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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

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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).

Cardiovascular Effects

Once the external pressure is released, cellular contents, including potassium, phosphorous, and urate, are released into the circulation and accelerate metabolic derangements. Reciprocally, the breakdown of cell walls allows calcium and sodium to rush into the cell, leading to hypocalcemia and hyponatremia. The immediate life-threatening finding of crush syndrome for many patients is hyperkalemia leading to dysrhythmias, often developing < 1 h after extrication (14).

Patients frequently develop shock in the first few hours after extrication, likely with a significant hypovolemic component (5). The etiologies of hypovolemic shock are numerous, including lack of oral intake, hemorrhage from associated injuries, and profound third spacing. With release, intravascular fluids flow into the intracellular compartments, and the injured area begins to sequester large volumes of fluids. Limbs can hold up to 12 L of fluid in their large compartments. Distributive shock can also develop from the release of inflammatory mediators due to reperfusion injury (2). Hypovolemia can then lead to further ischemia and cell death. Acidemia often accompanies these metabolic derangements, due in large part to ongoing hypoperfusion.

Renal Effects

Although dysrhythmias are the most immediate concern in crush syndrome, renal effects are the most serious complication. The catastrophe of the Armenian earthquake in 1988 was recognized as a "renal disaster" because crush syndrome was found to be the leading cause of death for those who reached medical care (3,4). The initial injury to the kidney before extrication is largely due to decreased circulating blood volume exacerbated by third spacing of fluid into the injured limb.

Additionally, release of myoglobin into the systemic circulation contributes to further kidney injury. The heme protein found in myoglobin has several nephrotoxic effects. First, the nitric oxide-scavenging effect of myoglobin causes vasoconstriction, exacerbating a prerenal state (5). The heme also leads to direct cytotoxicity, as the iron in myoglobin likely catalyzes the formation of more free radicals, leading to intrarenal failure, especially in the proximal tubule (5). Lastly, the kidneys readily filter the myoglobin and it precipitates with Tamm-Horsfall proteins in the tubules, leading to intraluminal cast formation, as seen in the original autopsies of crush syndrome patients (2,15). These tubular casts lead to obstruction and are hypothesized to increase intraluminal pressure, thereby decreasing glomerular filtration, although this is not completely supported by animal models (5,15).

Although myoglobin is responsible for the damage to nephrons, CK levels are commonly followed as a marker of muscle damage. CK begins to rise within 12 h of the injury and has a half-life of 1.5 days, peaking in 1–3 days, and CK concentrations can reach levels > 30,000 U/L. Once the CK has reached a level of > 5000 U/L, the patient has a nearly 20% chance of developing AKI (15,16).

Respiratory Effects

Patients with crush injuries are at risk for acute respiratory distress syndrome (ARDS) from inflammatory mediators (17,18). Additionally, during the course of treatment of both the renal failure from rhabdomyolysis and shock, patients often receive very large volumes of crystalloid, increasing the risk for pulmonary edema. Lastly, crush injuries can be complicated by long-bone fractures, and these patients may develop fat emboli syndrome (6).

Hematologic Effects

Due to systemic inflammation, crush syndrome may be associated with disseminated intravascular coagulation (DIC), possibly due to increased tissue thromboplastin levels. Patients may present with depressed platelet levels because of disseminated intravascular coagulation–related consumption (5,18). Coagulation factor synthesis can also be reduced as a consequence of the development of shock liver (19).

Infection

Patients are also at risk for subsequent infections and sepsis, especially when patients require fasciotomies as treatment for compartment syndrome, or in the context of poor nutritional status (10,18). In a study conducted after the Wenchuan county earthquake, 26 of 58 patients with severe crush syndrome developed severe sepsis, with open fasciotomies, duration of renal impairment, and time under the rubble, but not age, to be factors associated with the development of sepsis (20). In this study and the study by Kazancioglu et al., wound inoculation was the most common source of infection, with pseudomonal and acinetobacter organisms being commonly isolated (20,21).

Compartment Syndrome

Once the external force is released and the limb begins to swell, the pressures in noncompliant compartments may exceed perfusion pressures, leading to compartment syndrome. Compartment syndrome is a distinct clinical entity from crush injury and crush syndrome, although the

conditions are closely related and often co-exist. Compartment syndrome refers to loss of perfusion due to increased pressures within a closed space, leading to muscle ischemia (10,22,23). Normal compartment pressures are < 10–15 mm Hg (5,23). Compartment pressures of 30 mm Hg or a difference between diastolic pressure and compartment pressure of < 30 mm Hg leads to critical ischemia of the tissues (2,5). Compartment syndrome leads to additional ischemia of the limb, resulting in increased muscle breakdown, with release of more myoglobin and potassium into the circulation.

Prehospital Treatment of Crush Syndrome

The initial presentation of the patient with crush syndrome may be benign, as conscious patients might only complain of extremity pain. Immediately upon release from entrapment, the physical examination can range from only mildly ecchymotic or erythematous extremities, to diffusely swollen, tense limbs with skin breakdown (4).

