Benzene hexachloride poisoning with rhabdomyolysis and acute kidney injury

Sir,

A 21-year-old male was referred to our hospital for evaluation and management of renal failure. He presented with oliguria, dark color of urine, pedal edema, and muscular pain since 2 days. He had a history of ingestion of insecticidal liquid containing gamma benzene hexachloride (BHC) with a suicidal intention 3 days back. He had received gastric lavage and supportive treatment in the form of fluids at a primary care center. At the time of admission his blood pressure was 120/80 mmHg, pulse rate 90/min, respiratory rate was 24/min, and urine showed dipstick-positive proteinuria (3+) with myoglobinuria. He had tenderness of muscles (more in lower limbs) with normal tendon reflexes and altered mentation. Hematological parameters (total leukocytes 10,500/mm^3, platelets 1.9 lac/mm^3, hemoglobin 11.7 g/dL) and liver function tests were normal except for a mild elevation in aspartate aminotransferase (AST 94 U/L) without any evidence of hemolysis. The presence of metabolic acidosis (pH 7.18, HCO_3^- 9) and rise in creatinine from 0.9 to 3.1 mg/dL prompted us to initiate him on hemodialysis. Additional evaluation revealed total serum creatine phosphokinase (CPK) of 2890 U/L (range: 20-230 U/L), lactate dehydrogenase of 360 U/L, and a rise in potassium to 6.3 mEq/L. Immunological tests like complement levels (C3), anti-nuclear antibodies, and anti-neutrophilic antibodies were unremarkable. A clinical diagnosis of BHC-induced rhabdomyolysis leading to acute kidney injury (AKI) was made. Though myoglobinuria ceased over the next 3 days and total CPK decreased to 146 U/L by the end of 1 week, he required hemodialysis on alternate days for 1 week. After 2 weeks of hospitalization and supportive care, there was a gradual improvement in urine output, and a declining trend was noticed in creatinine. Similarly, his muscular tenderness gradually improved with the disappearance of uremic symptoms and creatinine decreased to 1.2 mg/dL on the 21st day.

Poisoning caused by industrial chemicals is an important cause of community-acquired AKI in Asia.[1,2] Our case demonstrates AKI associated with ingestion of BHC which is very unusual. BHC is an organochlorine insecticide and acute poisoning usually leads to features of neurotoxicity like altered mentation and seizures. Other features like liver dysfunction, metabolic acidosis, and hematological and gastrointestinal toxicities have occasionally been described.[3] BHC is known to be nephrotoxic as per reports of experimental animal models,[4,5] but reports of renal involvement in humans with myoglobinuria have not been convincingly reported in the literature. Development of muscle necrosis and AKI have been reported only in one case in 1977.[6] Rhabdomyolysis is a life-threatening condition resulting from the breakdown of skeletal muscles and commonly occurs with trauma, burns, viral infections, polymyositis, convulsions, exertion, ischemia, alcohol abuse, and various toxins.[7,8] Other causes of myoglobinuria were safely excluded in our patient as per clinical history and temporal ingestion of BHC leading to acute presentation suggesting its etiological role. The role of dehydration and sepsis leading to AKI was ruled

out in our case. Though the exact mechanism cannot be determined, generation of free radical-related oxidative damage in skeletal muscles and renal tubular cells may lead to renal toxicity.

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