Case report

Crit Care Shock (2026) 29:65-68

Role of charcoal hemoperfusion in reducing ICU stay in a rare case of glufosinate ammonium poisoning

Himalini Subbiah[1], Siva Prakash[1], Vaishnavi S.[1], Vengatesan Kowshik[1], Sudagar Singh RB[1]

Abstract

Glufosinate ammonium has emerged as an important cause of poisoning in agricultural regions. It can lead to severe clinical manifestations. Due to the absence of an antidote and the limited efficacy of the conventional hemodialysis alternative, detoxification such as charcoal hemoperfusion is gaining acceptance. We present a case of a young male who had ingestion of the herbicide, presenting to us with severe respiratory depression, bradycardia, and neurological symptoms, and the use of charcoal hemoperfusion as a mainstay treatment option in reducing the mortality, morbidity, and also reducing the course ICU stay.

Introduction

Glufosinate ammonium is a widely used herbicide. Accidental or deliberate ingestion of it can pose significant neurological and cardiovascular complications. It can cause respiratory arrest and multi-organ dysfunction syndrome (MODS). In recent years, the use of charcoal hemoperfusion for the treatment of poisoning has gained attention due to its advantages. This paper will explore the crucial role of charcoal hemoperfusion as a main treatment in cases of life-threatening and severe poisonings, particularly mitigating the neurotoxicity of hyperammonemia, enhancing the outcome in this patient.

[1] Department of General Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

Address for correspondence:
Dr. Himalini Subbiah
Department of General Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India
Tel: +919445695306
Email: himalinidgrt@gmail.com

Case report

A 26-year-old male, hailing from Andhra Pradesh, came with the history of consumption of 250 ml of 13.5% w/w undiluted glufosinate ammonium (33.75 grams) (**Figure 1**), (1) mixed with 100 ml of alcohol, the night before, and was taken to the hospital after 30 min of consumption. The patient started having gastrointestinal symptoms and developed multiple episodes of vomiting. Gastric lavage was performed at a nearby hospital. The patient was drowsy, which was initially attributed to alcohol. The patient had a persistent drop in heart rate and was given an atropine injection, but still had persistent bradycardia. (2) He was still drowsy after 12 hours of ingestion and was brought to our hospital for further management. The patient was given an activated charcoal wash in the Emergency Department (ED) at a dose of 1 mg/kg. The patient's GCS on arrival was 9 and was drowsy. Pupils were normal in size and sluggishly reactive to light. He had multiple episodes of loose stool. On arrival at the ED, the patient had a regular bradycardia of 52 beats/min (bpm). The electrocardiogram (ECG) showed sinus bradycardia, with normal PR and QTc intervals. The cardiac markers were negative. The echocardiogram was normal. Blood pressure was 110/70 mmHg with a reduced respiratory rate of 11/min. Gradually, the patient developed a buildup of carbon dioxide. Serial blood gas analysis was

monitored, and he was admitted to the intensive care unit (ICU) for further management. The patient's heart rate was regularly monitored; the lowest HR documented was 39 bpm. Hence, the patient was started on orceprenaline tablet 5 mg. Despite having a baseline heart rate of 45 bpm, he did not report chest pain or palpitations. The patient had fluctuating saturation with a reduced respiratory rate, (3) had hyperbilirubinemia during hospital stay—admission total bilirubin 3.79 mg/dl (indirect 2.93 mg/dl). Additional significant laboratory findings are presented in **Table 1**. Abdominal ultrasonography showed a normal liver, and the fibroscan was normal. It was attributed to alcohol, while hyperbilirubinemia settled during discharge. Serum ammonia levels were serially monitored and gradually increased, reaching a maximum of 96.1 μg/dl. (4)

Hence, the patient was given hepatic encephalopathy prophylaxis. In view of persistent drowsiness, respiratory depression, and bradycardia, the patient was planned for elective intubation. A nephrology consult was also obtained, and the patient was given one cycle of charcoal hemoperfusion with a resin cassette. Post-hemoperfusion, serum ammonia levels started to decrease, (5) and the patients' sensorium began to improve, while his Glasgow coma scale (GCS) improved to 14. Ammonia levels reduced to 53.4 μg/dl, heart rate improved to 62 bpm, and respiratory rate improved to 18/min. Given the patient's marked improvement, elective intubation was deferred. Heart rate progressively improved, and orciprenaline was stopped. The patient was transferred out of the ICU and monitored in the ward for a few days. The patient had episodes of anterograde amnesia during the hospital stay, which gradually improved during discharge.

