Cardiac Arrest Caused by Reperfusion Injury after Lumbar Paraspinal Compartment Syndrome

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Reperfusion of ischemic tissue is associated with a characteristic pattern of cellular injury that can result in serious systemic and local effects. Ischemia-reperfusion syndrome (IRS) has been documented in a number of clinical situations, including the release of tense fascial compartments in the extremities. Although the lumbar paraspinal musculature is susceptible to fascial compartment syndrome, there are no reported cases of a paraspinal compartment syndrome initiating ischemia-reperfusion injury.

We report a case of postoperative cardiorespiratory arrest and multiple organ failure in a 43-year-old woman who underwent operative release of a tense paraspinal compartment after a two-story fall. Her premorbid health was good, and investigations failed to show a cause for her arrest. We conclude that her decompensation was caused by ischemia-reperfusion syndrome. The pathophysiology of this phenomenon is discussed.

CASE REPORT

A 43-year-old schizophrenic woman jumped from a second-story window. She did not lose consciousness and was stable in transit. On arrival at the emergency department, she was floridly psychotic but complained only of shoulder pain. On examination, she had a broken clavicle but no other sign of thoracic trauma. There was no respiratory distress, and breath sounds were normal. Oxygen saturation was excellent on room air. Cardiac examination was normal. There was no abdominal distention or tenderness, and bowel sounds were normal. Chest radiograph showed a broken clavicle but was otherwise normal, and a pelvic film showed no pelvic injury. The patient was discharged from the emergency department.

Two days later, the patient presented once again to the emergency department. Her care givers reported that she was lethargic and was not voiding or moving her bowels. She denied dyspnea, chest pain, or palpitations. Pulmonary and cardiac examinations were normal. Her abdomen was markedly distended and tympanic, with mild diffuse tenderness. Perianal sensation and sphincter tone were normal. Bladder catheterization produced 2 L of clear urine. A computed tomographic scan of the abdomen showed a burst fracture of the second lumbar

vertebrae as well as colonic dilatation. Serum electrolytes were within normal limits.

She was admitted to the hospital and underwent computed tomography-guided needle decompression of her cecum, and ileus resolved. On her 5th hospital day, she underwent posterior spinal fusion. At the time of surgery, it was noted that the paraspinal muscle compartment was tense. When the compartment was opened, dusky, poorly perfused muscle herniated into the wound. The remainder of the procedure was uneventful, except for a transient decrease in systolic blood pressure to 75 mm Hg, which responded to crystalloid and packed cell transfusion. It was believed at the conclusion of the procedure that the paraspinal muscles were viable. Fifteen minutes after arrival at the postanesthesia care unit, she was noted to have a weak, thready pulse of 90 beats per minute, with a systolic blood pressure of 100 mm Hg. Her serum potassium was 7.2 mEq/L, and serum bicarbonate was 16 mEq/L. Arterial blood gas analysis showed a serum pH of 7.124, with a base deficit of -15.5 mmol/L She was given crystalloid fluid and sodium bicarbonate and was transferred to the intensive care unit. Repeated measurements showed persistent acidosis and hyperkalemia. Three hours postoperatively, her systolic blood pressure decreased to 75 mm Hg and she began to have periods of ventricular tachycardia. Approximately 5 hours postoperatively, she became profoundly hypotensive and progressed rapidly from bradycardia to asystole. She was intubated, advanced cardiac life support protocol was initiated, and after 20 minutes of chest compressions, she recovered a spontaneous sinus rhythm.

