**A Lethal Mix-Up: Fatal Neurological Consequences of Accidental Rodenticide Ingestion**

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**Introduction**

Bromadiolone, a potent rodenticide known as a superwarfarin, typically causes multi-organ haemorrhage in acute poisoning cases. Rarely, acute intoxication can lead to neurological symptoms ranging from mild dizziness to life-threatening status epilepticus.

**Case description**

A previously well 19-year-old Myanmar man ingested two sachets of bromadiolone-containing rodenticide (25g 0.005%/sachet). He presented to the emergency department two hours later with symptoms of vomiting and decreased consciousness. Initial assessment revealed restlessness, drowsiness, hypertension, tachycardia, tachypnoea, and hypoxemia. Lung examination revealed transmitted sounds, while neurologic examination was hindered, and other physical examinations were unremarkable. Within 20 minutes of arrival, he experienced refractory generalized tonic-clonic seizures unresponsive to anti-epileptic treatment, resulting in further deterioration of consciousness and necessitating intubation for airway and cerebral protection. Laboratory investigations indicated significant leucocytosis, polycythaemia, severe lactic acidosis, renal derangement, hyperCKemia, and transaminitis, with an unaltered coagulation profile. Radiological examinations showed no abnormalities. Treatment included activated charcoal via a Ryles tube, aggressive hydration with 4 litres of normal saline, and vitamin K infusion. Resuscitation resulted in notable improvements in blood gas samples (initial pH of 6.915, HCO3 of 8.6, to pH of 7.290, HCO3 18.8) and lactate levels (initial lactate 24.0 to 1.4). Despite plans for plasma transfusion and an MRI of the brain, the patient's condition deteriorated, leading to refractory seizures and eventual death.

**Discussion**

Neurological manifestations, though rare, are life-threatening complications following acute bromadiolone intoxication. Cases by Wang et al. and Jia et al. indicate that symptoms can range from mild to severe, including acute demyelination in the corpus callosum. The rapid progression to refractory seizures highlights the neurotoxic effects of bromadiolone, exacerbating metabolic derangements and leading to death. Immediate resuscitation and vitamin K administration are crucial for addressing both neuropathology and coagulopathy, but in this case, the toxicity was too severe to be counteracted by standard treatments.

**Conclusion**

The rapid progression to refractory seizures and multi-organ failure underscores the potent toxicity of bromadiolone. This outcome emphasizes the need for heightened awareness, early detection, and the development of more effective treatment strategies for bromadiolone poisoning.

**Keywords**

Rodenticide, status epilepticus, vitamin K