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Case Report

Internal Medicine Section

Fatal Glyphosate Poisoning Presenting with Myocarditis and Multiorgan Failure: A Case Report

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ABSTRACT

Glyphosate-based Herbicides (GBHs) are widely used agricultural chemicals linked to increasing reports of intoxications. Disruption of oxidative phosphorylation, along with cardiotoxic effects related to polyoxymethylene amines, is considered to play a role in the toxic actions of Glyphosate (GLY). The present case reports reveal a fatal case of GLY poisoning in a 34-year-old male who ingested approximately 100 mL of Roundup herbicide containing 41% GLY. Despite aggressive medical and supportive management, the patient's condition rapidly deteriorated, leading to multi-organ failure and death. Laboratory investigation revealed severe leukocytosis, electrolyte derangements, hepatic dysfunction, as well as cardiac injury, with elevated troponin I and CK-MB levels, and 2D echocardiography presented global Left Ventricular (LV) hypokinesia with an Ejection Fraction (EF) of 25%. This case highlights the potential lethality of GLY poisoning and the importance of prompt recognition and treatment, as in this case patient reported approximately 25 hours after ingestion. The findings suggest that GLY toxicity can cause severe systemic inflammation, myocardial injury, and multi-organ failure, leading to a poor prognosis. This case report aims to increase awareness about the severity of GLY poisoning and the emphasis the early intervention and aggressive management.

Keywords: Cardiac arrest, Electrolyte derangements, Leukocytosis

CASE REPORT

A 34-year-old male presented to the casualty department with a history of alleged ingestion of approximately 100 mL of Roundup herbicide that contains GLY 41% as an active constituent at home one day prior to admission. The patient was first discovered by his grandmother, and his brother subsequently brought him to the hospital. According to the informant, the patient was not under the influence of alcohol at the time of ingestion. The patient was brought the next day to the hospital approximately 25 hours after ingestion.

Post-ingestion, the patient experienced three episodes of vomiting approximately 10 minutes after ingestion, accompanied by persistent nausea. There was no history of haematemesis, melena, or blood-stained vomitus. The patient had a history of chronic alcohol use, consuming around 100 mL of country liquor daily for the past six years, with the last intake reported the day before ingestion. There was no history of hypertension, diabetes mellitus, bronchial asthma, tuberculosis, or any other significant comorbid illness.

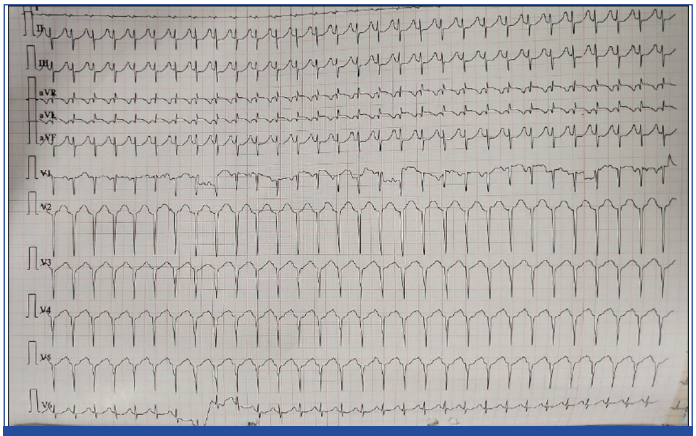
On presentation to the casualty department, the patient was immediately managed with gastric lavage as part of the initial decontamination protocol. Upon examination, the patient's general condition was poor, was afebrile with a pulse rate of 118 beats per minute, blood pressure of 90/60 mmHg, and Oxygen Saturation (SpO_2) of 90% on room air. Cardiovascular examination was within normal limits. Respiratory system examination showed bilateral air entry, and the abdomen was soft and non-tender. The patient was conscious but drowsy. The patient was immediately started on supportive treatment after admission. The patient was initiated on broad-spectrum intravenous antibiotics and was stepped-up during treatment as the White Blood Cell (WBC) kept on rising post-admission, including meropenem (1 g i.v. twice daily), piperacillin-tazobactam (4.5 g i.v. thrice daily), and linezolid (600 mg i.v. twice daily). Supportive medications included lorazepam (4 mL i.v. thrice daily), pantoprazole (40 mg i.v. once daily), and ondansetron (4 mg i.v. thrice daily), along with other symptomatic and supportive therapy.

