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Earthquake-related acute kidney shut down; an overview of pre-existing associated factors

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ABSTRACT

Acute kidney injury (AKI) is a critical complication that can arise in the aftermath of earthquakes, particularly due to crush syndrome, which is the second most frequent cause of mortality in such disasters. The causes of AKI in earthquake victims are multifactorial, including direct renal trauma, hypovolemia, rhabdomyolysis and hemodynamic alterations. Rhabdomyolysis, the breakdown of damaged muscle tissue, is a primary contributor, releasing intracellular components into the bloodstream that can overwhelm the kidneys. Patients with chronic kidney disease (CKD), diabetes, or hypertension are more susceptible to AKI. These conditions impair renal function, making the kidneys more vulnerable to additional stressors such as dehydration or rhabdomyolysis.

Keywords: Acute kidney injury, Chronic kidney disease, Acute renal failure, Rhabdomyolysis, Earthquake, Chronic renal failure, Hypovolemia

Review

Implication for health policy/practice/research/medical education:
In earthquake-related acute kidney injury (AKI), the pre-existing conditions such as diabetes mellitus, chronic renal failure, hypertension, and dehydration significantly exacerbate the risk and severity of earthquake-related kidney injury. These conditions impair renal function and reduce the kidney's ability to handle additional stressors, leading to worse outcomes.

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Introduction

Earthquake, as a devastating natural disaster, often leads to numerous medical complications, with acute kidney injury (AKI) being a significant concern among crush syndrome casualties (1). Crush syndrome, or traumatic rhabdomyolysis, arises from muscle destruction products entering the systemic circulation after prolonged compression and subsequent reperfusion (2). Acute kidney injury, is a life-threatening complication of crush syndrome, and a common cause of death following earthquakes (3). The incidence of acute renal failure ranges from 2% to 50% across earthquakes, influenced by building resilience, rescue speed, and local healthcare capacity (4). Earthquake-related AKI is preventable with rapid rescue, early hydration, and monitoring of clinical/laboratory markers like myoglobin and creatine kinase (5,6). Predictive tools like SAFE-QUAKE enhance triage efficiency, particularly in resource-limited settings (7,8). Previous studies showed that, crush syndrome-induced AKI arises from three primary pathways, consisted of hypovolemia due to dehydration, hemorrhage, or third-spacing into damaged muscles, across with direct tubular injury from myoglobin toxicity, cast formation, or oxidative stress and in some condition, obstruction of pelvic trauma or urinary tract damage (9, 10). At this condition, the released myoglobin from crushed muscles accumulates in renal tubules. Then in proximal tubules, heme from myoglobin generates reactive oxygen species by iron-mediated pathways, causing oxidative stress and tubular necrosis (11). Myoglobin then binds to Tamm-Horsfall protein in distal tubules, forming obstructive casts that impair urine flow (11). Pharrell to the above conditions, damaged muscles absorb sodium and water, causing third-spacing and hypovolemia, which followed by reduced renal blood flow leads to prerenal acute renal failure (9). Additionally, severe hypovolemia can progress to shock, exacerbating ischemic kidney injury (9,12). It should remember that, prolonged muscle compression also leads to rhabdomyolysis, releasing myoglobin and other toxic substances that cause tubular injury and obstruction (13,14). Similarly, reduced renal perfusion during entrapment followed by reperfusion upon rescue

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generates reactive oxygen species, exacerbating renal damage (14,15). Correspondingly, systemic inflammation from trauma and infections triggers cytokine release, further impairing renal function (14,16). This review aims to provide an overview of the factors associated with acute renal failure in earthquake-related crush syndrome, focusing on the clinical and laboratory parameters that can aid in predicting and managing this critical condition.

