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Herbicide poisoning causing multiple colonic perforations: case report

Tesfaye Birhanu Abebe[1,2*], Daba Iticha Ayana[1*], Dereje Gebisa Bedada[1] and Eyob Girum Weldeyes[1]

Abstract

Introduction Herbicide poisoning, particularly from 2,4-dichlorophenoxyacetic acid (2,4-D), is an uncommon yet significant cause of systemic toxicity, especially in agricultural regions such as Ethiopia. Patients exposed to 2,4-D frequently exhibit symptoms associated with gastrointestinal, muscular, renal, and neurological toxicity. In this report, we describe a case of 2,4-D ingestion that resulted in systemic complications and the exceptionally rare occurrence of multiple colonic perforations.

Case presentation A 19-year-old male farmer presented with a loss of consciousness three hours after attempting suicide by ingesting 25 ml of 72% 2,4-D solution. This was preceded by vomiting and epigastric pain. The patient was treated for hypovolemic shock, aspiration pneumonia, and coma secondary to herbicide poisoning. Despite initial stabilization and recovery, the patient developed signs of peritonitis 14 days post-ingestion, which led to the discovery of multiple colonic perforations. Surgical intervention included total colectomy with ileostomy, which was later reversed. This case is notable as the first documented instance of multiple colonic perforations due to 2,4-D poisoning in humans, as confirmed through an extensive literature review.

Conclusion This case highlights the importance of vigilance in patients presenting with herbicide poisoning, as early detection and prompt management are essential for improving outcomes in individuals with multiple colonic perforations. Raising community awareness and establishing guidelines for the safe use of herbicides are vital to preventing such incidents.

Keywords 2,4-Dichlorophenoxyacetic acid, Colonic perforation, Herbicide poisoning, Surgery, Case report

Introduction

Herbicides, commonly known as weed killers, are utilized during the planting season to eliminate unwanted vegetation in agricultural environments [1]. Among these substances, 2,4-dichlorophenoxyacetic acid (2,4-D) is frequently available in the region and may be ingested either intentionally or accidentally [2]. Despite its widespread use and rarity of poisoning incidents, the ingestion of 2,4-D can lead to significant systemic toxicity, particularly affecting neuronal function, gastrointestinal tissues, and renal systems. Initial gastrointestinal symptoms of toxicity may manifest as vomiting and abdominal pain, whereas neurological effects can range from confusion

*Correspondence:
Tesfaye Birhanu Abebe
tesfu13b21@gmail.com
Daba Iticha Ayana
dabaiticha45@gmail.com
[1]Department of Surgery, School of medicine, collage of health sciences, Salale University, P.O.BOX: 245, Fiche, Ethiopia
[2]Department of internal medicine, School of medicine, collage of medicine and health sciences, University of Gondar, Gondar, Ethiopia

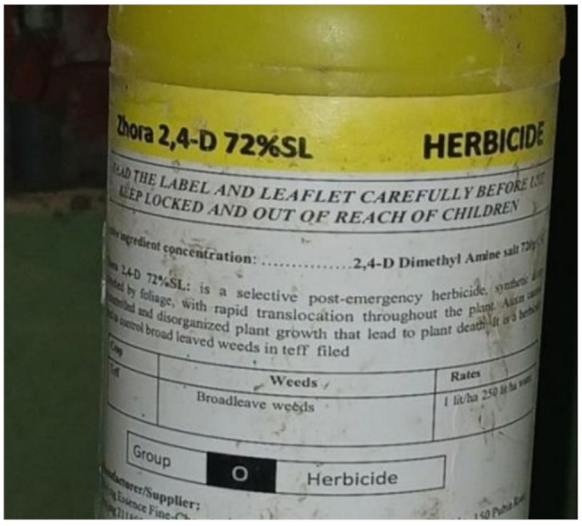


Fig. 1 Image of the herbicide bottle brought in with the patient

to coma, indicating alterations in mental status [3]. The hypotension observed in affected individuals is primarily due to intravascular volume depletion, with additional contributing factors, including vasodilation and direct myocardial toxicity [4]. The proposed mechanisms for the toxic effects of 2,4-D include dose-dependent damage to cellular membranes, uncoupling of oxidative phosphorylation, and disruption of acetyl coenzyme A metabolism [5, 6]. Although there are documented cases of 2,4-D ingestion resulting in various systemic complications and fatalities, there have been no prior reports of multiple colonic perforations associated with this compound, making this case exceptionally rare. Furthermore, this case report is the first of its kind and is anticipated to significantly contribute to the fields of toxicology, clinical practice, and public health.

