Crush Injuries and the Crush Syndrome

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Abstract

Combat related injuries bring untold misery to the victims and their loved ones. While injuries to vital organs cause immediate death, delayed mortality after reaching a hospital may occur due to several reasons, rhabdomyolysis or the crush syndrome being the most important. Crush syndrome predominantly affects the kidneys leading to renal failure, but the clinical picture may include acute respiratory distress syndrome, dyselectrolytaemia, disseminated intravascular coagulation, hypovolemic shock, arrhythmias and psychological trauma. Rescue, resuscitation and rehabilitation are onerous tasks and so a coordinated strategy with well trained team of professionals through various chains of evacuation is advocated. This article addresses these issues drawing upon the experience of the writer in combat casualty care in active operations.

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Introduction

Life in the modern world means existing with the evolving face of modern warfare and global terrorism besides disasters, both natural and man-made alike. Man has devised weapons of mass destruction, which in the hands of warring nations and urban terrorists can cause blasts, building collapses etc., resulting in a unique pattern of damage to the human body namely, crush injuries. They not only cause immediate mortality, but also widespread complicated haemodynamic and metabolic disturbances over a period of time in those who survive, that requires active management.

Historical perspective

World War II became a watershed in the study of crush injuries with the path breaking paper in 1941, wherein Bywaters and Beall described crush syndrome as a phenomenon that occurs in some survivors of crush injuries [1]. Mubarak et al identified a spectrum of disorders from compartment syndrome to crush syndrome [2]. Haemodialysis as a primary modality of treatment took shape in the European Continent. Hyperbaric oxygen therapy is firmly established in the treatment armamentarium since three decades [3]. Present attempts are aimed at streamlining the early detection of crush syndrome, standardising a fluid regimen pattern and identifying casualties for renal replacement therapy [4]. Over a period of five decades various conflicts and natural disasters across the globe have made the medical community wiser in the management of crush injuries and crush syndrome.

Definition of terminologies

The spectrum includes

(a) *Crush injury*: Injury caused as a result of direct physical crushing of the muscles due to something heavy.

(b) *Crush syndrome:* Also termed rhabdomyolysis, involves a series of metabolic changes produced due to an injury of the skeletal muscles of such a severity as to cause a disruption of cellular integrity and release of its contents into the circulation.

(c) *Compression syndrome:* An indirect muscle injury due to a simple, slow compression of a group of muscles leading to ischaemic damage and thus causing crush substances to enter the blood.

(d) *Compartment syndrome:* A localized rapid rise of tension within a muscle compartment, which inevitably leads to metabolic disturbances akin to rhabdomyolysis.

Pathophysiology

It is recorded that upto 80% of crush injury patients die due to severe head injuries or asphyxiation. Of the 20% that reach hospital, 10% make an uneventful recovery. The other 10% go into crush syndrome [1]. Our whole focus has to be centered on tackling this 10%, which has severe and extensive metabolic disturbances.

Crush and rupture of muscle cells releases myoglobin, which gets converted to methmyoglobin and finally acid haematin, which is released into the circulation. Muscles

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also contain potassium, magnesium, phosphate, acids, enzymes like creatine phosphokinase (CKMM) and lactate dehydrogenase (LDH). Though essential for cell function, they are toxic when released into the circulation in large amounts. Regional ischemia caused by occlusion of micro and macrocirculation to muscles following crush, releases sodium, calcium and fluids leading to raised muscle volume and tension. CK and ATP are exhausted. Nitric oxide system is activated and this further contributes to muscle vasodilatation and aggravation of hypotension [5].

Casualties deteriorate only after being rescued out of the debris of collapse or entrapment, because once the tissue tension is released, reperfusion to the ischaemic damaged muscles disrupts sodium-potassium-ATPase mechanism. In turn, this releases myoglobin degradation products, lactic acid, uric acid and muscle enzymes like creatinine phosphokinase and aldolase, besides ions like calcium, potassium and phosphate into the circulation [1]. Myoglobin is filtered out of the glomerulus, but once the renal threshold is exceeded, it precipitates in the distal convoluted tubules causing obstruction. It has also been noticed that an element of vasoconstriction of the afferent arterioles induced by myoglobin degradation products adds to this setting of tubular destruction.

