Case Report | Journal of Anaesthesia and Critical Care Case Reports | 2025 September-December; 11(3): 17-20

Severe Glufosinate Ammonium Toxicity and Clinical Recovery With Dual Detoxification: A Case Report

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Abstract

Glufosinate ammonium (GLA), a non-selective herbicide, is considered to have low mammalian toxicity. However, ingestion can lead to severe neurological and cardiovascular effects, primarily due to hyperammonemia and myocardial depression. A 25 year old male presented with altered sensorium and cardiovascular compromise following ingestion of unknown quantities of Basta® (GLA-containing herbicide). He developed bradycardia, hypotension, and severe LV dysfunction. Subsequently, patient developed sudden unresponsiveness and was found to have hyperammonemia. Management included hemodialysis for hyperammonemia and intravenous lipid emulsion (ILE) therapy for cardiotoxicity. Clinical improvement was observed with normalization of Glasgow coma scale and cardiac function. Hemodialysis and ILE therapy led to rapid clinical recovery. The patient was extubated on day two and discharged to ward while he remained clinically stable. GLA toxicity can result in life-threatening complications. Multimodal detoxification, including hemodialysis and ILE, may improve outcomes.

Keywords: Glufosinate ammonium, Herbicide poisoning, Hyperammonemia, Intralipid therapy, Cardiotoxicity

Introduction

Poisoning from agricultural chemicals is a major health hazard especially in developing countries like India. Among these agricultural chemicals, herbicides like paraquat and glyphoshate are the most commonly ingested poisons with high morbidity and mortality. Due to easy availability, lack of proper storage techniques and lack of awareness of the hazards of these agricultural chemicals, poisoning from these chemicals are commonly observed [1]. Management of poisoning of these highly toxic compounds requires a profound knowledge of proper management procedures.

Glufosinate ammonium (GLA) is a glyphosate herbicide which is marketed as "Basta®". This is a non-selective herbicide often labeled as having low human toxicity [2]. However, reports have documented delayed-onset neurotoxicity, cardiotoxicity, and fatal hyper-ammonemia. There are no structured guidelines nor a specific antidote for glyphosate herbicide poisoning in India and treatment is mainly supportive [3, 4]. In this case report, we present a rare case of severe GLA poisoning with both neurological and cardiac manifestations which was successfully managed with hemodialysis and intralipid therapy.

Case presentation

A 25-year-old male with no pre-existing co-morbidities presented to a local hospital following ingestion of Basta® (herbicide containing glufosinate ammonium and phosphinothricin) on June 9, 2025. Initially, he underwent gastric lavage followed by administration of activated charcoal. He was clinically stable and was admitted in the Intensive Care Unit (ICU) for monitoring. However, on the following day, he developed sudden unresponsiveness, bradycardia (heart rate of 38/min), hypotension and ST depression on Electrocardiogram (ECG). Mechanical ventilation was initiated via an endotracheal tube and after initial stabilisation he was transferred to our hospital (tertiary care center) for further management.

On arrival at the emergency room in our hospital, he had a Glasgow Coma Scale (GCS) of E4VTM5 and continued to be on controlled mode of mechanical ventilation. Computed Tomography (CT) was done in view of low GCS which showed no significant abnormality and he was subsequently shifted to the ICU for further management. He had persistent hypotension with noradrenaline infusion which was on going from the referral centre at 0.26 mics/kg/min. Bedside echocardiography revealed severe left ventricular dysfunction with an ejection fraction of approximately 20%, and he was initiated on dobutamine infusion at 5 mics/kg/min. An arterial line was placed in the right radial artery. Arterial Blood Gas analysis (ABG) showed raised serum lactate (5 mmol/L). Laboratory investigations revealed elevated serum ammonia of 157 µg/dL along with deranged liver function test. He had an oliguric acute kidney injury (AKI) with 15 to 20 ml/hour of urine output. A diagnosis of severe glufosinate poisoning causing cardiac toxicity while affecting multiple other organs in the form of AKI, deranged liver function tests and elevated lactate levels was made.