Search strategy

For this review, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ) and Embase, using different keywords like; acute kidney injury, chronic kidney disease, acute renal failure, rhabdomyolysis, earthquake, chronic renal failure and hypovolemia

AKI in crush syndrome

Prior investigations found that, the extent of muscle injury is directly correlated with the risk of developing acute renal failure (17). The anatomical location of injuries also matters, with lower extremities being more frequently affected during earthquakes. Moreover, the presence of additional injuries, such as fractures to the pelvis, acetabulum, or vertebra, is associated with increased levels of creatinine, potassium, and myoglobin, although no direct relationship was detected amongst these additional injuries and the happening of renal failure (18,19). These clinical observations highlight the importance of assessing the overall injury burden in predicting AKI risk among earthquake survivors (20). Accordingly, the total entrapment time, defined as the duration under debris plus the time to evacuation, is a critical factor influencing the severity of crush syndrome and the likelihood of acute renal failure (20,21). Moreover, prolonged immobilization, even without direct compression, can result to crush syndrome (22). Studies have demonstrated significant correlations amid total entrapment time and initial serum concentrations of potassium, creatine kinase, myoglobin, lactate dehydrogenase, uric acid, creatinine, and blood urea nitrogen (23,24). These correlations suggest that longer entrapment times exacerbate muscle damage and increase the risk of acute renal failure (25). Furthermore, a noteworthy difference was observed in total entrapment time exceeding 6 hours amongst individuals with kidney failure and those without, indicating that prolonged entrapment substantially elevates the risk of kidney injury (18,19). This finding emphasizes the need for rapid evacuation and treatment to mitigate the detrimental effects of prolonged compression and ischemia on kidney function (26,27).

Pre-existing factors associated with earthquake-related AKI

Patients with pre-existing chronic kidney disease (CKD) are at a significantly higher risk of developing AKI following crush injuries (28). Chronic renal failure compromises renal reserve, making the kidneys more susceptible to further damage (29). Studies have shown that CKD patients are more likely to experience severe acute renal failure and have poorer outcomes compared to those with normal kidney function (30). Moreover, elderly individuals are more vulnerable to acute renal failure due to age-related decline in renal function and reduced physiological resilience (31). The combination of trauma, dehydration, and metabolic imbalances during earthquakes exacerbates this vulnerability (32). Diabetes is also a well-established risk factor for AKI. Hyperglycemia, endothelial dysfunction, and microvascular complications impair renal autoregulation, increasing the likelihood of acute renal failure in earthquake victims with diabetes (33). Accordingly, earthquake-induced stress and trauma can elevate blood pressure and worsening renal outcomes (34). In addition, earthquake victims often suffer from dehydration due to limited access to water and prolonged entrapment. Hypovolemia reduces renal perfusion, leading to pre-renal AKI, which can progress to intrinsic renal injury if not promptly addressed (20). Similarly, earthquake-related injuries often lead to infections, which can precipitate AKI through systemic inflammation, hemodynamic instability, and direct renal damage (35). Sepsis is a particularly potent risk factor for AKI in this context (36). Prior studies also showed, obesity and metabolic syndrome are associated with chronic inflammation, insulin resistance, and endothelial dysfunction, all of which predispose individuals to acute renal failure (37,38). The metabolic stress induced by earthquakes further compounds these risks (37,38). Finally, certain medications, such as non-steroidal anti-inflammatory drugs, renin-angiotensin system inhibitors, and diuretics, can exacerbate acute renal failure by impairing renal hemodynamics or causing direct nephrotoxicity. Earthquake victims on these medications are at higher risk (39).

Electrolyte and metabolic derangements

Potassium released from muscle cells causes cardiac arrhythmias, reducing cardiac output and worsening renal ischemia (40). Elevated phosphate binds calcium, forming nephrotoxic calcium-phosphate crystals that obstruct tubules (acute phosphate nephropathy). Further, muscle breakdown releases lactate and sulfate, lowering blood pH and impairing renal function (41). One of the important features of this condition is cytokine storm, while proinflammatory mediators (e.g., tumor necrosis factor alpha [TNF-α] and interleukin 6) from injured muscles and activated macrophages amplify systemic inflammation, worsening endothelial and tubular damage (42,43). Additionally, leukocyte extracellular traps contribute to intraperitoneal and systemic inflammation, accelerating muscle and kidney injury in experimental models (44). Notably, untreated compartment syndrome