Elevated lactic acid levels are well coordinated with muscle ischaemia time, but it is generally accepted that serum creatinine phosphokinase is the most important index of muscle damage [4]. The appearance, duration and gravity of oliguria and kidney damage are not related to the severity of muscle damage [6]. The muscles are grossly swollen, hard, cold, insensitive and necrotic. Kidneys also tend to be edematous and show an increase in volume. The released potassium in the circulation causes alteration in cardiac rhythm. Ultimately, patients go into shock affecting respiratory gas exchange due to lung edema. It is a ripe setting for ARDS.

Crush injuries are not common after head and chest injuries because the prolonged pressure necessary to cause this syndrome often results in death [7,8]. So most of such patients are conscious at rescue and the chest injuries are relatively trivial. However studies show that upto 10% chest trauma is associated with crush injuries [9].

Clinical features

Casualties normal at rescue, however, soon go into shock. Petechiae, blisters, muscle bruising, and superficial injuries are seen. Myalgia, muscle paralysis and sensory deficit are common. Fever, cardiac arrhythmia, pneumonia, 'tea or cola' coloured urine, oliguria and renal failure are the sequence of events. Nausea, vomiting, agitation and delirium are seen in the delayed rescue patients.

Investigations

Serum creatinine kinase (CKMM) levels greater than 1000 IU/l with associated clinical features is generally taken as an indicator of crush syndrome. Normal range is 25-175 U/l, usually rises 2 to 12 hrs after a crush, peaks in 1 to 3 days and declines after 3 to 5 days.

Among the other investigations, serum aldolase may be of some help; serum myoglobin and myoglobin degradation products are highly sensitive tests; serum lactic acid, AST, ALT and LDH show a steady rise; serum uric acid -moderate rise may be noticed; serum urea and creatinine – steep rise is seen especially after a prolonged crush; serum potassium levels show an early rise and is a predictor for dialysis [11,12].

Hypocalcaemia and stress related hyperglycaemia may be seen. Urine RE may show presence of myoglobin products. Blood gas analysis, haemogram and ECG are also helpful. Intracompartmental pressure monitoring is useful as it is generally accepted that levels greater than 30 mm Hg point towards the need for a fasciotomy [13]. Doppler studies are done to look for limb ischaemia, and the body weight is recorded.

Treatment

In scenario of crush injuries, we are dealing with rescue, resuscitation, recognition of the syndrome, treatment and rehabilitation.

Rescue should be a highly coordinated effort, and patient be transported to a higher level of care with dialysis facilities [14,15]. Apart from the usual modes of combat care principles, some workers advocate use of potassium binders like oral polystyrene sulfonate before transportation to avoid renal damage [12]. Noriaki et al have attempted a predictive model for estimating risk of crush syndrome [16].

Resuscitation should ideally commence at the site of injury. Casualties are often in shock, and may lose litres of extracellular fluid into the injured extremity. Clear history is not always available in combat, and the syndrome may appear insidiously in patients who initially appear well. Paramedical personnel should be taught to suspect this condition and treat aggressively with fluid therapy.

Recognition of crush syndrome and treatment involves a close link amongst trauma surgeons, physicians, biochemists and radiologists..

Rehabilitation should be not only physical but also psychiatric and is a long term process [17].

Fluid replacement and monitoring

Though this is the mainstay of treatment [18], no

clearly enunciated formula exists. Early fluid resuscitation, within the first 6 hours, preferably before the victim is extricated is essential [19].There is a wide variation in the quantity of fluids infused. There are reports of greater than 25 litres of saline being given in one day. Of course, there is consensus on the fact that saline is the fluid to be given. To counter the metabolic acidosis, both bicarbonate and lactate or even oral citrate is essential. Gunal et al advocate 50 mmol of bicarbonate for every lit of isotonic saline [4]. Close check is kept on the CVP, BP, pulmonary status and urinary output [20, 21]. Insulin glucose drip to reduce large rise in serum potassium concentration has been used. Patients with crush syndrome need numerous blood product transfusions and the inevitable logistic problem of collection, storage and transportation should be correctly addressed [12].