The day-wise laboratory investigations demonstrated a progressive deterioration in the patient's clinical and biochemical parameters as detailed in [Table/Fig-1] over the hospital course, indicating worsening systemic condition and multi-organ involvement. Haematologically, haemoglobin levels remained largely stable within normal limits, indicating no evidence of anaemia. However, the Total Leukocyte Count (TLC) showed a marked and persistent leukocytosis, rising from 8,800/mm^3 on Day 2 to 29,000/mm^3 by Day 8, suggesting severe ongoing infection or sepsis. The platelet counts initially increased, showing possibly reactive thrombocytosis, but sharply declined on Day 9, reflecting disease progression and possible sepsis-related consumption. Renal function tests remained within normal range, indicating preserved renal function despite critical illness. Serum electrolytes, however, showed fluctuations- hyponatraemia and hypokalaemia, both clinically significant and potentially contributing to cardiac instability. Liver function tests revealed mild transaminitis and hyperbilirubinaemia by Day 7, suggestive of hepatic stress or ischaemic injury. Serum calcium, phosphorus and albumin were below normal, indicating metabolic and nutritional compromise. Additionally, serum ammonia and low Thyroid Stimulating Hormone (TSH) suggested hepatic dysfunction and possible stress-related endocrine imbalance. Urine abnormalities (albumin and sugar initially present, later absent) reflected transient renal involvement. Overall, the laboratory profile revealed progressive leukocytosis, electrolyte derangements, rising inflammatory markers, hepatic dysfunction, and cardiac enzyme elevation, all correlating with clinical deterioration leading to multi-organ failure and death.

Inflammatory and cardiac markers were grossly abnormal. C-Reactive Protein (CRP) was markedly elevated at 185.1 mg/L, and procalcitonin at 1.82 ng/mL, both indicating significant systemic inflammation and sepsis. Troponin I (288.8 ng/L) and CK-MB (35 IU/L) were raised, consistent with myocardial injury. Further, a two-dimensional echocardiogram (2D Echo) was conducted on Day 5 and Day 8 [Table/Fig-2], which demonstrated global Left Ventricular (LV) hypokinesia with an Ejection Fraction (EF) of 25%, indicating

Laboratory parameter (units)	Day-1 (Day of admission)	Day-2	Day-3	Day-4	Day-5	Day-6	Day-7	Day-8	Day-9 29/9
Haemoglobin (gm/dL)	-	12.3	11.8	12.2	13.1	13.4	13.9	13.5	12
Total leucocyte count (cells/ mm^3)	-	8800	10600	18200	20500	23900	23500	29000	28500
Platelet count (lacs/ mm3)	-	1.44	1.67	2.08	2.52	2.57	2.71	2.08	1.43
D-dimer (ng/mL)	-	359	-	-	-	-	-	-	-
Serum calcium (mg/dL)	-	8.8	-	7.9	-	-	7.3	-	-
Creatinine kinase (mg/dL)	-	-	-	320	-	-	-	-	-
CKMB (ng/mL)	-	-	-	35	-	-	-	-	-
CRP (mg/L)	-	-	-	-	185.10	-	-	-	-
Blood urea (mg/dL)	-	29	27	23	18	21	20	30	33
Serum creatinine (mg/dL)	-	0.5	0.5	0.6	0.8	0.7	0.8	1.1	1.0
Serum sodium (mEq/L)	-	142	142	144	139	141	140	137	134
Serum potassium (mEq/L)	-	2.9	3.8	3.5	2.5	3.2	2.6	3.6	3.6
Total protein (g/dL)	6.1	-	-	5.9	-	-	6.4	-	-
Serum albumin (g/dL)	3.3	-	-	2.8	-	-	2.9	-	-
Total bilirubin (mg/dL)	1.4	-	-	1.1	-	-	2.1	-	-
AST (IU/L)	56	-	-	72	-	-	57	-	-
ALT (IU/L)	72	-	-	50	-	-	33	-	-
ALP (IU/L)	124	-	-	95	-	-	118	-	-
Serum lipase (U/L)	-	517	-	293	-	-	-	-	-
Serum magnesium (mg/dL)	-	1.7		1.7	-	1.9	-	-	-
Serum phosphorous(mg/dL)	-	2.6		3.9	-	-	-	-	-
Cortisol (µg/dL)	-	-	5.83	-	-		-	-	-
Ammonia (µmol/L)	-	-	-	-	-	81	-	-	-
Procalcitonin (ng/mL)	-	-	-	-	1.82		-	-	-
Serum cholinesterase (U/L)	-	-	-	-	-	2.7	-	-	-
Troponin I (ng/mL)	-	-	-	288.8	-	-	-	-	-
TSH (µIU/mL)	-	-	0.133		-	-	-	-	-
Urine albumin	Present	-	-	-	-	-	-	Nil	-
Urine sugar	Present	-	-	-	-	-	-	Nil	-

