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Management of the crush syndrome in critical patients: 10 cases

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Abstract

Introduction: Crush trauma may be life threating in extremities. Crush syndrome leads to a systemic disorder through muscle cytolysis and the spread of metabolic substance into the circulatory system. In the present study, we summarized the follow-up and treatment of 10 cases with intensive care unit (ICU) crush injury.

Meterials and Methods: We have analyzed the clinical data of 10 patients with crush injury who were under treatment in the intensive care unit of our clinic. Age, sex, diagnosis, APACHE II score, sepsis, intensive care complications, treatment parameters in intensive care and arterial blood gases parameters, routine blood biochemistry, alanine aminotransferase, lactate dehydrogenase, creatine kinase, creatinine, existence of blood urea nitrogen, urinary protein and severity score of the patients have been recorded. Patients have been closely monitored for symptoms of crush injury, changes, crush area, urination and dangerous complications.

Results: The mean age of 10 patients (10 male) was 41.3 ± 8.7 years. APACHE II score was 21,7.8 out of 10 patients had traumatic shock, one showed acute renal failure and one presented with multiple organ dysfunction syndrome (MODS). In 3 patients presenting the criteria for crush syndrome, the symptoms of extremity distension and sensory function disorder were regulated with rapid surgical operation and hemodialysis, and urination increased, even in some patients, it reached the normal level. Serologic parameters were regulated in most of the patients after application. Amputation was applied to 5 (50%) patients in our group for serious infection and crush. 2 (20%) patients died, one because of MODS and one because of acute renal failure.

Conclusion: Early and aggressive resuscitation, emergency treatment and close monitoring of serious complications are of great importance for saving the lives of the patients with crush syndrome under intensive care.

Keywords: Crush Syndrome; Hemodialysis; Critical Illness.

INTRODUCTION

Crush syndrome, determined by Bywaters and Beall in 1941, generally presents when a part of the body is exposed to a high level of force or pressure, and is most commonly seen in extremities (1). Today, traffic accidents, earthquakes and mine accidents are among the leading causes.

Myoglobin, LDH, CK, potassium, phosphate, Mg, acids and enzymes, occurring as a result of cell lysis in the extremities exposed to trauma, are released into the systemic circulation. Potassium released into the systemic circulation may cause cardiac arrest together with the occurring metabolic acidosis. Myoglobin is the number one reason of acute renal failure, directly or indirectly (2). In the management of such patients under

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Corresponding Author Buğra Karakaş İnönü University Faculty of Medicine Department of Anesthesilogy and Reanimation Malatya, Turkey E-mail: karakasbugra@gmail.com intensive care requires a multidisciplinary approach. Therefore, the aim of the present study is to discuss the management of the patients with crush syndrome under intensive care.

MATERIALS and METHODS

In this study, 10 patients with dominant isolated extremity trauma, crush trauma characteristics secondary to multitrauma, who were treated in Intensive Care unit between 2013-2014, were retrospectively analysed. These 10 patients in the unit were those taken into operation from the emergency and then hospitalized. In the retrospective analysis, blood tests, arterial gas analyses, radiological analysis, monitoring, clinical analysis of internal, neurological and surgical examinations of the patients were evaluated. APACHE 2 and SAPS 2 scores were analysed within the first 24 hours. Blood, urine and tracheal cultures taken at the first hospitalization were analysed. Moreover, hospitalization duration under intensive care, blood gas, complete blood count and biochemical follow-ups, intensive care treatments, complications, intubation and mechanical ventilation renal replacement support, Erythrocyte suspension given to opened tracheostomy patients, Fresh Frozen Plasma, Albumin, Fibrinogen replacement, antibiotherapy and prognosis were evaluated.

RESULTS

The mean age of the 10 male patients were 41,3 (19-76). Mean body weight was 75,9 (57-91) kg. 7 patients experienced injury as a result of traffic accident in vehicle, and 3 patients as a result of traffic accident out of the vehicle. All patients had injuries and fractures in lower extremities. Totally 5 patients had leg amputation, and one of them was during the accident. Of the amputations, 3 were on the upper side of the knee and 2 were on the lower side of the knee. Only one of the patients had fasciotomy. The mean hospitalization duration under intensive care was 19,2 (3-68) days. 4 patients had tracheostomy opened. One of the patients did not need mechanic ventilation. 3 patients had hypertension as co-morbid disease. 3 sessions of hemofiltration was applied to one of the patients. The mean APACHE 2 score of the patients was recoded as 23,9 (9-37), while mean SAPS 2 score was 42,2 (17-71). Mean potassium value was measured as 3,9 (3,3-4,2), while mean phosphor value was 4,3 (6,6-3,2). Mean thrombocyte value of the patients was determined as 127500 (73000-153000). Totally, blood was injected to the patients from blood components; as 11 units of whole blood (WB), 125 units of erythrocyte suspension (ES), 81 units of fresh frozen plasma (FFP), 1 unit of thrombocyte apheresis; albumin 13500 ml and fibrinogen 20gr were also injected. Their distribution according to patients is shown in in the table (Table 1). 4 patients were discharged, 3 patients were transferred to orthopaedics, and 1 patient was transferred to plastics, while 2 patients died as a result of organ failure and acute renal failure.