Case presentation

A 19-year-old male farmer presented with herbicide poisoning three hours after quarreling with his family and attempting suicide by ingesting approximately 25 ml (72%) of 2,4-D herbicide. His family observed him fleeing with the herbicide bottle, trying to avoid intervention. They later discovered that he had experienced multiple episodes of vomiting and complained of epigastric abdominal pain, which was followed by a loss of consciousness within an hour of ingestion. He had no known history of psychiatric illness, previous suicide attempts, depressive episodes, or substance abuse. The patient also had no history of cardiac, renal, or metabolic disorders or any prior surgical or medical history related to the abdomen. His family took him to a local health center, where gastric lavage was performed, and fluid resuscitation was initiated. He was subsequently referred to a tertiary hospital. Upon arrival, with the herbicide bottle in hand (Fig. 1), he was acutely ill and in respiratory distress. His vital signs were as follows: blood pressure (BP) was 82/43 mmHg (hypotensive), pulse rate (PR) was 136 beats per minute (tachycardic), oxygen saturation (SpO2) was 88% on room air (hypoxic), respiratory rate (RR) was 28 breaths per minute (tachypneic), and temperature (T) was 37.9 °C (febrile). Neurological examination revealed a Glasgow Coma Scale score of 6/15 (eye = 1, verbal = 2, motor = 3), mid-sized but non-reactive pupils, hypertonic and hyperreflexic lower extremities, no motor preferences, and an upgoing plantar reflex. Other systemic evaluations revealed no abnormal findings. Upon initial investigation, blood tests revealed normal random blood sugar levels, a left shift without leukocytosis on the complete blood count (CBC), mild hypokalemia, elevated creatinine levels, and increased liver enzymes. Urinalysis revealed 3 + hemoglobin and many red blood cells. The electrocardiogram revealed regular narrow complex tachycardia (sinus) (Fig. 2). Owing to resource

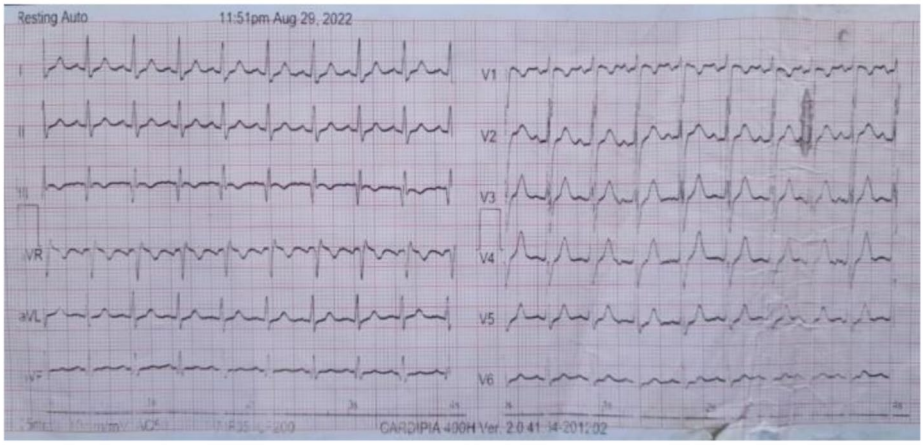


Fig. 2 electrocardiogram showed sinus tachycardia

Table 1 Summary of the investigation (H = High, n = normal, L = Low)

