SAS Journal of Medicine
Abbreviated Key Title: SAS J Med
ISSN 2454-5112
Journal homepage: https://saspublishers.com

OPEN ACCESS

Medicine

Crush Syndrome: Biological Profile of Patients Victims of the Al Haouz Earthquake - About 100 Cases

A. Belmekia[1,3*], Y. Akrim[2,3], S. Kamissoko[2,3], S. Chellak[2,3], A. Boukhira[2,3]

[1]Faculty of Medicine and Pharmacy of Agadir, Morocco
[2]Faculty of Medicine and Pharmacy of Marrakech, Morocco
[3]Avicenne Military Hospital of Marrakech, Morocco

DOI: https://doi.org/10.36347/sasjm.2024.v10i10.004 | **Received:** 11.08.2024 | **Accepted:** 19.09.2024 | **Published:** 02.10.2024

***Corresponding author:** A. Belmekia
Faculty of Medicine and Pharmacy of Agadir, Morocco

Abstract **Original Research Article**

Crush syndrome encompasses local and systemic manifestations secondary to prolonged ischemia of significant muscle masses, resulting from intense and sustained compression [1]. This massive rhabdomyolysis poses a life-threatening prognosis due to the onset of either hypovolemia with shock, sudden hyperkalemia, or acute renal failure. Following the earthquake that struck Al Haouz on September 8, 2023, in El Haouz, 71.8 km southwest of Marrakech, with a magnitude of 6.9, a prospective study was conducted on 100 patients admitted to the emergency department of the Avicenne Military Hospital in Marrakech. These patients presented various traumas and muscle compressions, with varying severity levels and locations. Upon admission, levels of myoglobinemia, creatine phosphokinase, creatinine, and kalemia were measured. Hyperkalemia was detected in 4 individuals with concentrations exceeding 5.5 mmol/L. Analysis of creatine phosphokinase (CPK) revealed that 27% of the patients suffered from rhabdomyolysis, with CPK levels exceeding 1000 IU/L. Among these patients, 5 had severe rhabdomyolysis with CPK levels surpassing 5000 IU/L. 25% of the patients with rhabdomyolysis developed acute renal failure, a complication also present in all cases of severe rhabdomyolysis. In terms of myoglobinemia, 16% of the patients had levels exceeding 500 µg/L, and 11 patients had levels above 1000 µg/L. Notably, all patients with severe rhabdomyolysis exhibited myoglobinemia levels exceeding 1000 µg/L, correlating with the development of acute renal failure.

Keywords: Missing.

INTRODUCTION

The term rhabdomyolysis defines a clinical and biological syndrome caused by the lysis of skeletal striated muscle fibers, whose contents are released into the general circulation. Any situation causing an imbalance between metabolic supply and demand can lead to rhabdomyolysis.

However, Crush syndrome encompasses the local and general manifestations secondary to prolonged ischemia of significant muscle masses, due to intense and sustained compression [1]. This syndrome was first described by Bywaters and Beall in 1941 after the Battle of London [2].

This massive rhabdomyolysis is life-threatening due to the onset of hypovolemia with shock, sudden hyperkalemia, acute renal failure (caused by hypovolemia and the precipitation of myoglobin in the tubules), and most often a combination of these three consequences [3, 4].

The biological analysis of various parameters such as creatine phosphokinase, creatinine, myoglobinemia, and kalemia is of paramount importance in the diagnosis, monitoring, and prognosis of patients affected by Crush syndrome.

MATERIALS AND METHODS

Following the Al Haouz earthquake that occurred on September 8, 2023, in El Haouz, 71.8 km southwest of Marrakech, with a magnitude of 6.9, causing 2,960 deaths and 6,125 injuries, a prospective study was conducted on 100 patients admitted to the emergency department of the Avicenne Military Hospital in Marrakech. These patients presented with traumas and muscle compressions of varying severity and locations. The levels of myoglobinemia, creatine phosphokinase, and kalemia were measured upon their admission.

The measurements were performed using the Atellica® Solution analyzer by Siemens. The blood samples were collected without the use of a tourniquet. Patients with minor abrasions or minor injuries requiring only superficial care or simple sutures were excluded from the study.

RESULTS

In a sample of 100 patients admitted to the emergency department following the Al Haouz earthquake, presenting with various traumas, we observed the following results: The average age of the patients was 31 years, with a range from 5 to 74 years. There was a female predominance with a sex ratio of 1.3.

Hyperkalemia was detected in 4 individuals with concentrations exceeding 5.5 mmol/L. Notably, one patient had moderate hyperkalemia at 6.7 mmol/L, while another had severe hyperkalemia at 7.18 mmol/L.

The analysis of creatine phosphokinase (CPK) revealed that 27% of the patients suffered from rhabdomyolysis, with CPK levels exceeding 1000 IU/L. Among these patients, 5 had severe rhabdomyolysis with CPK levels surpassing 5000 IU/L. Notably, 25% of the patients with rhabdomyolysis developed acute renal failure, and all patients with severe rhabdomyolysis also presented this complication.