For the next 3 days, the patient required dopamine and epinephrine to maintain a mean blood pressure of 60 mm Hg, despite pulmonary capillary wedge pressures of 16 to 20 mm Hg. She required mechanical ventilation for 7 days. Her chest radiographs demonstrated mild bilateral fluffy infiltrates, and 70% inspired oxygen with 10 cm H_2O of positive end-expiratory pressure produced a PaO_2 of 106 mm Hg. She became severely coagulopathic, with a prothrombin time of greater than 35 seconds, a partial thromboplastin time of 55 seconds, plasma fibrinogen of 119 mg/dL, and elevated D dimers. Her platelet count was 39,000/mm^3. She manifested oliguric renal failure, with peak blood urea nitrogen and creatinine of 116 and 8.5 mg/dL, respectively, and required daily dialysis for 1 month. Repeated sets of blood cultures were negative. Electrocardiogram and pulmonary ventilation/perfusion scan were normal. Serum creatine phosphokinase reached a peak of 10,581 IU/L, but MB index was negative. Serum lactate was 6.3 standard IU. Adrenocorticotropic hormone stimulation testing showed normal adrenal function. She eventually recovered renal function, had complete resolution of cardiovascular, coagulative, and respiratory function, and was discharged in satisfactory condition on postoperative day 39.

DISCUSSION

The ill effects caused by reperfusion of ischemic tissue have come under increasing scrutiny by cardiac, vascular,

and transplant surgeons and are also recognized as an important cause of local and systemic complications after crush injury and compartment syndromes. [1] Reperfusion injury has been shown to contribute to the failure of transplanted and revascularized organs [2] and to exacerbate muscle damage after extremity trauma. [1,3] Furthermore, the damage is not limited to the reperfused tissue, and reperfusion of ischemic extremities is associated with serious systemic complications.

At the organ level, reperfusion may cause tissue damage or may exacerbate the effects of ischemia. Xanthine concentrations can increase during periods of ischemia because xanthine cannot be broken down in the absence of oxygen, and when tissue is reperfused xanthine oxidase catalyzes a "burst" of toxic oxygen species production as xanthine is oxidized. These oxygen species then react with endothelial cells to increase the production of cytokines and adhesion molecules, which can further contribute to organ dysfunction. [4] Studies with a feline intestinal model showed more organ damage with 3 hours of ischemia plus 1 hour of reperfusion than with 4 hours of ischemia alone. [5] Several studies have shown that reperfusion with anoxic blood lessens tissue reperfusion damage, suggesting that oxygen itself may precipitate at least some of the injury. [6]

The systemic consequences of IRS result from washout of blood from the ischemic tissue into the systemic circulation. This blood is frequently low in pH and high in potassium. [7] It may also be rich in bradykinin, lactate, myoglobin, and prostaglandins as well as activated leukocytes. In addition to these mediators of tissue damage, there may also be a neural component to the vascular changes in IRS. [8] The clinical consequences of IRS include renal failure, [8-10] lung injury, [11] and sudden death. [12]

Lumbar paraspinal compartment syndrome was first described in a 1985 case report of a young man with postexertional back pain. [13] The diagnosis was made on the basis of myoglobinuria, elevation in serum creatine phosphokinase, and computed tomographic findings suggestive of ischemia and necrosis. The authors subsequently demonstrated with cadaveric dissection and pressure measurements in healthy volunteers that the paraspinal muscles are ensheathed in a fascial envelope that is anatomically and physiologically similar to other muscle compartments that are known to be susceptible to compartment syndrome. Subsequent studies have shown a possible relationship between paraspinal compartment pressures and lower-back pain. [14-16] A case has been reported of rhabdomyolysis resulting from lumbar paraspinal compartment syndrome in a 20-year-old man. In this case, T1-weighted and T2-weighted magnetic resonance images showed hemorrhagic necrosis in paraspinal muscles. [17]

IRS after paraspinal compartment syndrome has not been reported. In this case, the diagnosis was based on the intraoperative visualization of a tense paraspinal compartment, followed by an unexplained hemodynamic collapse after release of the compartment and subsequent multiple organ failure. Based on this case,

consideration should be given to the potential for a lumbar paraspinal compartment syndrome after lumbar trauma, particularly when decompression is delayed, and to the possibility of systemic reperfusion injury after compartment release.

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