UDK 616-001.32-083.98
COBISS.SR-ID 110170889

ISSN 2466-2992 (Online) (2022) br. 2, p. 15-24

TREATMENT OF PATIENTS WITH CRUSH INJURY

TRETMAN PACIJENATA SA KRAŠ POVREDAMA

Biljana Stošić [1,2], Vesna Marjanović [1,2], Marija Stošić [3], Jelena Živadinović[2], Ivana Budić [1,2]

[1]University of Niš, Faculty of Medicine, [2]Clinic for Anesthesiology and Intensive Care, University Clinical center Niš, [3]Clinic for Cardiac Surgery, University Clinical Center Niš

Summary: It was observed that, in road traffic accidents extremities were the most affected parts of the body (41% lower extremity and 21% upper extremity). Superficial injuries were found to be most common (47%), followed by fractures (20%), crush injuries (14%) and concealed injuries (12.4%). Major reason of morbidity and prolonged hospital stay after traffic accidents is musculoskeletal injury. Victims who survive after the major traumatic injuries can succumb to the various life threatening complications. Most of these injuries are directly related to the bone and soft tissue injuries. Goal of an anesthesiologist is to address initial musculoskeletal insult and treat or avoid secondary complications. Every trauma patient has a possibility of crush injury and they should be searched for the same thoroughly. Early diagnosis and treatment of problems like hypovolemia, crush syndrome, rhabdomyolysis can avoid further metabolic, cardiovascular and renal complications. Thus early diagnosis and management of crush injury can modulate the overall outcome of a trauma patient. This management of crush injury patient is a multidisciplinary work and requires good interdepartmental coordination for successful outcome. More sophisticated biochemical studies and devices to measure intercompartmental pressures are required to avoid unnecessary fasciotomies.

Key words: Crush injury, treatment, hypovolemia, rhabdomyolysis

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 2

INTRODUCTION

It was observed that, in road traffic accidents extremities were the most affected parts of the body (41% lower extremity and 21% upper extremity). Superficial injuries were found to be most common (47%), followed by fractures (20%), crush injuries (14%) and concealed injuries (12.4%) [1]. Major reason of morbidity and prolonged hospital stay after traffic accidents is musculoskeletal injury. Victims who survive after the major traumatic injuries can succumb to the various life threatening complications. Most of these injuries are directly related to the bone and soft tissue injuries. Goal of an anesthesiologist is to address initial musculoskeletal insult and treat or avoid secondary complications. Every trauma patient has a possibility of crush injury and they should be searched for the same thoroughly. Early diagnosis and treatment of problems like hypovolemia, crush syndrome, rhabdomyolysis can avoid further metabolic, cardiovascular and renal complications. Thus early diagnosis and management of crush injury can modulate the overall outcome of a trauma patient.

PATHOPHYSIOLOGY OF CRUSH INJURY

Crush injuries are caused by continuous prolonged pressure on the body. It involves mainly lower and upper extremities. Chest, abdomen or face can also have crush injuries. The direct pressure after crush injury causes muscle cell to become ischemic. Continuous pressure causes muscle damage resulting in an influx of fluid into the muscles resulting in edema and elevation in compartment pressure [2]. The cells then switch to anaerobic metabolism, generating large amounts of lactic acid. Prolonged ischemia then causes the cell membranes to leak. Continuous inflow of fluids (edema fluids, bleeding) or too little outflow (venous obstruction) can cause increased pressure in the compartment. This increased compartment pressure will cause tissue pressure to increase more than total capillary pressure and ultimately cause capillary collapse. All this will cause increased diffusion of fluid from the intravascular space to the extravascular space and lead to further increase in compartmental pressure. This edema-outflow obstruction – more edema vicious cycle if continued will hamper oxygenation of muscle tissue. If not treated early this will proceed to tissue ischemia, muscle necrosis and nerve dysfunction, eventually irreversible cell damage/death [3].

TOXINS RELEASED IN CRUSH INJURY

The toxin leak may continue for as long as 60 hours after the crush injury [4,5]. Some of these substances and their consequences are listed in Table 1.

