

Pathophysiology of Crush Syndrome and Its Prehospital Management, with Preventive Strategies for Earthquake Victims: A Literature Review

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ABSTRACT

Earthquakes are major public health disasters that frequently cause building collapses and prolonged entrapment, resulting in crush injuries that may progress to crush syndrome (CS), a life-threatening systemic complication of traumatic muscle damage. This literature review aims to summarize the pathophysiology of CS and evaluate current prehospital management and preventive strategies to improve survival among earthquake victims. A comprehensive search of PubMed, ScienceDirect, and ResearchGate was conducted using the PICO framework and Boolean operators. Open-access English articles published between 2017 and 2026 were screened, yielding 17 eligible studies from 2,530 initial records for qualitative synthesis. The findings indicate that CS develops through ischemia during compression followed by reperfusion injury after extrication, leading to rhabdomyolysis, hyperkalemia, and acute kidney injury. Effective prehospital management includes rapid triage and early administration of potassium-free isotonic saline before extrication to reduce systemic complications. Adjunctive measures, including urine alkalinization with sodium bicarbonate, calcium gluconate for myocardial stabilization, and cooling therapy, may further improve outcomes. Early recognition and aggressive pre-extrication fluid resuscitation remain the cornerstone of CS management, minimizing reperfusion-related complications, preventing acute kidney injury, and improving survival in earthquake mass-casualty settings.

Keyword : Crush syndrome; Earthquake; Fluid resuscitation; Prehospital care; Rhabdomyolysis.

Introduction

Earthquakes remain a catastrophic global public health threat, frequently causing building collapses and mass casualties. Victims trapped under debris for extended periods often sustain severe crush injuries, which are defined by sustained mechanical compression of skeletal muscle leading to extensive ischemia and necrosis^{2,5,6}. Consequently, crush syndrome (CS), the systemic manifestation of this traumatic muscle damage is a leading cause of preventable death in survivors after direct trauma⁶⁻⁸. The pathophysiology of CS involves a lethal transition from the ischemia phase during compression to a systemic reperfusion injury immediately upon extrication^{5,6,9,10}. Physical pressure causes sarcolemma rupture and rhabdomyolysis, triggering a sudden release of toxic intracellular constituents primarily potassium, myoglobin, creatine kinase (CK), and phosphate into the systemic circulation^{6,7,10-12}. Modern insights further implicate Damage Associated Molecular Patterns (DAMPs), such as mtDNA, and iron dependent ferroptosis in propagating systemic inflammatory response syndrome and multi organ failure^{4,7,8,10}. The most immediate threats include hyperkalemia-induced fatal cardiac arrhythmias and acute kidney injury (AKI) caused by myoglobin precipitation and uric acid nephrotoxicity^{6,7,10,11}.

Evidence from major earthquake disasters underscores the massive clinical burden of CS, where high incidences of AKI frequently overwhelm local healthcare resources^{1,2,6,13-16}. In this context, pre-hospital management is pivotal for survival^{2,5,11}. Effective field response relies on rapid triage (e.g., START or MARCH systems) and initiating aggressive fluid resuscitation before extrication (pre-extrication) to dilute systemic toxins^{2,11,16}. Standardized preventive strategies, including using warmed isotonic saline and avoiding potassium containing solutions, are fundamental to reducing mortality^{2,6,12,16,17}. Although CS is extensively researched, management protocols remain dispersed across various disciplines, leaving gaps in the implementation of critical pre-hospital interventions. Therefore, this literature review aims to provide a comprehensive overview of the pathophysiology of crush syndrome and evaluate current pre-hospital and preventive strategies to optimize earthquake victim survival.

Material and Methods

The method used in this study was a literature review focusing on gathering, analyzing, and synthesizing data regarding crush syndrome. To ensure the validity and reliability of the findings, this review prioritized primary research studies, including randomized controlled trials, cohort studies, observational studies, and experimental laboratory models,

while secondary literature was utilized as supporting references. The PICO framework was employed to facilitate the literature search, formulate the research questions, and establish a structured reference layout that aligns with the study objective. The PICO components were defined as follows:

- Population: Earthquake victims experiencing crush syndrome.
- Intervention: Pre-hospital management and preventive strategies.
- Comparison: None or standard emergency care without early field intervention.
- Outcome: Comprehensive understanding of the pathophysiology, effective execution of pre-hospital first aid, and successful prevention of systemic complications.

Literature sources were retrieved from multiple international scientific databases, including PubMed, ScienceDirect, ResearchGate, Springer, PubMed Central (PMC), BioMed Central (BMC), and the National Institutes of Health (NIH) database. The comprehensive search was conducted exclusively in English. Based on the publication criteria, we selected scientific literature published within the last 10 years, specifically between 2017 and 2026, to ensure the inclusion of contemporary medical data and updated clinical guidelines.

