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REVIEW

A Comprehensive Review for Refreshing the Crush Syndrome Knowledge After the Devastating Earthquake in Türkiye

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ABSTRACT

On February 6, 2023, the southeastern region of Türkiye suffered from two consecutive devastating earthquakes with 7.6 and 7.8 magnificence. Since then, at least 44,324 deaths and approximately 200,000 wounded survivors have been reported. Clear evidences of mortality reasons have not been revealed; yet, we explicitly know from Marmara Earthquake in 1999 that many survivors died due to crush syndrome and associated complications. Crush syndrome is a systemic response of severe crush injury with hyperkalemia, acute kidney injury, and deep tissue damage. In crush syndrome, muscle damage initially occurs with the direct effect of trauma (baromyopathy), and later on, it develops with increased intramuscular pressure. Clinical findings consist of local inflammation findings in the traumatized area and systemic findings due to substances released from crushed muscle tissue into the systemic circulation. Earthquakes and other mass accident events leading to Crush Syndrome can place severe demands on local health-care systems. Excessive demand for dialysis machines, ventilators, and intensive care units can be necessary.

Keywords: Crush syndrome, disaster medicine, earthquake

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INTRODUCTION

On February 06, 2023, the southeastern region of Türkiyesuffered from two consecutive devastating earthquakes with 7.6 and 7.8 magnificence. Since then, at least 44,324 deaths and approximately 200,000 wounded survivors have been reported.[1] Clear evidences of mortality reasons have not been revealed; yet, we explicitly know from MarmaraEarthquake in 1999 that many survivors died due to crush syndrome and associated complications.[2] Crush syndrome is a systemic response of severe crush injury with hyperkalemia, acute kidney injury (AKI), and deep tissue damage.[3] This review comprehensively defined the pathophysiology and clinical manifestations of crush syndrome patients. Moreover, we expressed the treatment strategy both on the field and in hospital settings.

PATHOPHYSIOLOGY

In crush syndrome, muscle damage initially occurs with thedirect effect of trauma (baromyopathy), and later on, it develops with increased intramuscular pressure. Ischemia develops with compartment syndrome. The main defect is the decrease in Na-K ATPase and Ca ATPase activity in the cellmembrane. Baromyopathy causes occlusion of micro and macrocirculation in muscles therefore the muscle cell membrane loses permeability. Extracellular calcium and sodium shift into the cell causing an increased passage of sodium and calcium into the cell. It is the main reason for the hypertonicity and swelling of the cell. The increase in cytosolic calcium activates proteolytic enzymes and further increases ATPconsumption. As a result of proteolytic enzyme activation andcell swelling, rhabdomyolysis develops in muscle cells. Skel-

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etal muscle ischemia to edema and lysosome degranulation begins in about 30 min and reaches the maximum level in 4–6 h. It causes irreversible morphological changes that lead to necrosis. In addition, muscle ischemia and ischemic reperfusion injury develop during the recovery of this ischemia (as a result of the formation of reactive oxygen metabolites) and play a role in the pathogenesis of rhabdomyolysis.[3]

Various factors affect the emergence of AKI during rhabdomyolysis. These factors are shown in Table 1.

Renal vasoconstriction is a characteristic feature of rhabdomyolysis-induced AKI and is the result of several mechanisms. Fluid sequestration in the damaged muscle, inability to reach water under rubble, and bleeding reduce the renal blood supply.[4] The renin-angiotensin system is activated by hypovolemia, vasopressin, and sympathetic nervous system activation. Cytokines such as endothelin-1, thromboxane A2, tumor necrosis factor α, and vasodilator nitric oxide deficiency contribute to the pathogenesis of AKI. In addition, oxidation of iron ions in myoglobin, formation of hydroxy radical, reperfusion injury, endotoxins, hyperphosphatemia, hyperuricemia, and disseminated intravascular coagulation are also associated. It plays a role in the pathogenesis of AKI that occurs in the course of crush syndrome.[5]

CLINICAL MANIFESTATIONS

Clinical findings consist of local inflammation findings in the traumatized area and systemic findings due to substances released from crushed muscle tissue into the systemic circulation. Open or traumatic closed bone fractures, bleeding, muscle laceration, and contusion may be seen. The most important finding is the development of compartment syndrome. The findings of compartment syndrome can be summarized with the 6 P signs of ischemia.

