

Clinical Case Seminar

CCS6 (1-5)

Intensive management of a crush syndrome case

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Abstract

We here describe the case of a 16-year-old boy who was victim of a car accident, and developed a crush syndrome with exponential increase in CPK, myoglobin and with worrying levels of potassium in the blood and initial contraction of diuresis. Early start of treatment with hyperhydration and continuous hemofiltration, begun 4 hours after the car accident; it has been effective in reducing lactic acidosis, serum levels of K⁺ and myoglobin and it also significantly attenuated the renal insult by preventing myoglobin deposit damage.

Key-Words: rhabdomyolysis; crush syndrome; renal failure; hyperkalemia; massive fluid resuscitation; preventive dialysis

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Introduction

The crush syndrome, also known as traumatic rhabdomyolysis, is the systemic manifestation of the breakdown of muscle cells with release of their contents into the circulation (1-2)

The trauma to the extremities, even if not involving vital organs, can be life threatening.

One of the most feared complications of crush syndrome is acute renal failure, a condition that can be prevented or reversed. (3)

The first descriptions of the crush syndrome were in the German-language literature following the earthquake of Messina in 1909 and The World War II (4).

Only in 1923 Seigo Minami described the cases of three soldiers who died of kidney failure caused by a crush injury during the First World War. Using microscopic studies to investigate the pathology of their kidneys, he discovered that the soldiers had died of "auto-intoxication" caused by rhabdomyolysis (5).

Eric Bywaters in 1941 described the crush syndrome after studying the victims of the London Blitz during The World War II. He defined "auto-intoxication" the release of rhabdomyolysis products after reperfusion of tissues. He established the need to replace the emergency fluid to treat the crush syndrome. Therefore the current guidelines are based on these findings. (6)

Early diagnosis and treatment in the very first hours since the traumatic insult reduces progressive renal failure and therefore mortality.

Pathogenesis

It is now known that cell death is caused by the compression of muscle fibers. Reduced blood perfusion of the tissue with consequent ischemia occurs, and this together with the trauma leads to a metabolic disequilibrium and organ failure.

It has been observed that skeletal muscle usually tolerates up to 2 hours of ischemia without permanent injury. However, at 4-6 h, tissue necrosis develops (7). By 24 hours, histologic changes caused by ischemia - reperfusion injury are maximal (8)

The traumatic insult damages the sodium-potassium sarcolemmic ATPase (Na / K), allowing calcium efflux. The increase in intracellular calcium stimulates the activity of intracellular proteases, leading at cell rupture.

The cells swell and their intracellular Ca ++ concentration increases with an increase of cytoplasmic protease activity leading to the degradation of myofibrillar proteins. Tissue reperfusion causes the release of free radical superoxide produced by ischemic lesions (9). Hypovolemia is often the first manifestation of the crush syndrome; clinical observations have shown that large volumes of fluid can leak into the interstitial space and cause hypotension (10-11).

In addition to intravascular depletion, there is a large load of toxins that can lead to even fatal electrolyte abnormalities. Hypocalcemia results from the influx of Ca ++ becoming dangerous when combined with hyperkalemia and acidemia. Hyperkalemia and its associated cardiotoxicity are the second most common cause of early deaths due to crush injuries (12).

Tissue thromboplastin levels also increase and can lead to disseminated intravascular coagulation (11). The high concentrations of myoglobin released into the blood represent a danger to the kidney. Whether intravenous fluid reinstatement is inadequate or delayed for more than 6 hours, it may cause renal failure (13) Rhabdomyolysis leads to decreased renal function through three mechanisms: reduction of renal perfusion, tubular obstruction and direct toxic effects of myoglobin on the renal tubules.

