ASIA PACIFIC JOURNAL of MEDICAL TOXICOLOGY
APJMT 15;2 http://apjmt.mums.ac.ir June 2026

CASE REPORT

Acute glyphosate poisoning: a case report on fatal poisoning

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Abstract

***Introduction*:** Herbicidal compounds are one of the common types of poisonings used for Deliberate Self Harm in Developing countries, associated with high morbidity and mortality with multisystem toxicity The rare manifestations involve Cardiovascular and Neurological system.
***Case Report*:** In 2025, a 30-year-old male presented to emergency department at Lata Mangeshkar hospital, India, one hour after ingestion of 150 ml of 41% glyphosate (Glycel) with vomiting, tachycardia (140/min), hypotension and hypoxia. Within hours, he developed metabolic acidosis, type 1 respiratory failure, refractory hypotension requiring noradrenaline, left ventricular dysfunction (EF 35%), and neurological signs (horizontal/vertical nystagmus, diplopia). Management included gastric lavage, mechanical ventilation, Inotropic support (Noradrenaline, Dobutamine), N-Acetylcysteine and Antibiotics for aspiration pneumonia. MRI Brain was normal. By day 6, cardiac, neurological and respiratory parameters gradually normalized; the patient was extubated and discharged without sequelae after psychiatric evaluation.
***Discussion*:** The severe multisystem toxicity observed in this case, particularly the transient cardiomyopathy and neurological deficits, is primarily driven by the surfactants in commercial glyphosate formulations rather than the glyphosate moiety itself. These surfactants likely induce cellular damage through mitochondrial dysfunction and the uncoupling of oxidative phosphorylation. The reversibility of these manifestations suggests a transient toxic-metabolic process rather than permanent structural injury, emphasizing the critical role of surfactant-induced cardiotoxicity and neurotoxicity.
***Conclusion*:** This case highlights rare cardiovascular and reversible neurotoxic effects of glyphosate poisoning which is mainly due to mechanism of uncoupling of oxidative phosphorylation. The most common clinical symptoms are gastrointestinal like erosions and gastrointestinal hemorrhages. Increased risk of mortality is seen patients developing acute kidney injury, pulmonary edema and hyperkalemia. This case report underscores the efficacy of aggressive supportive care in achieving favorable outcomes despite absence of an antidote.

Keywords: Herbicide, Glyphosate, Cardiotoxicity, Neurotoxicity, Poisoning

How to cite this article: Kolse S, Deoke S, Holay M. Acute glyphosate poisoning: a case report on fatal poisoning. Asia Pac J Med Toxicol. 2026; 15(2):81-83.

INTRODUCTION

Herbicidal poisoning is a significant problem in developing nations like, India, particularly in rural areas. Mortality is seen in most cases with pulmonary, cardiovascular and renal toxicity. We report a case of glyphosate ingestion with predominant cardiovascular and neurotoxicity which are rare manifestations, where the patient survived with aggressive supportive management. The key clinical events, finding and the necessary interventions done have been highlighted in Table 1.

CASE PRESENTATION

In 2025, a 30-year-old male farmer was brought to the emergency department of Lata Mangeshkar hospital, India, after ingestion of approximately 150 mL of the herbicide Glycel (isopropylamine salt of Glyphosate 40.6% w/w as active ingredient) one hour prior. He presented with multiple episodes of vomiting without hematemesis, diarrhoea, sweating, or excessive secretions.

On examination, the patient was drowsy but arousable with inflamed oral mucosa, heart rate 140/min (regular), blood pressure 100/70 mmHg, oxygen saturation 94% on 4 L/min oxygen, and respiratory rate 26/min. Pupils were 2 mm and reactive to light. No fasciculations were noted; deep tendon reflexes were brisk. Cardiovascular examination revealed tachycardia.

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Respiratory system showed throat-conducted sounds and crepitations in both lung fields (right & left).

