REVIEW ARTICLE
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Crush Syndrome and Nursing Care Management*

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ABSTRACT

Crush syndrome is a medical condition that occurs after a crushing injury to skeletal muscle, and is characterized by major shock and kidney failure. The key clinical features of crush syndrome are crushing injury to a large mass of skeletal muscle, increased permeability of the cell membrane, and the release of potassium, enzymes, and myoglobin from within cells. The symptoms of crush syndrome include myalgia, generalized weakness, and darkened urine. To prevent the complications that may occur due to rhabdomyolysis and compartment syndrome, importance is had in ensuring the casualty maintains adequate circulatory volume and providing adequate diuresis. Nursing care is very important in planning the treatment and care to be applied to the patient and in monitoring treatment effectiveness.
Keywords: Nursing, crush syndrome, rhabdomyolysis, compartment syndrome, management

INTRODUCTION

Crush syndrome is when an environmental factor exposes the torso, extremities or other parts of the body to direct physical trauma through a crushing or compressing force (1). As a result of the deterioration of muscle tissue integrity regarding crush syndrome, myoglobin, potassium, and phosphorus are released into the bloodstream. This syndrome is typically characterized by hypovolemic shock and hyperkalemia. If left untreated, acute renal failure can also accompany this (2).

While traffic accidents, wars, avalanches, and landslides are among the causes of crush syndrome, it is also often seen to result from being trapped in a void after an earthquake (3). Crush syndrome, is one of the complications seen after an earthquake, and was first recorded in 1909 with the Messina earthquake. Fatigue, muscle swelling, and brown urine were reported to have been observed in the survivors of the earthquake, and most of these people died in the hospital (4, 5). The cause of this condition, known as crush syndrome, was first reported by nephrologist Dr. Bywaters in 1940. Bywaters followed up on patients who'd survived the London bombing of 1941 after being under the rubble for 3-4 hours and who had no injuries other than a limb impingement. In his observations of these patients, he noticed a decrease in blood pressure, a decrease in the amount of urine, and an increase in the level of blood urea to cause crush syndrome, as well as the release of some harmful substances in the oppressed muscle groups (6). In the years following Bywaters' determinations, other important developments were recorded in the fight against crush syndrome. The death rate due to crush syndrome, which had been %91 during World War II, was observed to decrease to 50% during the Vietnam War years (7, 8).

Crush syndrome is assumed will develop in approximately 2-5% of all injuries, and with acute renal failure developing in 1.5% of all injuries resulting from earthquakes (9). Türkiye is on significant fault lines and has experienced many earthquakes over the last century, in particular the 1939 Erzincan Earthquake, the 1999 Adapazarı and Düzce Earthquakes, and the very recent Kahramanmaraş Earthquake, with many lives lost in all of these. Although the crush syndrome rate has not yet been determined regarding the Kahramanmaraş Earthquake, this rate was reported as 1.4% in the 1999 İzmit Earthquake (10). The importance of nursing and multidisciplinary approaches has been understood once again due to the sequelae and psychological support needs of injured people (11).

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Pathophysiology

Rhabdomyolysis is defined as the occurrence of various pathologies in the systemic circulation as a result of the breakdown of muscle cells due to crush syndrome and involves the mixing of intracellular electrolytes, myoglobin, and other sarcoplasmic proteins into extracellular spaces. Although crush syndrome is the main cause of rhabdomyolysis, rhabdomyolysis may not always occur as a result of muscle trauma and may not lead to acute renal failure (12).

The etiology of rhabdomyolysis can be divided into two main groups non-physical and physical causes.

Non-physical causes: These include alcohol and drug use (e.g., statins), electrolyte disturbances (especially hypokalemia and hypophosphatemia), and infections.

Physical causes: These include earthquakes, traffic and mining accidents, electric shocks, excessive exercise, and muscle compression (baromyopathy) as a result of staying in certain positions for extended periods of time. Baromyopathy involves impairment of the permeability of the muscle cell membrane. While substances such as potassium, myoglobin, and creatine, which are found in large amounts in the muscle, pass into the extracellular environment, sodium, chloride, calcium, and water are filtered into the cell; thus, cell edema develops that leads to compartment syndrome (9,13).

