

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Crush injury and syndrome: A review for emergency clinicians

Brit Long ^{a,*}, Stephen Y. Liang ^b, Michael Gottlieb ^c

^a SAUSHEC, Emergency Medicine, Brooke Army Medical Center, Fort Sam Houston, TX 78234, USA

b Divisions of Emergency Medicine and Infectious Diseases, Washington University School of Medicine, 660 S. Euclid Ave, St. Louis, MO 63110, USA

^c Ultrasound Director, Assistant Professor, Department of Emergency Medicine, Rush University Medical Center, Chicago, IL, USA

ARTICLE INFO

Article history: Received 28 February 2023 Accepted 17 April 2023

Keywords: Crush injury Crush syndrome Acute kidney injury Disaster Earthquake

ABSTRACT

Introduction: Primary disasters may result in mass casualty events with serious injuries, including crush injury and crush syndrome.

Objective: This narrative review provides a focused overview of crush injury and crush syndrome for emergency clinicians.

Discussion: Millions of people worldwide annually face natural or human-made disasters, which may lead to mass casualty events and severe medical issues including crush injury and syndrome. Crush injury is due to direct physical trauma and compression of the human body, most commonly involving the lower extremities. It may result in asphyxia, severe orthopedic injury, compartment syndrome, hypotension, and organ injury (including acute kidney injury). Crush syndrome is the systemic manifestation of severe, traumatic muscle injury. Emergency clinicians are at the forefront of the evaluation and treatment of these patients. Care at the incident scene is essential and focuses on treating life-threatening injuries, extrication, triage, fluid resuscitation, and transport. Care at the healthcare facility includes initial stabilization and trauma evaluation as well as treatment of any complication (e.g., compartment syndrome, hyperkalemia, rhabdomyolysis, acute kidney injury).

clinicians must understand the pathophysiology, evaluation, and management of these conditions to optimize patient care.

Published by Elsevier Inc.

1. Introduction

Millions of people worldwide face primary natural or human-made disasters every year. Primary disasters may be natural (e.g., earthquakes, cyclones, hurricanes, flooding, landslides) or human-made (e.g., terrorist attacks, air or railway crashes, wars) [1-6]. Approximately 800 million people currently live in areas prone to earthquakes or at high risk of severe tropical cyclones [3,6-9]. This number is likely to increase due to continued population growth in urban centers. Several major cities including Istanbul, Mexico City, Tehran, and Tokyo have a high risk of seismic activity, and a single severe earthquake could impact millions [7-10]. The initial magnitude of the disaster, population density affected by the disaster, time of day, building standards, community and individual preparedness, and the emergency response all determine the scope and severity of disaster [9,10]. Secondary hazards including power failures, fires, inadequate access to clean water and food, and communicable diseases in the setting of poor sanitation and crowding in emergency shelters can further amplify the damage inflicted [9,10].

* Corresponding author at: 3551 Roger Brooke Dr, Fort Sam Houston, TX 78234, USA. E-mail addresses: Brit.long@yahoo.com (B. Long), syliang@wustl.edu (S.Y. Liang).

https://doi.org/10.1016/j.ajem.2023.04.029 0735-6757/Published by Elsevier Inc. Severe earthquakes can cause massive injury and death, with the majority of deaths occurring immediately after the earthquake from major trauma and entrapment, with risk of crush injury and crush syndrome [3-7,9-11]. Crush injury occurs with direct physical trauma to the body from an external force [3,6,11,12]. Crush syndrome is the severe systemic manifestation of muscle and end-organ injury leading to complications including dysrhythmias, rhabdomyolysis, acute kidney injury (AKI), and sepsis [12-18]. Data from prior disasters suggest that approximately 80% of entrapped victims die rapidly due to severe injury, while 10% sustain crush injury and 10% mild trauma [3-6,16]. Of the 10% who experience crush injury, 40–70% experience crush syndrome [3-5].

The 2023 earthquake affecting Turkey and Syria has resulted in over 47,000 deaths and displaced millions [19]. Emergency clinicians and emergency medical services personnel are at the frontline of evaluating and managing patients affected by natural and human-made disasters, including severe earthquakes, and are likely to care for patients with crush injury and crush syndrome. This review provides a focused overview of crush injury and crush syndrome for emergency clinicians.



2. Methods

The authors searched PubMed and Google Scholar for articles using keywords "crush injury", "crush syndrome", and "disaster". Article types included case reports, case series, retrospective studies, prospective studies, systematic reviews and meta-analyses, clinical guidelines, narrative reviews, and online resources. Guidelines and information from governmental and non-governmental health organizations were included, where relevant. The literature search, restricted to studies and resources published in English, was conducted on February 17, 2023, and retrieved a total of 12,547 articles. Emergency clinicians with experience in critical appraisal of the literature reviewed all of the resources and decided which resources to include for the review by consensus, with a focus on emergency medicine-relevant articles. A total of 106 references were selected for inclusion in this review.

3. Discussion

3.1. Mechanism and definitions

Crush injury is due to direct physical trauma and compression of the human body [3,6,11,12]. The compressive force directly damages soft tissues, muscles, bones, nerves, and other tissues based on the site of the injury [3-6,13,18]. Anywhere from 3 to 20% of mass casualties during an earthquake sustain crush injury from building collapse and body entrapment [9,20]. The most commonly injured sites include the lower extremities (74%), the upper extremities (10%), and the trunk (9%) [3,6,9,11,16,20]. Other forms of crush injury include severe traumatic brain injury or airway compromise, which can result in immediate death, as can injuries to the trunk including solid organ damage with severe hemorrhage or chest injury (e.g., pneumothorax, cardiac tamponade) [3-6,18]. In the extremities, muscle, bone, and soft tissue damage associated with crush injury result in edema and intravascular hypovolemia. This increases intracompartmental pressures, reducing capillary, lymphatic, and venous outflow, further increasing tissue damage and intracompartmental edema [3,11,21]. Intracompartmental pressures continue to increase and can eventually result in decreased arteriolar perfusion and compartment syndrome [21].

Crush syndrome is the systemic manifestation of a crush injury with organ dysfunction [13-15]. Cellular damage and myonecrosis from crush injury releases myoglobin, potassium, phosphorus, and uric acid into the blood [2,3,6,12-16]. These can lead to a number of complications, including acute kidney injury, hypotension, and acidemia. More severe complications include acute respiratory distress syndrome (ARDS), cardiac dysrhythmia, and disseminated intravascular coagulation (DIC) [13,22-25]. Delayed effects include venous thromboembolic events (VTE), hemorrhage, sepsis, and psychiatric disorders (e.g., anxiety, depression, and post-traumatic stress disorder) [23,25-30]. Table 1 lists manifestations of crush syndrome.