Patients may experience pain (pain), increased pressure (pressure), paresthesia (paresthesia), pulselessness, paresis (paresis), and pallor (pallor) in the trauma area.[6] While severe and continuous pain is an early sign of irreversible ischemia, pulselessness can be seen in late phase. The simplest and most effective treatment for compartment syndrome are the surgical opening of the fascia (fasciotomy).

In traumatic rhabdomyolysis, the skin and subcutaneous tissue in the traumatized area are generally intact, although not the rule. Local clinical symptoms caused by trauma (myalgia, weakness, and stiffness in the muscles) are in the foreground. Pain may be much more than expected according to clinical findings.[7] Ischemia develops after trauma, causing hypoxia. As a result of hypoxia, edema and tissue necrosis develop. However, recovery occurs if the ischemia in the tissue is stopped during the hypoxia and edema phase. Therefore, the first 6–8 h, which are considered golden hours, are very important. Functional deterioration in muscle tissue takes 2–4 h, irreversible functional deterioration occurs after 4–12 h. Abnormal function in nerve tissues begins after 30 min, then it takes 12–24 h for irreversible deterioration. Systemic findings differ according to the affected organ. Hypovolemic shock, hypotension, AKI, arrhythmia, heart failure, respiratory failure, infection, disseminated intravascular coagulation, and sepsis can be seen as a severe outcome after systemic failure. In general, oliguria and sometimes anuria may be seen in patients due to AKI.[8]

Clinical Course

The clinical course in patients with crush syndrome is more complicated than in AKIs due to other causes. In these patients, both surgery (for example, bleeding, trauma, and other complications due to surgery), as well as medical complications (e.g., various infections, cardiac arrhythmias due to hyperkalemia and hypocalcemia, other organ, and system deficiencies) lead to high morbidity and mortality. Hyperalbuminemia or hypoalbuminemia are also poor prognostic indicators. Other biochemical anomalies caused by rhabdomyolysis include hyperuricemia, elevated aspartate aminotransferase, alanine aminotransferase, and lactic dehydrogenase. High anion gap metabolic acidosis and related deaths can be seen in these patients due to various reasons.

Acute kidney failure is one of the most important elements of crush syndrome. However, laboratory findings of the uremic syndrome may occur before this picture develops. This is due to laboratory findings of rhabdomyolysis. The symptoms of kidney failure and kidney failure are very similar to each other. In such patients, blood urea nitrogen (BUN) and creatinine levels are increased when AKI occurs, while in patients with rhabdomyolysis, the creatinine/BUN ratio may be higher than the expected value. In patients with rhabdomyolysis, acute renal failure (ARF) is prerenal or functional in the initial stage. In the next stage, acute tubular necrosis (ATN) occurs. ATN is considered to occur in patients who do not respond to volume replacement, Mannitol and bicarbonate administration, and persistent oliguria despite adequate fluid administration regardless of urinary tract obstruction.

There is no indication for biopsy in every patient who develops ARF in the background. However, it can be applied in cases that cannot be cured after a certain period of time (1 month). In some patients, it has been reported that while the compartment syndrome that occurred in the initial phase improves, the findings worsen again on the 2nd or 3rd day and a more

Table 1. Crush syndrome associated risk factors of acute kidney injury

1. Amino acids and other organic acids: Contributes to the development of acidosis, aciduria and dysrhythmias.
2. Creatine phosphokinase (CPK) and other intracellular enzymes: Crush is one of the diagnostic laboratory findings of injury.
3. Free radicals, superoxide and peroxides: They occur after ischemic tissue reperfusion and increase tissue damage
4. Histamines: They cause vasodilation and bronchoconstriction
5. Lactic acid: It has a major contribution to acidosis and dysrhythmias
6. Leukotrienes: They are responsible for ARDS and hepatic damage
7. Lysozymes: They are responsible for cell digestion, increasing cell damage
8. Myoglobin: Precipitates in renal tubules (especially in low urine pH and presence of acidosis); Causes the development of Acute Kidney Injury (AKI)
9. Nitric oxide: Vasodilation worsens hemodynamic shock
10. Phosphate: Causes hyperphosphatemia, hypocalcemia, and dysrhythmias leading to precipitation of serum calcium
11. Potassium: Causes hyperkalemia, dysrhythmias
12. Prostaglandins: Causes vasodilation, lung damage
13. Purines (uric acid): It is nephrotoxic
14. Thromboplastin: Released in Disseminated Intravascular Coagulation (DIC).