Agent	*Effect*
Amino acids and other organic acids	Acidosis, Aciduria, Dysrhythmia
Creatinine phosphokinase (CPK)	Laboratory marker for crush injury
Free radicals, superoxides, peroxides	Secondary tissue damage
Histamine	Vasodilation, Bronchoconstriction
Lactic acid	Acidosis, Dysrhythmia
Leukotrienes	Lung injury
Lysozymes	Cellular injury
Myoglobin	Renal failure
Potassium	Renal failure, Dysrhythmia, Cardiac arrest
Thromboplastin	Disseminated intravascular coagulation

Table 1: Toxins released after crush injury

CLINICAL PRESENTATION

- Pain is the main presenting symptom and it is deep and aching in nature and is worsened by passive stretching of the involved muscles. Pain can be out of proportion to the injury [2].
- Immediately following extrication, a severe neurologic deficiency, mainly flaccid paralysis of the injured limb, may be present. Sensory loss to pain and touch is seen in a patchy pattern.
- Pallor, pulselessness and poikilothermia (hypothermia) may be present but they are usually late signs [6].
- Limb edema is initially not present. Gross edema takes time to develop and can progress to compartment syndrome.
- Distal pulses may be present even in the presence of gross edema. Investigation for additional injuries is warranted if pulses are not present.
- Uncontrolled bleeding in mangled extremities may be present and it can lead to severe hypovolemic shock and death.
- Even if skin and subcutaneous layers are not injured still the underlying muscles can be severely damaged.
- Associated injuries elsewhere may be present.
- Unlike the adult, the signs of hypovolemia or significant hemorrhage in a child are subtle and difficult to identify. The best early sign of hypovolemia in pediatric victims is a weak pulse as opposed to tachycardia in adults.

PREHOSPITAL CARE

• Prehospital care begins with first assessing trauma scene safety by the care provider. It is important to move the victim away from the trauma scene and to get medical aid as early as possible.

• The primary focus for trauma resuscitation in the field is airway, breathing, circulation, (the ABCs of the primary survey) and spinal stabilization.

• Shock, respiratory distress, and altered mental status are associated with high mortality and must be rapidly identified in the field with subsequent rapid transport to the nearest appropriate trauma/medical center.

• For entrapped victims, venous access can sometimes be established during extrication. For trauma victims in shock, venous access should be attempted during transport to the medical center [7].

• Resuscitation fluid of choice at trauma scene is Lactated Ringers solution. But if one suspects crush injuries then it is prudent to use isotonic saline for resuscitation fluid till victim reaches hospital as there are chances of fatal arrhythmias because of hyperkalemia.

• Tying of obviously bleeding vessels or applying direct pressure bandages on crushed or mangled extremities can stop ongoing hemorrhage

• Application of tourniquets above the injured extremity can itself cause limb ischemia hence routine use of tourniquets should be avoided. Tourniquet can be used temporarily in victim who is actively bleeding so that hypovolemic shock can be avoided before reaching hospital.

• Spinal stabilization, i.e. securing of a victim to a rigid spine support, of not only the cervical spine but whole spine is an important aspect of the prehospital care of trauma victims.

• Remember that the aim of primary resuscitation is not to treat the trauma but stabilization of the patient till transfer to hospital for treatment.

EMERGENCY MANAGEMENT

Emergency management is aimed at stabilization of hemodynamic status, treatment of crush injury and prevention of its complications. As patient arrives at EMS, airway, breathing, circulation and hemodynamic status should be checked. Secondary survey follows primary survey and associated injuries should be evaluated and treatment plan decided accordingly. Crush injury in more than one extremity should raise anticipation of crush syndrome. All routine investigations including serum electrolytes and urine for routine and myoglobin should be sent to laboratory after patient arrives at hospital.

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 2

STABILIZATION OF HEMODYNAMIC STATUS

Intravenous Fluids

The mainstay of treatment for crush injury is administration of intravenous fluids. At least two 14 or 16 G intravenous access should be established as soon as patient arrives at emergency area and fluid resuscitation should be started immediately. Initially a colloid or crystalloid such as normal saline is used. Potassium containing fluid, e.g. lactated Ringer's solution should be avoided in suspected crush injury patients as it may worsen hyperkalemia [8]. Once the patient is rescued from trauma site, it is critical to maintain a high urine output. Foley catheter placement is very important as it allows more accurate measurements of urine output as well as urine pH.

