Acute kidney injury and rhabdomyolysis due to multiple wasp stings

Sir, We read the article, “Acute kidney injury and rhabdomyolysis due to multiple wasp stings” by Radhakrishnan et al.,[1] with great interest. We wish to highlight certain issues in this case.

Various case reports are available describing the development of systemic manifestations following wasp or bee stings in patients with two or three stings in contrast to at least 50 stings as described by the author.[2]

The authors have described pigmentary nephropathy as the cause of acute kidney injury (AKI) following rhabdomyolysis. But, this is only one of the various pathophysiologic mechanisms; the other important mechanisms are direct heme protein-induced oxidant injury and renal vasoconstriction with diminished renal circulation.[3]

Myoglobin levels were not performed in this case due to nonavailability, but serum myoglobin levels are not needed for the diagnosis or management of rhabdomyolysis. Myoglobin is cleared more rapidly than creatinine phosphokinase (CK) and therefore is less sensitive for detecting rhabdomyolysis, especially when presentation is delayed. Also, presence of myoglobin in urine is not specific for the development of AKI; therefore, it is not necessary for routine testing.[3]

We presume that the author must have monitored serum calcium levels in this case because, while managing a case of rhabdomyolysis with AKI, monitoring of serum calcium levels is very important. Calcium levels are often low initially, secondary to precipitation of calcium with phosphate in damaged muscles. Subsequently, in the recovery phase of rhabdomyolysis and AKI, calcium mobilization from damaged muscles may subsequently result in hypercalcemia.[3]

Forced alkaline diuresis is a common intervention in rhabdomyolysis, but evidence of clinical benefit is lacking. The largest retrospective study of bicarbonate and mannitol therapy versus no use of this therapy in trauma patients did not find any difference between groups in the incidence of renal failure, need for dialysis or mortality. A current consensus statement suggests that sodium bicarbonate or mannitol administration is not necessary and is not superior to normal saline diuresis in increasing urine pH.[4] Further, a large dose of bicarbonate may worsen the degree of hypocalcemia, especially if hypovolemia is corrected.

In severe rhabdomyolysis, as was evident in this case, intermittent hemodialysis (IHD) may be needed to address rebound hyperkalemia and acidosis. However, conventional hemodialysis may be relatively inefficient at removal of circulating myoglobin owing to its large molecular weight. Continuous renal replacement therapy modes, such as continuous venovenous hemofiltration (CVVH) or continuous venovenous hemodiafiltration (CVVHDF) have the advantage of aborting these rebound complications, and convection removes these larger molecular weight solutes better than diffusion. The Chinese case series as quoted by the author have also reported similar results. In this series, despite the lack of difference in mortality rates, the patients who began renal replacement therapy with CVVH or CVVHDF with plasma exchange experienced a better and more rapid recovery of kidney function than those initiated with IHD.[5]

Also, although controversial, myoglobin clearance could theoretically be augmented using novel super high-flow hemofilters (molecular weight cut-off 30-60 kDa).

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References
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Sir,

Admissions due to methanol poisoning are common among our poverty‑ridden population, mostly due to consumption of country liquor or exposure with toxic products like antifreeze agent etc. [1]

We are reporting a patient of Methanol poisoning subsequently developing Putamen necrosis.

A 50‑year‑old male patient presented to the emergency department with chief complaints of giddiness, vomiting, abdominal pain, and blurred vision, since 4 h. His history was negative except for consumption of country liquor, 6 h back. On examination, patient was irritable, in altered sensorium had labored breathing, heart rate was 70/min, noninvasive blood pressure (BP) was 84/50 mm Hg, respiratory rate was 40/min, and pupils were dilated and sluggishly reacting. Systemic examination was within normal limits. Arterial blood gas (ABG) analysis revealed severe high anion gap metabolic acidosis with co‑existing respiratory acidosis and mild hypoxemia. In view of the suggestive history, chief complaints, and the ABG reports, we considered it as a methanol poisoning, in the absence of definitive diagnostic facilities.

He was rehydrated with isotonic saline (1L intravenous [IV]) over 20 min and with continued rehydration his BP returned to 118/82 mm Hg within 30 min. Invasive positive pressure ventilation was started  (synchronized intermittent mandatory ventilation), gastric lavage was done and ethyl alcohol (10%, 10 mg/kg, IV) was infused over 10 min and thereafter continued on a maintenance dose of 2 mL/kg/h IV, for the next 36 h. Midazolam  (0.05 mg/kg/h) and fentanyl  (2.0 µg/kg/h) infusion was started.

Soda‑bicarbonate (100 ml IV) was given, and ABG samples were repeated every 2 hourly. Oral folic acid (1 mg/kg) was started and administered 4 hourly, for the next 7 days. On the follow‑up, acidosis got corrected over 24 h, but patient remained unconscious, so magnetic resonance imaging was planned. It revealed bilateral basal ganglia hyper‑intensity lesion, extensive edema over fronto‑temporal region and right basal ganglia hemorrhage [Figure 1]. Thereafter, injection mannitol (20% 100 mL, three times daily), syrup glycerine (30 mL, 3 times daily) and tablet acetazolamide (one tab twice daily) were started to decrease the intracranial edema. Gradually, the patient regained full sensorium and was extubated on seventh Intensive Care Unit day. His cardio respiratory parameters were within normal limits but had blurred vision and residual motor weakness in all the limbs. Thereafter patient remained stable and was shifted to the respective ward after 14 days.

Country liquor contain many impurities and varying amount of methyl alcohol. After ingestion, methyl alcohol is metabolized by alcohol dehydrogenize to form formaldehyde and subsequently to formic acid. Toxic effects of these metabolites usually manifest after 12–24 h of ingestion. Initial symptoms are nausea, vomiting,