In order to correct hyperammonemia, hemoperfusion or hemodialysis was planned for the patient. Due to limited access to hemoperfusion and patient's financial constraints, hemodialysis was

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Submitted: 11/10/2025; Reviewed: 23/10/2025; Accepted: 19/11/2025; Published: 10/12/2025

DOI: https://doi.org/10.13107/jaccr.2024.v11.i03.292

Table 1: Laboratory and other reports of the patient over the three day period in the Intensive critical care unit

Parameters	Day 1	Day 2	Day 3
Serum Ammonia (µg/dL)	157	135	80
Electrocardiogram (ECG)	ST segment depression in anterior leads	Resolved	NA
Echocardiogram	EF ~ 20%	EF ~ 40 - 45%	NA
Glasgow coma scale (GCS)	E4VTM5	E4VTM6	E4V5M6
Serum Urea (mg/dL)	26	35	NA
Serum Creatinine (mg/dL)	0.65	0.59	NA

Table 2: Contents of Basta® (a commercial glufosinate ammonium herbicide formulation)

Component	Function	Toxicological role
Glufosinate Ammonium (18.5%)	Active Herbicidal Ingredient	Inhibits glutamine synthetase → hyperammonemia
Alkyl ether sulphate (AES)	Spreader / Penetrant	Cardiovascular toxicity (vasodilation, bradycardia)
Propylene glycol	Solvent / Stabilizer	Cardiotoxicity and CNS depressant in high doses
Water (Q.S.)	Diluent	Non-toxic carrier
Other adjuvants / emulsifiers	Enhance stability and plant uptake	May add to GI and systemic irritation

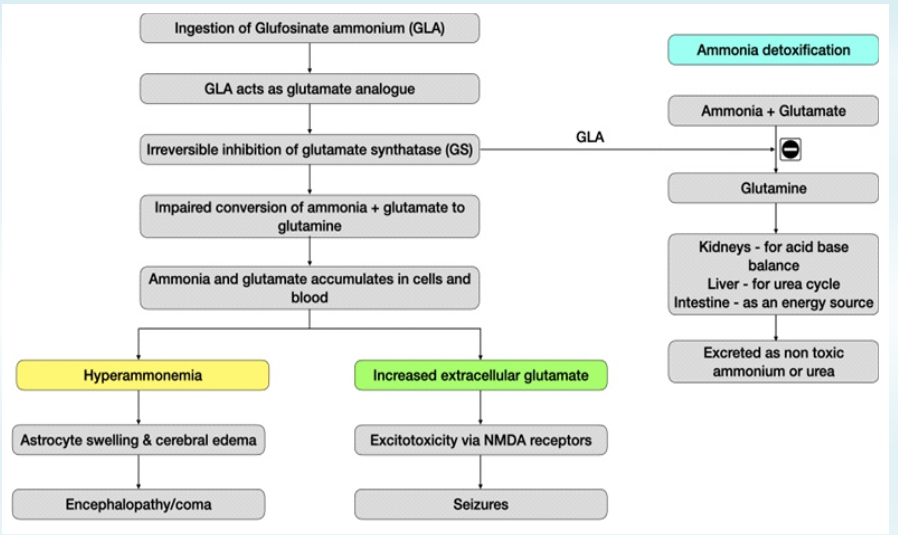

Figure 1: Mechanism of Toxicity of GLA

planned for hyperammonemia after a detailed discussion with the family. A 16 Fr (French) Dialysis catheter was secured in the right Internal Jugular Vein (IJV) under ultrasound guidance and hemodialysis was initiated for a duration of 4 hours. Intralipid therapy was administered for suspected cardiac toxicity. Intralipid emulsion (ILE) was given as a 1.5 mL/kg intravenous bolus followed by 0.25 mL/kg/min intravenous infusion over 1 hour. By the following day, the patient improved hemodynamically. Echocardiography also showed improved left ventricular function (EF 40–45%), and vasopressors were slowly tapered and discontinued. Urine output improved to 50-70 ml/hour with reducing lactate trend. Ammonia level reduced to 130 µg/dl. Further, his neurological status improved (GCS - E4VTM6) with reactive pupils. Considering improved hemodynamics with good neurological status, a weaning trial from the mechanical ventilation was attempted. As the patient tolerated the weaning trial, he was successfully extubated and was put on oxygen support using nasal prongs transitioning to room air subsequently. The patient was then transferred to the ward in the following days. (Table 1)

Discussion

GLA inhibits glutamine synthetase which leads to ammonia accumulation and neurotoxicity. Additionally, surfactants such as Alkyl ether sulphate (AES) which is present in the formulation contribute to cardiovascular collapse [3, 4]. This case is notable for early cardiac dysfunction and a positive response to Hemodialysis (HD) along with Intralipid emulsion (ILE). The components which are usually found in Basta® are described in Table 2 and the mechanism of toxicity of GLA is depicted in Figure 1.