Discussion

Glufosinate ammonium is an herbicide used in India, more commonly in Andhra Pradesh. Usually considered safe, whereas undiluted ingested forms remain highly neurotoxic and cardiotoxic. The mortality rate is 6.1% to 17.7%. It has been banned in 29 countries. It is a structural analogue of glutamate, irreversibly inhibiting glutamine synthetase, which leads to the accumulation of glutamine and ammonium. In humans, accidental ingestion leads to hyperammonemia. Intracellular accumulation of ammonia in neurons causes oxidative injury. Hence, hepatic encephalopathy prophylaxis, such as lactulose and rifaximin, can be given to reduce these symptoms. Primarily, glutamate is an excitatory neurotransmitter that can lead to seizures. (6) But in this case, hyperammonemia has caused decreased GCS and drowsiness. Initial levels of hyperammonemia can serve as a biomarker, indicating the severity of central nervous system symptoms, according to some studies.

The most common post-ingestion system involved is the gastrointestinal system, which can cause nausea and vomiting. It is also cardiotoxic, as witnessed in this case, which caused severe bradycardia. Hence, pulse rate should be regularly monitored. Atropine injection and orciprenaline can be used based on heart rate. It can cause vasodilation and shock, which should be managed with vasopressors, and causes respiratory arrest in many cases. Here, the patient had multiple episodes of desaturation and hypopnea, which improved after charcoal hemoperfusion. (7) It commonly affects the hippocampus, which is involved in memory. Elderly patients carried an increased risk of developing amnesia. Severe cases lead to MODS. The cause of death is mostly respiratory arrest.

There is no specific antidote available. Median toxic dose is 30.4 grams, and a few studies have shown 13.9 grams as the toxic dose. (1) Hence, supportive management is the mainstay of treatment. Gastric lavage performed within 6 hours reduces the absorption of the toxin. But removing the toxin from circulation also plays a vital role in management. It is a lipophilic toxin; hence, charcoal hemoperfusion has an important role in treatment by acting as an adsorbent, removing the toxin from circulation. Conventional hemodialysis does not have a role. Hemoperfusion reduces worsening symptoms, leading to the patient's recovery. It has the highest efficacy when done within 24 hours, but can be done up to 48 hours. Studies and case reports show that charcoal hemoperfusion reduces neurological symptoms. (8) As seen in our case, there was a drastic improvement in the patient's symptoms, reducing the need for intubation, ICU stay, and enabling earlier recovery. The neutrophil-to-lymphocyte ratio can also be used to predict neurotoxicity and outcomes. (9) Age >70 years and a GCS<9 are poor predictors. Intravenous lipid emulsions can also be used in patients where hemoperfusion is not feasible. Other manifestations include 6th nerve palsy.

Conclusion

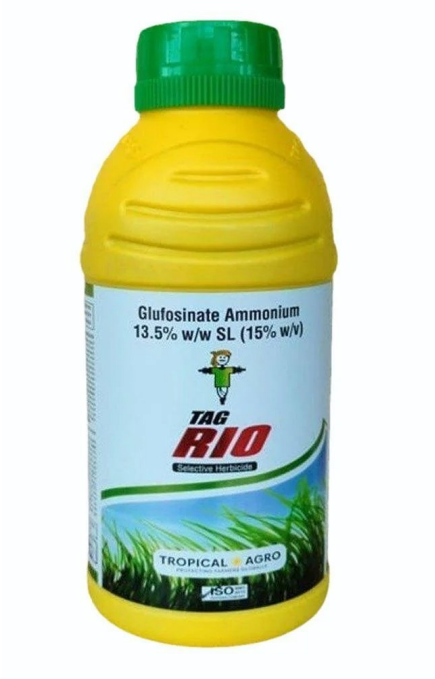
It is important to know that there is no antidote for glufosinate ammonium intoxication, though not commonly encountered in urban settings. However, earlier charcoal hemoperfusion has been shown to reduce morbidity, mortality, and ICU course by facilitating rapid removal of the toxin directly from the circulation, reducing systemic toxicity, helping prevent MODS if initiated early, and enhancing the patient's recovery.