Rhabdomyolysis has multiple causes. The main cause is direct myocyte damage or disruption of the energy supply in muscle cells. Muscle cells keep intracellular calcium levels low through the sodium-potassium adenosine triphosphatase (Na-K ATPase) pump and voltage-dependent sodium-calcium (Na-Ca) exchange channels. The Na-K ATPase pump provides active transport of intracellular sodium to the extracellular space. The gradient that occurs through the excretion of sodium into the extracellular space causes calcium to concentrate in the sarcoplasmic reticulum and mitochondria. In addition to the muscle necrosis that occurs under an overwhelming load, traumatic rhabdomyolysis may also occur, especially with reperfusion after the removal of the overwhelming load. Reperfusion causes blood, sodium, and inflammatory mediators in particular to reach the damaged tissue and form free radicals. The resulting free radicals and ATP supply-demand mismatch cause an increase in intracellular calcium levels and disruption of cellular transfer mechanisms. Meanwhile, increased calcium levels, activate proteolytic enzymes and phospholipases, disrupting muscle cell structure. Apoptosis and cell lysis occur through induced hypoxia. Due to cell destruction, enzymes and electrolytes such as potassium, myoglobin, creatinine kinase, phosphate, uric acid, lactate dehydrogenase, and aspartate transferase increase in the serum. When increased myoglobin levels exceed the plasma binding capacity, myoglobin transforms into glomerular precipitates that can cause acute kidney injury. Acute kidney injury and an accompanying electrolyte imbalance occur due to traumatic rhabdomyolysis (13, 14).

Another important consequence of crush syndrome is compartment syndrome, which is an acute condition that may require surgical intervention, and result in increased pressure in the area surrounded by facial membranes and the disruption of tissue function in that space (15, 16). Acute compartment syndrome (ACS), is the general name of the group of major traumas that include increased pressure in the muscle compartment of the relevant extremity, impaired extremity vascularization, and long bone fractures. In addition to major trauma, ACS can occur following minor trauma or non-traumatic causes and is usually seen on the forearm, gluteal region, thigh, and foot. The common symptom seen in all cases is cellular anoxia (15).

Clinical signs

In patients with crush syndrome, clinical findings mainly occur in the form of traumatic rhabdomyolysis with localized symptoms occuring in the oppressed muscle, as well as systemic findings due to substances released from these muscles.

The increase in the number of enzymes such as myoglobin, potassium, magnesium, phosphate, acids, creatine phosphokinase (CPK), and lactate dehydrogenase (LDH) leaking into the circulation as a result of crushing has a toxic effect on the body. Traumatic asphyxia, hypovolemia, crush injury to the extremities, and organ injury may occur in adults with a severe crush injury. In addition, acute kidney injury and acute respiratory distress syndrome can be seen as the sequelae of crush injury.

Traumatic asphyxia: This is seen to occur due to severe crushing of the chest. An increase in thoracic pressure and pressure in the superior vena cava can cause ruptures in the head and neck capillaries. Cervicofacial cyanosis, edema, subconjunctival hemorrhage, and petechial eruptions on the face, neck, and trunk may also occur.

Hypovolemia: Hypovolemic shock may occur, usually within the first few hours after a crush injury. This can occur as a result of a damaged blood vessel from an injured extremity or as a result of occult bleeding due to an organ injury. Understanding and treating the cause of hypovolemic shock is very important for preventing the development of acute kidney injury.

Crush injury to the extremities: Crush injury to the extremities may present clinically in a spectrum ranging from diffuse swelling and erythema to blisters and purpura, open fractures, and ischemia. In addition to the management of the anticipated orthopedic and vascular problems associated with such injuries, awareness of the potential these patients have for developing acute compartment syndrome and rhabdomyolysis should be considered. Compartment syndrome has typical findings that can be summarized as 6P (i.e., pain, pressure, paresthesia, pulselessness, paresis, and pallor). Prolonged compression can lead to cell death, leading to muscle necrosis and rhabdomyolysis.

Organ injury: Blunt injury to the thorax or abdomen may cause injuries such as pulmonary contusion, cardiac contusion, rib fractures, pelvic fracture, hemothorax, and pneumothorax.

Acute kidney injury: In addition to the direct nephrotoxic effects of the breakdown products resulting from crush syndrome, acute kidney injury may also occur as a result of myoglobin and urate crystals contributing to acute tunnel necrosis or from and due to hypotension and hypoperfusion. Crush-related acute kidney injury manifests as rhabdomyolysis and myoglobulinemia, hyperkalemia, hyperphosphatemia, and myoglobinuria. Elderly individuals and those with chronic kidney disease are at increased risk for progression to end-stage kidney disease. Even under optimal conditions, the risk of dialysis is about 10%. Indicators of the need for dialysis are shown in Table 1 (14, 17, 18).