3.2. Scene management and extrication

Crush injury is commonly associated with entrapment. Victims may be extricated by bystanders or prehospital personnel, but in large-scale trauma events including earthquakes or major building collapses, significant numbers of victims may be entrapped requiring rescue by specialized teams [3,4,6,18,24]. Time to extrication is also known as time period under the rubble (TPR) and depends on a variety of factors including disaster severity, population density, building structural quality, and rescue work efficacy [3,5,6]. TPR is highly associated with earthquake morbidity and mortality, with pediatric and elderly victims at greatest risk of severe injury and death [3-5,18,24]. TPR >24 h is strongly associated with risk of death, with a low number of survivors extricated after 48 h [24]. However, cases of victims rescued after being entrapped for as much as 13 days have been reported [24,35]. Literature is controversial regarding the risk of AKI with TPR; severity of

Table 1

M	anifestations of	crush syndrome	[3-6,18,21,31-34].
---	------------------	----------------	--------------------

Manifestation Considerations	
Acute kidney injury	 Increase in serum creatinine/decrease in glomerular filtration rate Due to a variety of causes, including rhabdomyolysis, hypotension, and renal hypoperfusion Direct nephrotoxic effects from myoglobin, potassium, phosphorus, and uric acid may also occur
ARDS	 Associated with volume resuscitation, distributive shock, fat embolism from long bone fractures, and severe tissue necrosis
Asphyxia	 Initial crush injury to the chest can result in asphyxia Due to a significant increase in pressure in the thoracic cavity and superior vena cava The increased pressure in combination with attempts of inspiration with a closed glottis results in rupture of capillaries in the head/neck Patients may have cyanosis, edema, and petechial eruptions in the head, neck, and torso proximal to the site of the
	compression - Accompanied by other injuries such as liver and splenic laceration, pulmonary and myocardial contusions, rib fractures, and brain injury
Extremity crush injury	 Presents along a spectrum, from swelling, blisters, and purpura to closed or open fractures, neurovascular injury, and mangled or amputated extremities Patients are at risk of severe orthopedic/neurovascular injuries that compromise limb function, as well as acute compartment
Hypotension	syndrome and rhabdomyolysis - Hypotension occurs in the first several hours after the initial injury, associated with organ injury, bleeding, distributive shock, third spacing, severe inflammation, and reperfusion injury
Organ injury	 Direct injury to the torso can result in hemothorax, pneumothorax, pulmonary contusion, myocardial trauma, solid organ injury (e.g., liver or splenic laceration), hollow viscus injuries, rib fracture, pelvic fractures, and spinal cord injury Penetrating injury from projectiles can also occur

muscle damage and degree of hypovolemia are likely the greatest predictors of AKI rather than TPR [35-40].

Care in this setting is dangerous and requires specialized training. Dust, extremes of temperature, uncontained fires, hazardous materials and gases, and risk of explosions pose significant threats to rescuer safety [3-6]. Heavily damaged buildings are at risk of secondary collapse and may further endanger victims and rescuers alike, leading to additional casualties. Rescuers should be trained in extrication and providing patient care while donning personal protective equipment [3,24].

Life-threatening complications are common during entrapment, but secondary injuries may also occur during rescue. Therefore, rescue team members and healthcare professionals must coordinate initial victim assessment as well as the timing and method of extrication [3,4,6,41]. Frequent reassessment of the victim is paramount. Between 13 and 40% of early deaths at the scene can be prevented with simple treatment interventions including airway management, hemorrhage control, fracture stabilization, fluid resuscitation, and prevention of hypothermia (Table 2) [3,41]. In major earthquakes, close to 20% of all deaths occur shortly after victim extrication [41]. These deaths include patients who were stable prior to extrication but then hemodynamically deteriorated shortly thereafter, known as rescue death [3-6,18,41]. It is believed that reperfusion of a severely traumatized extremity leads to systemic circulation of tissue breakdown products, resulting in lifethreatening cardiac dysrhythmias [42,43]. To prevent this, victims should be medically evaluated while they are still entrapped and stabilized prior to extrication [3,6,23,27,28,30]. A full primary and secondary trauma survey is often not possible, but the rescuer should evaluate the body site compressed, patient airway and respiratory status, sources of severe bleeding or other injuries, volume status, and presenting symptoms [41].

Crush injury to the chest and inhalation injury can lead to respiratory failure and death [3,5,17]. Victims may require respiratory support

Table 2

Survey	Problem	Intervention
Airway	Airway compromise	Maintain airway patency
Breathing	Ventilation may be impaired due	Protect patient by applying a
	to dust, noxious gas, or direct	dust mask
	trauma	Space limitation may interfere
		with oxygenation and intubation
		Analgesia may assist breathing
		in those with rib fractures
Circulation	Exclude dehydration	Control external bleeding
	Assume crush injury	Assess volume status and
	If victim is alive but trapped for an	administer fluid considering
	extended period, major active	medical and logistic issues
	bleeding is unlikely	
Disability	Neurologic examination may not	Install/maintain cervical
	recognize all abnormalities	protection
Exposure	Consider hypothermia	Cover exposed patients to avoid
	Expose body parts only if	hypothermia
	necessary	~ *

with supplemental oxygen, oral or nasal airway placement, intubation, and portable ventilators. Pneumothorax may require chest decompression.

Dehydration is common with prolonged entrapment and is a major cause of morbidity and mortality. Volume resuscitation is recommended if possible, as inadequate fluid replacement for >6 h after a crush injury increases the risk of AKI [3,6,27,29,30]. AKI can be prevented in many victims with early fluid resuscitation while entrapped [3,5,6,25,27,29,30]. If an extremity vein is found, intravenous (IV) access should be obtained and fluids administered at a rate of 1 L/h [3-6]. If peripheral IV cannulation is not possible, intraosseous (IO) infusion is recommended, followed by subcutaneous infusion of isotonic fluids at 1 mL/min if IV or IO access is not available [3,5,6]. Fluid resuscitation should be continued throughout the extrication; if the duration of extrication exceeds 2 h, the infusion rate should be lowered to 0.5 L/h or less [3-6]. The initial volume of fluid infused should consider environmental conditions (e.g., higher ambient temperatures may result in greater insensible fluid losses), entrapment and extrication time (more fluid is required if rescue will be delayed), volume status (as evidenced by signs of hypovolemia), and patient factors (age, comorbidities, body mass index) [3,4,6]. Patients with known comorbidities such as congestive heart failure or renal failure should receive smaller fluid volumes (e.g., 10 mL/kg) [3-5]. Urine output should be monitored if possible, with a goal >50 mL/h [3-5]. While placement of a urinary catheter allows for accurate measurement of urine output, volumes can also be approximated using a container with a known volume or qualitatively assessing for dampness in the patient's undergarment [3,4,6]. Importantly, victims entrapped with their pelvis or lower extremities positioned above the level of the heart are at greater risk of pulmonary edema from volume resuscitation, and their respiratory status should be frequently reassessed [3,5]. Of note, other causes of hypovolemia include hemorrhage, burns, and third spacing. Hemorrhage in particular can occur with severe solid organ injury (e.g., liver or splenic laceration) or limb injury [3,18].

Fluid type varies based on available resources. Isotonic saline is the first line option in mass casualty events for volume replacement and can prevent AKI [3-6,18]. Isotonic saline with 5% dextrose or infusion of sodium bicarbonate added to half-isotonic saline may reduce metabolic acidemia and hyperkalemia and may be used in smaller-scale disasters [28,30,44]. Mannitol is not recommended in patients with volume depletion, congestive heart failure, and electrolyte abnormalities [3,5,6].