Diuresis

There is need to underscore the fact that maintaining effective kidney function is the cornerstone in our management of crush injuries. In established crush syndrome, urinary output should be at least 300 ml / hr – that means at least 12 lit of fluid/day as fluid sequestration inside damaged muscles may be as high as 4 lit [12]. Mannitol diuresis achieves this in a large measure [4,12]. However, Holt et al suggest that mannitol is not superior to IV fluids alone, though it is indicated in a setting of compartment syndrome [22]. Brown showed that bicarbonate and mannitol do not prevent renal failure in patients with CK >5000 U/l [23]. Loop diuretics are avoided unless it is to ensure a short burst of kidney activity. Dopamine has been tried as a primary mode in one Japanese study with limited success though this also has the added advantage of ensuring a steady blood pressure [7].

Dialysis

It now has a firm place in the management of crush syndrome. The Russians had a tremendous success in the Armenian earthquake where 23% required dialysis [24,25]. It is therefore essential to include a nephrologist or at least a physician in the team. The important predictive factors for dialysis include anuria, fluid overload, serum creatinine levels, BUN and bicarbonate levels [4]. Potassium levels above 7 meq/l is an independent and important predictive factor of dialysis [11]. At least twice or even thrice daily dialysis may be needed for upto15 days. Prophylactic dialysis may be called for in patients at high risk for hyperkalaemia [26].

Hyperbaric oxygen

At high pressures, physically dissolved levels of oxygen increases in the plasma, tissue viability is enhanced, some vasoconstriction occurs and so fluid outflow from the vascular compartments decrease thus reducing tissue edema. It directly assists wound healing by fibroblast proliferation. Finally it can reduce anaerobic bacterial growth in necrosed muscles [27]. The usual dose is about 2.5 atmospheres for about one and half hours twice a day for a week [3].

Antibiotics: Multiple broad spectrum non nephrotoxic antibiotics may be needed.

Surgery: Laparotomy and thoracotomy may be called for wide and ruthless debridement of all necrosed muscles is indicated followed by delayed primary or secondary suturing. Signs of a possible rise in compartmental pressure call for a fasciotomy [13]. Early fasciotomy is better, as fasciotomies after 8 -10 hours of crush have necessitated amputations [28]. Kantarci et al [29] feel that when a patient needs a fasciotomy, in all probability he will need a renal replacement therapy. Fractures need fixation and conservative amputations may have to be performed either as emergencies or as an elective measure. Immunisation against tetanus is indicated. Late complications of muscle contracture require good rehabilitation therapists [30].

Conclusion

The hospital management of crush injuries is essentially that of rhabdomyolysis. Surgical management is akin to that of any case of trauma. Very energetic fluid replacement and diuresis with timely fasciotomies and monitoring of enzymes and electrolytes will salvage many limbs and lives.

Conflict of Interest

None identified

References

1. Bywaters EGL. 50 years on – the crush syndrome. BMJ 1990: 301; 1412-5.
2. Mubarak S, Owen CA. Compartmental syndrome and its relation to the crush syndrome – a review of 11 cases of prolonged limb compression. Clin Orthop 1975; 113: 81-9.
3. Bouachour G, Cronier P, Gouello JP, Toulemonde JL, Talha A, Alquier P. Hyperbaric oxygen therapy in the management of crush injuries: a randomised double blind placebo-controlled trial. J Trauma 1996; 41: 333-9.
4. Gunal AI, Celiker H, Dogukan A, Ozalp G, Kirciman E, Simsekli H et al. Early and vigorous fluid resuscitation prevents acute renal failure in the crush victims of catastrophic earthquakes. J Am Soc Nephrol 2004; 15: 1862-7.
5. Rubinstein I, Abbassi Z, Coleman R, Milman F, Winaver J, Better OS. Involvement of nitric oxide system in experimental muscle crush injury. J Clin Invest 1998; 101: 1325-33.
6. Oda J, Tanaka H, Yoshiaka T, Iwai A, Yamamura H, Ishikawa K et al. Analysis of 372 patients with crush syndrome caused by the Hanshin-Awaji earthquake. J Trauma 1997; 42: 470-6.
7. Yoshimura N, Nakayama S, Nakagiri K, Azami T, Ataka A, Ishii N et al. Profile of chest injuries arising from the 1995

Southern Hyogo Prefecture earthquake. Chest 1996; 111: 750 - 61.