severe rhabdomyolysis picture emerges; at this stage, plasma creatine kinase (CK) level also rises again (second wave phenomenon). After the Marmara Earthquake, the mean oliguria was observed in patients with crush syndrome in the clinical course of which oliguria was observed that the duration was 10.8±7.2 (lower and upper limits 1–37) days; this period is slightly shorter than the oliguria period (15.2±5.6 days) in patients with crush syndrome after the Kobe Earthquake in Japan. The mean oliguric period is longer in patients who need dialysis support than in patients who do not have dialysis.[9]

PULLING THE PATIENTS OUT WITH CRUSH INJURIES AND ON-SITE MANAGEMENT

Patients with crush injuries are often stranded at the scene. Individual victims or a small number of people who have sustained injuries can be rescued by eyewitnesses or professional search and rescue teams. However, events of massive trauma such as earthquakes and the collapse of major consequences in natural or man-made disasters can trap thousands of sprawling.

Recovery time is strongly associated with earthquake mortality. Children and elderly individuals are quite vulnerable. Crush syndrome, which occurs in trauma patients who cannot be rescued from the scene for more than 24 h, is highly associated with mortality. A limited number of cases are excavated from 48 h to 14 days. The survivors are often found in cavities of the collapsed structure. Most deaths occur in victims trapped under rubble too heavy to withstand; other causes of death include dust inhalation, traumatic asphyxia, head injury, or multiple injuries. In addition, rescuers can account for a significant percentage or even the majority of casualties in many cases. Removing survivors from collapsed structures is dangerous and caution should be exercised at the risk of secondary collapse.[10]

Close Field Medicine

The extent of Crush syndrome requires initial medical attention by rescuers inside rubbles or closed areas. Tunneling and digging may be required to rescue victims. Hazards such as explosion and collapse need to be well-predicted. The safety of rescuers is the most important point. Most of the mass crush damage is erosion from earthquakes. Explosions, fires, or landslides are also can be a reason for crush damage.[11]

Airway, Breathing and Circulation

Chest crush injury is an important source of respiratory failure and death. Patients may require oxygen, airway, advanced airway preservation techniques, and the use of portable ventilators. Inhalation injury associated with dust or hot gases in situations such as fire or bombing may also require improved airway management. Pneumothorax, hemothorax, flail chest, and pulmonary contusion may require chest decompression (e.g., needle or finger decompression, chest tube) during rescue care.

In patients who are stuck at the scene for a long time, circulation may be impaired and hypovolemia may occur. Hypovolemia may be caused by several reasons. The most common cause of hypovolemia in long-term stranded patients is dehydration and is the leading cause of death. Bleeding or burns from traumatic injuries can also cause hypovolemia. In the emergency management of hypovolemia, large-di-

ameter intravenous catheters and isotonic saline administration are required to prevent AKI and to prevent death from hyperkalemia after rhabdomyolysis due to Crush syndrome. Liquids containing potassium should be avoided. In the treatment of hypovolemia, urine output is monitored for response to treatment. Crush injuries without adequate fluid resuscitation develop into Crush syndrome.

Typically, adults are given a 1000 mL/hr bolus of normal saline for 2 h initially, then reduced to 500 mL/hr. Smaller volumes, such as 10 mL/kg, are recommended for those with known heart failure, kidney failure, or chronic obstructive pulmonary disease.[12,13]

Reperfusion of Extremities and Hyperkalemia

After salvaging a trapped limb, patients may have minimal symptoms, the limb may be only mildly erythematous or ecchymotic, or it may be clearly mottled and ischemic. Any pain can be treated with opioids or ketamine.[14] Release of potassium from injured and necrotic muscles and other tissues can lead to a rapid onset of hyperkalemia and ventricular fibrillation. Pre-hospital administration of intravenous crystalloid is the mainstay of treatment and is associated with better outcomes. Pre-hospital electrocardiogram, tall peaked T-wave, and wide QRS can provide early recognition of hyperkalemia and early treatment of fatal hyperkalemia can be provided by paramedics on physician's order.

