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Management of Crush-related Acute Kidney Injury After Disasters

Recently, on February 6, 2023, a 7.8 magnitude earthquake struck southeastern Turkey and neighboring states. It has become the worst disaster ever recorded in the region, with over 40,000 deaths, and the rescue operations have not been finalized yet. This incident should stress the importance of preparedness, foresight, and organization of our disaster management system for and during natural or artificial disasters. The experiences of these mournful days are hard-learned lessons for the future.

As tens of thousands of survivors suffered minor or major injuries from direct trauma or ensuing situations such as being trapped under the rubble, the healthcare system has been under tremendous pressure to triage, care, and safely discharge these victims to closely affected locations, frequently lacking water, food, communication, transportation, clothing, shelter, undamaged hospitals, medical professionals, and equipment, especially during the first few days when morbidity and mortality rates were highest. In this editorial, we would like to focus on acute kidney injury (AKI) related to crush syndrome, one of the many physical and psychological health consequences of the earthquake,

Crush syndrome, also known as traumatic rhabdomyolysis, involves, but is not limited to, a sequence of metabolic changes that occur when the skeletal muscles are injured to the extent that their contents are released into the circulation, causing organ dysfunction. Rhabdomyolysis can be detected by measuring creatine phosphokinase (CPK) levels five times the upper limit of normal (approximately 1,000 U/l).[1] Depending on the intensity of the earthquake and the amount of time spent under the rubble, the incidence of AKI related to crushes ranges from 0.5% to 25% in earthquake victims.[2] The rate of dialysis required varies among different incidents, with some reaching as high as 75%, as seen in the Marmara earthquake.[3]

Crush-related AKI refers to the various pathophysiological causes of kidney damage that can occur in victims of crush injuries. As in any AKI, these are classified within three main etiologies: prerenal, intrarenal, and postrenal. Prerenal causes of AKI are common in these patients since their fluid intake is severely or entirely restricted while entrapped, they have hemorrhagic shock caused by trauma, and third-spacing into reperfusion-damaged muscle tissue after extrication. As pelvic traumas are common in these patients, postrenal obstructive pathologies should also be considered. More commonly, crush-related AKIs are due to intrarenal causes, especially three main myoglobin-related injury mechanisms seen in rhabdomyolysis: decreased renal perfusion, tubular obstruction following cast formation, and direct injury of myoglobin to tubules.[4] Elevated serum CPK levels, dark/tea-colored urine, and oliguria in a patient with apparent crush injury highly suggest rhabdomyolysis-related AKI.

The mainstay of preventing AKI in patients with rhabdomyolysis is aggressive fluid replacement with intravenous crystalloids not containing any amount of potassium as early as possible, even before extrication if possible. Fluid administration helps to prevent hypovolemia, increase urinary flow to prevent cast formation, and wash out toxins damaging tubules.[5] If venous access is established before extrication, 1 l/h of infusion is started and continued for up to 2 hours of the extrication process. If prolonged, the rate may be reduced to 0.5 l/h. The subsequent fluid infusion rate should be tailored to the urine output, ongoing fluid losses, and comorbidities to prevent hypervolemia associated with dialysis requirements. Raising the urine pH over 6.5 by alkalinization with the administration of sodium bicarbonate may reduce tubular cast formation, uric acid precipitation, and hyperkalemia. However, close monitoring of pH, calcium, and potassium is needed to avoid alkalosis-related complications. Hyperkalemia is one of the major causes of mortality in these patients because of potassium released from the crushed extremity or as a consequence of AKI. Potassium levels should be monitored and treated rapidly to prevent fatal arrythmias. Extremity amputation only to prevent crush syndrome is not recommended and should be considered only when it will make extrication possible, or the extremity is an apparent infection/inflammation source.[6]

Once AKI develops, renal replacement therapy indications, such as refractory hyperkalemia, hypervolemia, and severe uremic toxicity, should be rechecked frequently to initiate the appropriate modality on time. Since patients with crush syndrome are prone to rapid elevations of potassium to levels beyond pharmacological interventions and require intermittent hemodialysis to prevent the development of fatal arrhythmias. Continuous dialysis strategies may be limited by the need for large amounts of sterile replacement fluid that can be difficult to obtain in a disaster. As hemodynamic instability is common due to various etiologies such as hypovolemia from restricted fluid intake, hemorrhage, and third-spacing fluids, anemia, and septic shock, continuous renal replacement therapy

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(CRRT) may be provided.[7] In recent years, blood purification for treating trauma-related injuries is becoming common. Combination therapy of extracorporeal techniques, such as hemoadsorptive (HA) therapies with CRRTs, seem to be promising treatments for rhabdomyolysis-related AKI by absorbing circulating myoglobin proteins and inflammatory cytokines to prevent further injury to the kidneys.[8]

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