Case Report

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Dose-dependent Clinical Spectrum and Management of Glyphosate-surfactant Herbicide Poisoning: A Case Report

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ABSTRACT

Glyphosate-Surfactant Herbicide (GlySH) poisoning, though less common than Organophosphate (OP) poisoning, poses significant clinical challenges due to its dose-dependent toxicity and the lack of specific antidotes. This report presents two cases with contrasting presentations. Case 1 involved a 32-year-old male with diabetes who ingested 150-200 mL of GlySH in a suicide attempt. He developed gastrointestinal symptoms, severe lactic acidosis, hypotension, and multiorgan dysfunction, requiring intensive care, intubation, and aggressive treatment. In contrast, in Case 2, a 27-year-old female with no comorbidities, ingested around 20-50 mL of GlySH and exhibited milder symptoms such as throat burning, hypoglycaemia, and tachycardia, which resolved with symptomatic treatment and intravenous fluids. For both cases, the cornerstone of treatment included fluid and electrolyte correction, along with supportive management. These cases underscore the dose-dependent nature of GlySH toxicity. Misdiagnosis is most common, as OP poisoning results in delayed treatment and exacerbation of such conditions. Treatment remains primarily supportive, following poisoning guidelines. Therefore, these cases call for better diagnostic guidelines and preventive measures to reduce the mortality associated with GlySH, especially in agricultural settings.

Keywords: Multi-organ dysfunction syndrome, Organophosphorus compound, Suicide

CASE REPORT

Case 1

A 32-year-old male patient, who had been a known diabetic for three years and was on Metformin (500 mg) with Glimepiride (1 mg), presented to the Emergency Medical Service (EMS) after three hours with an alleged history of suicidal consumption of pesticide. Bystanders stated that the patient had ingested a pesticide but were unsure of the type. Upon arrival, the patient exhibited symptoms including vomiting, abdominal pain, a burning sensation in the stomach, and increased salivation. Based on these symptoms, Organophosphorus (OP) poisoning was suspected. His vital signs upon arrival were a Pulse Rate (PR) of 125/min, Oxygen Saturation (SpO_2) of 94% on Room Air (RA), and Blood Pressure (BP) of 140/90 mmHg. The pupils were 1.5 mm and reactive to light, and his Glasgow Coma Scale (GCS) score was 11/15 (E2V4M5).

As part of the emergency management protocol, immediate gastric lavage with activated charcoal was performed. Gastric lavage was conducted using 500 mL of Normal Saline (NS) with 50 g of activated charcoal in 400 mL of NS, administered via a Ryles tube. This was given as a loading dose {150 mg/kg Intravenous (IV) over one hour in 200 mL of NS) and maintenance doses (50 mg/kg IV over four hours in 500 mL of NS, followed by 100 mg/kg IV over 16 hours in NS}. After two hours of presenting symptoms, it was determined that the patient had consumed Glyphosate (GLY) (kallaikolli), approximately 150-200 mL, a herbicide. By the time the final diagnosis was made, the patient had developed hypotension (80/60 mmHg) and worsening tachycardia (134 bpm). The patient's condition further deteriorated, exhibiting breathlessness with SpO_2 levels dropping to 70% on RA. The patient was intubated and started on inotropes with noradrenaline (0.4 µg/kg/min), titrated according to BP.

Arterial Blood Gas (ABG) analysis revealed severe lactic acidosis (9 mmol/L). Capillary Blood Glucose (CBG) was charted regularly, and routine investigations showed deranged Renal Function Tests (RFT) (urea 120 mg/dL and serum creatinine 3.5 mg/dL) and Liver Function Test (LFT) derangements (aspartate aminotransferase 316 U/L, alanine transaminase 354 U/L, total bilirubin 2.8 mg/dL), along with dyselectrolytaemia, with sodium at 122 mmol/L and potassium at 2.9 mmol/L [Table/Fig-1]. The patient received symptomatic treatment with IV fluids (NS at 100 mL/hour) and potassium correction using sodium bicarbonate (100 mEq bolus, then 10 mL/hour, adjusted based on ABG levels). Additional supportive medications included intravenous pantoprazole 40 mg, Emeset 4 mg IV, and vitamin C 500 mg twice daily.