Acute respiratory distress syndrome. Acute respiratory distress syndrome can also occur following a severe crush injury. Large-volume crystalloid resuscitation may be associated with an inflammatory response to injury, tissue necrosis, or fat embolism syndrome associated with long bone fractures.

Managing Crush Syndrome

Medical care of the injured is handled in three stages: removal from the wreckage, removal from the wreckage (on-site intervention), and post-removal from the wreckage (intervention in a health facility). In addition, three main methods are applied to prevent or minimize the severity of acute kidney injury: intravenous (IV) fluid hydration, alkalization, and forced diuresis under special conditions. In the treatment of crush syndrome, administering IV sodium bicarbonate ($NaHCO_3$), increasing the urine pH above 6.5, and preventing renal toxicity are among the main goals (19).

On-site management

The ABCDEs of first aid

Severe respiratory failure may occur once the injured has been exposed to blunt chest trauma. In this case, they may need oxygen, airway patency, and the use of a ventilator. Circulatory collapse and hypovolemia may also be seen in injured who've been unable to be removed from the wreckage for a long time (20).

First aid for the injured involves the following steps:

A (Airway): Airway patency should be checked, and the cervical spine should be protected by wearing a collar.

B (Respiratory): Breathing should be checked, and the casualty should be kept away from dust by having them wear a dust mask. Safe intubation can be difficult in the field and oxygen may not be given for safety reasons. Administering analgesia to injured persons with vertebral fractures may facilitate breathing.

C (Circulation): Circulation should be checked, and the bleeding should be brought under control if present; vascular access should also be opened immediately. The casualty should be hydrated. Liquids containing potassium should be avoided.

D (Defect): Stabilization of the spine should be ensured, and a brief neurological evaluation should be made.

E (Full Body Examination): Injuries may be overlooked when examining a clothed victim. If one is to save the wounded's life, they should be stripped down. In order to prevent hypothermia, the casualty should be redressed and covered after the examination.

Triage and transport

Triage in the field usually consists of three categories, giving priority to individualized care and transport.

• The first category involves –urgent- casualties. Injuries in this category are treated and transferred to a health institution before others.

• The second category involves –delayed- casualties.

• The third category involves –minor- injuries. The casualties in this category are those who can be discharged with on-scene treatment and are the last to be transported (21).

In order to reduce death rates, injured are triaged with color codes according to the severity of the injury and their medical condition (Figure 1).

Triage is repeated at hourly intervals and the injured are referred to the appropriate triage area as their clinical status changes (16).

Vascular access should be established from an appropriate extremity as an emergency response while the injured is under debris. If a vein is not found and the casualty can tolerate it, oral fluid intake should be provided. After establishing the appropriate vascular access, an infusion rate of 0.9% NaCl (saline) should be started at a rate of 1000 cc/h in adults and 10 cc/kg in the elderly until 2 cc/kg/h of urine is produced. If more than 2 hours is required to remove the injured from the wreckage, the infusion rate should be reduced to 500 cc/h (1, 14).

When the injured is removed from the debris, a 1000 cc/h infusion of 0.9% NaCl should be continued. The casualty should be observed for 6 hours from the start of the infusion. If anuria is observed and the casualty is normovolemic, infusion (at 500-1000 cc/day plus insensible losses) should be continued and dialysis should be prepared. If the casualty has sufficient urine output and close observation is possible, the infusion should be continued (at ≥6000 cc/day,) and dialysis should be prepared (1). However, if a close observation environment is not possible, the infusion rate should be continued at 3000-6000 cc/day and the casualty should be made ready for dialysis (Figure 2) (14).

Table 1: Indications of the Need for Dialysis

1. Rapid elevation of serum potassium of 6.5 mmol/L or more or unresponsive to other measures
2. Metabolic acidosis: blood pH ≤7.1
3. BUN level ≥100 mg/dL (30 mmol/L) or serum creatinine ≥8 mg/dL (700 mmol/L)
4. Uremic syndromes such as hypervolemia, pericarditis, bleeding, or other unexplained disturbances of consciousness

Figure 1. Triage colors.

Hospital management

The first aid given in the field should be transferred to the health institution where the injured is sent. Extra trauma to the casualty should be avoided by following the trauma management procedure during the referral. Until the casualty is stabilized, several clinical examinations should be performed and laboratory findings (e.g., sodium, potassium, calcium, phosphate, bicarbonate, creatinine and lactate, and/or arterial blood gases) should be checked (22).