The application of a tourniquet to prevent the potassium released from the crushed extremity from entering the systemic circulation is not supported by evidence and is not recommended.[12,13] Tourniquets should only be used to control extremity bleeding. Similarly prophylactic fasciotomy should not be performed in the field due to infection risks.[13] In addition, prehospital amputation of severely crushed or dismembered limbs simply to avoid Crush syndrome is also not supported by evidence and increases the risk of stump infection. However, amputation may be needed as a last resort to save the patient.

Field Amputation

The limbs of some trapped patients may not be able to be removed from the debris or structure in a timely manner. In such cases, field amputation may be required as a last resort for rescue. Manual or chainsaws can be used depending on the space available, resources, and risk of explosive gas. A surgical team should be dispatched whenever possible to perform this procedure, if available.

Triage and Transport

Field triage generally uses three categories to prioritize on-scene care and transport:

"Immediate" patients are previously treated and transported, followed by the "delayed" category.

The "minor" category patient may be discharged from on-scene treatment or transferred last.

An "expectant" category is generally used for patients with a low probability (<%10) of survival, such as post-cardiac arrest outcomes, severe head injuries, patients in permanent shock, and patients with major burns; it can provide palliative care in these patients. Most patients, generally 80–85% in mass accident episodes such as earthquakes, only require basic therapeutic care without major surgery or organ support. This is more seriously injured patients removing the stuffing from the hospital before they leave. Only about 15–20% of survivors are earthquake sensitive to the need for hospitalization, major surgery, or organ support. This second group carries the risk of death and the mortality rate in this group is called the "critical death rate.[14–16]"

HOSPITAL MANAGEMENT

Initial Trauma Management

In patients with crush injuries who come to the emergency department, isotonic saline administration initiated in the field should be continued with close monitoring of urine output. If there is no contraindication, a Foley catheter should be inserted. Trauma management should be performed in accordance with the trauma management procedure.

Clinical Monitoring

Clinical examination and laboratory studies should be performed several times a day until stabilized. These include electrolytes (especially serum sodium, potassium, calcium, phosphate, and bicarbonate), creatinine and lactate, and/or arterial blood gases. Urine myoglobin and CK can be obtained to detect rhabdomyolysis that may occur.

Crushed Extremity

The need for amputation varies greatly (3–59%) depending on delays in recovery, associated injuries, and local resources. Guidelines recommend that amputation should be limited to cases where a limb cannot be saved or where injuries to the limb cause sepsis, systemic inflammation, or uncontrollable bleeding.[12] Decision-making regarding limb salvage is clinical; the crushed limb severity score (MESS) can be looked at or it can be decided according to the management of the experienced surgeon.

When mass events occur, resources such as blood transfusions to salvage a limb may be insufficient and held at a

Table 2. Indications of dialysis in patients with crush syndrome

1. Serum potassium of 6.5 mmol/L or more or rapid elevation of serum potassium 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 consciousness disorders
5. Persistent oliguria or anuria despite adequate fluid administration.

higher threshold where amputation is more likely necessary. Severe crush injury is a known cause of acute limb compartment syndrome. Early recognition, evaluation, and treatment of compartment syndrome reduce the risk of Crush syndrome and limb loss. Fasciotomy to completely decompress all affected compartments is the definitive treatment in the vast majority of cases.[17] Delays in performing a fasciotomy increase morbidity, including the need for amputation.

Controversy exists regarding the role of prophylactic fasciotomy in severe crush injury. Based on the authors' experience at a level 1 trauma center, we do not perform prophylactic fasciotomy for severe crush injury. We typically perform fasciotomies only if clinically established acute compartment syndrome is present at admission or if measured compartment pressures show a difference of 30 mmHg or less between diastolic blood pressure and compartment pressure (delta pressure). In indeterminate cases, impending compartment syndrome may be suspected by increased measured compartment pressures and is managed by serial examinations, and may lead to fasciotomies when delta pressure drops to 30 mmHg or less. Prophylactic fasciotomy is also not recommended for mass crush injuries in the absence of measured compartment pressure increase.