The literature search was conducted to identify publications addressing the pathophysiology, prehospital management, and preventive strategies for crush syndrome in earthquake and mass casualty settings, using an iterative Boolean operator strategy to ensure comprehensive and relevant article retrieval. The search combined the terms ("Crush Syndrome" OR "Crush Injury") AND ("Earthquake") AND ("Pre-hospital" OR "Management" OR "Prevention"), with further refinement using expanded medical terminology. Only English-language, open-access articles published between 2017 and 2026 were included. The initial search identified 2,530 records, of which duplicates and ineligible studies were removed through database automation and preliminary screening, leaving 375 articles for title and abstract review. After excluding irrelevant studies, 37 full-text articles were assessed for eligibility, and 20 were excluded due to inappropriate outcomes, irrelevant topics, or duplicate publication. Ultimately, 17 studies met the inclusion criteria and were included in this review. The study selection process is presented in the PRISMA flowchart.

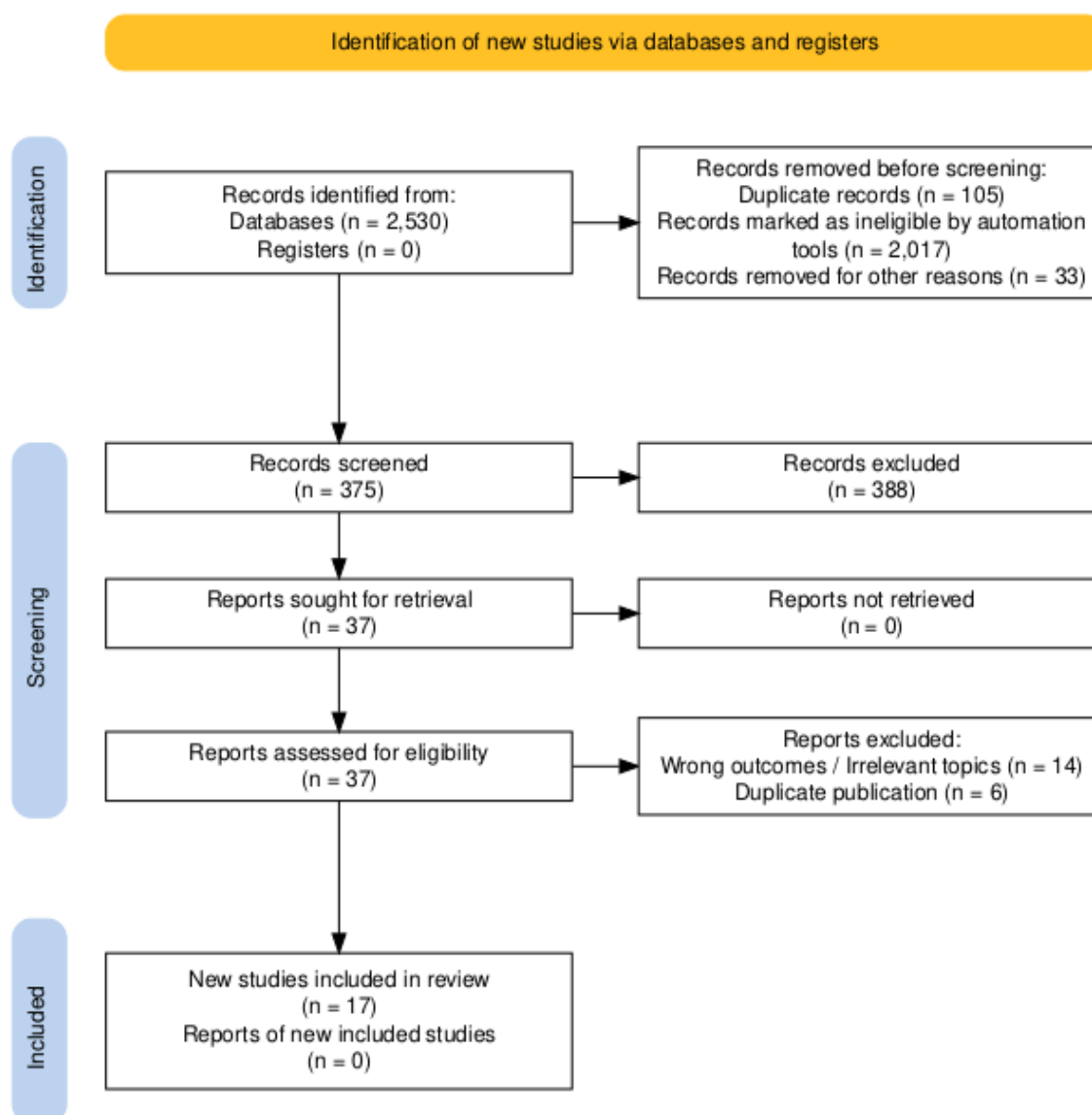


Figure 1. PRISMA Flowchart

Data extracted from the 17 included studies comprised author, publication year, study design, pathophysiological findings, pre-hospital fluid resuscitation protocols, and clinical outcomes. The findings were synthesized using a tabulation framework to compare key evidence. Included studies were required to discuss the pathophysiology, biological mechanisms, pre-hospital management, or preventive fluid resuscitation strategies for crush syndrome in earthquake or mass-casualty settings, while studies lacking these topics were excluded. The synthesized evidence was then analyzed to provide an evidence-based overview of crush syndrome management in emergency and disaster settings.