Treatment of Hyperkalemia

Mode of treatment used to treat hyperkalemia depends upon its severity. Administration of calcium gluconate is one of the fastest method to decrease blood potassium, but it will act only for a short period of time. Usual dose is 10 ml of 10 percent solution infused over 3 to 5 minutes. Insulin will shift extracellular K+ to intracellular side. Infusion of 50 g of dextrose combined with 10 units of insulin will decrease blood K+ immediately and effect will last for some hours. Sodium bicarbonate can be used in cases with severe hyperkalemia associated with metabolic acidosis. Other modalities which can be used are β2 agonists, loop diuretics, cation exchange resins like sodium polystyrene sulfonate and ultimately hemodialysis as last resort [9].

Alkaline Diuresis

Alkalinization of urine will increase solubility of myoglobin and promote its excretion. It also prevents oxidative damage resulting from cycling of myoglobin by stabilizing the more reactive ferryl form. Sodium bicarbonate will reverse the pre-existing acidosis that is often present and also treat hyperkalemia. It will also increase the urine pH, thus decreasing the amount of myoglobin precipitated in the kidneys. Ion trapping via alteration of urine pH may prevent the renal reabsorption of poisons that undergo excretion by glomerular filtration and active tubular secretion. Since membranes are more permeable to nonionized molecules than to their ionized counterparts, acidic (low-pH) poisons are ionized and trapped in alkaline urine, whereas basic ones become ionized and trapped in acid urine. Urine output of 3-6 ml/kg/hr and urinary alkalinization by adding sodium bicarbonate to an IV solution enhances the excretion of acidic toxins. Infusion of 12 L/day of normal saline with 50 mEq of sodium bicarbonate per liter of fluid will maintain an alkaline urine output of 8 L/day [8]. This can be achieved with intravenous fluids, mannitol, and sodium bicarbonate and furosemide at 1 mg/kg. Acetazolamide, 250 to 500 mg, may be used if the patient becomes too alkalotic. Alkalinization of urine is contraindicated in patients with congestive heart failure, renal failure, and cerebral edema. Acid-base, fluid, and electrolyte parameters should be monitored carefully. The patient with crush injury syndrome should maintain a urine output of at least 300 ml/h with a pH higher than 6.5.

Mannitol

Intravenous mannitol has several beneficial actions for the victim of crush injury. It protects the kidneys from the effects of rhabdomyolysis, increases extracellular fluid volume, and increases cardiac contractility. Mannitol can be given in doses of 1 gm/kg added to the patient's intravenous fluid as a continuous infusion. The maximum dose is 200 gr/24h; doses higher than this can cause renal failure. Mannitol should be given only after good urine flow has been established with IV fluids. Mannitol should be avoided in patients with congestive heart failure and pulmonary congestion as it may cause frank pulmonary edema. It is contraindicated in patients with active cranial bleeding and in patients with anuria. Electrolyte monitoring is essential during mannitol administration as it will cause excretion of many electrolytes including Na+, K+, Ca++, Mg++, Cl–, HCO3 and phosphate [10].

SURGICAL MANAGEMENT OF CRUSH INJURY

Wounds should be cleaned, debrided, and covered with sterile dressings in the usual fashion. Splinting the limb at heart level will help to limit edema and maintain perfusion. Application of the pneumatic anti-shock garment (PASG) should be avoided. The use of PASG has been reported to cause compartment syndrome and crush injury syndrome [11]. There are case reports of hyperbaric oxygen improving the outcome of victims of crush injury [12]. Treatment of closed crush injuries is conservative. They should not be routinely explored since the intact skin acts as a barrier against infection. The use of fasciotomies is controversial. Routine use is not to be advocated. Fasciotomies will not reverse muscle necrosis in the absence of compartment syndrome.