In environments where resources are limited, good results from fasciotomy are less likely. Avoiding unnecessary fasciotomy can reduce the risk of infection and eliminate the need for post-fasciotomy wound care.[18,19] Some physicians recommend that fasciotomy be performed on severely crushed limbs only when the distal pulses are significantly reduced or absent.[12,18,20] Fasciotomy for late compartment syndrome after 8 h is also controversial. Extensive myonecrosis is most likely to be present, and performing fasciotomy without amputation obliges the clinician to perform serial surgical debridements and carefully monitor for systemic problems such as wound sepsis, rhabdomyolysis, and renal failure.

Liver and spleen lacerations can be seen in such traumas. Mortality rates are higher for those associated with chest or abdominal injuries. In this respect, it is necessary to be alert.

Once AKI has occurred, aggressive intravenous fluid resuscitation is no longer appropriate. If conditions such as excessive volume overload, hyperkalemia, severe acidemia, and uremia have occurred, hemodialysis should be performed. A crush injury can cause rapid and severe hyperkalemia, requiring two or more daily hemodialysis.

Earthquakes and other mass accident events leading to crush Syndrome can place severe demands on local healthcare systems. Excessive demand for dialysis machines, ventilators, and intensive care units can occur. This can exceed local resources and threaten access to chronic hemodialysis patients. When an insufficient number of hemodialysis machines are available, peritoneal dialysis can be used despite the slower clearance of potassium.

Crush Syndrome Treatment and Dialysis Indications

The most critical and first thing to do in the management of crush syndrome can be counted as rapid and aggressive fluid therapy and elimination of the factors that cause it, management and prevention of complications that may arise. Intravenous fluid therapy should be started as soon as possible to correct hypovolemia and prevent the development of AKI. If possible, intravenous access should be opened and fluid therapy should be started before the debris is removed. Because recovery from debris usually takes about 90 minutes.

Adults are initially given a 1000 mL/h normal saline bolus initially for 2 h, then reduced to 500 mL/h. Smaller volumes such as 10 mL/kg are recommended for those with known heart failure, renal failure, or chronic obstructive pulmonary disease.[12,13] In children, isotonic fluids should be started at a rate of 15–20 ml/kg/h. Fluids containing potassium or lactate should not be given. Isotonic fluid at a rate of 1000 ml/h should be started immediately after removal from the wreckage for patients who cannot be started on fluid therapy under the rubble. Systemic alkalization is in the background to reduce intracompartmental pressure, reduce acidosis, and hyperkalemia. If any patients met those criteria counted in Table 2, urgent dialysis would be considered in emergency settings.

CONCLUSION

We know from anatolia's devastating earthquakes and fault movements in the previous centuries that a magnificent earth is expected in the future.[21] The mortality rates of crush syndrome are still very high despite the developments in critical care of crush syndrome victims. Initial treatment is the most important measure to reduce mortality. Conventional treatment methods including fluid resuscitation, correction of electrolyte imbalance, diuresis, and hemodialysis are still the most applied approaches.[22] Urgent treatment and interventions-even fasciotomy-can immediately be performed for selected victims on-site to avoid mortality.[23] Crush syndrome-related hyperkalemia and AKI are the most frequent cause of hospital deaths but luckily the most important reversible complication after earthquakes.[24]