Upon extrication of a previously stable patient, the systemic exposure to the metabolic milieu created by the crush syndrome causes a rapid clinical deterioration unless appropriately combatted (4). First responders and prehospital providers should be aware of the risk for development of hyperkalemia with extrication and during transport. Rapid rise in serum potassium levels can lead to electrocardiograph (ECG) changes and eventually ventricular fibrillation or asystole, if untreated. Dysrhythmias can be further exacerbated by rapid development of lactic acidosis and calcium shifts (17). Prompt ECG monitoring is therefore a higher priority in crush patients than in other trauma patients (4).

The most critical therapy in crush syndrome is early and aggressive fluid resuscitation with 0.9% saline to restore circulating blood volume. Traditionally, patients were treated with i.v. fluids several hours after arrival at the hospital. However, more recent practices of starting i.v. fluid resuscitation at the scene, even before extrication, have demonstrated improved outcomes, including reducing the need for renal replacement therapy (RRT) and mortality (24). A report of early field hydration of patients trapped by the Bingol, Turkey earthquake before extrication found that their small group of patients had a 25% need for RRT as compared with the need for dialysis in 60% and 77% of the patients in the Kobe and Marmara earthquakes, respectively, in whom early i.v. fluid was not started (24). An initial rate of 1–1.5 L/h is suggested, which can be adjusted depending on the clinical status of the patient (3,4).

Although tourniquets have been considered to isolate areas of crush injury and prevent release of cellular contents into the circulation, no data currently support this practice and it is not recommended by guidelines (1). Similarly, there is no evidence to support prehospital amputation of severely crushed limbs, even in disaster scenarios (1).

ED Management of Crush Syndrome

As with all trauma patients, evaluation, monitoring, and treatment occur simultaneously after Advanced Trauma Life Support protocols. In addition to standard trauma evaluations including the primary and secondary survey, these patients should quickly have an ECG performed to evaluate for effects of hyperkalemia. Electrolyte levels should be obtained, with a special interest in potassium, calcium, and phosphorous. A blood gas, either arterial or venous, is useful to evaluate for metabolic acidosis, check a base deficit, and might allow for rapid analysis of the potassium and calcium (1). Potassium levels should be monitored 3–4 times a day and rechecked frequently in the early phases of resuscitation (3).

Urinalysis should be performed, specifically noting the color of the urine, and urine microscopy can detect casts. Myoglobin can be measured in both the serum and the urine, and many authors recommend following these levels. Additionally, CK should be checked on presentation and followed serially. Liver function tests should be sent to evaluate for shock liver and coagulopathy.

Fluid resuscitation initiated in the field should be continued in the ED, with a goal of continuing to restore circulating blood volume with normal saline. Ideally, the urinary output should be maintained at > 200–300 mL/h (3,5). Patients might require 6–12 L per day to maintain this output, due to ongoing third spacing in the crushed limb. Once the patient has been resuscitated, changing to hypotonic saline may be appropriate (3). The data regarding the utility of bicarbonate infusions are unclear. The hypothesis is that by alkalinizing the urine to a pH > 6.5, precipitation of the myoglobin with Tamm-Horsfall proteins and urate crystals are reduced, thereby minimizing cast formation and further tubular obstruction.

Additionally, most published protocols call for the addition of mannitol for three purposes, as an osmotic diuretic, as a free-radical scavenger, and as an osmotic agent, to reduce the swelling of the affected limb and decrease the risk of compartment syndrome. If urinary flow exceeds 20 mL/h, 50 mL 20% mannitol (total of 1–2 g/kg/day, given at a rate of 5 g/h) may be added to each liter of infusate (3). Mannitol should not be given to patients who are anuric (2).

Much of the data supporting these practices come from animal data and small retrospective, noncontrolled human studies (11). Several centers and authors have published treatment algorithms focusing on aggressive

hydration with subsequent addition of bicarbonate infusions and mannitol diuresis, but none of these algorithms have been tested in randomized controlled trials (3,5). A retrospective study comparing patients with CK levels of > 5,000 U/L who received bicarbonate and mannitol infusions to those who did not found no difference in rates of renal failure, need for dialysis, or mortality (16). Similarly, a recent systematic review found no high-quality evidence to support alkaline diuresis over saline alone (25). Regardless, many authors continue to recommend this protocol (2,3,13,17). It is likely reasonable to provide bicarbonate and mannitol to patients with traumatic rhabdomyolysis, unless the patient has a contraindication. This resuscitation should continue until the clinical and biochemical evidence of myoglobinuria resolves (3).

Despite adequate resuscitation, up to one third of patients with rhabdomyolysis will develop acute renal failure (ARF) (5). Providers need to be prepared to initiate dialysis as needed, for standard dialysis indications of hyperkalemia, acidemia, uremia, or hypervolemia (3). Given the likelihood of severe renal injury, early discussions with renal consultants from the ED can be considered.