Results

Table 1. Characteristics of the Reviewed Journals

No	Author & Year	Article Title	Study Design / Article Type	Population / Context	Pathophysiological Aspects	Pre-Hospital Management	Complication Prevention Strategies	Key Findings	Limitations
1	Khan et al. (2025)	Pathophysiology and management of crush syndrome: A narrative review	Narrative Review	Traumatic rhabdomyolysis patients	Sarcolemma rupture triggers release of myoglobin, potassium, and uric acid; roles of ferroptosis and oxidative stress.	Rapid extrication, severity assessment, adherence to BLS and ATI protocols.	Aggressive debridement of necrotic muscle and fasciotomy for compartment syndrome.	AKI is currently associated with iron retention; mental and social rehabilitation are keys to recovery.	Study heterogeneity; challenges in translating animal model results to humans.
2	Daniş et al. (2025)	Pre-earthquake kidney function is a predictor of outcomes in earthquake-related crush syndrome	Multicenter Retrospective Analysis	469 patients in 46 nephrology clinics (Kahramanmaraş Earthquake)	Muscle damage releases myoglobin and electrolytes, triggering systemic inflammation and AKI.	Quality and timeliness of pre-hospital care significantly influence clinical outcomes.	Screening of vulnerable populations; baseline renal function assessment for prioritized triage; aggressive hydration.	Pre-earthquake renal function (creatinine/eGFR) is a significant predictor of mortality.	Retrospective design; lack of data regarding time elapsed between injury and hospital arrival.
3	Usuda et al. (2023)	Crush syndrome: a review for prehospital providers and emergency clinicians	Narrative Review	Field providers and emergency clinicians	Ischemia-reperfusion (IR) injury following decompression releases intracellular toxins into circulation.	Aggressive resuscitation prior to extrication; avoidance of potassium-containing solutions; immediate flu	Icing therapy, hyperbaric oxygen therapy (HBOT), and tourniquet use (controversial) to delay reperfusion.	Immediate initiation of treatment is the most critical factor in reducing mortality.	Knowledge relies heavily on historical data; no established evidence-based treatment exists to date.

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4	Seah & Wulandari (2025)	Importance of early prehospital intervention in crush injury and compartment syndrome: a case report	Case Report	49yo male trapped in a drain for 15 hours	Compression phase (ischemia/necrosis) followed by a reperfusion phase (sudden toxin release).	initiation. Early fluid resuscitation, analgesia (morphine), a pre-extrication IV calcium gluconate.	Proximal tourniquet to delay reperfusion injury onset; calcium gluconate to mitigate hyperkalemia risk.	ED-level field interventions successfully managed severe metabolic disturbances before becoming critical.	Single case report; findings may lack generalizability.
5	Orlyk (2025)	Emergency Medical Care for Victims with Crush Syndrome at the Pre-Hospital Stage	Systematic Review	War and disaster casualties (military/civilian)	Blood supply disruption triggers toxin accumulation; potassium release causes cardiac arrest.	CABDE algorithm, upstream tourniquet before extrication, tight bandaging, cooling, and bolus infusion	Stepwise triage (red/yellow/green zones); massive blood transfusion for critical hemorrhage.	Proper pre-hospital care reduces lethality from 80% down to 15–20%.	Primary focus on tactical and combat conditions.
6	Yeşiloğlu et al. (2023)	Approach to Earthquake Victims at Emergency Services: A Review	Review & Analysis	Earthquake victims in Emergency Departments	Traumatic rhabdomyolysis due to persistent pressure; myoglobin/uric acid obstruct renal tubules.	Pre-earthquake disaster planning, structured triage (START), and direct transport to secondary hospitals.	Early wound debridement, irrigation, antibiotics, and myoglobin screening via urine dipstick.	Accurate triage and multidisciplinary teams (including surgical) optimize patient outcomes.	Resource limitations and destroyed infrastructure impede the response.
7	Abu-Zidan et al.	Prehospital management of earthquake	Narrative Collective Review	Healthcare providers in earthquake	Ischemia-reperfusion induces	MARCH priority (massive	Prevention of hypothermia (hot packs);	The MARCH approach is more appropriate than	Lack of prospective data; need for a

