



Treatment and Follow-up for Earthquake Victims in the Intensive Care Unit

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ABSTRACT

Our world has been afflicted by natural disasters numerous times throughout the ages, and consequently, several morbidities and mortality have been reported. Earthquakes are natural disasters with widespread and long-lasting effects on the affected population and different clinical complications. Crush syndrome (CS) is observed in most patients followed up in the intensive care unit (ICU) after earthquakes. In CS, traumatic rhabdomyolysis, hypotension due to decreased fluid volume in the intravascular space, hyperkalemia, and renal failure due to hypovolemia can ensue. Rhabdomyolysis releases intracellular potassium, phosphorus, nucleic acids, and myoglobin into the circulation. The most important laboratory finding was hyperkalemia. Acute renal failure, compartment syndrome, disseminated intravascular coagulation, and metabolic disorders may be observed because of rhabdomyolysis. Pulmonary complications, infections, and other medical complications may develop in patients with CS. It is important to start treatment at the site of trauma before reaching the hospital, to closely monitor the patient during treatment in the ICU, and to treat patients with a multidisciplinary approach to reduce mortality and morbidity after earthquakes.

Keywords: Anesthesiology and reanimation, Crush syndrome, earthquake, intensive care unit

Introduction

Our world has been afflicted by natural disasters numerous times throughout the ages and consequently has suffered materially and psychologically (1). Several definitions summarize the wide-ranging effects of disasters. The World Health Organization defines disaster as an event that causes suffering at a level to a society exceeding its capacity to cope and disrupts normal functioning. The common point of the different definitions is that a disaster disrupts normal functioning and causes suffering and losses that exceed the coping capacity of a region (2). Earthquakes are major natural disasters that cause a range of widespread and

long-lasting effects on the affected population, from injuries and infectious diseases in the acute period to disability and diseases such as anxiety and depression affecting mental health (3). The provision of appropriate care to critically ill patients in disaster situations is of vital importance. In intensive care unit (ICU) practice, it is important to define critical patients and to recognize and identify patients who will be prioritized for admission to the ICU to minimize patient mortality during both resuscitation and treatment. It is important to determine the emergency status of patients for admission to the ICU according to acute needs and risk levels. Disease severity, functional status, comorbidities, age, and other factors are important in the decision for

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admission. The Society of Critical Care Medicine admission, discharge, and triage guidelines recommend hospitalization of patients admitted to the ICU according to 2 priority levels (4). Higher priority is given to patients who require active life support for organ failure, including mechanical ventilation vasopressor invasive hemodynamic monitoring, and other similar treatments. This priority level includes patients requiring critical care such as continuous renal replacement therapies, extracorporeal membrane oxygenation, and intra-aortic balloon pumps, as well as patients with high urgency who have not yet developed organ failure. Lower priority patients include those with a significantly lower likelihood of recovery and those with advanced malignancy or chronic advanced organ failure (e.g. patients with metastatic cancer and respiratory failure secondary to pneumonia or septic shock requiring vasopressors).

In the immediate period following earthquakes, some disaster victims die in the disaster area because of the trauma suffered. Other victims die in the early and late periods depending on the degree and localization of the injury. Some are rescued and continue their treatment in the hospital. It is important that the treatment of earthquake victims starts at the site of the disaster. Prevention of metabolic acidosis and resulting hyperpotassemia is of vital importance for the survival of earthquake victims (5). Older age, admission to the ICU, severe traumatic brain injury, crush syndrome (CS), multiple organ failure and presence of cardiac/respiratory disease are known factors associated with the risk of mortality in patients hospitalized after an earthquake (6). The Injury Severity Score (ISS) has been used after disasters to define the severity of earthquake trauma. According to the ISS scoring, scores between 1 and 24 indicate mild to moderate injuries, scores between 25 and 75 indicate serious injuries, and a score of 76 indicates a fatal injury (5). After earthquakes, the types of trauma can differ depending on the infrastructural characteristics of the affected region. Different types of traumas, such as laceration, contusion, extremity, thorax, or closed abdomen traumas, can be observed. CS has been very frequently reported after the Marmara earthquake (7).