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REFERENCES

1. AFAD. Kahramanmaraş'ta Meydana Gelen Depremler Hk. Basın Bülteni-36. Available at: https://www.afad.gov.tr/kahramanmarasta-meydana-gelen-depremler-hk-36. Accessed Mar 1, 2023.
2. Demirkiran O, Dikmen Y, Utku T, Urkmez S. Crush syndrome patients after the Marmara earthquake. Emerg Med J 2003;20:247–50. [CrossRef]
3. Peck SA. Crush syndrome: pathophysiology and management. Orthop Nurs 1990;9:33–40. [CrossRef]
4. Michaelson M. Crush injury and crush syndrome. World J Surg 1992;16:899–903. [CrossRef]
5. Ochoa-Cacique D, Córdoba-Mosqueda ME, Aguilar-Calderón JR, García-González U, Ibarra-De la Torre A, Reyes-Rodríguez VA, et al. Double crush syndrome: epidemiology, diagnosis, and treatment results. Neurochirurgie 2021;67:165–9. [CrossRef]
6. Akdam H, Alp A. Crush syndrome. J Tepecik Educ Res Hosp [Article in Turkish] 2015;25:71–7. [CrossRef]
7. McHowell WD. Crush syndrome: pathophysiology and management. Orthop Nurs 1990;9:78. [CrossRef]
8. Qiao O, Wang X, Wang Y, Li N, Gong Y. Ferroptosis in acute kidney injury following crush syndrome: a novel target for treatment. J Adv Res 2023 Jan 24. doi: 10.1016/j.jare.2023.01.016. [Epub ahead of print]. [CrossRef]
9. Turgut N, Adaş G, Akçakaya A, Mıngır T, Topuz C, Ay A. Earthquake; trauma, crush syndrome and cardiopulmonary resuscitation. Okmeydanı Med J [Article in Turkish] 2012;28 (Suppl 2):135–47. [CrossRef]
10. Macintyre AG, Barbera JA, Smith ER. Surviving collapsed structure entrapment after earthquakes: a "time-to-rescue" analysis. Prehosp Disaster Med 2006;21:4–17. [CrossRef]
11. Jensen J, Thompson S. The Incident Command System: a literature review. Disasters 2016;40:158–82. [CrossRef]
12. Sever MS, Vanholder R; RDRTF of ISN Work Group on Recommendations for the Management of Crush Victims in Mass Disasters. Recommendation for the management of crush victims in mass disasters. Nephrol Dial Transplant 2012;27(Suppl 1):i1–67. [CrossRef]
13. Sever MS, Vanholder R. Management of crush victims in mass disasters: highlights from recently published recommendations. Clin J Am Soc Nephrol 2013;8:328–35. [CrossRef]
14. Tanaka H, Oda J, Iwai A, Kuwagata Y, Matsuoka T, Takaoka M, et al. Morbidity and mortality of hospitalized patients after the 1995 Hanshin-Awaji earthquake. Am J Emerg Med 1999;17:186–91. [CrossRef]
15. Sever MS, Erek E, Vanholder R, Akoğlu E, Yavuz M, Ergin H, et al; Marmara Earthquake Study Group. The Marmara earthquake: epidemiological analysis of the victims with nephrological problems. Kidney Int 2001;60:1114–23. [CrossRef]
16. Frykberg ER, Tepas JJ 3rd. Terrorist bombings. Lessons learned from Belfast to Beirut. Ann Surg 1988;208:569–76. [CrossRef]
17. Farber A, Tan TW, Hamburg NM, Kalish JA, Joglar F, Onigman T, et al. Early fasciotomy in patients with extremity vascular injury is associated with decreased risk of adverse limb outcomes: a review of the National Trauma Data Bank. Injury 2012;43:1486–91. [CrossRef]
18. Reis ND, Better OS. Mechanical muscle-crush injury and acute muscle-crush compartment syndrome: with special reference to earthquake casualties. J Bone Joint Surg Br 2005;87:450–3. [CrossRef]
19. Better OS, Rubinstein I, Reis DN. Muscle crush compartment syndrome: fulminant local edema with threatening systemic effects. Kidney Int 2003;63:1155–7. [CrossRef]
20. 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.
21. Kayaalp E, Arslan O. Earth in practice: uncertainty, expertise and the expected Istanbul earthquake. Environ Plan E Nat Space 2022;5:1579–96.
22. Li N, Wang X, Wang P, Fan H, Hou S, Gong Y. Emerging medical therapies in crush syndrome - progress report from basic sciences and potential future avenues. Ren Fail 2020;42:656–66. [CrossRef]
23. Lutter C, Schöffl V, Hotfiel T, Simon M, Maffulli N. Compartment syndrome of the foot: an evidence-based review. J Foot Ankle Surg 2019;58:632–40. [CrossRef]
24. He Q, Wang F, Li G, Chen X, Liao C, Zou Y, et al. Crush syndrome and acute kidney injury in the Wenchuan Earthquake. J Trauma 2011;70:1213–7.