Case Study

Management of Lifethreatening Multiorgan Dysfunction Due to Glufosinateammonium Poisoning: A Rare Case Report

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Abstract

Glufosinate ammonium is an herbicide that is considered one of the most hazardous substances and is prohibited in 29 countries worldwide. The mechanism involves inhibition of the enzyme glutamine synthase, leading to an increase in blood ammonia levels and subsequent damage to various organs, particularly the nervous system. Hyperammonaemia is considered one of the main mechanisms of glufosinate ammonia toxicity in humans. Commercial preparations also contain surfactants ranging from 30% to 70%, which can contribute to cardiovascular depression. The most striking feature is late-onset amnesia; this is due to the toxin-induced damage to the hippocampus.

This case of a 31-year-old female who consumed 500 ml of a glufosinate ammonium-based herbicide highlights the entire clinical management. She presented to us after 48 hours with a GCS of 8. She was intubated and ventilated for 4 days, during which she developed seizures, arrhythmias, myocarditis, and pulmonary oedema. She was started on vasopressors and antiarrhythmic, and we used colchicine for myocarditis, as it inhibits the activity of NLRP3 (NOD-, LRR-, and pyrin domain-containing protein 3), mediating its anti-inflammatory properties. We have also used Intralipid emulsion therapy, as lipid emulsions can isolate the lipophilic toxins, reducing their bioavailability and toxicity.

The timely use of amiodarone infusion, colchicine and the intralipid emulsion therapyreflected in the clinical outcome of the patient. She was extubated after 4 days and discharged in a stable hemodynamic state after two more days of general ward observation.

Keywords: Glufosinate ammonium, myocarditis, neurotoxicity, colchicine, lipid sink theory.

Introduction

Glufosinate-ammonium, a broad-spectrum herbicide, is highly hazardous according to the Pesticide Action Network (PAN) India. It is banned in 29 countries, but it is used to control weeds in India. The bulletin published by the Indian Council of Agricultural Research (ICAR) reports

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several unauthorised uses, including in food crops. This organophosphate/phosphonoglycine herbicide is classified as Class II by WHO, which means that it is moderately hazardous, and it falls under the Herbicide Resistance Classification (WSSA) 10[1]. As there is no antidote for this, the supportive therapy is the base of the treatment. The literature on this poisoning is limited, including reports of fatalities.

The primary mechanism of glufosinate ammonia is inhibition of theenzyme glutamine synthase, leading to an increase in blood ammonia levels and subsequent damage to various organs, particularly the nervous system, and tissue necrosis. The proposed mechanism of hyperammonemia secondary to glufosinate exposure is inhibition of glutamine synthetase in human cells[6]. Commercial formulations often contain surfactants that contribute to cardiovascular depression and vasodilation.

This case of 31-year-old glufosinate ammonium poisoning illustrates the urgency and complexity of treatment. Despite the severity and major organ involvement, timely medical intervention resulted in remarkable recovery of our patient.

Case report

A 31-year-old female with no prior comorbid illness presented to our hospital after 48 hours of consuming 500 ml of glufosinate-ammonium herbicide. The empty bottle of poison was brought by the attenders. According to them, she had intentionally consumed the poison due to its easy availability.

Initially, she was admitted to a nearby hospital withmild symptoms of nausea and abdominal pain. First aid measures to remove the poison were given, but details are not clearly provided by the attendants. She was later discharged.

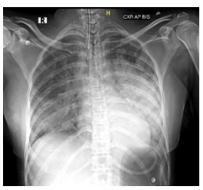

Fig 1: Chest X-ray in ICU

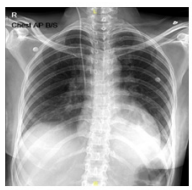

Fig 2: Chest X- ray on Day 3

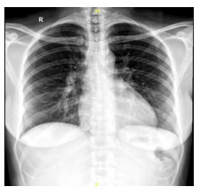

Fig 3: Chest X- ray on discharge

Upon admission to our emergency department, she appeared drowsy (E3V1M5), with 3 mm reactive pupils and no focal neurological deficits. Her Glasgow Coma Scale(GCS) dropped to 8, accompanied by bronchospasm and hypoventilation. She was intubated in emergency and started onmechanical ventilation. A respiratory examination revealed bilateral diffuse crepitations, and a chest x-ray was suggestive of pulmonary oedema. Blood gas showed metabolic acidosis with a high anionic gap. Later she was moved to ICU for further management.

In the ICU all the necessary examinations and evaluations were done. Her ammonia level was 181, and she started to have facial twitching. Hyperammonemia is considered one of the potential risk factors for neurotoxicity[7]. An EEG was done which showed seizure-like activity. Other laboratory parameters showed elevated Trop I and severe LV dysfunction with bilateral pulmonary oedema. She was in severe cardiogenic shock and in need of high vasopressors and inotropes. Her arrhythmias were addressed with amiodarone infusion.