Tourniquet placement prior to extrication remains controversial. The Joint Trauma System (JTS) Clinical Practice Guideline (CPG) states that if fluid resuscitation and monitoring is not immediately available, a tourniquet may be placed on the affected extremity prior to extrication if entrapment time has exceeded 2 h to help prevent crush syndrome [18]. The best means of completing this is to apply two tourniquets side by side proximal to the injury immediately prior to extrication. If this is not possible, tourniquets can be applied immediately after extrication [18]. However, this is in contrast to the Renal Disaster Relief Task Force and International Search and Rescue Advisory Group recommendations, which recommend applying a tourniquet only for lifethreatening hemorrhage as a last resort when direct pressure or other hemostatic measures have failed, as tourniquet placement increases the risk of neurologic damage, thrombosis, abscess, blisters, contusions, and abrasions [3,4,6,18]. They do not recommend tourniquet placement to reduce the risk of crush syndrome if the limb can be salvaged [3,4,6,18]. If a tourniquet is applied, it should be removed as soon as possible to limit the risk of limb loss [3,4,6,18].

In some situations it may be impossible to free an entrapped limb, or the victim may need to be extricated rapidly [18,45,46]. In this setting, a field amputation may be necessary. A manual or powered saw is typically recommended, with the amputation performed by a surgical team if possible [18]. After a tourniquet is applied, a guillotine amputation should be performed at the most distal site that will facilitate expedient extrication. This approach allows preservation of bone stock for formal revision of the amputation on transfer to definitive medical care [6,18]. Analgesia and sedation should be administered, with ketamine serving as an optimal agent due to its dissociative and analgesic effects while preserving spontaneous ventilation [3,6,47]. While a field amputation may prevent crush syndrome, it is associated with significant mortality due to the risk of hemorrhage and infection and should only be performed for life-saving indications [22,43,45].

3.3. Assessment after extrication

Following extrication, the victim should be moved away from the damaged area to a safe location or collection point for assessment and transport. A systematic assessment should be completed (e.g., a primary and secondary trauma survey) to identify and treat life-threatening injuries, as well as to prioritize therapeutic needs and resources [3-6]. Victims who are alert, oriented, talking, breathing normally, and moving all extremities likely do not have life-threatening injuries. However, if the victim is unresponsive or has visible potentially life-threatening or penetrating injuries, rescuers and healthcare personnel at the scene must consider available resources and medical factors (e.g., extent of traumatic injuries, physical findings, and victim characteristics) to decide whether to triage and treat the victim. When sufficient numbers of healthcare personnel and transport resources are available, optimal medical care should be provided to all victims, regardless of illness severity. In this setting, the patient who is unresponsive or with lifethreatening or multisystem injuries should have their spine immobilized, any life-saving procedure rapidly performed (e.g., needle decompression for tension pneumothorax), and then transported to the nearest safe, well-equipped healthcare facility [3,4,6,18]. Of note, severe neurologic deficiencies including sensory loss or flaccid paralysis after extrication do not indicate spine injury in all cases. These findings may also be seen with peripheral neuropathy from direct compression or compartment syndrome [11,29]. The secondary survey is essential to assess for severe abdominal and thoracic trauma, which are major contributors to morbidity and mortality [12,22,48,49]. The clinician conducting the secondary survey should also assess major muscle groups for trauma and pain, as rhabdomyolysis is common following crush injury. Areas at high risk of rhabdomyolysis include muscles of the lower extremities and trunk (e.g., latissimus dorsi) [3,12].

In the setting of limited resources or a mass casualty event, the goal of triage is to allocate limited medical resources to patients for whom the most benefit can be expected [3,4,6,18]. Guidelines recommend treating cases with at least 50% probability of survival in the field [3-5]. If resources including healthcare personnel or transport at the scene are limited, a dedicated triage system at the scene is recommended, such as the Simple Triage and Rapid Treatment (START)

Descargado para Lucia Angulo (lu.maru26@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en julio 19, 2023. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2023. Elsevier Inc. Todos los derechos reservados.

system, which categorizes patients as immediate, delayed, minor, and expectant. Immediate patients are treated and transported first, followed by delayed and then minor [50]. Minor patients are treated last and may not require transport to the ED. Expectant patients include those with a low likelihood of survival, such as post cardiac arrest patients, those with persistent shock, massive burns, or severe head injuries [51]. These patients should receive palliative care including analgesia if possible with reevaluation, as patients initially classified as expectant may be triaged to a different category after reevaluation.

3.4. Management after extrication

Pain control should be ensured and reassessed post-extrication. Visible bleeding should be stopped with direct pressure and/or topical hemostatic agents [3,18]. Wet clothing should be removed and the patient covered with warm sheets with consideration of active rewarming based upon the patient's temperature given the risk of hypothermia [3,4]. Volume status should be assessed rapidly, including vital signs, skin color, turgor, capillary refill, and urinary output [3-6]. These factors should be considered in combination with environmental factors (e.g., high ambient temperatures) and logistical factors. In the majority of crush victims, continuous fluids should be administered to reduce the risk of AKI. If fluids were not administered prior to extrication, a rate of 1 L/h is recommended [3-6,18]. A maximum fluid volume of 3–6 L/day is recommended [3-5]. If not already in place, a urinary catheter should be inserted to track urine output, provided there is no concern for urethral trauma. Clinicians should target a urine output >50 mL/h to help prevent crush-related AKI [3-5]. If the patient remains anuric 6 h after fluid initiation, two scenarios may be present: the patient may still be volume depleted and require further fluid, or they have renal failure. If the patient has evidence of volume overload or if anuria is present despite volume resuscitation, no further fluid should be administered [3-5]. If hypotension is believed to be due to blood loss from hemorrhage, then blood products should be administered if available [3,18].

Hyperkalemia is strongly associated with crush injury and crush syndrome with AKI. Many crush victims die from hyperkalemia at the scene, during transport, or after initial hospitalization [3,4,52]. Portable electrocardiography (ECG) and laboratory assessment at the incident scene is recommended if possible, though a normal ECG is insufficient to exclude hyperkalemia in isolation [53-56]. If hyperkalemia is found or suspected, insulin/dextrose and nebulized albuterol should be administered. Calcium gluconate or calcium chloride should be administered if there are ECG changes or the patient is hemodynamically unstable [56,57]. If hyperkalemia in the setting of crush syndrome is found, sodium bicarbonate infusion is recommended [3-5].

3.5. Transport to healthcare facility

A dedicated transport process involving emergency medical services from the incident scene or collection point to the nearest wellequipped, safe healthcare facility should be established, preferably prior to a mass casualty event [3,58]. While transport from the incident scene to a healthcare facility is paramount, several factors can affect patient transport. Entrapped patients often require significant medical care at the scene, including fluid resuscitation and even procedures such as field amputation [3,4,18,59]. Patient numbers and scene issues may overwhelm local resources. Triage and transport decisions must take into account available resources, the number of patients, and type of injuries. Patients categorized as immediate should be transported first if possible, followed by delayed and minor patients. If the transport time will be short, prolonged field stay should be avoided, but if it will be delayed or prolonged, then patients may benefit from more dedicated care at the scene (fluid resuscitation, blood transfusion, etc.) [3,4,18].

Table 3 lists primary components of focus and responsibility for personnel at the scene.

Table 3

Primary components of rescuer focus and responsibility at the scene.