Complications after crush injury

- Hemorrhage and shock
- Hypothermia
- Hyperkalemia
- Acute compartment syndrome
- Rhabdomyolysis
- Acute renal failure
- Hepatic dysfunction
- DIC.

COMPARTMENT SYNDROME

Richard von Volkmann in 1872 was first to describe compartment syndrome. He proposed that "The paralysis is caused by too long continued isolation of the arterial blood" [13]. Compartment syndrome may occur in the abdomen, chest and face but the majority of cases are diagnosed within the extremities. The majority of cases of compartment syndrome (roughly 45%) are due to tibial fractures. These fractures generally involve high levels of energy with many being open fractures [3]. Compartment syndrome develops when increased tissue pressure in a myofascial compartment increases to a point that blood flow to the muscles and nerves is impaired. The resultant ischemia causes tissue and nerve damage leading to cellular death. Symptoms worsen acutely, and if the condition is not quickly reversed, individuals develop irreversible damage to nerves and muscles leading to permanent deficits. Treatment of compartment syndrome is emergency fasciotomy. Ideally it should be done before appearance of painlessness or paralysis in extremity [4].

MEASUREMENT OF INTRACOMPARTMENTAL PRESSURES

Mainly Stryker STIC Device (Stryker Corporation, Kalamazoo, Michigan) is used to measure the intracompartmental pressures [3]. The normal pressures within a compartment range from 0 to 4 mm Hg when muscle is at rest but during exertion it can rise up to 8 to 10 mm Hg. In normotensive patients cut off point for emergency fasciotomy is taken generally as 30 mm Hg. In hypotensive or hypertensive patients comparison with diastolic pressures is more justified. Whitesides postulated that a patient could be hypotensive and have a value less than 30 mm Hg but still have an elevated compartment pressure being within 20 mm Hg from the diastolic number [14]. Some authorities consider that field fasciotomy can increase chances of infection, bleeding and sepsis. It converts a closed injury to an open one, risking infection and sepsis. Several studies indicate a worse outcome in patients who received fasciotomy compared with those who did not. Hence it is advocated that fasciotomies should be done only in patients not responding to conservative and medical line of treatment [2,8,15].

RHABDOMYOLYSIS

Crush syndrome is a form of traumatic rhabdomyolysis that occurs after prolonged continuous pressure and characterized by systemic involvement [16]. Rhabdomyolysis is the breakdown of muscle fibers with leakage of potentially toxic cellular contents, e.g. in Table 1 into the systemic circulation [17].

PATHOPHYSIOLOGY

In rhabdomyolysis, there is extensive muscle breakdown and release of toxins in systemic

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 2

circulation, mainly myoglobin. Two crucial factors for development of myoglobinuric renal failure are hypovolemia and aciduria. Renal vasoconstriction with diminished renal circulation, intraluminal cast formation and direct heme protein induced cytotoxicity are main mechanisms behind heme protein induced renal toxicity [18]. It has been suggested that ARF is caused by tubular obstruction causing increased intraluminal pressures and thus opposing glomerular filtration. Other mechanism suggested is heme protein precipitated in kidneys itself providing substrate for generating toxic free radicals. The propensity for cast formation is determined by the pH, the filtered load of myoglobin and the flow through the renal tubules [19-20]. Heme causes free radical induced oxidative damage to the renal tubule. It has been suggested that myoglobin is central to the oxidative injury manifested as lipid peroxidation, and that this may be inhibited by an alkaline pH. Other reasons implicated in acute renal failure are, renal vasoconstriction secondary to circulatory shock and pigment nephrotoxicity [8].

CLINICAL FEATURES

- General: Malaise, fever, tachycardia, nausea and vomitings.
- Musculoskeletal: Pain, tenderness, paraesthesia, weakness
- Complications: Dark urine, oliguria, anuria, hepatic dysfunction, disseminated intravascular coagulation. Hepatic dysfunction occurs in 25 percent of patients with rhabdomyolysis. Proteases released from injured muscle lead to hepatic injury. ARF and diffuse intravascular coagulation are late complications, developing 12 to 72 hours after the acute insult [21].

