Presented at the ACMT Annual Scientific Meeting 2022 – Virtual

Published in J Med Tox 2022; 18:81-82

021. Drug And Toxicant-associated Rhabdomyolysis: Clinical and Epidemiologic Trends

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Background: Rhabdomyolysis is the breakdown of muscle tissue that may develop from many underlying causes (e.g. Toxicants, environmental, traumatic, exertional). Toxicant-induced rhabdomyolysis may occur from sedation with compressive ischemia of muscles or from psychomotor agitation with muscular flexion. Little research exists to describe the clinical aspects of toxicant-associated rhabdomyolysis and differences between rhabdomyolysis associated with sedation or agitation.

Research Question: What are the epidemiologic and clinical features of toxicant induced rhabdomyolysis?

Methods: We identified cases of rhabdomyolysis from the American College of Medical Toxicology's (ACMT) Toxicology Investigators Consortium (ToxIC). Cases were identified and reported by 50 medical toxicology consultative services throughout the United States and Canada. We report the toxicants that are associated with rhabdomyolysis and their epidemiologic and clinical characteristics. Within the "nervous system symptoms" data field, cases were divided into those with agitation ("agitation", "rigidity", "clonus", "seizures") and sedation ("CNS depression" & no agitation symptoms). The means of nominal variables were compared using Chi Square and Fisher exact test when data was sparse.

Results: Between 2014-2020, there were 1812 cases of rhabdomyolysis with a known toxicant source. Most cases were adults (19-65 years) (82%) and male (67%). Most common agents associated with rhabdomyolysis were sympathomimetics (21%), opioids (17%), antidepressants (8.5%) and sedatives/hypnotics (7.1%). Most common laboratory findings included hyperlactatemia (32%), troponinemia (30%), AKI (25%), metabolic acidosis. A very small percentage developed hyperkalemia > 6.0 mmol/L (.66%), Overall mortality in patients with toxicant associated rhabdomyolysis is 4.1%. Sedated patients were more likely to develop AKI (45% vs. 7%; $p < 0.01$), hypotension (30% vs. 10%; $p < 0.01$), metabolic acidosis (28% vs. 13%; $p < 0.01$) and had a higher mortality (8.1% vs. 2.2%; $p < 0.01$) than those with agitation.

Conclusion: Toxicant induced rhabdomyolysis is associated most commonly with sympathomimetics and opioids. Patients with sedative-associated rhabdomyolysis had higher rates of critical illness, organ failure, and mortality.