Global Task	Specifics
Determination of rescuer ability to respond	 Resolve personal and family disaster-related issues and have a plan for family Inform authorities coordinating response i unable to respond
Patient interventions prior to extrication	 Consider rescuer safety when approaching scene/any damaged structures Begin medical evaluation of entrapped victims Start 1 L/h infusion of isotonic saline prior to extrication
Patient interventions during extrication	 Reevaluate victims during extrication at regular intervals if possible Continue isotonic saline infusion at 1 L/h for first 2 h in adults Adjust fluid rate if extrication takes longer than 2 h, not to exceed 500 mL/h
Patient care after extrication	 Remove the victim as quickly as possible from the site to a dedicated area for evaluation Evaluate vital signs; perform primary survey Perform triage Treat any life-threatening emergencies Perform secondary survey
Fluid administration and urine output monitoring after extrication	 Continue or start isotonic saline at 1 L/h in adults, considering environmental factors (ambient temperature) Insert urinary catheter to monitor urine output
Other patient care considerations prior to, during, or after extrication	 Treat other issues including airway obstruction, respiratory distress, severe pair Diagnose and manage severe hyperkalemia as soon as possible Prepare patient for transport to dedicated healthcare facility

3.6. ED management

3.6.1. Initial triage and evaluation

Data suggest that 80–85% of overall patients in mass casualty events require only basic medical care without interventions [3,4,20,59,60]. In these scenarios, approximately half of patients arriving at the healthcare facility in the first hour after the incident have low acuity and high like-lihood of survival [20,59,60]. The lower acuity patients should be triaged and treated rapidly at the healthcare facility, followed by discharge if possible to ensure sufficient capacity for the higher acuity patients that may follow. An alternate space to treat patients with minor injuries should be established as soon as possible to further increase ED capacity to manage critically ill patients [3,4].

Prior to patient arrival, a triage or initial evaluation system should be in place at the healthcare facility. This may include specific zones for patients, including red (life-threatening but treatable conditions), yellow (less serious but urgent conditions), green (mild conditions and ambulatory), gray (terminal patients to be observed and kept comfortable), and black (dead on arrival) [61]. Patients triaged to the red zone require immediate intervention and resuscitation, preferably in a resuscitation area if possible, with primary and secondary surveys completed to assess for and intervene on life-threatening emergencies (e.g., decompression and tube thoracostomy for pneumothorax). Healthcare professionals should assume these patients have severe traumatic injuries until proven otherwise. Any active major bleeding must be stopped, with transfusion recommended to restore volume [18]. Hypothermia must be avoided, which is associated with poor prognosis and increased mortality [62]. Imaging should be obtained based on the injury (e.g., radiographs for injured extremity, head computed tomography [CT] non-contrast for severe head injury, etc.). Laboratory analysis should include complete blood cell count (CBC), extended

electrolyte panel (e.g., calcium, magnesium, phosphate), renal and liver function, creatine kinase, venous blood gas, lactate, and urinalysis. ECG is also advised. Maintaining clear patient records is vital during any mass casualty event, so it is important to develop a system for tracking patients (e.g., paper chart) as many may not be registered until hours later in their hospital care [3].

3.6.2. Crush injury

Crush injuries affecting the torso can result in hepatic and splenic laceration/rupture and hollow viscus injuries. The likelihood of these injuries increases in a structural collapse, and they are associated with increased mortality [3,5]. Following the primary and secondary trauma survey, ultrasound can be used to assess for intra-abdominal fluid. If present and the patient is unstable, surgical intervention is likely necessary. If free fluid is not present on ultrasound but the patient has signs/ symptoms of abdominal trauma (e.g., bruising, pain with palpation), dedicated imaging including CT is recommended if possible. However, CT is a limited resource in mass casualty events. Immediate surgical control with damage control surgery (DCS) may be necessary depending on the injury (liver or splenic laceration) [63].

3.6.3. Crush syndrome

Crush syndrome is common in patients with significant damage to large muscle groups but may develop irrespective of trauma severity [3,4,12,18]. Thus, all crush victims are at risk of crush syndrome, which is associated with significant morbidity (including renal failure) and mortality [3,4,6,12,22]. The mortality rate among those with crush syndrome approaches 20%, though this rate is even higher in those with multiorgan failure [64,65]. Crush syndrome should be suspected if the patient has oliguria, dark urine, hypertension, edema, dyspnea, nausea, or vomiting [3,4,6]. Significant predictors include tachycardia (>120/min), abnormal urine color, white blood cell count >18,000/mm³, and hyperkalemia [66].

Fluid resuscitation in crush victims is essential to reducing the risk of AKI as previously discussed [3-6,18]. Volume status must be assessed at the healthcare facility after transport, based on physical examination including vital signs, mucous membranes, capillary refill, and skin turgor [67-69]. The overall target for fluid resuscitation is euvolemia and urine output >50 mL/h [3,4]. If hemorrhage is not present, IV fluids should be continued to prevent hypoperfusion and reduce the risk of AKI or crush syndrome. If hemorrhage is present resulting in hemodynamic compromise, direct pressure should be applied and blood products transfused [3,18]. If the patient is hypotensive and volume depleted, isotonic saline should be administered. Isotonic bicarbonate infusion (150 mEq bicarbonate in 1 L D5W) is recommended if the patient is hyperkalemic and acidemic [70-72]. Importantly, loop diuretics such as furosemide are not beneficial in treating crush syndrome with AKI, as many patients are hypovolemic, and may increase mortality and delay renal recovery [73-78]. Loop diuretics may be considered if the patient is hypervolemic [3-5].

AKI associated with crush syndrome typically presents with oliguria or anuria in first stages, ranging between 7 and 21 days [22,28]. This is associated with poor prognosis [79,80]. The length of oliguria depends on the degree of initial ischemia, recurrence of ischemia, and nephrotoxic insults. Nephrotoxic agents (e.g., aminoglycosides, nonsteroidal anti-inflammatory drugs) should be avoided during this phase, and hemodynamics should be optimized to ensure renal perfusion [3,5].

Several other metabolic disorders frequently occur in crush syndrome. One of the most common abnormalities in crush syndrome is severe and rapid onset hyperkalemia [3-6,18]. ECG and laboratory testing should be used to assess for hyperkalemia. However, the absence of ECG findings should not be used to exclude hyperkalemia [3,5,53,54]. If hyperkalemia is present, the previously discussed treatments are recommended [56,57]. However, calcium, insulin/glucose, sodium bicarbonate, and beta agonist therapy have temporary effects [56,57]. Sodium bicarbonate has a controversial effect on reducing serum

potassium in the absence of acidemia but may be administered [56,57,81]. Metabolic acidemia can occur due to cellular necrosis, uremia, lactic acid release, and shock [3,5]. Severe acidemia may result in reduced myocardial contractility, decreased cardiac output, arrhythmias, and hypotension [70-72]. If present, isotonic sodium bicarbonate infusion as previously discussed is recommended [3-5]. Hemodialysis may be required. Alkalemia is more rare in crush victims and is most commonly due to excessive sodium bicarbonate infusion or high bicarbonate dialysates [3,5,82]. Alkalemia can increase calcium protein binding and reduce ionized calcium levels. If serum pH exceeds 7.45, acetazolamide can be considered. Hypocalcemia may occur but should not be treated unless the patient is symptomatic, as calcium may precipitate in muscles with phosphate [83-86]. Signs and symptoms of hypocalcemia may include Chvostek and Trousseau signs, tetany, paresthesias, hypotension, carpopedal spasm, seizures, bradycardia, reduced cardiac contractility, and prolonged QT interval [3]. If these are present, calcium gluconate 1-2 g IV over 10-20 min is recommended [3].