Crush Syndrome

Crush literally means compressing, being compressed, getting stuck, or being under pressure. Although these definitions only refer to the trauma itself, CS is a complex clinical state with muscle destruction (rhabdomyolysis) that occurs as a result of this trauma and surgical or medical signs and symptoms that develop afterward (1). In our country, 17,479 people died after the Marmara earthquake, and acute renal failure (ARF) developed in 639 of the 43,953 injured due

to crush injury (1,8). In both the Marmara earthquake and the great Turkey-Syria earthquake that occurred in 2023, it was determined that the highest mortality rate occurred in patients who developed CS (1,9). Crush syndrome after the rescue of disaster victims and the reperfusion of destroyed muscle cells that release substances into the systemic circulation with toxic effects on the other organs (10,11). The pathophysiology of CS occurs in 2 stages. The first stage is traumatic rhabdomyolysis or muscle cell destruction. In the second stage, hypotension, hyperkalemia, and renal failure resulting from the loss of fluids in the intracellular space and metabolic events following the release of muscle cell contents to the extracellular space ensue. In addition to the damage from volume deficiency, renal injury from rhabdomyolysis is the basic component of CS (12).

Different clinical presentations can be observed before and after admission to the ICU. The clinical presentations of CS can be analyzed in two components: local symptoms in the crushed muscles and systemic findings due to the substances released from these muscles. In the early period, compartment syndrome may be observed, especially in patients who receive intensive fluid therapy before admission to the hospital, while under debris, because of increased fluid passage into the damaged muscle groups. Newly occurring swellings may develop over the course of treatment in areas that were observed to be normal before admission to the hospital/ICU. This is also a sign of an increased risk of rhabdomyolysis. The findings of compartment syndrome can be summarized with the 6P signs of ischemia: pain, pressure, paresthesia, pulselessness, paresis, and pallor that may be observed in the limb under trauma (11,13). Different substances are released into the circulation in CS, with different effects on the body. A summary of the effects of the released substances is shown in Figure 1. Systemic findings vary according to the affected organ. The most common findings are hypotension/shock, cardiac and respiratory failure, and acute renal failure (ARF). ARF observed in these patients is more complicated than that observed due to other etiologies, with high morbidity and mortality resulting from both surgical and medical complications. Although the classical triad of symptoms of rhabdomyolysis is defined as weakness, myalgia, and tea-colored urine, this triad is observed in only 10% of all patients (14). In addition to these symptoms, non-specific findings may accompany rhabdomyolysis.

Intracellular potassium, phosphorus, nucleic acids, and myoglobin pass into the circulation because of rhabdomyolysis seen in CS. The most important laboratory finding was hyperpotassemia. The resulting arrhythmia



Figure 1. Systemic effects of substances released during Crush syndrome (5)

and heart failure are one of the most common causes of death. Hypocalcemia occurs as a result of the precipitation of released phosphorus and calcium crystals precipitating in soft tissues. Blood myoglobin values may be normal because of its short half-life, but they cause darkening in urine color, which we describe as cola-colored or dark red urine, and pigmented granular silt in urine sediment. Increased serum creatine kinase and lactate dehydrogenase (LDH) levels are signs of rhabdomyolysis. A correlation exists between creatine kinase levels and the degree of muscle damage. Anemia, leukocytosis, and thrombocytopenia are frequently observed. Anemia indicates traumatic bleeding or hemodilution, and leukocytosis indicates rhabdomyolysis and infections (10,15,16). In blood gas analysis, metabolic acidosis with an anion gap is often observed. Acute kidney injury (AKI) manifests as elevated serum urea and creatinine levels in patients with CS. Serum creatinine levels rise more rapidly than those in other renal pathologies. Serum creatine kinase (>5 times the upper limit of normal or >1000 IU/L), myoglobin, LDH, potassium, creatinine, and aspartate aminotransferase levels were elevated during rhabdomyolysis during ICU follow-up.

Serial creatine kinase measurements are important in patient follow-up, where peak concentrations are generally reached at 24-72 hours. Follow-up of creatine kinase can be stopped when a safe downward trend is observed (17,18). For treating rhabdomyolysis, early and etiology-directed treatment is important in terms of preventing the development of ARF, preventing extremity loss, and preventing life-threatening complications (Table 1).