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	(2023)	crush injuries: A collective review		fields	sodium/water influx (edema) and potassium efflux (cardiac arrest).	hemorrhage first), medica evaluation before extrication, an isotonic saline	avoidance of potassium fluids; documentation of entrapment duration.	ABCDE in the field; fluid resuscitation is key to AKI prevention.	national registry for earthquake victims.
8	Abu- Zidan et al. (2024)	Surgical and critical care management of earthquake MSK injuries and crush syndrome	Collective Review	Hospitalized earthquake patients	Reperfusion post-extrication releases toxic metabolites; muscle cell lysis (rhabdomyolysis) triggers AKI.	Principles of first aid, triag and rapid transportation to the hospita	Wound management (NWPT), antibiotics, external fixation for open fractures, and early RRT (dialysis).	Fasciotomy should not be routine; ORIF is the most common fracture surgery; intermittent dialysis is the first choice.	Low-quality evidence regarding indications for fasciotomy and HBOT.
9	Nola et al. (2023)	Crush Syndrome in Earthquakes – Stay and Play or Load and Go?	Professional Paper	19,167 trauma patient records	Muscle ischemia caused by building material pressure; reperfusion syndrome follows load release.	"Greatest goo for the greater number" triag isotonic saline (1 L/h) during extrication.	Avoid tourniquets unless life- threatening bleeding occurs; treat hypocalcemia only if symptomatic.	Crush syndrome patients should "load and go" if parameters worsen; second- line hospitals are primary targets.	Ethical challenges in conducting randomized controlled trials (RCTs) in disasters.
10	Hamlah et al. (2023)	Mini Review of Biochemical Basis, Diagnosis and Management of Crush Syndrome	Mini Review	Natural disaster and conflict casualties	Striated muscle destruction releases enzymes (CK, LDH, ALT) and myoglobin into the bloodstream.	IV hydration early as possible at the injury site, ideally before weight removal.	Calcium gluconate for hyperkalemia; insulin/glucose to shift potassium; bicarbonate for urine.	CK levels >5x the upper normal limit is the primary diagnostic criterion; myoglobinuria is an early AKI indicator.	Experimental therapies (antioxidants/ir on chelators) require translation to humans.

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11	Wood et al. (2025)	Consensus Statement on the Pre-Hospital Management of Crush Injury	Consensus Statement	UK pre-hospital providers	Significant fluid redistribution and release of lactic acid and potassium post-reperfusion.	Earliest possible safe load release, IV/IO access, and warmed saline (500ml bolus for adults).	Interval cardiac monitoring; avoid potassium-elevating drugs (suxamethonium); individualized hydration.	Concerns regarding hyperkalemia have been de-emphasized; prophylactic treatment is not supported without ECG signs.	High-quality evidence is extremely limited; recommendations are based on low certainty.
12	Akrivos et al. (2025)	Crush injury and crush syndrome: a comprehensive review	Comprehensive Review	Mass disaster casualties (earthquakes, floods, etc.)	Muscle ischemia triggers calcium influx damaging mitochondria; free radical release during reperfusion.	Aggressive isotonic saline resuscitation as early as possible; urine alkalization (pH > 6.5).	Electrolyte correction; pain management (opioids); prophylactic antibiotics; continuous ECG monitoring.	Early recognition of rhabdomyolysis risk is vital; AKI is the most common cause of death in the first 2 weeks.	Controversy regarding mannitol use, fasciotomy indications, and optimal dialysis timing.
13	Nepali et al. (2017)	Rhabdomyolysis in Earthquake Victims in Nepal	Retrospective Analysis	1812 victims (572 hospitalized) in Nepal	Muscle necrosis releases cellular constituents obstructing renal tubules (myoglobin precipitation).	Delays in hospital transport increase the risk of AKI development.	Vigilance for CPK thresholds (>18,000 IU/L); aggressive fluid resuscitation in high-risk victims.	AKI occurred at lower CPK levels than in other studies due to delayed medical assistance.	Small AKI sample size (n=23); difficulty accessing data in remote mountainous areas.
14	Yang et al. (2026)	Advances in rhabdomyolysis: A review of pathogenesis, diagnosis, and treatment	Comprehensive Review	Pre-hospital responders and clinicians	Intracellular calcium overload triggers cell membrane lysis and mitochondrial oxidative stress.	Rapid identification of pathogenic factors; immediate establishment of intravenous	Fluid resuscitation (3–24L in first 24h); urine alkalization; AI-assisted diagnostics.	CK >5,000 IU/L is a better AKI predictor in trauma; Machine Learning outperforms traditional SOFA	Primarily retrospective studies; lack of standardized biomarker data; subjectivity in

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						access.		scores.	diagnosis.
15	Thanapa ul et al. (2026)	Pathophysiolo gy-guided biomarkers and therapeutics for precision trauma medicine	Scoping Review	Military and civilian casualties	Mechanical triggers induce DAMPs (mtDNA, HMGB1), endothelial collapse, and cellular senescence.	Use of wearable biosensors an Point-of-Care (POC) platforms for real-time triag	Multi-analyte biomarker panels (FABP3, Syndecan-1); endothelial stabilizers; senotherapeutic s.	Conventional ISS fails to capture molecular dynamics; mtDNA acts as a unifying trauma alarmin.	Most next- generation biomarkers are still in early- stage clinical development.
16	Cao et al. (2025)	Crush syndrome: review of experimental models and emerging therapeutic strategies	Comprehensi ve Review	Experimenta l animal models focus	Reperfusion injury triggers SIRS and Multiple Organ Dysfunction Syndrome (MODS).	Large-volume resuscitation : often impractical in large-scale natural disasters.	Gas interventions (O2, CO, H2S); stem cell therapy (MSCs); biological agents (rhEPO).	Rats are the most commonly used species (70%); need for standardized digital animal models.	Animal anatomy differs from human fascial compartments; challenges in extrapolating data.
17	Abu- Zidan et al. (2025)	Management of earthquake- related acute renal injury	Frontier / Review	Earthquake victims with renal impairment	AKI mechanisms: Prerenal (hypovolemia), Renal (uric acid nephrotoxicity), Postrenal (pelvic fractures).	Medical and fluid therapy must be initiated starting in the field.	Hydration to ensure euvolemia; hyperkalemia prevention; dialysis for K+ > 6.5 mEq/L or pH ≤ 7.1.	AKI is a fatal but preventable complication; intermittent dialysis is preferred over peritoneal.	Demand for hemodialysis often exceeds available resources following major disasters.

