**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. On rare occasions, acute intoxication can lead to neurological symptoms ranging from mild dizziness to life-threatening refractory status epilepticus.

**Case description**

A previously healthy 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 vomiting and unresponsiveness. Clinically, he was restless, drowsy, hypertensive, tachycardic, tachypneic, and hypoxemic. Lung examination revealed transmitted sounds, neurologic examination was hindered, while other physical examinations were unremarkable. Within 20 minutes of arrival, he experienced refractory generalized tonic-clonic seizures unresponsive to intravenous valium thus necessitating intubation for airway and cerebral protection. He was further loaded with intravenous phenytoin and started on propofol infusion, in addition to sedations, to cease the seizures. Laboratory investigations indicated significant leucocytosis, polycythaemia, severe lactic acidosis, renal derangement, hyperCKemia, and transaminitis, with an unaltered coagulation profile. The CT brain was normal. Initial 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). However, despite the antiepileptic and sedation, refractory seizures recurred a few hours later, leading to cardiac arrest, and patient succumbed.

**Discussion**

Beyond haemostasis disturbances, neurological manifestations are rare but life-threatening complications following acute bromadiolone intoxication. Acute demyelination of the central nervous system and severe neurotoxic effects leads to refractory seizures, exacerbating metabolic derangements and ultimately causing death. Immediate resuscitation, seizure control and prevention, decontamination, and reversal (vitamin K) administration are crucial for addressing both neuropathology and coagulopathy. However, in this case, the toxicity was too severe to be counteracted by standard treatments.

**Conclusion**

Despites the rarity of neurological manifestation, the rapid progression to refractory seizures with multi-organ failure underscores the potent neurotoxicity of bromadiolone. This emphasizes the need for early detection, heightened awareness, and timely treatment strategies to counteracts the neurotoxicity of bromadiolone poisoning.

**Keywords**

Rodenticide, status epilepticus, vitamin K