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Novel Therapeutic Use of Hemoperfusion in Glufosinate Ammonium Poisoning: An Emerging Extracorporeal Strategy

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Clinical Image

Keywords: Glufosinate ammonium poisoning, Hemoperfusion, neurological impairment, hyperammonaemia.

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BACKGROUND

Glufosinate ammonium 13.5% SL is a non-selective post-emergence herbicide. Accidental or suicidal ingestion of this compound can cause severe neurological impairment as well as cardiovascular and respiratory failure. Clinical signs and subsequent neurological impairment are often associated with hyperammonaemia.

CASE DESCRIPTION

Mr. S, a 41-year-old male presented with an alleged history of Glufosinate ammonium 13.5% poisoning approximately 20ml. He got admitted to our centre 6 hours post insult. Initially, he was stable with normal hemodynamics, respiration and consciousness. As time progressed, respiratory distress developed subsequently respiratory failure with altered level of consciousness and an episode of seizure. Arterial blood gas revealed respiratory acidosis. Rapid sequence intubation was implemented and started on antiepileptics. Laboratory investigations were unremarkable. He developed intermittent episodes of myoclonic seizures and excitability phenomenon. He underwent three cycles of Hemoperfusion. Gradually, he improved with fading of seizure episode, then slowly weaned off and extubated successfully. He was discharged in a clinically fit condition without any disabilities.



CONCLUSION

This case report highlights in spite of normal serum ammonia levels and low volume consumption, prompt initiation of hemoperfusion is crucial.

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