Parameters	Case-1	Case-2
Vitals	PR 125/min SpO_2 94% at Room Air (RA) BP 140/90 mmHg GCS 11/15 (E2V4M5)	PR 126 bpm BP 90/60 mmHg SpO_2 95% at Room Air (RA) GCS 15/15
Laboratory investigation	Lactic acidosis (9 mmol/L) RFT: Urea 120 mg/dL and serum creatinine of 3.5 mg/dL LFT: Aspartate Aminotransferase 316 U/L, Alanine transaminase 354 U/L, Total bilirubin 2.8 mg/dL Electrolytes: Sodium- 122 mmol/L, potassium- 2.9 mmol/L	Blood glucose levels (54 mg/dL) RFT: Urea 14 mg/dL and serum creatinine 0.8 mg/dL LFT: Aspartate aminotransferase 31.3 U/L, Alanine transaminase 40 U/L, Total bilirubin 1.0 mg/dL Electrolytes: Sodium 140 mmol/L, potassium- 4.0 mmol/L

[Table/Fig-1]: Comparison of both cases based on the vitals and other parameters.
PR: Pulse rate; SpO_2: Oxygen saturation; BP: Blood pressure; GCS: Glascow coma scale; RFT: Renal function test; LFT: Liver function test

The patient's vitals and other parameters stabilised. After 48 hours, the patient was extubated and discharged with vitamin supplementation (vitamin C 500 mg once daily for two weeks). Psychiatric counselling was provided at the time of discharge, along with a prescription for tablet alprazolam 0.5 mg for two weeks. After one week, follow-up investigations were normal, and continued vitamin supplementation was recommended.

Case 2

A 27-year-old female reported to the Emergency Medical Service (EMS) with a history of ingesting 20-50 mL of kallaikolli (41% glyphosate) with suicidal intent. The patient had no known comorbidities. She arrived four hours after consumption and had experienced one episode of vomiting, presenting with moderate dehydration. On further examination, the patient exhibited a

severe burning sensation in her throat and epigastrium, along with tachycardia (126 bpm) that was regular. Her BP was 90/60 mmHg, SpO_2 was 95% on RA, the GCS score was 15/15, and her blood glucose levels were low (54 mg/dL) [Table/Fig-1].

Gastric lavage of 500 mL was performed, followed by the administration of activated charcoal (50 g in 400 mL of normal saline) through a Ryles tube. The patient was immediately started on normal saline due to her low BP, which improved to 100/60 mmHg after a bolus of normal saline (300 mL), followed by 150 mL/hour. This was later changed to Dextrose and Sodium Chloride (DNS) at 100 mL/hour in view of the low CBG levels.

She was treated with pantoprazole (40 mg IV twice), Emeset (4 mg IV three times), and supportive medications, including a vitamin C 500 mg tablet. After stabilising her vital parameters, she was kept Nil Per Os (NPO) for 24 hours and was subsequently given clear liquids, which she tolerated well. The following day, she was started on a semisolid diet. Once stable, she was discharged with psychiatric counselling and prescribed alprazolam 0.5 mg for two weeks. She followed-up after one week with normal investigations and continued vitamin supplementation for another two weeks.

DISCUSSION

These two cases highlight the spectrum of GLY poisoning, influenced by dose, patient factors, and timely management. In case 1, a 32-year-old male with diabetes who ingested 150-200 mL in a suicide attempt presented with gastrointestinal symptoms and progressed to severe systemic toxicity, including hypotension, lactic acidosis, and Multi-Organ Dysfunction Syndrome (MODS), which required intubation and intensive care. In contrast, case 2 involved a 27-year-old female with no comorbidities who ingested 20-50 mL, experienced mild symptoms such as throat burning, tachycardia, and hypoglycaemia, and recovered with symptomatic treatment. These cases underscore the dose-dependent nature of toxicity, with comorbidities such as diabetes mellitus exacerbating outcomes. Early intervention and tailored supportive care were crucial for recovery, while the suicidal intent highlights the importance of psychiatric evaluation in poisoning cases.

India, with its strong agricultural background, commonly uses pesticides and herbicides such as GLY to combat crop loss, where such rampant destruction poses a global issue affecting farmers and food security [1]. N-phosphomethylglycine, or GLY, an organophosphorus derivative and phosphonic acid salt, is a widely used non-selective herbicide due to its effectiveness and versatility in controlling weed growth [2,3]. Genetically modified formulations enhance its utility but also increase associated risks [4]. It was discovered in 1950 by Swiss scientist Dr. Henri Martin and introduced in 1971, becoming the third most-used herbicide globally [5-7]. GlySH, marketed as 'Roundup', 'Glycel', or 'Lagaam', has a fatality rate of 3.2-29.3% [3]. The US Environmental Protection Agency (EPA) classifies GLY under Category III for relatively low oral and dermal acute toxicity [8]. While generally safe with proper use, ingestion can cause significant morbidity and mortality, depending on the dose, concentration, and the individual patient's physiological state [2,3,6]. Although its effects on the central nervous system are limited compared to organophosphate poisoning, potential risks cannot be ignored.