We have also given colchicine (0.5 mg twice daily) for myocarditis, as the studies suggest that it works by reducing NLRP3 activity and has been shown to improve several cardiac conditions, including acute coronary syndrome and pericarditis. Hepatoprotective agents were added for ischaemic transaminitis. Intralipid emulsion therapy was given for 2 days (20% bolus of 1.5 ml/kg followed by 0.25–0.5 ml/kg/min) as the studies showed that the use of lipid sink theory is promising in reducing the risk of cardiotoxicity. Using it in the initial period was a crucial role. On monitoring for 4 days in ICU, her parameters improved and shock was resolved.

She was successfully extubated, switched to nasal oxygenation and finally discharged in a stable haemodynamic state after two more days of general ward observation.

Monitoring for late-onset amnesia, a known feature of this poisoning, showed a favourable outcome, where she initially experienced the symptoms which started after a month of discharge, but later, upon following up, she completely recovered with no such episodes.

Table 1: Trend of vitals and other lab parameters

Parameters	Day 1	Day 2	Day 3	Day 4	Discharge
Vitals					
BP (mmHg)	130/90	87/63	112/68	92/74	110/80
HR	90 bpm	140 bpm	100	98 bpm	80bpm
RR	22	18 bpm	22	24bpm	20 bpm
ABG					
pH	7.15				
PaCO2	31	-	-	-	-
PaO2	58				
HCO3	14				
Lactate	5.3				
Ammonia	-	54	181	60.5	-
Cardiac marker					
Trop I	-	3286	10430	-	-
Liver function test					
ALP	54	46	54		60
AST	63	486	344	202	85
ALT	17	298	305	265	135
GRBS	259	164	-	-	-
CBC					
Hb	15.1	12.2	12.4	12.6	12.6
RBC	5.52	4.42	4.40	4.52	4.52
Platelet	202	198	183	176	176
Renal parameter & electrolytes					
Creatinine	1.16	0.66	0.79	0.6	0.59
Sodium	136	150	146	144	138
Potassium	3.8	3.69	4.5	3	3.9
Chloride	111	98	97	100	100
Calcium	7.5	7.7	8	7.3	8.3
CUE		-	-	-	-
Color	Pale yellow				
pH	5.5				
Appearance	Hazy				
Proteins	1+				
Glucose	Absent				
Blood	3+				
Pus cells	Plenty				

Discussion

Glufosinate is an analogue of glutamate, which is an excitatory amino acid in the human central nervous system (CNS). It inhibits the enzyme glutamine synthetase, leading to intracellular accumulation of ammonia, where hyperammonaemia is considered to be one of the main mechanisms of glufosinate ammonia toxicity in humans[1]. Based on the mechanism of glufosinate poisoning, serum ammonia level could increase when glufosinate activity is irreversibly inhibited in acutely poisoned patients.

Initial symptoms will be nausea, vomiting, abdominal upset, pain and dizziness. Moderate symptoms will be dyspnoea, confusion and agitation. Severe symptoms will be respiratory failure, coma, seizure and shock[2].

Due to toxin exposure and its metabolites, which lead to an imbalance between glutamate, ammonia and glutamine, there is neurological toxicity. Among the various neurotoxicological effects, late-onset amnesia is the most noticeable feature due to toxin-induced damage to the hippocampus[6].

Haemodynamic effects are observed after the intoxication, such as changes in vascular resistance or cardiac output. Cardiovascular manifestations were also observed, like tachycardia and hypotension. These cardio-suppressive effects were likely due to the surfactant [2], which is anionic in nature, sodium polyoxyethylene alkyl ether sulfate, and also its concentration varies from 30 % to 70 % [6].

Studies have shown that colchicine prevents the progression of the disease in viral myocarditis. This anti-inflammatory drug works in part by reducing NLRP3 activity and has been shown to improve several cardiac conditions, including acute coronary syndrome and pericarditis [4].

There have been documented fatalities resulting from the ingestion of glufosinate-containing herbicide. Pathological autopsy revealed that the cause of death was acute cardiorespiratory failure due to glufosinate intoxication characterised by multi-organ congestion without specific pathological findings [5].

The lipid sink theory is a therapeutic approach in which intravenous lipid emulsion (ILE) is used as a potential antidote for certain intoxications. This theory states that the administration of a lipid emulsion can scavenge lipophilic toxins [3]. Surfactant in the glufosinate ammonium herbicide is lipophilic in nature, so intravenous lipid emulsion is found to be promising therapy [6].

Conclusion

Careful evaluation, examination and to look for complications like myocarditis, pulmonary aspiration and neurotoxicological effects along with quantification of intoxication is important inguiding the treatment. Use of colchicine to improve cardiac conditions and lipid sink theory is used in our study, which showed promising result in the treatment. Patients should be hospitalised until fully recovered, and follow-up is also required for late manifestations.

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