Table 1. Complications due to rhabdomyolysis

Mechanical complications
Peripheral neuropathy
Compartment syndrome
Acute renal failure
Disseminated intravascular coagulation
Metabolic irregularities
Hyperkalemia
Hyperuricemia
Hyperphosphatemia
Hypercalcemia (late period)
Hypocalcemia
Hypophosphatemia (late period)

Table 2. Acute kidney injury kidney disease improving global outcomes classification

	Serum creatinine level	Urine output
Stage 1/risk	1.5-1.9 fold increase or ≥ 0.3 mg/dL increase	< mL/kg/hour during the 6-hours block
Stage 2/damage	>2-2.9 fold increase	< mL/kg/hour over two 6-hours blocks
Stage 3/failure	≥ 3 fold increase or >4 mg/dL	< mL/kg/hour for more than 24 hours or anuria for ≥ 12 hours

1. Acute Renal Failure

The treatment of ARF includes treatment of the underlying disease, prevention of rhabdomyolysis, fluid resuscitation, and renal replacement therapies. RIFLE and 2012 KDIGO (Kidney Disease: Improving Global Outcomes) criteria evaluated with serum creatinine (glomerular filtration rate) levels and urine output can be used in the diagnosis (19). It is recommended to start fluid resuscitation before the victims arrive at the hospital because fluid resuscitation provides adequate renal perfusion by increasing renal tubule flow and diluting nephrotoxins such as myoglobin. However, the best type of crystalloid to administer to patients remains controversial. Potassium-containing fluids are not particularly recommended. Because CS causes hyperkalemia, there is a concern that potassium in the fluid to be administered may worsen this condition. Lactated Ringer's solution and saline (0.45% or 0.9%) administered as crystalloids have not been found to be superior to one another. Because lactated Ringer's solution contains relatively high potassium, it is theoretically not recommended in CS with hyperkalemia. On the other hand, prolonged isotonic saline infusion may cause metabolic acidosis (16,18,20,21). In addition, clinical studies investigating the use of bicarbonate and/or diuretics to prevent the development of ARF or the role of mannitol in rhabdomyolysis are limited, with data lacking to reach a definitive recommendation. A recent comprehensive review emphasized that aggressive early-volume resuscitation with normal saline is currently recommended as the primary focus of treatment (18,22). Supporting data on the use of loop diuretics are limited to case reports (18). A central venous catheter should be inserted to appropriately administer fluid resuscitation to patients. Patient monitoring is also important. In addition, urine output is an important indicator in patients receiving fluid resuscitation. In cases where there is no urine output, the amount of fluid can be reduced to prevent volume overload. According to the KDIGO classification of ARF, conservative treatment is recommended in patients with stage 1 renal damage, conservative approach and renal replacement therapies should be considered in patients with stage 2 renal damage, and renal replacement therapies should be initiated in patients with stage 3 renal damage. Urgent dialysis indication should be considered in cases of uncorrectable metabolic acidosis, hyperkalemia that does not improve with treatment, other electrolyte disorders, the

presence of uremia symptoms, presence of hypervolemia with oliguria or anuria (Table 2) (16). Compartment syndrome and peripheral neuropathy are seen as mechanical complications of CS.

2. a. Compartment Syndrome

Massive transfer of sodium and calcium into damaged cells promotes ischemia, leading to local edema and an increase in intracompartmental pressure. With an increase in intracompartmental pressure, muscle tissue undergoes necrosis. The diagnosis of compartment syndrome is primarily clinical. The most reliable finding is pain. The presence of a pulse does not rule out the diagnosis. Medical fasciotomy with mannitol treatment for decompression may be performed, preventing or prolonging the need for a surgical fasciotomy. Surgical fasciotomy reduces intracompartmental pressure by making surgical incisions to the fascia of the injured muscle. Routine application of early fasciotomy may reduce the necrotic muscle mass, severity of renal failure, risk of ischemic contracture, and peripheral neuropathy, but increases the risk of infection. Compartment syndrome is a major risk factor for sepsis (16,22).

b. Peripheral Neuropathy

Because of edema of the affected muscle, peripheral nerves are compressed, which may cause ischemia of nerve tissues, leading to paralysis and paresthesia. Proximal nerves are frequently affected. Symptoms regress in a few days or weeks in most cases but may be permanent (16).

3. Metabolic Irregularities

Typical metabolic changes in rhabdomyolysis include hyperkalemia, metabolic acidosis, hypocalcemia or hypercalcemia, hyperuricemia, hyponatremia, and hyperphosphatemia with possible cardiac dysrhythmias (23). The most common and most important laboratory disorder in rhabdomyolysis is hyperkalemia, which can cause cardiac arrhythmias and heart failure. Hyperkalemia occurs in the early period of the disease, and close follow-up is required. Potassium levels should be checked serially and strictly. Cardiac monitoring with electrocardiogram is necessary in patients with high potassium levels (>6 mmol/L). Electrocardiogram changes that accompany severe hyperkalemia (Widening QRS, small p waves, and severe arrhythmias) should be evaluated.