8. Gonzalez TJ, Poza M. (Letter). Crush injuries to the head. Neuro Surg 1996; 39: 877- 8.
9. Ozdogan S, Hocaoglu A, Caglayen B, Imamoglu O, Aydiny D. Thorax and lung injuries arising from the two earthquakes in Turkey in 1999. Chest 2001; 120: 1163-6.
10. Iskit SH, Alpaz H, Tugtope H, Ozalemir C, Ayyildiz SH, Ozel K et al. Analysis of 33 paediatric trauma victims in 1999 Marmara, Turkey earthquake. Paed Surg 2001; 36: 368-72.
11. Sever MS, Erek E, Vanholder R, Kantarci G., Yavuz M, Turkmen A et al. Serum potassium in the crush syndrome victims of Marmara disaster. Clin Nephrol 2003; 59: 326-33.
12. Sever MS, Vanholder R, Lameire N. Management of crush related injuries after diasasters. New Engl J Med 2006; 354: 1052-63.
13. Oda Y, Shinloh M, Yukioka H, Nishi M, Fujimori M, Asada A et al. Crush syndrome – sustained in 1995 Kobe, Japan earthquake-treatment and outcome. Ann Emer Med 1997; 30: 507-12.
14. Peek Asa C, Kraus JF, Bourque LB, Vimalachandra D, Yu J. Fatal and hospitalized injuries resulting from the 1994 Northridge earthquake. Int J Epidemiol 1998; 27: 459-65.
15. Whittaker R, Fareed D, Green P, Barry P, Borge A, Fletes – Barrios R. Earthquake disaster in Nicaragua: reflections on the initial management of massive casualties. J Trauma 1974; 14: 37-43.
16. Noriaki A, Demson J, Zupan B, Mozine M, Pretto E, Oda J et al. Predictive model for estimating risk of crush syndrome: a data mining approach. J Trauma 2007; 62: 940-5.
17. Sharma R. Gujarat earthquake causes major mental health problems. BMJ 2002; 324: 259.
18. Shimazu T, Yoshioka T, Nakata Y, Ishikawa K, Mizushima Y, Morimoto F et al. Fluid resuscitation and systemic complications in crush syndrome: 14 Hanshin – Awaji earthquake patients. J Trauma 1997; 42: 641-8.
19. Better OS. The crush syndrome revisited (1940-90). Nephron 1990; 55: 97-103.
20. Slater MS, Mullins RJ. Rhabdomyolysis and myoglobinuric renal failure in trauma and surgical patients: a review. J Am Coll Surg 1998; 186: 693-716.
21. Better OS, Stein JH. Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. N Engl J Med 1990; 322: 825-9.
22. Holt SG, Moore KP. Pathogenesis and treatment of renal dysfunction in rhabdomyolysis. Intensive Care Med 2001; 27: 803-11.
23. Brown, Carlos VR, Rhee Peter. Preventing renal failure in patients with rhabdomyolysis: Do bicarbonate and mannitol make a difference? J Trauma 2004; 56: 1191-6.
24. Richards NT, Tattersall J, McCann M, Samson A, Mathias T, Johnson A. Dialysis foe acute renal failure due to crush injuries after the Armenian earthquake. BMJ 1989; 298: 443-5.
25. Collins AJ. Kidney dialysis treatment for victims of the Armenian earthquake. N Engl J Med 1989; 19: 1291-2.
26. Vanholder R, Sever MS, Erek E, Lameire N. Rhabdomyolysis. J Am Soc Nephrol 2000; 11: 1553-61.
27. Myers R. Hyperbaric oxygen therapy for trauma: crush injury, compartment syndrome and other acute traumatic peripheral ischaemias. Intl. Anaes Clinics 2000; 38: 139-51.
28. Finkelstein JA, Hunter GA, Hu RW. Lower limb compartment syndrome: course after delayed fasciotomy. J Trauma 1996; 40: 342-4.
29. Kantarci G, Vanholder R, Tuglular S, Akin H, Koc M, Ozener C et al. Acute renal failure due to crush syndrome during Marmara earthquake. Am J Kidney Dis 2002; 40: 662-89.
30. Matsuoka T, Yoshioka T, Tanaka H, Ninimiya N, Oda J, Sugimoto H et al. Long – term physical outcome of patients who suffered crush syndrome after the 1995 Hanshin – Awaji earthquake; Prognostic indicators in retrospect. J Trauma 2002; 52: 33-9.