This literature review encompasses 17 journal articles published between 2017 and 2026 (Table 1), reflecting the most recent clinical data and experimental advancements in disaster management. The composition of the reviewed studies includes various methodologies to ensure a balanced perspective: narrative, comprehensive, and frontier reviews (7 articles) providing foundational pathophysiological and therapeutic theory, and collective, systematic, or scoping reviews (5 articles) that synthesize multidisciplinary clinical data and management protocols. To provide empirical depth, the review integrates retrospective analyses (2 articles) that evaluate clinical outcomes and predictors of mortality among victims of major earthquakes. Additional sources include a case report detailing targeted pre-hospital interventions in entrapment scenarios (1 article), a professional paper evaluating survival strategies such as the "load and go" principle (1 article), and a consensus statement establishing standardized pre-hospital protocols (1 article). While the primary focus is on mass casualty earthquake victims, several articles also address tactical military trauma and non-disaster civilian incidents to provide a holistic and generalizable framework for the management of crush syndrome.

Demographic Characteristics and Clinical Conditions of Disaster Victims

Earthquakes consistently generate mass casualties with a distinctive pattern of traumatic injuries. Crush syndrome has been identified as the second leading cause of death following direct trauma, with an incidence ranging from 2% to 15% among surviving victims and mortality rates reaching up to 48% without adequate early intervention^{5,10}. The majority of victims who successfully reach healthcare facilities sustain crush injuries predominantly involving the extremities, as prolonged compressive injuries to the chest and abdomen are generally fatal prior to extrication². Epidemiological data from major seismic events further demonstrate that approximately 7.7% of all treated patients present with crush injuries, underlining the consistent and significant clinical burden this condition poses during mass-casualty incidents^{2,15}.

Entrapment duration represents the most consistently identified determinant of crush syndrome severity across the reviewed literature. The longer compressive force is sustained on skeletal muscle, the greater the volume of myoglobin, potassium, and uric acid that will be released into systemic circulation upon reperfusion^{5,7}. Research indicates that delays in hospital transport following extrication significantly increase the risk of Acute Kidney Injury (AKI), even among victims with relatively lower creatine phosphokinase (CPK) levels; this suggests that the time elapsed functions as an independent risk factor irrespective of the

initial degree of muscular damage¹⁴. Furthermore, clinical documentation has established that an entrapment period of 15 hours is sufficient to precipitate severe hyperkalemia and metabolic acidosis by the time of extrication⁹.

Pre-existing comorbidities further stratify the risk of adverse outcomes among earthquake victims. Pre-disaster renal function, measured by serum creatinine and eGFR, as a significant independent predictor of mortality among 469 crush syndrome patients across 46 nephrology clinics, demonstrating that even mild pre-existing renal impairment substantially elevates the risk of severe AKI and requirement for renal replacement therapy¹³. Additionally highlighted that post-compression systemic inflammatory response syndrome (SIRS) can synergistically exacerbate multi-organ damage through multiple organ dysfunction syndrome (MODS), explaining why victims who appear clinically stable immediately following extrication may undergo rapid and unexpected deterioration⁸.