Clinical Features and Predictors of Severity

GLA poisoning is associated with a high rate of delayed onset neurological complications which necessitates the need for prolonged observation of the patient after ingestion of the poison. Neurological complications can range from early onset nausea, vomiting, dizziness and delayed onset (12–48 hours) seizures, alterations in levels of consciousness, respiratory failure, hemodynamic instability. Long term neurological effects of GLA poisoning include amnesia and psychiatric sequelae as hippocampal injury may present after initial recovery. Persistent neurological and psychiatric sequelae necessitate long term follow-up [3].

Key predictors of severity of clinical symptoms include i) Serum ammonia levels - levels > 86 µg/dL; ii) Ingested dose ≥ 13.9 grams of GLA; iii) Age ≥ 61 years and iv) Ethanol co-ingestion. Serum hyperammonemia on admission is strongly predictive of neurological complications (e.g., GCS < 8, seizures, amnesia). High ingested dose of GLA is associated with significantly increased risk of severe toxicity and older patients demonstrate worse outcomes, likely due to reduced metabolic and physiological reserve. Interestingly, concurrent ethanol intake was associated with reduced severity in some studies, potentially due to altered absorption or metabolism [5, 6].

Management

Supportive therapy remains the cornerstone of management and includes gastrointestinal decontamination with activated charcoal if presentation is early (<1 hour), airway protection and mechanical

ventilation as needed for a low Glasgow coma scale (GCS < 8). Fluid resuscitation is essential for hemodynamic support. Vasopressors may be used if needed for stabilising the hemodyamics early. Ammonia lowering agents like lactulose may be considered to reduce the gut absorption and seizures prophylaxis may also be considered. Specific management involves hemoperfusion, which is a preferred modality for effectively countering GLA and surfactant induced toxicity. However, in limited resource settings or unavailability of hemoperfusion, hemodialysis may be an effective alternate in mitigating the effects of the compound especially in the presence of co-existing renal failure [7-9]. Table 3 provides the differences between management of GLA toxicity with hemodialysis and hemoperfusion. Figure 2 shows when hemodialysis and/or hemoperfusion can be initiated in patients with GLA toxicity.

