The Journal of Emergency Medicine, Vol. 46, No. 2, pp. 313–319, 2014

0736-4679/$ - see front matter

ELSEVIER

http://dx.doi.org/10.1016/j.jemermed.2013.08.052

Clinical Reviews

CRUSH SYNDROME: A CASE REPORT AND REVIEW OF THE LITERATURE

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□ **Abstract—Background: Crush trauma to the extremities, even if not involving vital organs, can be life threatening. Crush syndrome, the systemic manifestation of the breakdown of muscle cells with release of contents into the circulation, leads to metabolic derangement and acute kidney injury. Although common in disaster scenarios, emergency physicians also see the syndrome in patients after motor-vehicle collisions and patients "found down" due to intoxication. Objective: The objectives of this review are to discuss the pathophysiology of crush syndrome, report on prehospital and emergency department treatment, and discuss the relationship between crush syndrome and compartment syndrome. Discussion: We present the case of a young man found down after an episode of intoxication, with compartment syndrome of his lower extremity and crush syndrome. Although he eventually required an amputation, aggressive fluid resuscitation prevented further kidney injury and metabolic derangement. Conclusions: Early, aggressive resuscitation in the prehospital setting, before extrication if possible, is recommended to reduce the complications of crush syndrome. Providers must be aware of the risk of hyperkalemia shortly after extrication. Ongoing resuscitation with i.v. fluids is the mainstay of treatment. Compartment syndrome is a common complication, and prompt fasciotomies should be performed when compartment syndrome is present.** © 2014 Elsevier Inc.

□ **Keywords—rhabdomyolysis; crush syndrome; renal failure; resuscitation; hyperkalemia**

INTRODUCTION

Crush trauma to the extremities, even if not involving vital organs, can be life threatening. The term *crush injury* refers to the damage resulting directly from the crushing force. Conversely, crush syndrome, also known as traumatic rhabdomyolysis, is the systemic manifestation of the breakdown of muscle cells with release of contents into the circulation (1,2). Crush syndrome leading to acute kidney injury (AKI) is one of the few life-threatening complications of crush injuries that can be prevented or reversed (3).

Crush syndrome was first described after the Battle of London by Bywaters and Beall in 1941. Patients pulled from the rubble initially appeared to be unharmed, but then these patients developed progressive limb swelling and shock and died of renal failure a few days later (2). Postmortem examination revealed muscle necrosis and brown pigment casts in the renal tubules (4). Crush injuries are common in natural disasters such as earthquakes, but emergency physicians more commonly see the syndrome in patients after motor-vehicle collisions, especially with prolonged extrications, as well as in victims of assault (5,6). Crush syndrome also occurs in patients who compress a part of their own body, such as patients "found down" due to a stroke, intoxication, or mental illness (1). Any condition that results in prolonged

RECEIVED: 23 February 2013; FINAL SUBMISSION RECEIVED: 19 May 2013;
ACCEPTED: 15 August 2013

immobility can result in a crush injury (4,7). In the United States, heroin is a common etiology and alcohol has been found to be the most common etiology of crush syndrome, compartment syndrome, and rhabdomyolysis in many industrialized countries (5,7–12). Patients might regain consciousness within several hours, but due to pain in limbs are unable to get up off the floor, leading to ongoing compression.

CASE REPORT

A 23-year-old male with a history of bipolar disorder and polysubstance abuse was brought into the emergency department (ED) by emergency medical services (EMS) after being found down at home. EMS reported that no one had seen the patient for nearly 24 h when his mother came home and found him lying on the floor in the kitchen. He was lethargic and confused with a Glasgow Coma Scale score of 13. He was lying on his left side, with his left lower extremity curled underneath his body. He was boarded, collared, and brought to the ED.