In cases where serum electrolyte measurement is impossible, solutions containing potassium should not be administered under any circumstances. Due to the risk of hyperkalemia, the injured should be taken to cardiac monitoring, and the necessary analgesia should be provided. If hyperkalemia has developed in the injured, NaHCO3 can be given between 50-100 mEq/L, or 2.5 mg of albuterol can be administered in 3 ccs as another option (19).

When considering nephrotoxicity, antibiotic treatment should be started to provide optimum benefit and the urine output of the casualty should be checked at frequent intervals. If the injured is able to urinate, he should be asked to urinate first (1,23). If the casualty is unconscious, the Foley catheter should be inserted as quickly as possible. For urine alkalization, the urine pH is recommended to be kept above 6.5 with an IV of 50 mmol NaHCO3. Target urine output should exceed greater than 50 cc/hour for adults. After urine output has been established, 20% Mannitol at a dose of 1-2 g/kg for the first four hours should be given to maintain a urine output of at least 8 liters per day (300 ml per hour). The maximum dose of mannitol is 2 grams per kilogram in 24 hours and should not be used if the patient has severe heart or kidney failure (19).

For a casualty who is unable to urinate, the symptoms of hypovolemia (e.g., low blood pressure, filiform pulse, cold sweating) should be evaluated first. If these findings are present, hypovolemia should be treated, and if the patient is unable to urinate, IV fluids should be started (9). Intervention should be made to the relevant extremity of at wound with a suspected fracture (23).

In patients who develop compartment syndrome as a result of crush syndrome, an indication of fasciotomy may arise (24).

Irreversible nerve damage may occur after the fourth hour, and irreversible muscle damage may occur after the sixth hour. Another important point is tetanus toxoid. The administration of the tetanus vaccine to injured people should never be skipped (25).

Nursing approach

The nursing approach to the injured in the field and in the hospital is very important in crush syndrome. Nurses should deal with the injured individual and their family holistically and should provide appropriate nursing care from the first encounter to prevent the severity of crushing and death, accelerate the healing process and prevent complications.

Nursing approach to the injured in the field

• During the first intervention, the nurse should inform the conscious casualty about all the attempts that are to be made.

• The casualty's personal information should be recorded. Vital signs should be closely monitored.

• The casualty should be kept at a suitable temperature and in a suitable environment, and protected from the cold. Tight-fitting clothing should be avoided.

• The injured's state of consciousness should be closely monitored. Necessary permissions should be obtained from the relatives of an unconscious injured person.

• In order to prevent acute kidney failure that may develop on the basis of hypovolemic shock, crush syndrome, and rhabdomyolysis, 1000 cc of 0.9% NaCl solution IV per hour should be started upon the accessible free extremity of the casualty.