Regarding myoglobinemia, 16% of the patients had levels exceeding 500 µg/L, and 11 patients had levels above 1000 µg/L. Among the patients with myoglobinemia above 500 µg/L, 68% developed acute renal failure. Furthermore, 81% of the patients with myoglobinemia over 1000 µg/L also presented acute renal failure.

It is noteworthy that all patients with severe rhabdomyolysis had myoglobinemia levels exceeding 1000 µg/L and all developed acute renal failure. These results highlight the correlation between elevated levels of CPK and myoglobin and the development of acute renal failure in patients affected by the Al Haouz earthquake.

DISCUSSION

Crush injuries to the extremities, even when they do not involve vital organs, can be life-threatening. Crush syndrome, a systemic manifestation of muscle cell breakdown with the release of their contents into the circulation, leads to metabolic imbalance and acute kidney injury [5].

The mechanism of cell injury and death in crush syndrome originates from the compression of muscle fibers. In addition to the direct trauma from compression, the tissue is deprived of blood flow and becomes ischemic, with both mechanisms causing muscle cell lysis, resulting in significant metabolic imbalance and ultimately organ failure [6].

The timing of the onset of cellular damage and death varies depending on the crushing force applied. Skeletal muscle can generally tolerate up to 2 hours of ischemia without permanent damage. However, after 4 to 6 hours, tissue necrosis develops [6]. At the cellular level, crushing opens stretch-activated channels in the muscle cell membrane and disrupts the Na/K transporter, allowing calcium to freely enter the cell. The increase in intracellular calcium stimulates the activity of intracellular proteases, leading to cell degradation [7].

Once the external pressure is released, the cell contents, including potassium, phosphorus, and urate, are released into the circulation, accelerating metabolic imbalances. Conversely, the rupture of cell walls allows calcium and sodium to rush into the cell, leading to hypocalcemia and hyponatremia. For many patients, crush syndrome immediately results in hyperkalemia, leading to dysrhythmias, which often appear less than an hour after extrication [8].

Hyperkalemia is defined as a plasma potassium level > 5.5 mmol/L. It is considered moderate between 6.1 and 6.9 mmol/L and severe if potassium levels are > 7 mmol/L [9].

With the release, intravascular fluids flow into the intracellular compartments, and the injured area begins to sequester large volumes of fluids. The limbs can contain up to 12 L of fluid in their large compartments. Distributive shock can also develop as a result of the release of inflammatory mediators due to reperfusion injury [10].

Although dysrhythmias are the most immediate concern in crush syndrome, renal effects are the most serious complication. The initial kidney injury before extrication is largely due to decreased circulating blood volume, exacerbated by third spacing of fluids into the injured limb. Additionally, the release of myoglobin into the systemic circulation contributes to worsening renal injuries. The heme protein present in myoglobin has several nephrotoxic effects. Firstly, myoglobin's nitric oxide inhibitory effect causes vasoconstriction, exacerbating a pre-renal state. Heme also leads to direct cytotoxicity as myoglobin's iron likely catalyzes the formation of more free radicals, leading to intrarenal failure, especially in the proximal tubule [11]. Finally, the kidneys readily filter myoglobin, which then precipitates with Tamm-Horsfall proteins in the tubules [12]. These tubular casts lead to obstruction and are thought to increase intraluminal pressure, thereby decreasing glomerular filtration [13].

In rhabdomyolysis, the myoglobinemia level (myoglobin in the blood) exceeds 500 µg/L, which is 6 times the upper limit of normal [14]. However,

determining myoglobin in serum and urine is only useful for early diagnosis of rhabdomyolysis, and the absence of elevated serum myoglobin levels does not rule out the diagnosis of rhabdomyolysis [15].

While myoglobin is responsible for nephron damage, CK levels are commonly monitored as a marker of crush syndrome. Normally, creatine phosphokinase is found in cardiac tissues, skeletal muscles, the brain, etc. However, in the case of muscle injury, there is a leakage of CK into the blood. Thus, CK is indicative of muscle damage. CK-MB is a more specific indicator of cardiac muscle damage, while CK-MM is more indicative of skeletal muscle damage [16]. A CK level exceeding 1000 IU/L indicates rhabdomyolysis; values exceeding 5000 IU/L indicate severe rhabdomyolysis [17]. The analysis of creatine phosphokinase (CK) revealed that 27% of the patients suffered from rhabdomyolysis, with CK levels exceeding 1000 IU/L, while only 16% of the patients had myoglobinemia levels exceeding 500 µg/L. This can be explained by the fact that the increase in serum myoglobin precedes the increase in CK; therefore, serum and urinary levels may have returned to normal by the time of hospitalization. Similarly, myoglobin elimination from plasma is rapid, so within 1 to 6 hours, both renal excretion and metabolism into bilirubin occur [18].