[Table/Fig-1]: Day wise laboratory investigation reports of the patient
CKMB: Creatinine kinase MB; AST: Aspartate transaminase; ALT: Alanine transaminase; ALP: Alkaline phosphatase; TSH: Thyroid stimulating hormone.



[Table/Fig-2]: ECG findings on Day-8.

severe LV systolic dysfunction. Additional findings included trivial mitral regurgitation, a dilated left ventricle, and no evidence of infective endocarditis or pericardial effusion, suggestive of possible myocarditis. Ultrasonography (USG) of the abdomen and pelvis revealed no significant abnormality.

In view of progressive desaturation, tachycardia (142/min), tachypnoea (36/min), and gasping respiration on the morning of Day 6, the patient was intubated with a 7.5 mm endotracheal tube, ensuring bilateral air entry. The patient was subsequently placed on mechanical ventilation with the following settings: mode-volume control, tidal volume- 380 mL, PEEP- 6 cmH2O, FiO_2-100%, respiratory rate- 24 breaths/min, and I:E ratio- 1:2. Arterial Blood Gas (ABG) analysis revealed metabolic acidosis, which was corrected accordingly.

In view of persistent hypotension, vasopressor support was initiated with noradrenaline, vasopressin, and dopamine infusions. The patient's vital parameters, fluid balance, and urine output were closely monitored throughout hospitalisation. Despite aggressive medical and supportive management, the patient's condition continued to deteriorate. On Day 9, the patient developed sudden cardiorespiratory arrest. Cardiopulmonary Resuscitation (CPR) was initiated immediately; however, despite all resuscitative efforts, the patient could not be revived and was declared dead on the same day.

DISCUSSION

The GBHs have widespread global distribution. As a commonly utilised agricultural chemical, primarily among farming communities, GBHs have been linked to an escalating number of reported intoxications [1]. GLY, a pioneering broad-spectrum herbicide, was discovered by Franz JE at Monsanto and introduced to the market in 1974. This groundbreaking chemical revolutionised pesticide development due to its distinctive mode of action and selective toxicity to plants. GLY's effectiveness lies in its ability to inhibit the enzyme 5-enolpyruvyl-shikimate-3-phosphate synthase, thereby blocking the shikimate pathway. This metabolic pathway is essential for plants, bacteria, fungi, and algae to synthesise aromatic amino acids, including tyrosine, phenylalanine, and tryptophan. Notably, the shikimate pathway

is absent in animals, making GLY relatively non-toxic to non-plant species [2]. Thus, GLY's unique mechanism of action, targeting a plant-specific biochemical pathway, has led to its assumed safety for mammals, including humans. This perception has driven the widespread use of GBHs since the 1970s [3]. The present case highlights the severe and rapidly progressive multisystem toxicity associated with delayed presentation after GLY-surfactant ingestion. Despite supportive management, the patient demonstrated progressive respiratory failure, worsening metabolic acidosis, rising inflammatory biomarkers, cardiovascular compromise consistent with myocarditis, and refractory shock leading to death on Day 10 post-ingestion. The markedly elevated cardiac enzymes, severe LV dysfunction (EF 25%), and global hypokinesia on echocardiography strongly suggested GLY-induced myocardial injury. Although additional investigations, such as cardiac Magnetic Resonance Imaging (MRI) or endomyocardial biopsy, would have strengthened diagnostic certainty, the clinical and biochemical profile was strongly indicative of toxic myocarditis. These findings reinforce that cardiovascular involvement- particularly myocardial dysfunction- is a key driver of mortality in severe GLY poisoning.