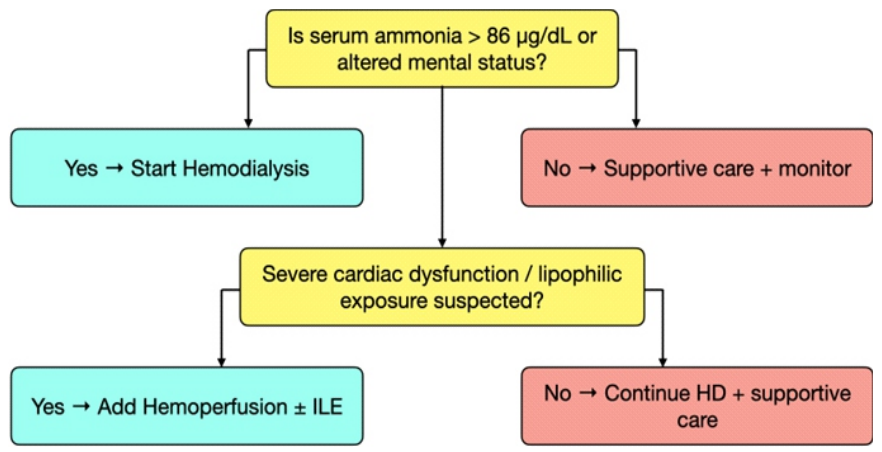

Figure 2: Management flowchart for GLA poisoning

Role of Intralipid Therapy In GLA Poisoning

Intralipid emulsion (ILE) acts like a lipid sink which creates a lipid phase in the blood, sequestering lipophilic toxins and reducing their tissue distribution. Intralipid also supplies fatty acids especially for cardiac ATP production and thus has a great potential for reversing cardiac toxicity. It also has a direct inotropic effect and may directly support myocardial function [10]. Intralipid may be used in GLA poisoning as well even when it is known that GLA is not highly lipophilic. The surfactants (AES) and solvents (propylene glycol) in Basta® are sequestered by the intralipid and thus may help to reduce CNS and cardiac effects caused by these co-formulants. Intralipid may be started in the presence of i) persistent hypotension or low ejection fraction unresponsive to vasopressors, ii) CNS depression or seizures not resolving with standard care and iii) signs of surfactant-related toxicity. Intralipid is usually administered as a bolus of 1.5 mL/kg of 20% ILE followed by an infusion of 0.25 mL/kg/min for 30 – 60 minutes. The maximum permissible intravenous dose of ILE is 10 mL/kg/day. It must be noted that ILE is

Table 3: Hemodialysis versus Hemoperfusion in Glufosinate Ammonium (GLA) Toxicity

Parameter	Hemodialysis (HD)	Charcoal hemoperfusion (CHP)
Mechanism	Diffusion and convection-based removal of water-soluble, low molecular weight substances	Adsorption of toxins onto activated charcoal columns; effective for lipophilic and protein-bound toxins
Target toxins	Water-soluble compounds with low protein binding	Lipophilic, protein-bound, or large molecular weight compounds (e.g., surfactants, solvents)
Effectiveness in GLA toxicity	Moderate for glufosinate ammonium (renal clearance); helps reduce hyperammonemia indirectly	More effective for removing lipophilic components like GLA and surfactants
Ammonia removal	Yes – Ammonia is water-soluble and dialyzable	No direct ammonia clearance; indirect benefit via toxin adsorption
Onset of action	Rapid – clearance starts immediately after connection	Rapid – adsorption begins immediately
Clinical scenario suitability	Useful when: i) Hyperammonemia is primary concern; ii) CHP not available; iii) Renal dysfunction	Useful when: i) Lipophilic herbicide ingestion; ii) Neurologic/cardiac toxicity; iii) No renal failure but altered GCS/seizures
Limitations	Less effective for surfactants/solvents; not lipophilic	Not effective for ammonia or small water-soluble toxins
Access and Equipment	More widely available	Less commonly available, particularly in non-tertiary centres
Adverse effects	Hypotension, bleeding, electrolyte shifts	Hypotension, thrombocytopenia, toxin rebound if short duration
Cost and complexity	Lower cost and easier initiation	Higher cost and needs specialized cartridges along with trained staff

not a substitute for dialysis or hemoperfusion and is to be used only as an adjunctive therapy in critically ill patients [10].

For follow up, patient and his family were contacted over telephone. Patient was clinically stable and also had rejoined his work. However, he complained of some cognitive impairment in the form of recent memory affection. Patient was advised to follow up with neurologist and psychologist for counselling.

Conclusion

Glufosinate ammonium poisoning is an under-recognised but serious toxicological emergency. It can manifest with delayed neurotoxicity, cardiovascular collapse, and hyperammonemia. Hyperammonemia is a critical prognostic marker and therapeutic target. This case report demonstrated that hemodialysis and intralipid therapy offer a synergistic approach for managing both hyperammonemia and surfactant-related cardiac effects. Early ICU-level supportive care can prevent mortality and multimodal detoxification, even when applied late, can reverse life-threatening symptoms.

Clinical message

This case report discusses the successful management of glufosinate ammonium poisoning which is an under-recognised but serious toxicological emergency. Early ICU-level supportive care can prevent mortality and multimodal detoxification, even when applied late, can reverse life-threatening symptoms.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his/her consent for his/her images and other clinical information to be reported in the Journal. The patient understands that his/her name and initials will not be published, and due efforts will be made to conceal his/her identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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How to Cite this Article

Itagi S, Rangappa R, Moola NS, Kuri HR. Severe Glufosinate Ammonium Toxicity and Clinical Recovery With Dual Detoxification: A Case Report. Journal of Anaesthesia and Critical Care Case Reports. September-December 2025; 11(3): 17-20.