Investigations

- Serum myoglobin levels
- Serial serum creatinine kinase (CK) levels
- Blood urea nitrogen
- Serum K+ levels
- Blood coagulation profile
- Urinalysis to determine myoglobin and CK

Management

Early diagnosis and treatment can prevent complications due to crush injury and rhabdomyolysis. Fluid replacement should start at the site of extrication of the trapped victim. Initial fluid should be preferably isotonic saline at the rate of 1.5 L/hr. It has been recommended as a prophylactic treatment [15]. Immediately after arrival of victim at hospital, we send all routine blood investigations to laboratory along with serum electrolytes. Hyperkalemia can develop within hours of crush injury and renal failure may develop. Patients often die of hyperkalemia unless they are treated rapidly. Other electrolyte imbalances which can be encountered are hypocalcemia and hyperphosphatemia. Arterial blood gases, blood and urine pH should be measured. Empirical 1 mEq/kg sodium bicarbonate can be given to decrease pre-existing acidosis; later alkaline diuresis can be instituted to avoid myoglobinuric renal failure. The patient with crush injury syndrome should maintain a urine output of at least 300 ml/h with a pH higher than 6.5 [8]. Mannitol can be given in doses of 1 gr/kg or added to the patient's intravenous fluid as a continuous infusion. The maximum dose is 200 gr/24h with continuous monitoring of urine output, urine pH, ABG, and serum electrolytes. Following algorithm can be used for a suspected rhabdomyolysis patient [22,23] (Figure 1).

ANESTHESIA IN CRUSH INJURY PATIENTS

Emergency fasciotomy, debridement, refashioning and amputation of involved extremity are some of the common surgical procedures required in crush injury patients. Muscle ischemia from acute crush injury can cause muscle necrosis within the 3-hour post-trauma period that was previously considered safe and irreversible tissue damage ensues after 8 hours of injury [5]. Hence it is very important to give emergency medical help at the earliest. If compartment pressures are elevated, fasciotomies should be performed. At the time of fasciotomy, extensive resection of all dead muscle should be performed at the first operation. Dead muscle cannot be identified by lack of bleeding.