Parameter (Reference range)	1st day of admission	3rd day of admission	10th day of admission	14th day of admission	16th day of admission	18th day of admission	At time of ileostomy reversal
Random blood sugar(70-140 mg/dL)	88–174(N)	106–149(N)	82–110(N)	73–117(N)	76–116(N)	73–109(N)	81–108(N)
Complete blood count							
WBC (3.0–11.0(10^6/ul))	9.20(N)	4.73(N)	10.8(N)	15.64(H)	11.4(N)	10.7(N)	9.87(N)
Neutrophil%(40–75)	85.2(H)	51.4(N)	72.5(N)	93.7(H)	89.1(H)	74.6(N)	70.7(N)
Lymphocyte%(20–50)	11.2(L)	39.1(N)	25.6(N)	3.9(L)	8.5(L)	21.5(N)	28.2(N)
Hemoglobin(12–21 g/dl)	15.6(N)	13.4(N)	10.89(L)	8.5(L)	9.9(L)	11.2(L)	14.7(N)
Hematocrit (36.0–63.0%)	45.0(N)	40.6(N)	32.9(N)	25.3(L)	29.8(L)	33.2(L)	44.6(N)
Platelet (140–460(10^3/ul))	105(L)	149(N)	128(L)	96(L)	126(L)	134(L)	252(N)
Renal function test							
Cr (0.6–1.2 mg/dL)	2.81(H)	1.23(H)	1.18(N)	1.37(H)	0.86(N)	0.78(N)	0.80(N)
Urea(7-30 mg/dL)	42.6(H)	31.3(H)	33.3(H)	28.6(N)	21.2(N)	21.9(N)	18.8(N)
Liver function test							
Aspartate Aminotransferase (0–40 u/l)	245(H)	233.1(H)	82.4(H)	62.3(H)	26.2(N)	28.4(N)	32.6(N)
Alanine Aminotransferase (0–41 u/l)	160.8(H)	107(H)	61.7(H)	39.0(N)	33.0(N)	31.2(N)	33.4(N)
Serum electrolyte (mmol/L)							
Sodium (136–145)	141(N)	138(N)	132.2(L)	139.5(N)	140.2(N)	139.9(N)	141.3(N)
Potassium (3.5–5.1)	3.04(L)	3.90(N)	3.12(L)	3.53(N)	3.70(N)	3.63(N)	4.25(N)
Calcium (2.15–2.5)	1.75(L)	2.08(L)	2.19(N)	2.21(N)	2.43(N)	2.39(N)	2.2(N)
Magnesium (0.6–1.1)	0.78(N)	0.72(N)	0.8(N)	0.87(N)	0.74(N)	0.89(N)	0.93(N)
Chlorine (98–107)	115.4(H)	102.9(N)	107(N)	103.3(N)	100.2(N)	104(N)	101(N)

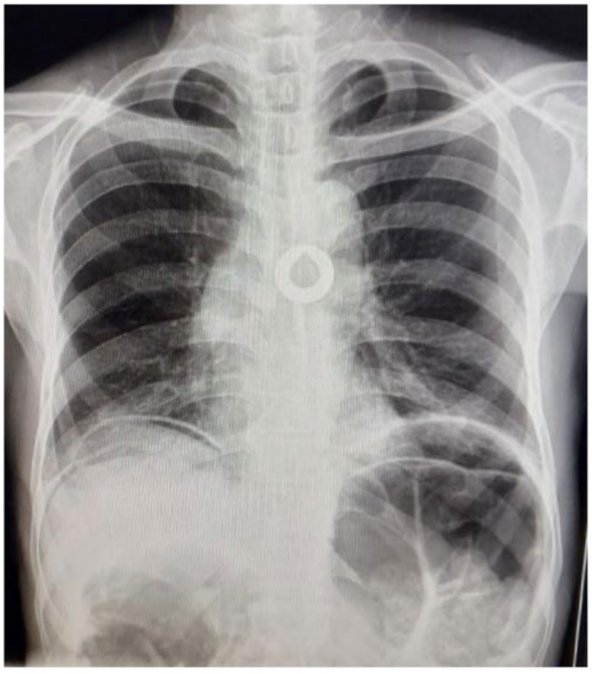
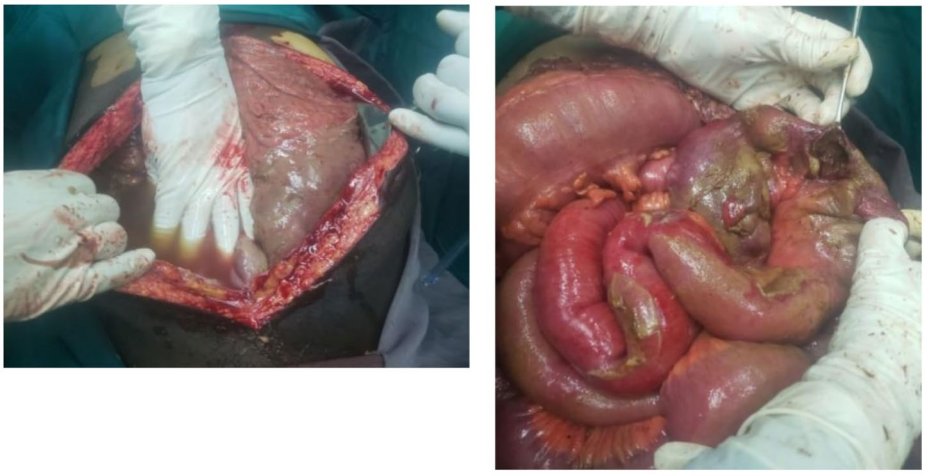


Fig. 3 Plain abdominal X-ray showed air under the diaphragm

limitations, arterial blood gas analysis, creatine phosphokinase levels, and serum toxin levels could not be determined. The patient was diagnosed with hypovolemic shock secondary to gastrointestinal loss, coma due to herbicide poisoning, acute kidney injury, and acute hepatitis.