In our study, we observed that acute renal failure, the most severe complication of rhabdomyolysis, occurred in 25% of the patients, a figure consistent with previously reported data in the general medical literature (15% to 33%) [19, 20]. All patients with severe rhabdomyolysis (CK > 5000 IU/L) also presented this complication. This aligns with the study by Melli [21], the study by Ward [20], and the study by Eneas *et al*., [22], which found a highly significant association between serum creatinine values, as a marker of renal function, and CK values. All patients with severe rhabdomyolysis had myoglobinemia levels exceeding 1000 µg/L. This has been demonstrated by Melli *et al*., showing that the amount of myoglobin released into the blood is correlated with the serum CK level [21].

CONCLUSION

Crush injuries to the extremities, even if they do not involve vital organs, can be life-threatening. Crush syndrome, also known as traumatic rhabdomyolysis, is the systemic manifestation of muscle cell breakdown with the release of their contents into the circulation. Measurements of creatine phosphokinase and myoglobin are crucial tools for diagnosing and assessing the severity of rhabdomyolysis. There is a highly significant association between serum creatinine values, as a marker of renal function, and CK and myoglobin levels. However, acute renal failure is one of the serious complications that can be avoided through optimal management.

BIBLIOGRAPHY

1. Liach, F., Fesenseld, A., & Hausser, M. (1981). The physiopathology of altered calcium metabolism in rhabdomyolysis induced acute renal failure. *N Engl J Med*, *305*, 117-123.
2. Smith, J., & Greaves, I. (2003). Crush injury and crush syndrome: a review. *Journal of Trauma and Acute Care Surgery*, *54*(5), S226-S230.
3. Better, O. S., & Stein, J. H. (1990). Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. *New England Journal of Medicine*, *322*(12), 825-829.
4. Bosch, X., Poch, E., & Grau, J. M. (2009). Rhabdomyolysis and acute kidney injury. *New England Journal of Medicine*, *361*(1), 62-72.
5. Genthon, A., & Wilcox, S. R. (2014). Crush syndrome: a case report and review of the literature. *The Journal of Emergency Medicine*, *46*(2), 313-319.
6. Sahjian, M., & Frakes, M. (2007). Crush injuries: pathophysiology and current treatment. *Nurse Pract*, 32, 13-18.
7. Malinoski, D. J., Slater, M. S., & Mullins, R. J. (2004). Crush injury and rhabdomyolysis. *Critical care clinics*, *20*(1), 171-192.
8. Sever, M. S., Erek, E., Vanholder, R., Kantarci, G., Yavuz, M. A. H. M. U. T., Turkmen, A., ... & Lameire, N. (2003). Serum potassium in the crush syndrome victims of the Marmara disaster. *Clinical nephrology*, *59*(5), 326-333.
9. Halperin, M. L., & Kamel, K. S. (1998). Potassium. *The Lancet*, *352*(9122), 135-140.
10. Smith, J., & Greaves, I. (2003). Crush injury and crush syndrome: a review. *Journal of Trauma and Acute Care Surgery*, *54*(5), S226-S230.
11. Penn, A. S. (1986). Myoglobinuria. In: Engel and Banker (Eds.). Myology. Vol. 2. McGraw-Hill, New York. 62. pp. 1785-1805.
12. Fleischer, R. (1881). Ueber eine neue form von haemoglobinurie beim menschen. *Berl Klin Wschr*, *18*, 691.
13. Koskelo, P., Kekki, M., & Wager, O. (1967). Kinetic behaviour of 131I-labelled myoglobin in human beings. *Clinica Chimica Acta*, *17*(3), 339-347.
14. Gabow, P. A., Kaehny, W. D., & Kelleher, S. P. (1982). The spectrum of rhabdomyolysis. *Medicine*, *61*(3), 141-152.
15. Poels, P. J. E., & Gabreels, F. J. M. (1993). Rhabdomyolysis: a review of the literature. *Clinical neurology and neurosurgery*, *95*(3), 175-192.
16. Panteghini, M. (1988). Serum isoforms of creatine kinase isoenzymes. *Clin Biochem*, *21*(4), 211-218.
17. Sauret, J. M., Marinides, G., & Wang, G. K. (2002). Rhabdomyolysis. *Am Fam Physician*, *65*(5), 907-912.
18. Reuter, J. D., Dysko, R. C., & Chrisp, C. E. (1998). Review of exertional rhabdomyolysis and a case in a rhesus monkey (Macaca mulatta). *Journal of medical primatology*, *27*(6), 303-309.
19. Gabow, P. A., Kaehny, W. D., & Kelleher, S. P.

(1982). The spectrum of rhabdomyolysis. *Medicine (Baltimore)*, 61, 141-152.

20. Ward, M. M. (1988). Factors predictive of acute renal failure in rhabdomyolysis. *Archives of internal medicine*, *148*(7), 1553-1557.
21. Melli, G., Chaudhry, V., & Cornblath, D. R. (2005). Rhabdomyolysis: an evaluation of 475 hospitalized patients. *Medicine*, *84*(6), 377–385.
22. Eneas, J. F., Schoenfeld, P. Y., & Humphreys, M. H. (1979). The effect of infusion of mannitol-sodium bicarbonate on the clinical course of myoglobinuria. *Archives of internal medicine*, *139*(7), 801-805.