Similar patterns of deterioration have been reported in the literature. Chhikara M et al., demonstrated a comparable trajectory of multisystem involvement, with patients developing metabolic acidosis, respiratory failure, escalating inflammatory markers, vasopressor dependence, and cardiac dysfunction [4]. Like the present case, their patient exhibited profound systemic inflammation and hemodynamic instability following GLY-surfactant ingestion. However, earlier hospital presentation and more timely initiation of organ support likely contributed to the survival observed in their case. Both reports emphasise that late presentation, combined with early cardiopulmonary involvement, significantly worsens prognosis in severe glySH poisoning.

In contrast, the case reported by Karlapudi P et al., described a patient who, despite multisystem involvement- including gastrointestinal irritation, metabolic acidosis, respiratory compromise, and early renal dysfunction- ultimately recovered with prompt medical intervention [5]. Renal impairment in their patient improved following dialysis, whereas renal function in the present case initially remained preserved but subsequently deteriorated alongside sepsis, systemic inflammation, and progressive multiorgan failure. These contrasting outcomes underscore the importance of early recognition, timely organ support, and aggressive management in improving survival after GLY ingestion. The present case further illustrates how delayed presentation (approximately 25 hours post-ingestion) can significantly limit the effectiveness of available supportive measures.

Severe toxicity after GLY ingestion has also been documented in other fatal reports. Stella J et al., described two lethal cases in which intensive care interventions failed despite early identification of poisoning severity, demonstrating that pulmonary oedema, metabolic acidosis, and hyperkalaemia frequently signal an irretrievable clinical course [6]. Recent epidemiological evidence by Zhu Y et al., further connects GLY exposure with adverse cardiovascular health, reporting a significant decline in Cardiovascular Health (CVH) score with rising GLY levels (β=-1.33; 95% CI -2.25 to -0.41), with no identifiable safe exposure threshold [7]. The clinical observations reported in the present study were also demonstrated by Lu J et al., who revealed GLY exposure promotes apoptosis in cardiac and vascular tissues [8], while Gress S et al., showed that GBHs disrupt cardiac electrophysiology in a dose-dependent manner through impaired calcium channel activity, potentially leading to arrhythmias, conduction blocks, and QT prolongation [1]. Together, these findings provide biological plausibility for the profound cardiac involvement observed in the present case and reinforce the need for heightened awareness of GLY's cardiovascular toxicity.

The GLY poisoning has no specific antidote and is managed with decontamination and aggressive supportive care; gastric lavage or activated charcoal may be considered within one hour of ingestion, although their efficacy in reducing systemic absorption remains uncertain, and intravenous fat emulsion has been suggested in severe cases, as GLY toxicity involves uncoupling of oxidative phosphorylation and direct cardiotoxicity [9]. However, in the present case, the patient presented approximately 25 hours after ingestion of Roundup herbicide, limiting the role of early decontamination. GLY formulations typically contain ≥41% isopropylamine salt and nearly 15% Polyoxyethyleneamine (POEA) surfactant and exert herbicidal activity via the shikimic acid pathway, which is absent in humans, accounting for relatively lower toxicity; nevertheless, uncoupling of oxidative phosphorylation and POEA-mediated cardiotoxicity are implicated in human toxicity, while the compound lacks anticholinesterase activity despite structural similarity to organophosphates [9-11]. Poor prognostic indicators include respiratory failure, metabolic acidosis, tachycardia, elevated creatinine, and hyperkalaemia, all of which mandate early identification and intensive management [12]. In suspected myocarditis, confirmatory investigations such as cardiac magnetic resonance imaging, endomyocardial biopsy, or coronary angiography- particularly CMR for detecting myocardial inflammation and fibrosis- could further strengthen the diagnosis [13].

CONCLUSION(S)

To conclude, despite the assumed safety of GBHs for mammals due to a plant-specific mechanism of action, GLY poisoning can have severe consequences. This case report highlights the devastating outcome of ingestion of 100 mL of GLY herbicide, resulting in multisystem organ dysfunction, myocarditis, and ultimately, death. The patient's presentation, characterised by vomiting, nausea, and altered renal and liver function, progressed to severe cardiac dysfunction, evidenced by global hypokinesia, reduced ejection fraction, and myocarditis. Despite aggressive supportive care, the patient succumbed to his condition on Day 9 of hospitalisation. This case underscores the importance of recognising the potential toxicity of GBHs and the need for prompt medical attention in cases of suspected poisoning. Further research is necessary to fully understand the mechanisms of GLY toxicity and to develop effective treatment strategies for managing exposure.

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