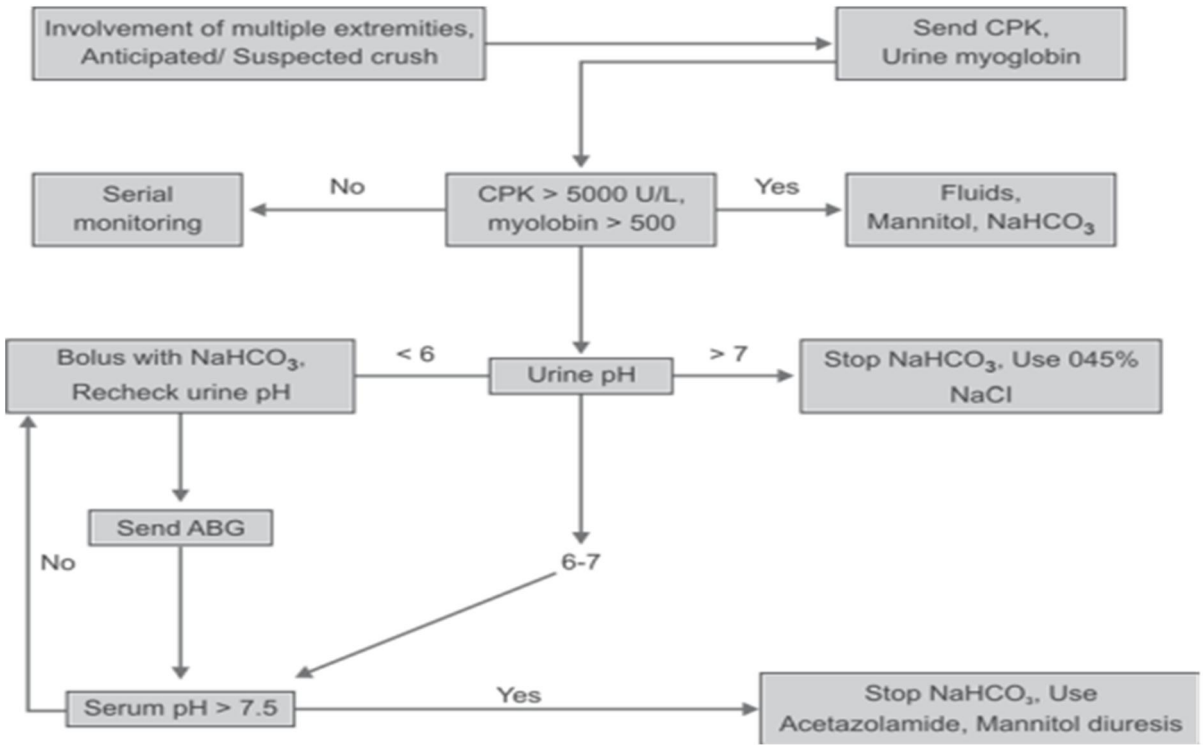

Figure 1. Treatment of rhabdomyolysis

Identification of dead muscle is by its reaction to direct physical or electrical stimulation. During preoperative assessment, along with general examination, volume status should be assessed. All patients with crush injury are required to be resuscitated and optimized well, before any surgical procedure. All patients are to be investigated with hemogram to estimate blood loss, serum electrolytes and renal functions.Hyperkalemia if present may lead to arrhythmias, which should be diagnosed and treated. Adequate cross matched blood and blood products are to be kept ready in blood bank. Additional investigations may be needed as per patients' medical status. Associated injuries should be noted. Regional anesthesia can be used for fasciotomy but patient may not cooperate because of associated injuries or deranged mental status. It may also be difficult to execute a regional block because of edematous extremities. Regional or Neuraxial blockade can be used for amputation cases where patient is hemodynamically stable and has no associated major injuries. Different peripheral nerve blocks like sciatico-femoral, popliteal, brachial plexus block can be used for such procedures. Neuraxial blockade is not advocated in hemodynamically unstable patients for both amputation and fasciotomy. One should avoid using adjuvants and intense blockade for fasciotomies as it is a short procedure, and it may obscure signs of inadequate fasciotomy for a long time. Hemodynamically unstable patient and associated major injuries will mandate general anesthesia with or without endotracheal intubation or laryngeal mask airway. Thiopentone sodium, propofol, ketamine can be used for induction of anesthesia before endotracheal intubation. These agents should be given in smaller titrated doses, in hypovolemic patients. One should avoid use of succinylcholine in extensive crush injury provided there is no anticipated airway difficulty as it can exacerbate existing hyperkalemia to dangerous levels. A short acting agent like atracurium or mivacurium may be used. Atracurium, cisatracurium, mivacurium are also beneficial in patients in ARF as these are least excreted by renal route. Maintenance of anesthesia is commonly done with inhalational agents or propofol infusion with opioids. It should be remembered that fasciotomy is a short procedure and hence

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 2

unnecessary long acting anesthetic agents should be avoided. Intraoperative monitoring includes continuous electrocardiogram, pulse oximetry, capnography, blood pressure and urine output. Central venous line helps in assessing adequacy of fluid status and also offers route for fluid and drugs administration, e.g. mannitol. Serum electrolytes and arterial blood gases estimations should be done during the procedure, if needed.

Intraoperatively, normovolemia and normal blood pressure should be maintained to optimize perfusion to ischemic tissues. Hypotension should be avoided. Hypertension, on the other hand, is associated with increased bleeding from muscles being excised. Most often, surgeries with crush injuries do not involve use of intraoperative tourniquets. When a patient arrives in the theater with a limb tourniquet, sudden blood loss and hypotension should be anticipated when they are removed at the start of surgical procedure. To avoid myoglobinuric renal failure intraoperatively, it is important to maintain central venous pressures, maintain alkaline pH of urine by producing alkaline diuresis, use of diuretics and mannitol during surgery. It is very important to monitor hemoglobin, serum electrolytes, serum myoglobin, serum CPK (if found raised initially) and blood coagulation during intraoperative period. Some authors suggest that serum myoglobin rather than CK levels should be used to guide therapy in such patients [23]. Thus early resuscitation, hemodynamic stability, adequate diuresis and prompt treatment of complications will help in better management of crush injuries.