Early treatment should be administered with insulin-glucose infusion and inhaled beta-2 adrenergic agents. If hyperkalemia is resistant to these treatments, it should be treated with cation exchange resins (kayexalate) or dialysis. Calcium gluconate or calcium chloride should be used to treat hyperkalemia in patients with serum levels of 7 mmol/L or in the presence of life-threatening dysrhythmias. Hypocalcemia occurs early in rhabdomyolysis because of the entry of calcium into damaged cells and the accumulation of calcium phosphate in necrotic muscle. Because hypoalbuminemia is common in these patients, corrected calcium levels should be calculated. Treatment of early hypocalcemia should be avoided unless patients are symptomatic or have severe hyperkalemia. Correction of hypocalcemia with calcium chloride or gluconate should be avoided because calcium deposition may occur in the injured muscle. In the recovery phase, serum calcium levels may return to normal, causing hypercalcemia due to calcium release from the injured muscle and secondary hyperparathyroidism due to ARF. Similar to hyperkalemia, hyperphosphatemia occurs as a result of phosphate release from damaged muscle cells. It can cause further problems when phosphate binds to calcium and accumulates in soft tissues (16,18,20).

4. Disseminated Intravascular Coagulation

Disseminated intravascular coagulation (DIC) syndrome is a syndrome characterized by systemic activation of intravascular coagulation, resulting in fibrin formation in the circulation and thrombus formation in small and medium-sized vessels. DIC is observed in the acute stages of trauma. Thrombotic plugs in the vessels may cause organ dysfunction and hemorrhagic complications with continuous depletion of platelets and clotting factors. The secretion of thromboplastin from the traumatized muscle is a probable cause of this complication. Sepsis is another stimulant in the development of DIC (5,16,24).

5. Infections

In CS, infections emerge in the late period and are a significant cause of late mortality. Patients with CS are prone to all kinds of infections. Infections may develop due to the presence of open and dirty wounds due to trauma in the early periods, immunosuppression developing because of rhabdomyolysis, surgical interventions performed under non-sterile conditions, prolonged ICU stay, or insufficient healthcare workforce. It is necessary to urgently clean the wounds of these patients, remove dead tissues, administer culture-guided antibiotherapy after the initial empirical treatment, and ensure that the selected antibiotics are not nephrotoxic. In addition, a tetanus toxoid booster dose should be administered to every patient (10,25).

6. Other Medical Issues

During the initial stages of trauma, the symptoms of pain may not be evident. However, narcotic analgesics and ketamine may be used for pain observed in patients in later stages and during ICU follow-up. The use of non-steroidal anti-inflammatory drugs is not recommended in this setting (10,26). Pulmonary complications are common after CS, especially with the effect of trauma. These may include infective (pneumonia, empyema) or non-infective conditions such as obstruction in the respiratory tract or hemopneumothorax. Acute respiratory distress syndrome (ARDS) is an important entity that increases mortality. ARDS may also develop due to sepsis. The most important gastrointestinal problems are peptic ulcer formation and subsequent bleeding, in which case ARF may worsen.

Patients who lose their relatives, homes, families, and sometimes their limbs develop very severe traumas because of the earthquake. Evaluations by different disciplines are necessary to solve the physical problems in these patients, as well as regular psychiatric evaluation and support for such patients who may have psychological traumas (10,26,27).

Conclusion

In conclusion, earthquakes are an important fact of our geography. To prevent earthquake-induced CS and related mechanical complications, (such as compartment syndrome, peripheral neuropathy), AKI, DIC, and other unwanted events, it is necessary to start treatment at the scene of trauma and then continue the treatment in the ICU. It is important that the patients treated and followed in the ICU who are closely monitored should have been assessed with a multidisciplinary evaluation that also addresses cognitive and psychological aspects. With these considerations, we can reduce morbidity and mortality.

Ethics

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: A.Y.A., Y.Y., Concept: A.Y.A., Y.Y., Design: A.Y.A., Y.Y., Data Collection or Processing: A.Y.A., Y.Y., Analysis or Interpretation: A.Y.A., Y.Y., Literature Search: A.Y.A., Y.Y., Writing: A.Y.A., Y.Y.

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