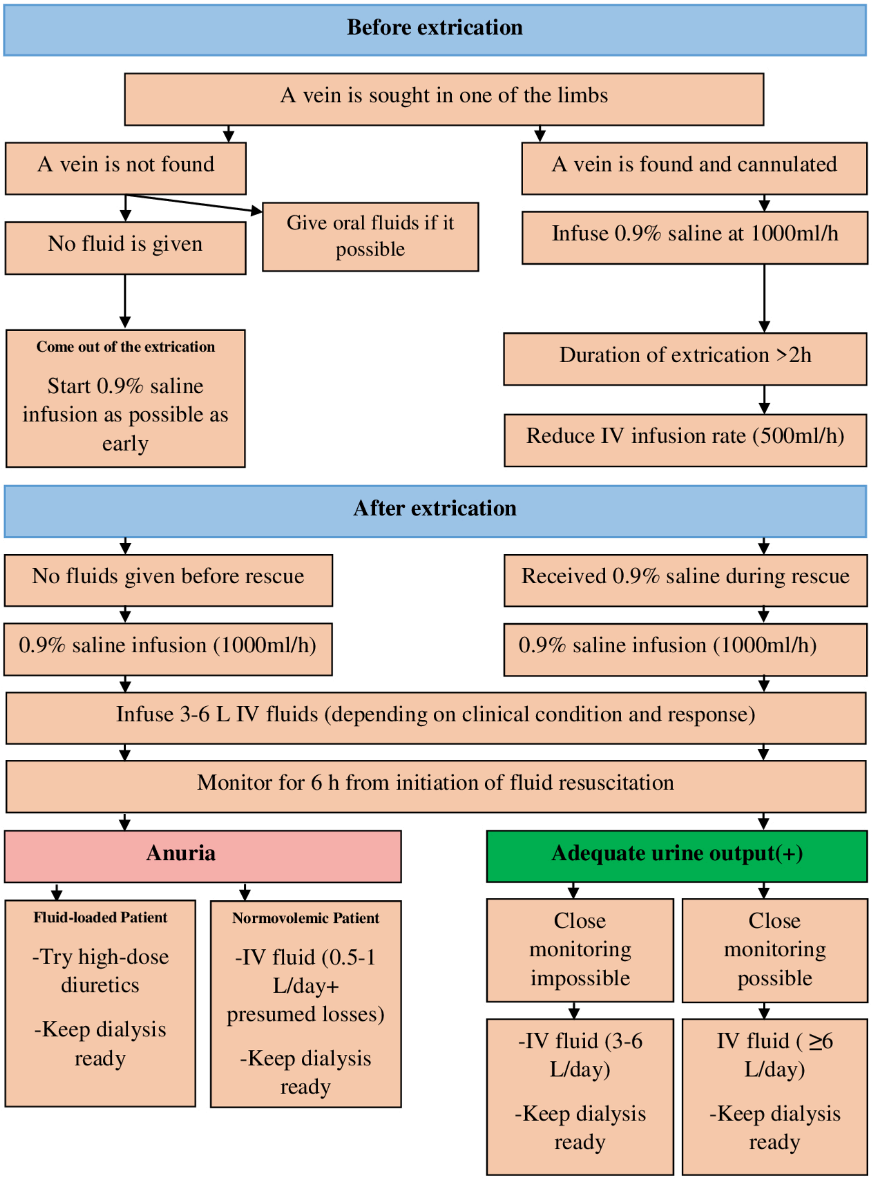

Figure 2. Algorithm for fluid resuscitation in crush injuries for mass disasters pre- and pos-extrication.

• Checking the extremities (e.g., skin color, temperature) at frequent intervals is important. The position of an extremity with an oppressed muscle should be such that it does not obstruct blood flow. In addition, position changes should be made at certain intervals, and active-passive exercises should be applied in a way that does not overexert the injured.

Nursing approach to the injured in the hospital

• Vital signs and laboratory findings (e.g., creatinine, BUN, hematocrit, serum myoglobin, sodium, potassium, SGOT, and SGPT) should be closely monitored.

• Monitoring the edema of the injured, performing skin care regularly, and keeping the skin moist is important.

• Care should be taken against the signs and symptoms of metabolic acidosis (i.e., apathy, disorientation, weakness).

• Culture samples (e.g., catheter tip, urine, blood) should be taken at regular intervals.

• ECG of the casualty, fluid intake and output, urine amount, color, and pH should be monitored and alkalization of urine pH should be ensured.

• Bleeding signs and symptoms should be monitored (e.g., petechiae, ecchymosis, hypotension, tachycardia, hematuria) and abnormal conditions should be reported to a physician.

• A diet low in protein and high in carbohydrates should be applied and foods containing phosphorus should be restricted (dairy and dairy byproducts, chocolate, meat and meat byproducts, legumes, and fruit).

• Oral care should be done at frequent intervals for the casualty's optimum comfort and nutrition.

• Maintenance of urinary catheter (every 72 hours), central venous pressure (every 72 hours), peripheral venous catheters (every 48-72 hours), and arterial catheter (every 96 hours) should be done regularly.

• The injured should be followed up frequently in terms of the risk of infection due to invasive procedures, catheters, and open wounds (e.g., redness, swelling, discharge, bad smells, fever at the catheter site, leukocytosis, and burning sensation during urination).

• In case of infection, antibiotic treatment should be started in line with the physician's request.

• If the casualty is fit for activity, their activities should be increased gradually, and a routine with rest periods should be established, early ambulation should be encouraged.

• Monitoring the nephrotoxic effects of the drugs on an injured who has undergone an intensive treatment program is also important (9, 17, 26).

CONCLUSION

Crush syndrome is an important health problem for both injured and healthcare professionals. In the management of crush syndrome, first aid and intervention in the field, as well as intervention in a health institution, should be handled with a multidisciplinary approach. Close monitoring of the casualty is required during removal from the wreckage, at the time of contact in the field, and from the emergency room to the intensive care environment. In order to prevent complications that may occur due to rhabdomyolysis and compartment syndrome, which are seen to result of from crush syndrome, maintaining an adequate circulatory volume and providing adequate diuresis are important. The nurses who, are an important part of the multidisciplinary team and the health professionals who are with the patient 24/7 should be knowledgeable about crush syndrome and its management and should be able to use this knowledge and experience in its proper place and time.

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