DISCUSSION

With the tissue damage during crush syndrome, renal blood stream decreases and if this decrease results in at glomerular filtration level, prerenal failure develops in the first place. Debridation of the wound occurred as a result of trauma develops as another problem, reperfusion damage, together with the decrease in the pressure and building up of blood in the tissue again. Myoglobin, LDH, CK, potassium, phosphate, Mg, acids and enzymes, occurring as a result of cell lysis in the extremities exposed to trauma, are released into the systemic circulation. Potassium released into the systemic circulation may increase the possibility of arrhythmia and cause cardiac arrest together with the occurring metabolic acidosis. Myoglobin is the number one reason of ARF, directly or indirectly (1). Free oxygen radicals bound to ferric ion as a result of persistence of renal ischaemia and haem metabolism cause cellular toxic damage. Moreover, these radicals and myoglobulin decrease the oxide amount and cause renal vasoconstriction. This, afterwards, leads to renal ischemic damage. As a result of impaired Na-K-ATPaz mechanism in damaged cells, myoglobin degradation products, lactic acid, uric acid, creatine kinase, aldolase and phosphate, calcium and potassium respectively get into circulations. All together, these physiopathological

changes result in acute tubular necrosis and acute renal failure. The development of acute renal failure was tried to be prevented with appropriate fluid treatment in all patients under intensive care. Urinations were monitored hourly. In diabetic patients, blood glucose was monitored and treated. Glucose insulin protocol was applied to hypercalcaemic patients ($K^+ > 6$ mEql), and hemofiltration (CVVH) treatment was applied to only one patient. Due to tissue factors arising from damaged muscular tissue and infection, common intravascular coagulopathy may develop. This, in turn, increases the weight of acute renal failure. Serum haptoglobulin binds myoglobin, however, bounding capacity ends when approximately 100 g muscular tissue dies. While myoglobin is directly filtered by kidneys, it damages capillary endothelium and causes volume loss. Precipitate occurring in distal tubule causes obstruction and leads to renal failure, which in turn, createshaemodialysis indication (2).

Clinical course is more complicated in the patients with crush syndrome compared to ARF caused by other reasons.In these patients, both surgical (for example, complications related to bleeding, trauma and operation) and medical (for example, infections, cardiac arrhythmia related to hyperpotassaemia and hypocalcaemia, other organ and system failures) complications cause high level of morbidity and mortality.Adverse prognostic indicators are argued to be present in hyperalbuminemia or hypoalbuminemia (3-5).

Patients with crush syndrome under intense care were reported to develop haematological complications. Among these complications, the most common is disseminated intravascular coagulation (DIC). Sometimes DIC develops only related to rhabdomyolysis; even it can be seen in the patients with very mild rhabdomyolysis. Secretion of thromboplastin from traumatized muscles probably remains in the pathogenesis of this complication. In this patient group quite prone to infection, sepsis has an important place among the factors stimulating DIC development (5-6). Even if DIC does not develop, the need for substantial transfusion of blood and blood components (Erythrocyte suspension, whole blood, human albumin, fibrinogen, thrombocyte apheresis and fresh frozen plasma) in the treatment underlines how frequent the haematological complications are during the clinical course.

Since head, neck or chest crush quickly results in death, after these traumas Crush syndrome is not quite seen. Only 10% of the patients with chest trauma can be associated with crush syndrome. However, crush substantially increases the number of respiratory tract complications. ARDS most frequently remains among these complications.The development of ARDS is one of the most critical pulmonary complications, and fat embolism may be added to it. Hypoxia, hypercarbia, tachycardia and petechia in chest and neck present with crush injury and long bone fractures.Thromboplastin oscillated from thrombocytes may trigger DIC and respiratory insufficiency. In elders and patients with coronary failure history, aggressive fluid treatment may lead to pulmonary oedema (7-8). All patients we treated due to crash, except for one, were on ventilatory support with mechanical ventilator, even we had to open tracheotomy in one patient because of elongated intubation. Due to the fact that the development of infection adversely affects the prognosis, blood, urine and tracheal microbial cultures were taken from these patients at the first hospitalization and appropriate antimicrobial treatment was applied.

As a result, in the management of such patients under intensive care requires a multidisciplinary approach. With early and effective treatment of possible complications and appropriate resuscitation of fluid and blood component, it is possible to save these patients lives.

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