Laboratory Data: Initial ABG showed metabolic acidosis (pH7.20, HCO3 15mEq/L, PaO2 63 mmHg). Complete blood count: TLC 13,200/cumm. Renal function: BUN

Table 1. Clinical timeline of events, findings and interventions

Time	Key Clinical events, Findings and Interventions
Within 1 hour of ingestion	Vomiting. Drowsy but arousable, HR- 140/min, BP-100/70 mmhg, SpO2 94% on 4Litres of oxygen. Gastric lavage was performed immediately upon arrival. Patient admitted in ICU.
2-4hours	Respiratory distress. ABG- pH7.20, HCO3 15, PaO2 63 (metabolic acidosis plus Type 1 respiratory failure). Patient Intubated and put on ACVC mode of ventilation. Empirical antibiotics (Piperacillin-tazobactum) was started.
12-24 hours	Hypotension developed, Intravenous fluids (crystalloid) were administered aggressively for resuscitation. Inotropic support Noradrenaline started at 0.5ug/kg/min and titrated up to 1.4ug/kg/min, had persistent hypoxia and acidosis. Patient had oliguria. Injectable N-Acetyl cysteine was given (1.2gm twice daily) for 3 days.
Day 2-3	Patient had diplopia plus nystagmus. Echo showed EF of 35% with global hypokinesia. Dobutamine was added at 5ug/kg/min and continued until hemodynamic stability was achieved.
Day 4-6	Improvement in hemodynamic, oxygenation and neurological resolution was seen. Extubated on day 6. Antibiotic therapy was de-escalated after negative cultures and stopped on day 7.
Day 10	Discharged. Psychiatry review was done.
1 month follow up	Complete neurological, cardiac, renal and functional recovery.

22mg/dL, creatinine 1.01 mg/dL (remained normal). Liver function: total bilirubin 1.7 mg/dL, SGOT105 U/L, SGPT 87 U/L (mild elevation, resolved). Electrolytes: hypomagnesemia corrected.

Cardiac biomarkers (troponin, CK-MB) not significantly elevated. Serial ABGs showed progressive resolution of acidosis and hypoxia. Imaging and Cardiac Evaluation: Chest X-ray on admission showed right lower zone haziness.

ECG: sinus tachycardia without ST-T changes. Echocardiography on Day 3 revealed global hypokinesia, moderate left ventricular dysfunction (EF 35%), consistent with transient cardiomyopathy likely due to surfactant (POEA) toxicity and uncoupling of oxidative phosphorylation. Repeat echocardiography showed recovery of LV function.

Neurological Examination: On Day 3, while conscious and oriented, the patient developed sudden-onset diplopia (worse on lateral gaze) and nystagmus (both horizontal and vertical components). Detailed neurological examination revealed: normal higher mental functions (alert, oriented to time/place/person, intact memory and attention); cranial nerves examination revealed normal pupillary reaction, no ptosis, full extraocular movements with diplopia on horizontal gaze, presence of horizontal jerk nystagmus on lateral gaze and vertical nystagmus on upward/downward gaze, no facial asymmetry or hearing loss; motor system showed normal bulk, tone, power 5/5 in all limbs, no drift; sensory system — intact to pinprick, temperature, vibration ,and proprioception; coordination had mild impairment on finger-nose and heel-shin testing due to nystagmus/diplopia but no dysmetria or intention tremor; gait — normal (no ataxia when eyes open); deep tendon reflexes brisk but symmetric; both plantars flexor. No meningeal signs or seizures. Brain MRI (performed on Day 4) was completely normal with no evidence of brainstem, cerebellar, or cortical lesions, ischemia, demyelination, or toxic encephalopathy changes on T1/T2/FLAIR/DWI sequences. These findings resolved completely by Day 6.

The patient was extubated on Day 6 and shifted to the ward. He was discharged on Day 10 after psychiatry consultation.