He was catheterized, and his urine output, which was initially oliguric, improved postresuscitation. Resuscitation with three liters of normal saline corrected the shock. Forced alkaline diuresis was initiated, and an oral airway was secured. He was placed on oxygen support before being transferred to the intensive care unit (ICU). There, he was intubated for airway protection and received coma care according to the protocol. During his ICU stay, his condition improved significantly, with notable recovery from acute kidney injury and acute hepatitis, as indicated by laboratory results (Table 1). He regained consciousness on the tenth day, was extubated on the eleventh day, and continued to be monitored for further recovery.

Starting on the 10th day of ICU admission, the patient experienced abdominal pain and mild distension, leading to a diagnosis of ileus. Despite medical management for mild hypokalemia, there was no improvement. By the 14th day after admission, his abdomen was rigid and tender, he had bloody stools, and he was febrile. A plain abdominal X-ray revealed air under the diaphragm (Fig. 3), and a complete blood count (CBC) indicated marked leukocytosis with a neutrophil shift. With a clinical impression of generalized peritonitis secondary to a perforated viscus due to herbicide poisoning, the patient was taken to the operating room for an emergency exploratory laparotomy under general anesthesia.



(A): Approximately 1000 ml of gastrointestinal content mixed fluid in the peritoneal cavity

(B): A 4×4 cm perforation in the distal sigmoid colon

Fig. 4 Intraoperative photograph showing gastrointestinal contents in the peritoneal cavity **(A)** and distal sigmoid colon perforation **(B)**

Intraoperative findings included approximately 1000 ml of gastrointestinal fluid mixed within the peritoneal cavity (Fig. 4), along with multiple colonic perforations, specifically, a 2 × 3 cm perforation at the cecal base, several small perforations at the splenic flexure, a 2 × 1 cm perforation in the proximal descending colon, and a 4 × 4 cm perforation in the distal sigmoid colon (Fig. 4). The bowel exhibited signs of edema, whereas other segments appeared normal. A total colectomy was performed, with the ileum exteriorized as an end ileostomy. The peritoneal cavity was meticulously irrigated with warm saline, a drain was placed in the left paracolic gutter, and the abdominal wall was closed in layers via retention sutures.

Postoperatively, the patient was transferred to the intensive care unit (ICU), where he required vasopressors and blood transfusions due to septic shock. The patient was administered 1 g of ceftriaxone IV twice daily, renal-adjusted vancomycin, 500 mg of metronidazole IV three times daily, and 40 mg of omeprazole IV twice daily. ICU monitoring was performed in accordance with established protocols. The patient demonstrated significant improvement and was moved to the surgical ward on the seventh postoperative day for nutritional rehabilitation and stoma care, ultimately being discharged on the twelfth postoperative day. Follow-up appointments were scheduled monthly at the Surgical Referral Clinic (SRC). Eight months post-surgery, the ileostomy was reversed through laparotomy, adhesiolysis, and ileorectal anastomosis. Intraoperative findings indicated extensive interloop adhesions, adhesions between the bowel and abdominal wall, and a deeply positioned distal stump. The patient's postoperative recovery was uneventful, and the patient was discharged on the seventh postoperative day and experienced no complications during the subsequent two years of follow-up.

Patient investigations revealed significant fluctuations in key parameters over the course of admission. The white blood cell (WBC) counts were initially normal (9.20 × 10^6/μL) but spiked on the 14th day (15.64 × 10^6/μL) before stabilizing (10.7 × 10^6/μL). Creatinine levels were high at admission (2.81 mg/dL) but normalized by the 10th day (1.18 mg/dL) and remained stable (0.80 mg/dL). Liver function tests initially revealed elevated Aspartate Aminotransferase (245 U/L) and Alanine Aminotransferase (160.8 U/L) levels, which gradually returned to normal by the 16th day (AST: 26.2 U/L, ALT: 33.0 U/L).