Hemodialysis (HD) may be necessary and is lifesaving in those with severe renal injury or failure resulting in uremia, volume overload, refractory electrolyte issues (e.g., hyperkalemia), and severe acidemia [3,5,87-89]. HD can remove 80-140 mmol of potassium every session, with plasma potassium levels falling 1-1.3 mmol/L in the first 60 min of HD [57,90]. Potassium levels can rebound after HD, and thus levels should be monitored following HD sessions [91]. Due to the severity of crush syndrome, HD may be needed two times per day after the initial injury [3,5]. Unfortunately, mass casualty events such as massive earthquakes can result in significant numbers of patients with crush syndrome [3,22]. These patients may require HD and rapidly overwhelm local resources. A surge plan should be in place, which may require transport of patients to other locations for HD or transport of equipment to the healthcare facility [3-5]. Peritoneal dialysis (PD) is not a first line option to treat hyperkalemia due to lower clearance rates, but it can be used in cases where HD machines are not available [22]. The Renal Disaster Relief Task Force can assist with advice, personnel, material, and psychosocial support for disasters with renal disease worldwide [3,5]. In the United States, the National Disaster Medical System (NDMS) can provide a Crush Injury Specialty Disaster Medical Assistance Team (DMAT) to provide advice and resources to local healthcare facilities [3-5].

Soft tissue injuries are common following natural disasters, including severe extremity injuries resulting in mangled tissue [3,4,6,18]. Assessment of the wound characteristics, tissues involved (e.g., vessels, nerves, bones), and neurovascular status is recommended. Wound infections are common in crush injury victims, with infections being a common cause of death [92,93]. Any open wound should be assumed to be contaminated. Wounds should be irrigated and covered with a clean dry dressing. Guidelines recommend against directly placing antibacterial agents such as betadine into the wound [3,18]. Severely contaminated, open wounds typically require washout and debridement in the operating room. Extensive debridement may be required due to the difficulty in differentiating necrotic and viable tissue [3,4,18]. Crush wounds are frequently contaminated with Gram-positive, Gram-negative, and anaerobic bacteria, and thus antibiotics are recommended [3-5,18]. If a wound appears infected, empiric broad-spectrum antibiotic therapy is warranted, and debridement should be performed as soon as possible [3,4,18]. Tetanus vaccination status should be assessed and updated if necessary [3,4,18].

Limb viability may be questionable in patients with severely mangled extremities, and treatment of limb injuries should focus on saving the patient's life first and then restoring or preserving function to the extremity [3]. Amputation can save the patient's life in the appropriate situation. Amputation rates range between 3 and 58%, varying based on duration of entrapment, other injuries, and local and healthcare facility resources [3,5,12,93,94]. Limb salvage is less likely with severe bone loss, extensive soft tissue loss, distal sensation and motor function loss related to peripheral nerve damage, and major vascular injury requiring reconstruction for flow restoration [3,6,18,95]. Despite this, guidelines recommend restricting amputation to non-salvageable limbs or if injuries to the limb have caused uncontrollable hemorrhage, sepsis, or severe systemic inflammation [3,6,18]. If amputation is indicated, it should be performed as soon as possible [3,6,13,18,42]. If the patient is critically ill and amputation is potentially life threatening, the extremity may be garroted and cooled to reduce pain, further infection, and release of toxins [3,6,18]. Once the patient has been stabilized, definitive traumatic amputation can then be performed [3,6,18]. Of note, a variety of scoring systems are available for assessing the possibility of limb salvage including the mangled extremity severity score, but these scores have not demonstrated high predictive ability [96,97].

Compartment syndrome may be present in those with severe crush injury by the time of arrival to the healthcare facility. Early recognition of this is essential to ensuring adequate treatment, including fasciotomy [3,21,98]. Clinicians should assess for swelling, severe pain with or without passive motion, paresthesias, color changes, and diminished or lost pulses [21]. Delays to fasciotomy are associated with risk of permanent neurovascular injury and limb loss [29,64,99,100]. However, prophylactic fasciotomy in patients with severe crush injury is not recommended [3,6,11,29,99,101]. Literature suggests prophylactic fasciotomy in the absence of objective elevated intracompartmental pressures is not beneficial and increases risk of amputation, bleeding, long-term motor and sensory nerve dysfunction, and infection [3,6,11,99,101]. Intracompartmental pressures should be evaluated if clinical signs or symptoms of compartment syndrome are present [21]. Fasciotomy is recommended if the intracompartmental absolute pressure is >30 mmHg or if the delta pressure is <20-30 mmHg [21]. If assessment of intracompartmental pressures is not available, fasciotomy should be considered in patients with significantly diminished or lost pulses [3,100,102]. Fasciotomy in late compartment syndrome (e.g., present for 8 h) is controversial due to the extensive myonecrosis and high likelihood of neurovascular compromise [3,4,18]. Other measures to reduce compartment pressures include administering IV fluids to ensure compartment perfusion, analgesia, raising the affected extremity to the level of the heart, reducing any fractures or dislocations, and removing any casts or splints [21]. Mannitol may reduce intracompartmental pressures and muscle edema, with literature suggesting mannitol can improve motor function and reduce limb swelling and pain [103-105]. It may be considered in patients with increased intracompartmental pressures who do not yet meet criteria for fasciotomy if there are no

Table 4

Primary components of focus and responsibility at the healthcare facility.

rform triage to designate victims to appropriate trees
form primary survey and manage any life-threatening ditions, followed by secondary survey aluate/manage fluid problems; if hypovolemic, find and it underlying cause (administer blood products for norrhage) wrrect hypothermia eat infection early with broad-spectrum antibiotics
eee appropriate records of patients eeek the type of fluid administered and fluid status etermine serum potassium and treat hyperkalemia cium, insulin/glucose, beta agonists, sodium irbonate) sert urinary catheter for urine output monitoring event and treat AKI; hemodialysis may be necessary patient is fluid overloaded and oliguric, restrict fluids and iate hemodialysis roid nephrotoxic medications and dose other medications renal function eat any other emergencies including acidemia, alkalemia,

Table 5

Complications associated	with crush syndrome.
--------------------------	----------------------

System	Complication	Consideration
Cardiovascular Gastrointestinal	Congestive heart failure, hypertension, myocardial infarction Bleeding, ulcers	Due to disaster-related stress, fluid overload, interruption of chronic medications Stress, drugs that disrupt epithelial lining or increase acidity, hemorrhage due to DIC or uremia
Hematologic	Anemia, leukocytosis, thrombocytopenia	Traumatic bleeding, hemodilution, rhabdomyolysis, infection, DIC
Infection	Sepsis, pneumonia, wound, urinary tract, tetanus	Foreign bodies, intravascular catheters, urinary catheters, aspiration, inadequate surgical debridement or antibiotics
Metabolic	Impaired glycemic control	Stress, irregular or poor nutrition, other medical/surgical complications, problems with regular therapy
Neurologic	Peripheral neuropathy, paresis/paralysis	Closed head injury, stretching, immobilization/compression of peripheral nerves, brain and/or spinal injury
Pulmonary	Bronchitis, asthma, pulmonary edema, acute respiratory distress syndrome	Stress, suboptimal living condition, inhalation of dust or noxious gas, aspiration, volume overload
Psychiatric	Depression, delirium, posttraumatic stress disorder	Disaster-related stress, loss of family/friends or property

DIC, disseminated intravascular coagulation.

contraindications (e.g., hypervolemia, hypovolemia, heart failure, hypertension) [105,106].