Pathophysiological Findings of Crush Syndrome

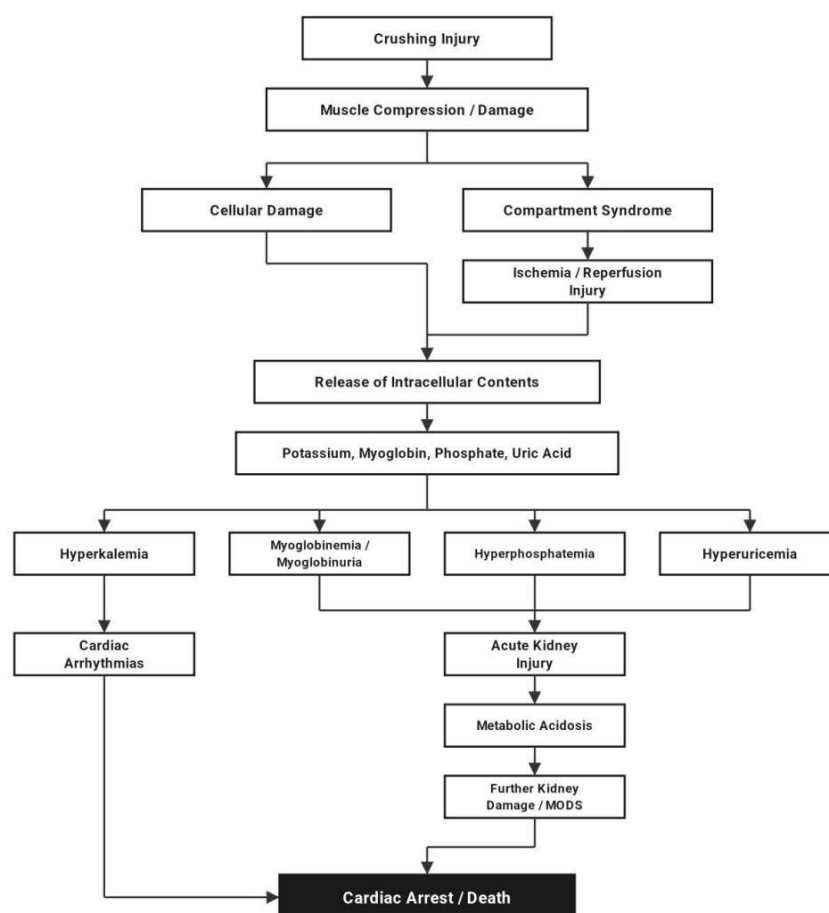


Figure 2. Pathophysiological Crush Syndrome

The pathophysiological mechanisms underlying crush syndrome are summarized in Figure 2. The condition is characterized by a transition from the ischemia phase (during

compression) to the reperfusion phase (following extrication)^{3,4,8,17}. Prolonged physical pressure causes sarcolemma rupture and traumatic rhabdomyolysis, leading to the release of intracellular constituents into the systemic circulation, including creatine kinase (CK), myoglobin, potassium, and phosphate. A CK level exceeding 1000 U/L or five times the upper normal limit is established as the primary diagnostic criterion (1,10,14,17). Recent insights highlight the role of Damage Associated Molecular Patterns (DAMPs), such as mtDNA and HMGB1, as well as ferroptosis (iron dependent cell death), in triggering systemic inflammatory response syndrome (SIRS) and multi organ failure^{1,12,15,16}. The most fatal clinical consequences are sudden cardiac arrest due to severe hyperkalemia and Acute Kidney Injury (AKI) caused by myoglobin precipitation and uric acid nephrotoxicity in the renal tubules^{1,12,17}.

Pre-Hospital Management in the Field

Field management prioritizes early identification and stabilization before and during the extrication process. Structured triage systems such as START (Simple Triage and Rapid Treatment) and the MARCH (Massive hemorrhage, Airway, Respiration, Circulation, Hypothermia) approach are considered more effective in earthquake zones than the traditional ABCDE algorithm because they prioritize life threatening hemorrhage control^{1,2,11,14}. The single most critical intervention is the initiation of aggressive fluid resuscitation before the load is removed (pre-extrication) to dilute toxins released during reperfusion^{2,9-12,16}. Establishing immediate intravenous (IV) or intraosseous (IO) access is essential, despite the challenges of treating entrapped patients^{6,9,16}. Emerging technologies, including wearable biosensors, point-of-care (POC) testing, and AI assisted diagnostics, are being introduced for real-time monitoring of physiological parameters during evacuation^{2,4,15,16}.

Discussion

Systemic Pathophysiology and Biological Consequences of Reperfusion

The pathophysiology of crush syndrome follows a biphasic progression consisting of an ischemia phase during active compression and a reperfusion phase following extrication^{7,10}. During the compression phase, sustained mechanical pressure impairs tissue perfusion and oxygen delivery to skeletal muscle, leading to ATP depletion, disruption of cellular membrane integrity, and sarcolemma rupture^{8,10}. These processes result in the intracellular accumulation of myoglobin, potassium, phosphate, and uric acid within the compressed tissue, while simultaneous calcium influx into myocytes triggers mitochondrial

dysfunction and oxidative stress, ultimately culminating in irreversible myocyte necrosis^{7,8,10}. Upon extrication and restoration of blood flow, the accumulated intracellular contents are released en masse into the systemic circulation, precipitating a spectrum of life-threatening complications through ischemia-reperfusion injury^{5,9,10}. This phase simultaneously triggers the release of lactic acid and inflammatory mediators, activating Systemic Inflammatory Response Syndrome (SIRS) and multi-organ involvement^{2,5,8}.

The sudden influx of sodium and water into damaged cells causes severe cellular edema and systemic hypovolemia, while concurrent potassium efflux into the circulation poses an immediate risk of fatal cardiac arrhythmia^{2,7,10}. Furthermore, Damage-Associated Molecular Patterns (DAMPs), including mitochondrial DNA (mtDNA) and HMGB1, function as key mediators that amplify systemic inflammation beyond the direct nephrotoxic effects of myoglobin alone^{4,8,10}. Among these systemic consequences, Acute Kidney Injury (AKI) represents the most prevalent and potentially fatal complication^{6,7,12}. A creatine kinase (CK) level exceeding five times the upper normal limit serves as the primary diagnostic criterion for rhabdomyolysis, with myoglobinuria functioning as an early clinical indicator of impending renal tubular injury^{12,17}. The mechanisms of AKI in this context are classified into three distinct categories: prerenal injury due to hypovolemia or cardiac depression, intrinsic renal injury caused by myoglobin and uric acid nephrotoxicity, and postrenal obstruction secondary to traumatic injuries such as pelvic fractures⁶. Effective management necessitates addressing these distinct therapeutic implications during both the pre-hospital and hospital-based phases to reduce disaster-related mortality^{2,5,6}.