PREVENTION AND TREATMENT OF ARF

Adequate intravascular volume expansion is the cornerstone of treatment of rhabdomyolysis. Exact amount of preloading is not known but it should be adequate to ensure good urine output. Alkalinization of as described above will prevent formation of pigmented casts in kidneys and thus prevent renal failure [22]. Diuretics can be used to maintain adequate urine flow as well mannitol can be used as described above. While using diuretics like mannitol patient should be adequately hydrated as these agents themselves can cause renal dysfunction otherwise. Early renal replacement therapy use in case of suspected acute renal failure had good results. Hemodialysis and continuous or intermittent peritoneal dialysis are modes available for renal replacement therapy [24].

SUMMARY

Thus management of crush injury patient is a multidisciplinary work and requires good interdepartmental coordination for successful outcome. More sophisticated biochemical studies and devises to measure intercompartmental pressures are required to avoid unnecessary fasciotomies.

REFERENCE

1. Verma PK, Tiwari KN. Epidemiology of Road Traffic-Injuries in Delhi: Result of Survey. Regional Health Forum- Volume 8, Number 1, 2004.
2. Gail T. Tominaga. Crush Syndrome. In: Demetrios Demetriades, Juan A. Asensio, (Eds). Trauma Management, Texas; Landes Bioscience 2000;61;618-22.
3. John FD. Compartment Syndrome of the Lower Extremity: Blast Injuries in a Ranger Battalion Don't Miss The Diagnosis. Journal of special operations medicine; ISSN 1553-9768;2006; Vol 6, Edition 2.
4. Darren J. Malinoski, Matthew S. Slater, Richard J. Mullins. Crush Injury and Rhabdomyolysis. Crit Care Clin 20 04;20: 171-92.
5. Christian Vaillancourt, Ian Shrier, Alain Vandal, Markus Falk, Michel Rossignol, Alan Vernec, Dan Somogyi. Acute compartment syndrome: How long before muscle necrosis occurs? CJEM 2004;6(3):147-54.
6. Wallace S, Goodman S. Compartment Syndrome, Lower Extremity. Emedicine 2005. Updated on Feb 9, 2009
http://www.emedicine.com/orthoped/topic596.htm
7. Samuel JS, Mark Eckstein. Prehospital Trauma Care; In: Demetrios Demetriades, Juan A. Asensio, (Eds). Trauma Management, Texas; Landes Bioscience 2000;1: 2-15.
8. Jason Smith, Tan Greaves. Crush Injury and Crush Syndrome: A Review; The journal of trauma 2003;54: S226-30.
9. Gary GS, Barry MB. Fluid and Electrolyte Disturbances. In: Anthony S Fauci, Eugene Braunwald, Dennis L Kasper, Stephen L Hauser, Dan L Longo, J. Larry Jameson, Joseph
10. Edwin K, Jackson Diuetics. In: Goodman LA, Gilman A, Hardman JG, Limbard LE (Eds). The Pharmacological