exacerbates muscle necrosis, perpetuating the release of toxic metabolites and increasing AKI risk (13). Here, there is also a crosstalk between inflammation and ferroptosis. Myoglobin degradation releases free iron, which promotes lipid peroxidation and ferroptosis as an iron-dependent cell death in renal tubules (45). Likewise, damage-associated molecular patterns (DAMPs) activate TLR4/NF-κB pathways, linking inflammation to ferroptosis and further exacerbating tubular damage (43,46). Similarly, DAMPs from crushed muscles activate the complement system (e.g., C5a), driving macrophage infiltration into kidneys and amplifying inflammation (47). Recent studies showed that, in crush syndrome models, the inflammatory mediators (e.g., TNFα, dsDNA) peak earlier in the peritoneal cavity than in serum, suggesting localized inflammation precedes systemic SIRS (systemic inflammatory response syndrome) (44,48,49). Then untreated intraperitoneal inflammation propagates to the bloodstream, amplifying renal oxidative stress and kidney tissue injury (35). It should remember that, SIRS-induced capillary leakage worsens renal hypoperfusion, exacerbating prerenal acute renal failure (18). Moreover, inflammatory cytokines impair mitochondrial function, worsening acidosis and tubular dysfunction (50).

Predictive laboratory parameters

Early and continuous monitoring of kidney function is crucial for detecting AKI in crush syndrome patients (5). While plasma creatinine and blood urea nitrogen levels are late indicators of kidney damage, other blood parameters such as creatine kinase, myoglobin, lactate dehydrogenase, and uric acid can provide earlier indications of AKI risk (13). The study by Köroğlu et al, suggests a myoglobin value beyond 2330 mg/dL upon hospital admission demonstrated high sensitivity for predicting AKI, while an initial plasma uric acid value more than 6.36 mg/dL had the utmost specificity (51). They also showed, a mean plasma creatine kinase level above 9290 U/L during patient follow-up predicted acute kidney failure with 79% sensitivity and specificity (51).

Focus on management

Adequate hydration can mitigate pre-renal acute renal failure and reduce the risk of progression to intrinsic renal injury (52). Elderly individuals, diabetics, and those with chronic renal failure or hypertension should be closely monitored for signs of AKI (33). Accordingly, adjusting or discontinuing nephrotoxic medications can reduce the risk of AKI (53). Besides, early treatment of infections and sepsis is essential to prevent acute renal failure (54). Amputation, while a difficult decision, may be required in cases of irreversible tissue damage or progressive clinical deterioration (25). Statistical improvements were observed in creatine kinase, myoglobin, creatinine, potassium, and uric acid levels among individuals who underwent amputation (24,25,55). However, the decision to perform an amputation must be carefully considered, as it carries its own risks and potential complications (25). Treatment protocols often consisted of sodium bicarbonate and mannitol to optimize myoglobin solubility and promote excretion in urine, although the efficacy of these adjunctive therapies remains controversial (21). Extracorporeal technologies such as dialysis are necessary once renal disturbance is established or significant hyperkalemia happens (21,25). Emerging mechanism-based treatments include inhibition of myoglobin endocytosis using cilastatin, which has shown renoprotective effects in preclinical studies (11). Oxidative damage amelioration strategies involve the use of antioxidants although their effectiveness varies (56). Immune-targeting treatments, such as inhibitors of IL-1β signaling and therapies targeting neutrophils and MET formation, are also under investigation (11). Kidney-targeted delivery systems, comprising microbubbles and nanoparticles are being explored to deliver drugs and proteins specifically to the kidney, enhancing bioactivity and reducing toxicity (57). Several investigations showed that, elevated serum uric acid levels are associated with kidney disease (58). Despite its antioxidant properties, uric acid can promote oxidative stress, inflammation, and endothelial dysfunction (59). Studies suggest that elevated uric acid levels in rhabdomyolysis patients may be associated with an increased risk of acute renal failure (60,61).

Conclusion

Earthquake-related crush syndrome poses a significant threat to kidney function, with AKI being a major cause of morbidity and mortality. Factors such as the number of affected extremities, total entrapment time, and specific laboratory parameters play critical roles in predicting the development and severity of AKI.

Authors' contribution

Conceptualization: Mohammad Reza Moonesan.
Data curation: Mehdi Yarahmadi.
Investigation: Mehdi Yarahmadi, Mohammad Reza Moonesan.
Resources: Mohammad Reza Moonesan.
Supervision: Mohammad Reza Moonesan.
Validation: Mohammad Reza Moonesan.
Visualization: Mohammad Reza Moonesan.
Writing–original draft: Mohammad Reza Moonesan.
Writing–review & editing: Mehdi Yarahmadi, Mohammad Reza Moonesan.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity (https://www.perplexity.ai) to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as

necessary, assuming full responsibility for the publication's content.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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