Primary components of focus and responsibilities for clinicians at the healthcare facility are listed in Table 4. Complications are common in patients with crush syndrome and include those present in Table 5.

4. Conclusions

Natural and human-made disasters affect millions annually. These disasters may result in mass casualty events and severe medical issues including crush injury and crush syndrome. Crush injury occurs with direct physical trauma to the human body. It may result in asphyxia, severe orthopedic injury, hypotension, organ injury, AKI, sepsis, and ARDS. Crush syndrome is the systemic manifestation of severe, traumatic muscle injury. Care at the scene requires trained individuals and focuses on treating life-threatening injuries, extrication, triage, fluid resuscitation, and transport. Clinicians at the healthcare facility must appropriately triage patients; perform initial primary and secondary surveys; and manage complications such as AKI, infection, severe chest or abdominal trauma, metabolic issues (e.g., hyperkalemia, acidemia), and compartment syndrome. A knowledge of these events and complications can improve the care of patients with crush injury and crush syndrome.

Declaration of Competing Interest

None. No authors have published a similar topic on crush injury or crush syndrome. No author is submitting a manuscript on this subject to another journal until AJEM makes a decision to reject or actually publishes (not just accepts) this submission.

Acknowledgements

All authors conceived the idea for this manuscript and contributed substantially to the writing and editing of the review. This manuscript did not utilize any grants, and it has not been presented in abstract form. This clinical review has not been published, it is not under consideration for publication elsewhere, its publication is approved by all authors and tacitly or explicitly by the responsible authorities where the work was carried out, and that, if accepted, it will not be published elsewhere in the same form, in English or in any other language, including electronically without the written consent of the copyright-holder. This review does not reflect the views or opinions of the U.S. government, Department of Defense, Defense Health Agency, U.S. Army, U.S. Air Force, or SAUSHEC EM Residency Program.

References

- National Center for Environmental Information. Billion-Dollar Weather and Climate Disasters. Available at: https://www.ncei.noaa.gov/access/billions/; 2023. Accessed 12 February 2023.
- [2] Our World in Data. Natural Disasters. Available at: https://ourworldindata.org/ natural-disasters. Accessed 12 February 2023.
- [3] 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 Apr;27 (Suppl. 1) i1–67.
- [4] Sever MS, Vanholder R. Management of crush victims in mass disasters: highlights from recently published recommendations. Clin J Am Soc Nephrol. 2013 Feb;8(2): 328–35.
- [5] Gibney RT, Sever MS, Vanholder RC. Disaster nephrology: crush injury and beyond. Kidney Int. 2014 May;85(5):1049–57.
- [6] International Search and Rescue Advisory Group. The medical management of the entrapped person with crush syndrome. Available at: https://www.insarag.org/ wp-content/uploads/2018/12/ATTACHMENT_C_The_Medical_Management_of_ the_Entrapped_Patient_with_Crush_Syndrome_10_2019_-_Final.pdf; October 2019. Accessed 17 February 2023.
- [7] Bilham R. Earthquakes and urban growth. Nature. 1988;336:625-6.
- [8] Lall SV, Deichman U. Density and disasters: Economics of urban hazard risk. https://gfdrr.org/docs/WPS5161.pdf; 2009. Accessed 16 February 2023.
- [9] Bartels SA, VanRooyen MJ. Medical complications associated with earthquakes. Lancet. 2012;379:748–57.
- [10] McKenna P. Earthquake engineer: earthquakes don't kill, but buildings do. N Scientist. 2011;210:23.
- [11] Michaelson M. Crush injury and crush syndrome. World J Surg. 1992;16(5): 899–903. Sep-Oct.
- [12] Oda J, Tanaka H, Yoshioka T, et al. Analysis of 372 patients with crush syndrome caused by the Hanshin-Awaji earthquake. J Trauma. 1997;42:470–5.
- [13] Slater MS, Mullins RJ. Rhabdomyolysis and myoglobinuric renal failure in trauma and surgical patients: a review. J Am Coll Surg. 1998;186:693–716.
- [14] Bywaters EG, Beall D. Crush injuries with impairment of renal function. 1941. J Am Soc Nephrol. 1998;9:322–32.
- [15] Better OS. History of the crush syndrome: from the earthquakes of Messina, Sicily 1909 to Spitak, Armenia 1988. Am J Nephrol. 1997;17:392–4.
- [16] Bywaters EGL 50 years on -the crush syndrome. BMJ. 1990;301:1412-5.
- [17] Centers for Disease Control and Prevention. After an earthquake: management of crush injuries & crush syndrome. Available at: https://stacks.cdc.gov/view/cdc/11 904; 14 January 2010. Accessed 21 February 2023.
- [18] Crush Syndrome Prolonged Field Care. Joint Trauma System Clinical Practice Guideline. Published. Available at: https://jts.amedd.army.mil/assets/docs/cpgs/ Crush_Syndrome_PFC_28_Dec_2016_ID58.pdf; 28 December 2016. Accessed 21 February 2023.
- [19] World Vision. Turkey and Syria Earthquake FAQs. Available at: https://www. worldvision.org/disaster-relief-news-stories/2023-turkey-and-syria-earthquakefaqs. Accessed 23 February 2023.
- [20] Tanaka H, Oda J, Iwai A, et al. Morbidity and mortality of hospitalized patients after the 1995 Hanshin-Awaji earthquake. Am J Emerg Med. 1999 Mar;17(2):186–91.
- [21] Long B, Koyfman A, Gottlieb M. Evaluation and Management of Acute Compartment Syndrome in the emergency department. J Emerg Med. 2019 Apr;56(4): 386–97.
- [22] Sever MS, Erek E, Vanholder R, et al. Clinical findings in the renal victims of a catastrophic disaster: the Marmara earthquake. Nephrol Dial Transplant. 2002;17: 1942–9.
- [23] Better OS, Abassi Z, Rubinstein I, et al. The mechanism of muscle injury in the crush syndrome: ischemic versus pressure-stretch myopathy. Miner Electrolyte Metab. 1990;16:181–4.
- [24] Macintyre AG, Barbera JA, Smith ER. Surviving collapsed structure entrapment after earthquakes: a 'time-to-rescue' analysis. Prehosp Disaster Med. 2006;21:4–17.
- [25] Ron D, Taitelman U, Michaelson M, et al. Prevention of acute renal failure in traumatic rhabdomyolysis. Arch Intern Med. 1984;144:277–80.
- [26] Kellum JA, Levin N, Bouman C, Lameire N. Developing a consensus classification system for acute renal failure. Curr Opin Crit Care. 2002;8:509–14.
- [27] Better OS, Stein JH. Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. N Engl J Med. 1990;322:825–9.
- [28] Better OS. Rescue and salvage of casualties suffering from the crush syndrome after mass disasters. Mil Med. 1999;164:366–9.
- [29] Reis ND, Michaelson M. Crush injury to the lower limbs. Treatment of the local injury. J Bone Joint Surg Am. 1986;68:414–8.
- [30] Better OS. The crush syndrome revisited (1940-1990). Nephron. 1990;55:97-103.