Patients should be examined on presentation and undergo serial examinations for development of compartment syndrome, especially in light of fluid resuscitation. Conscious patients might complain of pain disproportionate to the evident trauma, especially with passive motion. Patients might also complain of numbness or parasthesias (22,23,26). Many patients will have altered mental status or be unstable, which makes description of symptoms unreliable. The physical examination may reveal swollen, tense, erythematous limbs or back, but can also be insensitive for the syndrome. The affected compartments may not be obvious, such as compartment syndrome involving the gluteal muscles or the back, particularly in patients found down. Providers should remember that splinted limbs, especially in the unconscious patient, can deter a thorough physical examination of injured limbs and mask an area of compartment syndrome (10,11). Radiographic imaging with magnetic resonance imaging or CT may assist in making the diagnosis of compartment syndrome by delineating areas of edema and necrosis (10,27). Therefore, ED physicians should remain vigilant for compartment syndrome and have a low threshold to obtain imaging and measure compartment pressures. Any patient with compartment syndrome should have fasciotomies as soon as possible to restore perfusion and prevent permanent neurovascular injury (22,23).

Despite the high rate of infection among crush syndrome victims, empiric antibiotic therapy is not recommended unless open wounds are present. For patients with open wounds, empiric treatment with broad-spectrum cephalosporins with or without metronidazole, in addition to tetanus prophylaxis, is recommended (27).

Prognosis

Drawing firm conclusions about the prognosis for crush syndrome is difficult, as much of the data on this entity are derived from reports of earthquakes and other disasters. These reports are often from austere environments, and there is significant heterogeneity among the timing of treatment. Reports include broad ranges, with 4%–33% of patients with rhabdomyolysis developing ARF requiring some form of RRT, with an associated mortality rate of 3% to 50% (5,28,29). Several case reports and case series have reported on good outcomes with aggressive fluid algorithms (7,30–34). In a case series of 9 patients with crush syndrome after the collapse of a building in which an empiric mannitol-bicarbonate cocktail was initiated at admission, only two (28.6%) developed ARF, and no patients with crush syndrome had permanent kidney damage or mortality (31). One report of 7 patients pulled from earthquake rubble treated with a standard protocol of i.v. fluid resuscitation compared with 13 patients treated with variable resuscitation found that the standardized protocol was associated with a lower rate of dialysis (34).

A series of reports on 639 patients hospitalized with ARF due to crush injury after the Maramara earthquake in 1999 found that 477 patients (74.6%) required dialysis, and 15 patients died before dialysis could be instituted. Hyperkalemia was the most significant predictor of dialysis needs in the victims admitted within the first 3 days ($p = 0.008$; odds ratio = 3.33). Ninety-seven of the 639 patients with ARF as a result of crush injury died (15.2%), and mortality rates were 17.2% and 9.3% in dialyzed and nondialyzed patients, respectively. Findings significantly associated with mortality were sepsis, thrombocytopenia, DIC, ARDS, and abdominal and thoracic traumas (14,18,35).

Conversely, a report from the Wenchuan earthquake found a low rate of RRT requirements in hospitalized patients, but also a high rate of death (36). These authors drew the conclusion that patients were in such a remote area that they did not receive early i.v. fluids, leading to fewer survivors and fewer patients to treat with RRT. Although there are many confounders, these limited reports and observational data support the use of early, on-scene if possible, aggressive hydration. Fasciotomies are common in patients with crush syndrome, and should be performed urgently in patients with compartment syndrome (26,33).

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. Close monitoring of patients for development of shock, metabolic derangements, and rhabdomyolysis is critical. Ongoing resuscitation with i.v. fluids is the mainstay of treatment, and emergency physicians should consider early renal consultation. Compartment syndrome is a common complication, and prompt fasciotomies should be performed when compartment syndrome is present. Although no randomized studies of crush syndrome are available, observational data suggest that aggressive resuscitation and close monitoring can reduce the risk of renal failure and death.

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ARTICLE SUMMARY

1. Why is this topic important?

Although crush syndrome is commonly considered to be a consequence of disaster scenarios, emergency physicians working in all communities regularly encounter patients with crush syndrome, often as a consequence of being "found down" after an episode of intoxication or a stroke.

2. What does this review attempt to show?

Crush syndrome manifests as metabolic derangements due to the release of cellular contents into the circulation and the third spacing of large volumes of fluid in the injured muscle. The metabolic derangements and subsequent organ failure may be mitigated by monitoring and fluid resuscitation.

3. What are the key findings?

The early minutes to hours after the pressure is released are the most critical for a patient with crush syndrome, corresponding to the time the patient is in the prehospital and emergency department (ED) settings. Although life threatening, the effects of crush syndrome can be minimized or reversed with close monitoring and aggressive resuscitation. The need for prompt intervention makes this a critical subject for ED physicians to appreciate.

4. How is patient care impacted?

Providers must observe the patient carefully for development of hyperkalemia, acidosis, rhabdomyolysis, and subsequent dysrhythmias or shock. Saline infusions should be started in the prehospital setting, ideally before extrication, and continued in the ED. Patients should have a thorough physical examination to evaluate for compartment syndrome, and patients with compartment syndrome should have prompt fasciotomies.