Basis of Therapeutics, 10th edn. New York; The McGraw Hill 2006;29:757-87.
11. Godbout B, Burchard KW, Slotman GJ, Gann DS. Crush syndrome with death following pneumatic anti-shock garment application. J Trauma 1984;24:1052-6.
12. James PB. Hyperbaric oxygen treatment for crush injury. BMJ 1994;309(6967):1513.
13. Hatch W, Hatch V. Richard von Volkmann and surgical school of Halle from 1867 to 1889. Zentralbl Chir 2001;126: 822-7.
14. Whitesides TE, Heckman MM. Acute Compartment Syndrome: Update on Diagnosis and Treatment; J Am Acad Orthop Sueg 1996;4:209-18.
15. Demirkiran, Y Dikmen, T Utku, S Urkmez. Crush syndrome patients after the Marmara earthquake. Emerg Med J 2003;20:247-50.
16. Hiraide A, Ohnishi M, Tanaka H, et al. Abdominal and lower extremity crush syndrome. Injury 1997;28:685-6.
17. Ana L Huerta-Alardín, Joseph Varon, Paul E Marik. Bedside review: Rhabdomyolysis—an overview for clinicians. Critical Care 2005;9:158-69.
18. Flamenbaum W, Gehr M, Gross M, Kaufman J, Hamburger R. Acute renal failure associated with myoglobinuria and hemoglobinuria. In: Brenner B, Lazarus J (Eds). Acute Renal Failure. Philadelphia: WB Saunders 1983;269-82.
19. Salahudeen A, Wang C, Bigler S, Dai Z, Tachikawa H. Synergistic renal protection by combining alkalinediuresis with lipid peroxidation inhibitors in rhabdomyolysis: possible interaction between oxidant and nonoxidant mechanisms. Nephrol Dial Transplant 1996; 11:635-42.
20. Zager R, Burkhart K. Differential effects of glutathione and cysteine on Fe2+, Fe3+, H2O2 and myoglobin-induced proximal tubule cell attack. Kidney Int 1998;53:1661-7.
21. Akmal M, Massry S. Reversible hepatic dysfunction associated with rhabdomyolysis. Am J Nephrol 1990;10: 49-52.
22. Brown, Carlos VR, Rhee, Peter , Chan, Linda, Evans, Kelly, Demetriades, Demetrios, Velmahos, George C. Preventing Renal Failure in Patients with Rhabdomyolysis: Do
23. Bicarbonate and Mannitol Make a Difference? The Journal of Trauma: Injury, Infection, and Critical Care 2004; 56:(6)1191-6.
24. Helena Lappalainen, Eero Tiula, Lasse Uotila, Matti Manttari. Elimination kinetics of myoglobin and creatine kinase in rhabdomyolysis: Implications for follow-up. Crit Care Med 2002;30:2212-5

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 2

TRETMAN PACIJENATA SA KRAŠ POVREDAMA

Biljana Stošić [1,2], Vesna Marjanović [1,2], Marija Stošić [3], Jelena Živadinović[2], Ivana Budić [1,2]

[1]Univerzitet u Nišu, Medicinski fakultet, Klinika za anesteziologiju i intenzivnu negu, [2]Univerzitetski klinički centar Niš, [3]Klinika za kardiohirurgiju, Univerzitetski klinički centar Niš

Sažetak: Primećeno je da su u saobraćajnim nezgodama na putevima, ekstremiteti najviše pogođeni delovi tela (41% donji ekstremiteti i 21% gornji ekstremiteti). Utvrđeno je da su površne povrede najčešće (47%), a slede prelomi (20%), kraš povrede (14%) i prikrivene povrede (12,4 %). Glavni razlog morbiditeta i produženog boravka u bolnici posle saobraćajnih nezgoda su povrede muskuloskeletnog sistema. Žrtve koje prežive posle velikih traumatskih povreda mogu da podlegnu raznim komplikacijama opasnim po život. Većina ovih povreda je direktno povezana sa povredama kostiju i mekog tkiva. Cilj anesteziologa je da se pozabavi početnom muskuloskeletnom povredom i leči ili izbegne sekundarne komplikacije. Svaki pacijent sa traumom ima mogućnost za postojanjem kraš povrede i treba ih detaljno potražiti. Rana dijagnoza i lečenje problema kao što su hipovolemija, kraš sindrom, rabdomioliza mogu da izbegnu dalje metaboličke, kardiovaskularne i renalne komplikacije. Tako rana dijagnoza i tretman kraš povrede mogu da utiču na ukupan ishod traumatizovanog pacijenta. Tretman pacijenta sa povredama je multidisciplinarni rad i zahteva dobru koordinaciju timova u cilju uspešnog ishoda lečenja. Sofisticiranije biohemijske studije i uređaji za merenje interkompartmanskih pritisaka su potrebni da bi se izbegle nepotrebne fasciotomije.

Ključne reči: kraš povreda, lečenje, hipovolemija, rabdomioliza

Korespondencija/Correspondence

Biljana STOŠIĆ
Univerzitet u Nišu, Medicinski fakultet
Klinika za anesteziologiju i intenzivnu negu
E-mail: b.stosic@yahoo.com