Pre-Hospital Fluid Management and Field Protocols

Effective pre-hospital management of crush syndrome victims requires the implementation of a structured triage system that can be executed rapidly under conditions of severely limited resources. The MARCH approach (Massive hemorrhage, Airway, Respiration, Circulation, Hypothermia) is identified as more suitable for field application than the conventional ABCDE algorithm, as it prioritizes immediate hemorrhage control as the primary threat to survival². Concurrent implementation of the START system is recommended for rapid victim identification, combined with direct transport to secondary healthcare facilities equipped with trauma management capabilities¹. Furthermore, victims exhibiting progressively deteriorating clinical parameters should be managed following the "load and go" principle, prioritizing immediate evacuation while maintaining continuous fluid resuscitation throughout transport³.

The most critical determinant of pre-hospital management outcomes is the timing of

fluid resuscitation initiation. Evidence consistently demonstrates that fluid administration must be commenced prior to or concurrent with the extrication process; aggressive resuscitation before the compressive load is removed serves to dilute the toxic metabolites that will flood the systemic circulation upon reperfusion (5, 12). Intravenous hydration should be initiated at the injury site as early as possible, ideally before the weight of debris is lifted from the victim¹². In cases where conventional intravenous access cannot be established due to entrapment constraints, intraosseous (IO) access constitutes a clinically valid alternative that should be pursued without delay¹⁶.

Isotonic Normal Saline (0.9% NaCl) is the universally recommended resuscitation fluid, as potassium containing solutions such as Ringer's Lactate must be strictly avoided to prevent the exacerbation of rhabdomyolysis-induced hyperkalemia^{2,10}. Additionally, urinary alkalization targeting a urine pH exceeding 6.5 is recommended to prevent myoglobin deposition and subsequent injury within the renal tubules^{10,12}. Regarding fluid volumes, an initial rate of one liter per hour during extrication serves as a practical field guideline, although total requirements within the first 24 hours can vary significantly (ranging from 3 to 24 liters) depending on the severity of rhabdomyolysis^{3,17}. A target urine output of 200 to 300 mL per hour remains the primary parameter for ensuring adequate resuscitation^{3,17}. Collectively, evidence affirms that pre-extrication fluid resuscitation constitutes the single most critical field intervention for minimizing the risk of "rescue death" among earthquake survivors^{2,5,12}.

Beyond aggressive fluid replacement, effective pre-hospital care necessitates the early management of severe pain, electrolyte imbalances, and wound contamination. Analgesia should be initiated as soon as possible using agents such as intravenous morphine, fentanyl, or ketamine, the latter being particularly useful during extrication for its cardiovascular stability^{4,11}. In contrast, non-steroidal anti-inflammatory drugs (NSAIDs) must be strictly avoided due to the risk of exacerbating renal impairment¹¹. Myocardial membrane stabilization is vital if electrocardiographic (ECG) signs of hyperkalemia are present, requiring the prompt administration of intravenous Calcium Gluconate to prevent sudden cardiac arrest^{4,11,17}. To temporarily shift potassium intracellularly, medical teams may also utilize nebulized salbutamol or a combination of insulin and dextrose^{4,11}. Physical adjuncts such as localized cooling or icing of the crushed extremities can effectively suppress muscle metabolism and reduce the release of toxic metabolites into the circulation^{3,5}. While the application of a proximal tourniquet before load release remains controversial, it should be considered for life-threatening hemorrhage that direct pressure cannot control^{7,11}. Finally,

to prevent the transition from local injury to systemic sepsis, responders must initiate broad-spectrum antibiotics and tetanus prophylaxis for all contaminated open wounds as soon as possible^{8,17}.

Effectiveness of Preventive Strategies and Resuscitation Fluid Selection

The selection of resuscitation fluids and preventive adjuncts represents one of the most critical determinants in preventing the progression of local muscle injury into systemic crush syndrome. A summary of the recommended resuscitation fluids, adjunctive therapies, mechanisms of action, and expected clinical outcomes is presented in Table 2.

Table 2: Summary of Resuscitation Fluids and Preventive Modalities

Fluid / Adjuvant	Comparison & Mechanism		Clinical Outcome / Target
0.9% Isotonic Saline	Standard	potassium-free crystalloid.	Primary Outcome: Volume expansion and toxin dilution.
PlasmaLyte	Balanced solution;	prevents hyperchloremic acidosis.	Outcome: Equivalent to saline but with improved acid-base status.
Sodium Bicarbonate	Alkalinizes urine (Target pH > 6.5).		Outcome: Increases myoglobin solubility to prevent tubular casts.
Mannitol (20%)	Osmotic diuretic; scavenges free radicals.		Caution: Risk of osmotic nephrosis; use only in euvolemic states.
Calcium Gluconate	Stabilizes myocardial membranes against K ⁺ toxicity.		Outcome: Prevents fatal cardiac arrest if ECG changes occur.
Ringer's Lactate	Contains potassium; strictly avoided in CS.		Risk: Fatal exacerbation of hyperkalemia.