Various literature suggests that oral ingestion of GlySH typically causes mild, transient gastrointestinal disturbances but is often linked to suicidal ideation [1,2,7,9,10]. About 30-36% is absorbed, with peak concentration occurring in six hours, excreted unchanged in faeces initially and later in urine, with a half-life of 4-6 hours [9]. The corrosive effects of GlySH damage the mouth, throat, and oesophagus, causing dysphagia and bleeding [3,7,11]. GlySH metabolism can lead to renal disorders and metabolic acidosis, as observed by Hsu K, similar to our first case, which exhibited deranged renal function tests with lactic acidosis [10]. Even minimal exposure (0.3 mg/kg body weight per day) causes hepatorenal toxicity in animals [8,12], while prolonged low doses (50 ng/L) induce liver damage [13]. Diagnosis remains challenging due to insufficient data and guidelines, as the etiopathogenetic cycle of the agent remains cryptic [14]. It has been hypothesised that the GlySH herbicide inhibits oxidative phosphorylation, damaging mitochondria, DNA, and proteins, while Polyethoxyleneamine (POEA) exacerbates toxicity, causing cardiotoxicity and severe organ dysfunction [3,15].

In our case study, both cases demonstrated the dose-dependent nature of GlySH poisoning, with case 2 showing milder symptoms such as burning, hypoglycaemia, and tachycardia due to the intake of 20-50 mL. In contrast, case 1 exhibited severe, life-threatening complications such as hypotension and multi-organ damage, resulting from the consumption of 150-200 mL, which required intensive management. This implies the dose-dependent clinical characteristics of GlySH poisoning. The most common clinical manifestations, as per organ systems [3,5,11,15], are presented in [Table/Fig-2]. The involvement of all organ systems in its most severe form may ultimately manifest as MODS [9,15].

General	Dehydration
Gastrointestinal	Erosion of the Gastrointestinal (GI) tract, difficulty in swallowing and GI haemorrhage
Dermal	Eye and skin irritation have occasionally been reported from dermal exposure
Oro-nasal	Oral/nasal discomfort, tingling, throat irritation
Cardiovascular	Hypotension, dysrhythmias, acidosis, hyperkalemia, shock and malignant arrhythmia, abnormal chest X-ray, tachycardia
Respiratory system	Pneumonitis, acute lung injury, pulmonary oedema, pulmonary toxicity
CNS system	Altered level of consciousness, coma, epileptic seizures, and haemorrhage
Renal system	Oliguria, acute kidney injury, hepatic dysfunction, tubular necrosis, renal toxicity, and elevated creatinine levels

[Table/Fig-2]: Common clinical manifestations as per organ for Gly poisoning.

GlySH poisoning cases are significantly fewer compared to organophosphate poisoning, which is more commonly observed among farmers [3-5,9,11]. This often leads to misdiagnosis, as physicians may primarily consider any agricultural-related poisoning to be due to organophosphates, which was the similar kind of error observed in our first case. Additionally, there are no antidotes for GlySH poisoning, and treatment primarily involves supportive care based on the symptoms [3,9]. The documented mainstay of treatment for oral ingestion, according to various literature, includes gastric lavage or the use of activated charcoal within one hour of ingestion, which may help but does not prevent systemic absorption [3,9,11]. Haemodialysis can aid in clearing GLY, but not GlySH [9]. In both cases, gastric lavage was performed; however, it resulted in organ dysfunction, and correcting lactic acidosis with lactate-free fluids was crucial. In severe cases, treatments such as calcium channel blockers, tricyclic antidepressants, and beta-blockers may be considered [16-18]. These challenges underscore the need for accurate diagnosis and tailored management in cases of GlySH poisoning.

Hence, it is important to record precise data about the patient, with special emphasis on their history, whether it pertains to the presenting illness or is of a medical or personal nature. The accuracy of this data helps avoid errors in diagnosis and enables healthcare providers to deliver appropriate treatment, which will facilitate the patient's recovery at a faster rate and prevent the progression of the condition.

CONCLUSION(S)

These case reports highlight the dose-dependent nature of GlySH poisoning, with clinical outcomes ranging from mild symptoms to life-threatening complications. Accurate diagnosis and timely

intervention are critical, as misdiagnosis can delay appropriate treatment and exacerbate systemic toxicity. The lack of specific antidotes for GlySH poisoning necessitates tailored supportive care based on individual presentations. These cases underscore the importance of a thorough patient history, precise clinical assessment, and early management to improve outcomes and prevent complications. Strengthening diagnostic guidelines and raising awareness about GlySH toxicity can further enhance patient care in agricultural poisoning scenarios.

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