- [31] Eken C, Yigit O. Traumatic asphyxia: a rare syndrome in trauma patients. Int J Emerg Med. 2009 Aug 1;2(4):255–6.
- [32] Gulbahar G, Kaplan T, Gundogdu AG, et al. A rare and serious syndrome that requires attention in emergency service: traumatic asphyxia. Case Rep Emerg Med. 2015;2015:359814.
- [33] de Almeida MM, von Schreeb J. Human stampedes: an updated review of current literature. Prehosp Disaster Med. 2019 Feb;34(1):82–8.
- [34] Long B, Koyfman A, Gottlieb M. An evidence-based narrative review of the emergency department evaluation and Management of Rhabdomyolysis. Am J Emerg Med. 2019 Mar;37(3):518–23.
- [35] Sheng ZY. Medical support in the Tangshan earthquake: a review of the management of mass casualties and certain major injuries. J Trauma. 1987;27:1130–5.
- [36] Collins AJ, Burzstein S. Renal failure in disasters. Crit Care Clin. 1991;7:421-35.
- [37] Atef MR, Nadjatfi I, Boroumand B, et al. Acute renal failure in earthquake victims in Iran: epidemiology and management. Q J Med. 1994;87:35–40.
- [38] Hatamizadeh P, Najafi I, Vanholder R, et al. Epidemiologic aspects of the bam earthquake in Iran: the nephrologic perspective. Am J Kidney Dis. 2006;47:428–38.
- [39] Oda Y, Shindoh M, Yukioka H, et al. Crush syndrome sustained in the 1995 Kobe, Japan, earthquake; treatment and outcome. Ann Emerg Med. 1997;30:507–12.
- [40] Donmez O, Meral A, Yavuz M, et al. Crush syndrome of children in the Marmara earthquake. Turkey Pediatr Int. 2001;43:678–82.
- [41] Ashkenazi I, Isakovich B, Kluger Y, et al. Prehospital management of earthquake casualties buried under rubble. Prehosp Disaster Med. 2005;20:122–33.
- [42] Santangelo ML, Usberti M, Di Salvo E, et al. A study of the pathology of the crush syndrome. Surg Gynecol Obstet. 1982;154:372–4.
- [43] Noji EK. Acute renal failure in natural disasters. Ren Fail. 1992;14:245–9.
 [44] Zager RA. Studies of mechanisms and protective maneuvers in myoglobinuric acute
- renal injury. Lab Investig. 1989;60:619–29. [45] Jagodzinski NA, Weerasinghe C, Porter K. Crush injuries and crush syndrome – a re-
- view. Part 2: the local injury. Trauma. 2010;12:133–48. [46] Stewart RD. Young IC. Kenney DA. et al. Field surgical intervention: an unusual case
- [46] Stewart RD, Young JC, Kenney DA, et al. Field surgical intervention: an unusual case. J Trauma. 1979;19:780–3.
- [47] Bonanno FG. Ketamine in war/tropical surgery (a final tribute to the racemic mixture). Injury. 2002;33:323–7.
- [48] Peek-Asa C, Kraus JF, Bourque LB, et al. Fatal and hospitalized injuries resulting from the 1994 Northridge earthquake. Int J Epidemiol. 1998;27:459–65.
- [49] Yoshimura N, Nakayama S, Nakagiri K, et al. Profile of chest injuries arising from the 1995 southern Hyogo prefecture earthquake. Chest. 1996;110:759–61.
- [50] Clarkson L, Williams M. EMS Mass Casualty Triage. [Updated 2022 Aug 8]. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan Available from: https://www.ncbi.nlm.nih.gov/books/NBK459369/?report= classic.
- [51] Osler T, Glance LG, Hosmer DW. Simplified estimates of the probability of death after burn injuries: extending and updating the baux score. J Trauma. 2010;68: 690–7.
- [52] Sever MS, Erek E, Vanholder R, et al. Effect of gender on various parameters of crush syndrome victims of the Marmara earthquake. J Nephrol. 2004;17:399–404.
- [53] Wrenn KD, Slovis CM, Slovis BS. The ability of physicians to predict hyperkalemia from the ECG. Ann Emerg Med. 1991;20:1229–32.
- [54] Montague BT, Ouellette JR, Buller GK. Retrospective review of the frequency of ECG changes in hyperkalemia. Clin J Am Soc Nephrol. 2008;3:324–30.
- [55] Vanholder R, Borniche D, Claus S, et al. When the earth trembles in the Americas: the experience of Haiti and Chile 2010. Nephron Clin Pract. 2011;117:c184–97.
- [56] Long B, Warix JR, Koyfman A. Controversies in management of hyperkalemia. J Emerg Med. 2018 Aug;55(2):192–205.
- [57] Weisberg LS. Management of severe hyperkalemia. Crit Care Med. 2008;36: 3246-51.
- [58] Schultz CH, Koenig KL, Noji EK. A medical disaster response to reduce immediate mortality after an earthquake. N Engl J Med. 1996;334:438–44.
- [59] Sever MS, Erek E, Vanholder R, et al. The Marmara earthquake: epidemiological analysis of the victims with nephrological problems. Kidney Int. 2001;60:1114–23.
- [60] Frykberg ER, Tepas 3rd JJ. Terrorist bombings. Lessons learned from Belfast to Beirut. Ann Surg. 1988 Nov;208(5):569–76.
- [61] Ricci E, Pretto E. Assessment of prehospital and hospital response in disaster. Crit Care Clin. 1991;7:471–84.
- [62] Jurkovich GJ, Greiser WB, Luterman A, et al. Hypothermia in trauma victims: an ominous predictor of survival. J Trauma. 1987;27:1019–24.
- [63] Damage Control Resuscitation. Joint Trauma System Clinical Practice Guideline. Published. Available at: https://jts.amedd.army.mil/assets/docs/cpgs/Crush_ Syndrome_PFC_28_Dec_2016_ID58.pdf; 12 Jul 2019. Accessed 21 February 2023.
- [64] Malinoski DJ, Slater MS, Mullins RJ. Crush injury and rhabdomyolysis. Crit Care Clin. 2004 Jan;20(1):171–92.
- [65] Stewart IJ, Faulk TI, Sosnov JA, et al. Rhabdomyolysis among critically ill combat casualties: associations with acute kidney injury and mortality. J Trauma Acute Care Surg. 2016 Mar;80(3):492–8.
- [66] Aoki N, Demsar J, Zupan B, et al. Predictive model for estimating risk of crush syndrome: a data mining approach. J Trauma. 2007;62:940–5.
- [67] KDIGO Acute Kidney Injury Work Group. KDIGO clinical practice guidelines for acute kidney injury. Kidney Int. 2012;2(Suppl):1–138.
- [68] McGee S, Abernethy 3rd WB, Simel DL. The rational clinical examination. Is this patient hypovolemic? JAMA. 1999;281:1022–9.
- [69] Monnet X, Rienzo M, Osman D, et al. Passive leg raising predicts fluid responsiveness in the critically ill. Crit Care Med. 2006;34:1402–7.
- [70] Sabatini S, Kurtzman NA. Bicarbonate therapy in severe metabolic acidosis. J Am Soc Nephrol. 2009;20:692–5.

