is the most important tool a physician has for diagnosing CS. The most reliable clinical indicators of CS are pain that is out of proportion to the injury and pain that occurs when passively stretching the affected soft tissues. Moreover, a deep aching pain that is largely unresponsive to appropriately dosed analgesics should raise suspicion for evolving CS.

Traditionally, the 5 P's (pain, paraesthesia, pallor, pulselessness, and paralysis) have been described as clinical red flags for the development of CS. The sensitivity and specificity of these physical exam findings has been questioned. When present, the clinical findings indicate that CS has become entrenched and that tissue necrosis has started to occur. These are generally late findings in musculoskeletal trauma.

Direct compartment pressure measurement should be considered in patients at high risk of developing CS. The following features have been identified as potential risk factors for developing CS: high energy trauma, an unconscious patient, bleeding diatheses, forearm or low leg fractures, crush injuries, and males < 35 years of age. Invasive measurement of compartment pressure should be performed in the unconscious trauma patient and in those conscious patients where the clinical picture is still worrisome. An absolute compartment pressure > 30 mmHg, or a perfusion pressure (diastolic BP – compartment pressure) < 30 mmHg are indications for urgent fasciotomy. Detailed documentation of physical exam findings and serial pressure measurements should be done early and often to determine therapy and ensure adequate medical record.

3. What can be done in the ED?

The primary role of an ED physician is recognition of CS in a timely manner. If CS is

suspected consultation with an Orthopedic surgeon is warranted for emergent fasciotomy. The affected limb should be kept in neutral position. Elevation of the limb is controversial, with some authors advocating the dependent position to maximize perfusion pressure. Assessment of patients should include the removal of all compressive bandages and circumferential casts.

If these measures fail to stabilize or reduce the compartmental pressure, urgent fasciotomy should be performed to prevent potentially serious and permanent injury to the nerves and muscles of the compartment.

4. *What would happen if a fasciotomy were not performed?*

The natural history of untreated CS can be threatening to both life and limb. The two structures most affected by the rise in compartment pressure are muscles and nerves. As pressure in the compartment exceeds perfusion pressure of the muscle, anaerobic metabolism begins and the muscle tissue becomes necrotic. Necrotic tissue is not salvageable and eventually fibroses. Necrotic muscle is also a risk factor for the development of infection, which can lead to limb amputation or sepsis. Muscle cell death leads to elevated serum levels of creatine kinase (CK). When CK levels are markedly elevated, rhabdomyolysis is present. The circulating CK is a nephrotoxic substance that can induce renal failure. Adequate intravascular volume expansion with crystalloid solutions is the mainstay of treatment.

The peripheral nerves coursing through the pressurized compartments are also damaged by ischemia and physical compression. It is believed that complete nerve blockage occurs at an absolute intracompartmental pressure of 50 mmHg and that the likelihood of permanent paralysis goes up the longer the nerve is compressed within the compartment. As a general rule, a nerve compressed in a compartment with an absolute pressure of 30 mmHg for six to eight hours will incur a permanent conduction deficit. Dx

Back to Paul

Paul's splint was removed and his leg was positioned on supportive pillows. His clinical assessment revealed pain on passive stretching and a new dose of analgesia was not effective in relieving his pain. Compartmental pressures of his lower leg were evaluated and found to be 27 mmHg. The clinical diagnosis of CS was made. An urgent consultation to the orthopedic surgeon on-call was initiated. The patient was promptly taken to the operating theatre and underwent emergent fasciotomy.

Resources
1. Gourgiotis S, Villias C, Germanos S, et al: Acute Limb Compartment Syndrome: A Review. J Surg Educ (2007); 64(3):178–186.
2. Frink M, Hildebrand F, Krettek C, et al: Compartment Syndrome of the Lower Leg and Foot. Clin Orthop Relat Res (2010); 468(4):940–950.
3. Balogh ZJ, Butcher NE. Compartment Syndromes from Head to Toe. Crit Care Med (2010); 38(9):445–451.

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