When the circulating levels of myoglobin are low, it is eliminated from the circulation by the reticuloendothelial system. When the circulating levels of myoglobin are high, as occurs later in rhabdomyolysis and exceed 0.5-1.5 mg / dL, it is filtered by the kidney, with consequent myoglobinuria. The high concentration of myoglobin in the renal tubules causes tubular obstruction and acidification of the urine, myoglobin precipitates forming casts. Urinary alkalization with sodium bicarbonate has been shown to reduce precipitation (14)

Case report

We describe the case of a 16-year-old male victim of a car accident with a slightly displaced pluriframmentary fracture of the right temporal bone with a millimetric parenchymal hemorrhage in the ipsilateral temporal region, a multi-segmental exposed fracture to the right elbow, hepatic and pulmonary contusions. His GCS was 12. His diuresis was contracted.

His vital signs were: temperature 37.2 C, heart rate 147 beats / min, blood pressure 100/48 mm Hg, O2 saturation 99% on the ambient air.

His laboratory findings were: 7.7 g / dL hemoglobin, hematocrit 59.3% and platelets 171,000 / mm³. His sodium was 133 mmol / L, potassium was 7 mmol / L, chloride was 105 mmol / L, bicarbonate was 21 mmol / L, urea nitrogen in the blood was 30 mmol / L, creatinine was 1.2 mmol / L, glucose was 128 mmol / L and lactate was 20 mmol / L. Creatinine kinase (CK) was 9.919 IU / L, his myoglobin was 2,745 ng/ ml.

The patient was intubated and was admitted to the intensive care unit. He received aggressive fluid hydration at 20 ml / kg / h to maintain a urine output of at least 200 ml/h, and required an infusion of norepinephrine to maintain a mean arterial pressure > 65 mm Hg.

He received therapy withmannitol to increase diuresis (1.5 - 2gr intravenously in repeated doses) and bicarbonate at a dosage of 20 mEq/L.

He immediately started continuous hemofiltration, (started 4 hours after the accident) and after 12 h, his level of CPK was 5,919 IU/L, his urine myoglobin was 1,895 ng/ml and after 48 h the levels of myoglobin and cpk in the blood were normal. His diuresis was active and the PH of the urine has normalized. He has been extubated the following day from the start of dialysis. Since then he had surgery in a surgical clinic and is doing rehabilitation.

Discussion

Early diagnosis is crucial in patients with rhabdomyolysis. The patients who have suffered significant soft tissue injury (due to crushing or prolonged injury immobilization) or ischemia - reperfusion injury (due to vascular disruption) are at risk of developing rhabdomyolysis, myoglobinuria and renal failure. Patients often have painful and swollen extremities and need to be monitored for the development of a compartmental limb syndrome. The dark color of the urine in the absence of blood is suggestive of myoglobinuria and rhabdomyolysis. The fastest and the least expensive rhabdomyolysis screening test is the serum CPK level. To prevent systemic and renal complications of crushing syndrome, vigorous fluid supplementation is required, preferably started at the site of the lesionbefore extrication (15). The use of mannitol is recommended. The benefits of mannitol to induce a solute diuresis have been established in several experimental studies. It is an osmotic diuretic, promoting an increased urine production and dilution of tubular myoglobin, and it is a volume expander. Mannitol has also been shown to reduce the

intracompartmental pressure. (16). The alkalization of urine with sodium bicarbonate to prevent development of acute renal failure after crush injuries and rhabdomyolysis was supported by numerous studies (17-18-19-20).

Bicarbonate alkalizes urine and reduces the direct toxic effects of myoglobin (21). In fact it has been shown that myoglobin induces renal vasoconstriction only at acid pH (22). Daily Hemodialysis or Hemodialysis / continuous hemofiltration is useful in correcting fluids and electrolyte abnormalities that accompany rhabdomyolysis and kidney failure (23). The use of PPD (preventive peritoneal dialysis) at the onset of compression release, is beneficial for kidneys protection and for survival outcome in a rabbit model of CI (24). The use of early continuous hemofiltration (started 4 hours after the traumatic insult) was effective both in reducing lactic acidosis, serum levels of K⁺ and myoglobin it also significantly attenuated the renal insult by preventing myoglobin deposit damage in our patient.

Conflict of interest: The Authors declare no competing interest

Ethical approval This paper does not contain any studies with human participants or animals performed by the authors.

Informed consent No informed consent.

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