Descargado para Lucia Angulo (lu.maru26@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en julio 19, 2023. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2023. Elsevier Inc. Todos los derechos reservados.

B. Long, S.Y. Liang and M. Gottlieb

- [71] Kraut JA, Kurtz I. Use of base in the treatment of acute severe organic acidosis by nephrologists and critical care physicians: results of an online survey. Clin Exp Nephrol. 2006;10:111–7.
- [72] Kraut JA, Madias NE. Metabolic acidosis: pathophysiology, diagnosis and management. Nat Rev Nephrol. 2010;6:274–85.
- [73] Bagshaw SM, Delaney A, Haase M, et al. Loop diuretics in the management of acute renal failure: a systematic review and metaanalysis. Crit Care Resusc. 2007;9:60–8.
 [74] Mehta RL, Pascual MT, Soroko S, et al. Diuretics, mortality, and nonrecovery of renal
- function in acute renal failure. JAMA. 2002;288:2547–53. [75] Shilliday IR, Quinn KJ, Allison ME. Loop diuretics in the management of acute renal
- [75] Shiniday R, Quini K, Anson ME. Loop dureuss in the management of acute rehat failure: a prospective, double-blind, placebo controlled, randomized study. Nephrol Dial Transplant. 1997;12:2592–6.
- [76] Uchino S, Doig GS, Bellomo R, et al. Diuretics and mortality in acute renal failure. Crit Care Med. 2004;32:1669–77.
- [77] Ho KM, Sheridan DJ. Meta-analysis of furosemide to prevent or treat acute renal failure. BMJ. 2006;333:420.
- [78] Shimazu T, Yoshioka T, Nakata Y, et al. Fluid resuscitation and systemic complications in crush syndrome: 14 Hanshin-Awaji earthquake patients. J Trauma. 1997; 42:641–6.
- [79] Anderson RJ, Linas SL, Berns AS, et al. Nonoliguric acute renal failure. N Engl J Med. 1977;296:1134–8.
- [80] Parker RA, Himmelfarb J, Tolkoff-Rubin N, et al. Prognosis of patients with acute renal failure requiring dialysis: results of a multicenter study. Am J Kidney Dis. 1998;32:432–43.
- [81] Allon M, Shanklin N. Effect of bicarbonate administration on plasma potassium in dialysis patients: interactions with insulin and albuterol. Am J Kidney Dis. 1996; 28:508–14.
- [82] Better OS. Post-traumatic acute renal failure: pathogenesis and prophylaxis. Nephrol Dial Transplant. 1992;7:260–4.
- [83] Llach F, Felsenfeld AJ, Haussler MR. The pathophysiology of altered calcium metabolism in rhabdomyolysis-induced acute renal failure. Interactions of parathyroid hormone, 25-hydroxycholecalciferol, and 1,25-dihydroxycholecalciferol. N Engl J Med. 1981;305:117–23.
- [84] Knochel JP. Serum calcium derangements in rhabdomyolysis. N Engl J Med. 1981; 305:161–3.
- [85] Akmal M, Bishop JE, Telfer N, et al. Hypocalcemia and hypercalcemia in patients with rhabdomyolysis with and without acute renal failure. J Clin Endocrinol Metab. 1986;63:137–42.
- [86] Lane JT, Boudreau RJ, Kinlaw WB. Disappearance of muscular calcium deposits during resolution of prolonged rhabdomyolysis induced hypercalcemia. Am J Med. 1990;89:523–52.
- [87] Sever MS, Erek E, Vanholder R, et al. Serum potassium in the crush syndrome victims of the Marmara disaster. Clin Nephrol. 2003;59:326–33.
- [88] Better OS, Rubinstein I, Winaver J. Recent insights into the pathogenesis and early management of the crush syndrome. Semin Nephrol. 1992;12:217–22.

- [89] Collins AJ. Kidney dialysis treatment for victims of the Armenian earthquake. N Engl J Med. 1989;320:1291–2.
- [90] Evans KJ, Greenberg A. Hyperkalemia: a review. J Intensive Care Med. 2005;20: 272–90.
- [91] Blumberg A, Roser HW, Zehnder C, et al. Plasma potassium in patients with terminal renal failure during and after haemodialysis; relationship with dialytic potassium removal and total body potassium. Nephrol Dial Transplant. 1997;12: 1629–34.
- [92] Keven K, Ates K, Sever MS, et al. Infectious complications after mass disasters: the Marmara earthquake experience. Scand J Infect Dis. 2003;35:110–3.
- [93] Chen X, Zhong H, Fu P, et al. Infections in crush syndrome: a retrospective observational study after the Wenchuan earthquake. Emerg Med J. 2011;28:14–7.
- [94] Tattersall JE, Richards NT, McCann M, et al. Acute haemodialysis during the Armenian earthquake disaster. Injury. 1990;21:25–8.
- [95] LEAP Study group. Ability of lower-extremity injury severity scores to predict functional outcome after limb salvage. J Bone Joint Surg Am. 2008;90:1738–43.
- [96] Johansen K, Daines M, Howey T, et al. Objective criteria accurately predict amputation following lower extremity trauma. J Trauma. 1990 May;30(5):568–72. discussion 572–3.
- [97] Togawa S, Yamami N, Nakayama H, et al. The validity of the mangled extremity severity score in the assessment of upper limb injuries. J Bone Joint Surg (Br). 2005 Nov;87(11):1516–9.
- [98] Farber A, Tan TW, Hamburg NM, 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 Sep;43(9):1486–91.
- [99] Better OS, Rubinstein I, Reis DN. Muscle crush compartment syndrome: fulminant local edema with threatening systemic effects. Kidney Int. 2003;63:1155–7.
- [100] 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.
- [101] Michaelson M, Taitelman U, Bshouty Z, et al. Crush syndrome: experience from the Lebanon war, 1982. Isr J Med Sci. 1984;20:305–7.
- [102] Gunal AI, Celiker H, Dogukan A, et al. Early and vigorous fluid resuscitation prevents acute renal failure in the crush victims of catastrophic earthquakes. J Am Soc Nephrol. 2004 Jul;15(7):1862–7.
- [103] Daniels M, Reichman J, Brezis M. Mannitol treatment for acute compartment syndrome. Nephron. 1998;79:492–3.
- [104] Better OS, Zinman C, Reis DN, et al. Hypertonic mannitol ameliorates intracompartmental tamponade in model compartment syndrome in the dog. Nephron. 1991;58:344–6.
- [105] Better OS, Rubinstein I, Winaver JM, et al. Mannitol therapy revisited (1940–1997). Kidney Int. 1997;52:886–94.
- [106] Smith J, Greaves I. Crush injury and crush syndrome: a review. J Trauma. 2003;54: S226-30.