In the ED, he complained of pain in his left leg, but he was unable to provide any history. His vital signs were a temperature of 37.2°C, heart rate of 150 beats/min, blood pressure of 150/70 mm Hg, respiratory rate of 16 breaths/min, and an O_2 saturation of 99% on room air. His physical examination was notable for ecchymosis around his left orbit and numerous areas of skin breakdown on his left chest and abdomen. His left lower extremity had a noncircumferential macerated disruption to the skin on the posterior-lateral aspect, where it had been in contact with the floor, with surrounded blistering that appeared similar to a burn. The leg was cold with mottling, and the compartments of the lower leg were all tight to palpation. No pulses or capillary refill could be appreciated.

His laboratory results were notable for a white blood cell count of 26,000 cells/μL, hemoglobin of 19.4 g/dL, hematocrit of 59.3%, and platelets of 183,000/mm^3. His sodium was 132 mmol/L, potassium was 5.4 mmol/L, chloride was 105 mmol/L, bicarbonate was 14 mmol/L, blood urea nitrogen was 22 mmol/L, creatinine was 1.4 μmol/L, glucose was 128 mmol/L, and lactate was 2.8 mmol/L. The patient's toxicology screens, including ethanol, were negative. The patient's initial creatinine kinase (CK) was 41,669 IU/L. His head computed tomography (CT) and C-spine CT were negative for any acute injury or pathology.

The trauma team placed two large-bore i.v. lines and the patient was started on 2 L of 0.9% saline boluses. He was taken emergently to the operating room from the ED for fasciotomies of his left lower extremity.

After the procedure, the patient remained intubated and was admitted to the intensive care unit. After the fasciotomies, his left lower extremity pulses returned and the leg was monitored closely by the surgery department. He continued to receive aggressive fluid hydration at 200–500 mL/h to maintain a urine output of at least 200 mL/h, and he required a norepinephrine infusion to maintain a mean arterial pressure > 65 mm Hg. The following day, the patient's CK peaked at 50,867 IU/L. His urine myoglobin was checked, with a level of 32.9 μg/mL (reference range: < 0.025 μg/mL). With continued aggressive i.v. fluids his creatinine trended down during the following 3 days to 0.64 μmol/L. On hospital day 3, while he continued to receive high-volume fluid resuscitation, pulmonary edema developed. The i.v. fluids were reduced to 100 mL/h, mannitol and Lasix were added to maintain his urine output, and his ventilator was managed with low tidal volume ventilation for lung protection. The pulmonary edema resolved over the following day.

Despite the initial return of pulses with fasciotomy, the patient's left lower extremity suffered extensive soft-tissue damage and ischemia. He underwent a below-the-knee amputation (BKA) on hospital day 4. Shortly after the BKA, his CK dropped markedly, the shock improved, and aggressive fluid resuscitation was stopped. He was extubated the following day. The patient was not able to recall how he came to be lying on the floor, but did recall drinking a significant amount of alcohol the night before.

The patient was discharged to a rehabilitation hospital on post-injury day 20. He has since followed up in surgical clinic and has been doing well in physical therapy, learning to ambulate with his prosthesis.

DISCUSSION

The mechanism of injury and cell death in crush syndrome comes from the compression of the muscle fibers. In addition to the direct trauma of the compression, the tissue is deprived of blood flow and becomes ischemic, with both mechanisms causing lysis of muscle cells, leading to significant metabolic imbalance and eventual organ failure (13). The times to cellular injury and death vary with the crushing force involved. Skeletal muscle can generally tolerate up to 2 h of ischemia without permanent injury. However, at 4–6 h, tissue necrosis develops (13). At the cellular level, a crush insult opens stretch-activated channels in the muscle cell membrane and disrupts the Na/K transporter, allowing calcium to move freely into the cell. The increased intracellular calcium stimulates the activity of intracellular proteases, leading to eventual breakdown of the cell (5). Restoration of circulation to the damaged area results in ischemia-reperfusion injury. The post-ischemic tissues have high concentrations of neutrophil chemoattractants, leading to activation of neutrophils with release of proteolytic enzymes and generation of free radical superoxide anions once perfusion is restored (5,13).