Follow-up

At one-month follow-up, the patient demonstrated complete recovery. Neurologically, there was full resolution of diplopia and nystagmus with normal cranial nerve examination, motor/sensory function, coordination, and gait. No residual cognitive deficits were noted. Cardiac evaluation (repeat 2D echocardiography) showed normal left ventricular function with ejection fraction 55% and no regional wall motion abnormalities. Serial ECGs were normal. Renal function tests remained stable within normal limits. Functionally, the patient had returned to his pre-morbid level of activity as a farmer, with no exertional dyspnoea, fatigue, or limitations in daily activities. He resumed agricultural work without any sequelae. This objective recovery across neurological, cardiac, renal, and functional domains highlights the success of aggressive supportive care.

DISCUSSION

Acute glyphosate poisoning can lead to life-threatening multisystem toxicity, as illustrated by this case featuring cardiogenic shock with reversible myocardial dysfunction (EF 35% with full recovery), respiratory failure requiring mechanical ventilation, metabolic acidosis, and unusual neurological manifestations (diplopia with horizontal and vertical nystagmus) [1]. These rare features contributed to diagnostic complexity.

Glyphosate is a broad-spectrum, non-selective systemic herbicide. Toxicity in humans is primarily driven by surfactants (e.g., polyoxyethylene tallow amine/POEA) in commercial formulations such as Glycel, rather than the glyphosate moiety alone [2].

Key mechanisms include uncoupling of oxidative phosphorylation, mitochondrial dysfunction, and direct cellular membrane damage [3]. This explains the transient cardiomyopathy observed (global hypokinesia on

echocardiography), likely resulting from cardiotoxic surfactant effects [4]. Neurological features (nystagmus and diplopia) may arise from direct CNS toxicity, metabolic encephalopathy (acidosis, hypomagnesemia), or vestibular-ocular pathway disruption. The normal brain MRI supports a reversible toxic-metabolic process rather than structural damage. Differentials such as organophosphate poisoning, brainstem stroke, Wernicke encephalopathy, or demyelinating disease were excluded by the clinical course and resolution with supportive care. Similar rare cases of nystagmus, ataxia, and transient neurological deficits in severe glyphosate ingestions with favourable outcomes have been reported in literature [5].

Literature review shows that severe glyphosate surfactant intoxication commonly involves gastrointestinal corrosion, hypotension, pneumonitis, acute kidney injury, and metabolic derangements [1]. Prognostic factors associated with high mortality include oliguric AKI, persistent hyperkalaemia, pulmonary oedema, and large ingested volume [1]. Cases of cardiogenic shock and reversible myocardial dysfunction respond well to inotropes and, in refractory scenarios, intravenous lipid emulsion [4]. Respiratory failure is often due to aspiration or direct pulmonary toxicity.

There is no specific antidote. Treatment is entirely supportive, with emphasis on early airway protection, fluid resuscitation, hemodynamic stabilization, metabolic correction, and close monitoring. N-acetylcysteine was administered empirically for potential antioxidant and hepatoprotective benefits, although it is not standard therapy. The commercial formulation's surfactant content significantly contributed to the severity [2].

This case adds educational value by demonstrating uncommon but reversible complications and reinforces the critical role of aggressive, multidisciplinary intensive care in improving survival and functional outcomes in glyphosate poisoning, even in resource-limited settings. Strict cardio-neuro-respiratory follow-up is recommended at discharge.

CONCLUSION

This case highlights the rare but potentially life threatening cardiovascular and neurological toxicities associated with glyphosate ingestion. Despite the absence of specific antidote, early aggressive supportive care- including mechanical ventilation, dual inotropic support, correction of metabolic abnormalities, and intensive monitoring resulted in complete recovery without residual deficits. Clinicians should remain vigilant for uncommon manifestations such as reversible left ventricular dysfunction and transient neurotoxicity. Prompt recognition and multidisciplinary intensive care can significantly improve outcomes even in severe glyphosate poisoning.

ACKNOWLEDGMENTS

We gratefully acknowledge the involvement of physicians from the Medicine and Emergency departments along with nurses and hospital management for their valuable support.

Conflict of interest: The authors declare no conflict of interest.

Funding and Support: The authors received no financial support.

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