Table 1. Important investigations

Hemoglobin, g/dl WBC, cells/μl Platelets, cells/μl	15.2 8320 268,000
BUN, mg/dl Creatinine, mg/dl	7 0.7
Serum ammonia (1st value), μg/dl Serum ammonia (2nd value), μg/dl Serum ammonia (3rd value), μg/dl Serum ammonia (post hemoperfusion), μg/dl	75.3 75.6 96.1 53.4
Total bilirubin (1st value), mg/dl Total bilirubin (2nd vaue), mg/dl Total bilirubin (3rd value), mg/dl Total bilirubin (4th value), mg/dl Total bilirubin (5th value), mg/dl	4.12 (indirect 3.15) 3.79 (indirect 2.93) 3.46 (indirect 2.71) 2.44 (indirect 1.41) 1.72 (indirect 0.98)
SGOT, IU/l SGPT, IU/l	26 40
Serum magnesium, mg/dl Serum phophorus, mg/dl	1.9 4.4
TSH, mIU/l	1.4
Serum amylase, IU/l Serum lipase, IU/l Serum calcium, mg/dl	20 16 9.4

Legend: WBC=white blood cell; BUN=blood urean nitrogen; SGOT=serum glutamic-oxaloacetic transaminase; SGPT=serum glutamic-pyruvic transaminase; TSH=thyroid stimulating hormone.

Figure 1. Herbicide (glufosinate ammonium)



References

1. Shankar D, Thangaraj M, Gopinathan T, Varun S. A rare case of glufosinate ammonium poisoning [Internet]. 2022 [cited 2025 Oct 8]. Available from: https://www.jebmh.com/articles/a-rare-case-of-glufosinate-ammonium-poisoning-87795.html
2. Duc ND, Sinh NP, Anh LNH. Multi–organ damage due to glufosinate ammonium poisoning [Internet]. 2024 [cited 2025 Oct 8]. Available from: https://ijmshr.com/uploads/pdf/archivepdf/2024/IJMSHR_393.pdf
3. Mu HW, Hsieh PY, Hsiao SP, Chen CH, Yang KW, Pan CS, et al. Early Predictors of Delayed Respiratory Failure after Glufosinate Ammonium Ingestion [Internet]. 2023 [cited 2025 Oct 8]. Available from: https://www.ijpsonline.com/articles/early-predictors-of-delayed-respiratory-failure-after-glufosinate-ammonium-ingestion.pdf
4. Kim J, Cho YS, Chun BJ, Moon JM, Kim DK, Lee BK, et al. The trend of ammonia levels in patients with glufosinate ammonium poisoning with respect to neurotoxicity. Naunyn Schmiedebergs Arch Pharmacol 2023;396: 525–31.
5. Park J, Kwak S-J, Gil H, Kim S-Y, Hong S. Glufosinate herbicide intoxication causing unconsciousness, convulsion, and 6th cranial nerve palsy. J Korean Med Sci 2013;28:1687–9.
6. Wang P, Song C, Lu X, Lu Y. Seizure as the main presenting manifestation of three patients with acute glufosinate-ammonium poisoning. J Zhejiang Univ Sci B 2024 Apr 1;25:354–8.
7. Narendiran V, Challa Venkat SM, Venkatachalam B, Venkatramanan R, Rangaprasad G. Emerging indications for extracorporeal interventions: hemoperfusion for glufosinate ammonium poisoning [Internet]. 2024 [cited 2025 Oct 8]. Available from: https://www.ijccr.org/abstractArticleContentBrowse/IJCCR/36998/JPJ/fullText
8. Tanaka J, Yamashita M, Yamashita M, Matsuo H, Yamamoto T. Two cases of glufosinate poisoning with late onset convulsions. Vet Hum Toxicol 1998;40:219–22.
9. Kim J, Chun BJ, Moon JM, Cho Y. Prognostic value of neutrophil to lymphocyte ratio in the diagnosis of neurotoxicity after glufosinate ammonium poisoning. J Toxicol Environ Health 2022:85:511–9.