As shown in Table 2, the reviewed evidence consistently supports 0.9% isotonic saline as the first-line resuscitation fluid in earthquake disaster settings because of its high availability, low cost, and potassium-free composition^{2,6,10,11,16}. Potassium-containing solutions, particularly Ringer's Lactate, should generally be avoided because they may exacerbate hyperkalemia and increase the risk of fatal cardiac arrhythmias^{2,6,10,11}. Although

one study suggested that Ringer's Lactate may be appropriate for initial volume replacement in selected situations¹², current evidence predominantly favors potassium-free crystalloids to maximize patient safety^{10,11,16}.

However, it is critically noted that administering very large volumes of 0.9% NaCl risks inducing hyperchloremic metabolic acidosis due to high chloride intake, which theoretically may lower urinary pH and accelerate myoglobin precipitation within the renal tubules^{7,10,17}. To mitigate the impact of acidosis, balanced crystalloids such as PlasmaLyte have been studied in experimental models, showing efficacy equal to normal saline but without the risk of hyperchloremic acidosis^{5,7}. To protect renal filtration, the addition of Sodium Bicarbonate (NaHCO_3) to achieve targeted urinary alkalinization ($\text{pH} > 6.5$) is highly recommended, as it increases the solubility of myoglobin and uric acid, thereby preventing the formation of obstructive distal tubular casts^{6,12,16}. Nevertheless, some clinical evidence suggests that aggressive fluid resuscitation alone may be as effective as forced alkaline diuresis, necessitating close monitoring to avoid systemic alkalosis^{10,16}. Conversely, the use of osmotic diuretics like Mannitol is now strictly limited in recent consensus; it should only be administered once the victim's intravascular volume is fully restored (euvolemia) and urine output is established^{5,10,16}. Administration of Mannitol in dehydrated or oliguric states risks triggering osmotic nephrosis, which can accelerate the onset of acute kidney injury (AKI)^{10,16}. In addition to pharmacotherapy, physical interventions such as myocardial membrane stabilization using intravenous Calcium Gluconate must be promptly administered if electrocardiographic (ECG) signs of hyperkalemia (e.g., peaked T waves) are present^{7,9,12,16}.

Meanwhile, the application of a proximal tourniquet directly above the crushed extremity before load release is supported in certain reports to temporarily isolate metabolic toxins from the systemic circulation^{9,11}. However, other literature warns that tourniquet use remains controversial and should be limited to life-threatening hemorrhage due to the risk of exacerbating local ischemia and gangrene^{2,3,16}. Cooling therapy (icing) of the injured limbs is also reported as effective in suppressing the basal metabolic rate of muscle cells, thereby limiting the leakage of potassium and myoglobin during the evacuation process^{4,5,7,11}. Ultimately, for victims experiencing severe functional renal failure, intermittent hemodialysis is the preferred Renal Replacement Therapy (RRT) in field hospitals due to its high efficiency in correcting hyperkalemia and its ability to treat multiple patients per day compared to peritoneal methods^{6,7,10,14}. In conclusion, while adjuvant modalities such as urinary alkalinization, Calcium Gluconate, and cooling have strong

mechanistic foundations, rapid and aggressive volume expansion (1–1.5 L/h initially) using potassium free isotonic saline remains the gold-standard preventive strategy for ensuring renal perfusion and reducing disaster related mortality^{2,6,10,16}.

Conclusion

In conclusion, crush syndrome (CS) is a fatal systemic emergency and the second leading cause of preventable death after direct trauma in earthquake survivors. Its lethal pathophysiology transitions from ischemic compression to systemic reperfusion injury upon extrication, where traumatic rhabdomyolysis triggers a massive, sudden release of toxic intracellular constituents primarily potassium, myoglobin, and phosphate and inflammatory Damage-Associated Molecular Patterns (DAMPs) into the circulation. This influx poses immediate, life-threatening risks, including hyperkalemia-induced fatal cardiac arrhythmias and Acute Kidney Injury (AKI) caused by hypovolemia and direct myoglobin nephrotoxicity within the renal tubules. To minimize "rescue death" in resource-limited disaster zones, field protocols must prioritize rapid triage using the MARCH or START systems. Crucially, aggressive fluid resuscitation must be initiated before extrication using potassium-free 0.9% Isotonic Normal Saline to dilute circulating toxins and sustain renal perfusion, while potassium-containing solutions must be strictly avoided. Furthermore, implementing preventive adjuvants such as sodium bicarbonate for urinary alkalization (pH > 6.5) to prevent tubular obstruction, intravenous calcium gluconate for myocardial stabilization, and cooling therapy to lower muscle metabolic rates collectively bridges the gap to hospital care, ultimately optimizing victim survival rates in seismic disasters.

Declaration of Generative AI in Scientific Writing

The authors affirm that the usage of generative artificial intelligence tools, including Chat GPT, is intended solely for improve grammar, especially in material and methods. These tools were not used for generating content, data analysis, or interpretation. The authors are fully responsible for the accuracy and integrity of the content as well as the conclusions presented in this study

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