Discussion

2,4-Dichlorophenoxyacetic acid (2,4-D) is available in various formulations, including esters, acids, and salts [4]. Zhora 2,4-D 72% SL is a selective herbicide that effectively targets broadleaf weeds in both agricultural and non-agricultural settings. The designation "SL" refers to a soluble liquid formulation that contains 72% of the active ingredient 2,4-D. Human exposure to 2,4-D primarily occurs through dermal contact, inhalation, and oral ingestion [7]. Following oral intake, 2,4-D is rapidly absorbed in the gastrointestinal tract, with peak plasma concentrations observed between 10 min and 24 h, depending on the dosage and chemical form [8]. In individuals with occupational exposure, dermal absorption is rapid, with detectable levels of 2,4-D in urine within four hours [9]. Owing to its high solubility in water,

2,4-D is extensively distributed throughout the body; however, it does not accumulate and has a limited ability to cross lipid membranes, predominantly existing in an ionized state at physiological pH [10]. Active transport mechanisms facilitate its uptake into tissues and its passage across the blood–brain barrier [11]. 2,4-D is known to induce both necrosis and apoptosis, with oxidative stress potentially mediating the apoptotic process [12]. This oxidative imbalance can lead to the release of cytochrome c from the mitochondria, resulting in the activation of caspase-3. Mitochondria are integral to both apoptotic and necrotic pathways, with intracellular levels of ATP and glutathione (GSH) influencing the mode of cell death [13]. Consequently, the cytotoxic effects of 2,4-D may determine whether necrosis or apoptosis is induced. Notable adverse effects include significant muscle damage and ventricular fibrillation, with urinary excretion serving as the primary elimination pathway. Toxicity increases at doses that exceed the renal anion transport capacity [14, 15].

The ingestion of 2,4-D is associated with a wide range of symptoms that affect multiple organ systems, including the cardiovascular, respiratory, gastrointestinal, muscular, endocrine, and nervous systems [6]. Its corrosive properties can lead to gastrointestinal distress, which may manifest as nausea, vomiting, abdominal pain, throat discomfort, and diarrhea; in some cases, gastrointestinal hemorrhage has been reported [4, 16]. By inhibiting voltage-gated chloride channels in skeletal muscle, 2,4-D induces myotoxicity, resulting in muscle spasms, weakness, and rhabdomyolysis, as indicated by elevated creatine phosphokinase levels [16]. Direct toxicity to the myocardium can lead to tachycardia and hypotension, whereas central nervous system effects may include hallucinations, ataxia, miosis, fasciculation, paralysis, convulsions, and coma [4]. Additionally, cases of hepatitis and renal failure have been documented, with some patients experiencing respiratory failure due to central nervous system depression and respiratory muscle weakness [14]. Severe toxicity is characterized by extensive rhabdomyolysis, metabolic acidosis, respiratory failure, refractory hypotension, and coma [6]. Although organophosphates and various herbicides, including diquat, paraquat, and glyphosate, have been linked to intestinal perforation due to oxidative stress in both human and animal studies [17–19], our literature review did not reveal any documented cases of intestinal perforation attributable to 2,4-D poisoning. Furthermore, perforation of the large intestine following herbicide ingestion is exceedingly rare, as existing case reports primarily describe hemorrhagic events in the esophagus and stomach during the acute phase of exposure [20]. The mechanisms proposed to explain colonic perforations associated with 2,4-D herbicides remain somewhat ambiguous; however, they may involve direct damage to the mucosal lining, ischemic injury, and secondary infections arising from necrosis and oxidative stress [21]. These factors can adversely affect cellular membranes, disrupt oxidative phosphorylation, and interfere with acetyl coenzyme A metabolism, potentially leading to colonic perforation. Furthermore, research conducted by Nanni et al. indicates that 2,4-dichlorophenoxyacetic acid (2,4-D) induces neurotoxic alterations in the myenteric neurons of Rattus norvegicus [22]. The study reported an increase in overall neuron density and a stabilization of nitrergic neuronal density, which may be associated with elevated acetylcholine levels that stimulate intestinal smooth muscle, potentially facilitating the expulsion of harmful substances from the colon. In the case of the patient who developed colonic perforation following two weeks of 2,4-D ingestion, these findings may support the hypothesis that neurotoxic effects within the myenteric plexus could contribute to this complication.

Upon initial assessment and while administering supportive care, it is crucial to perform several laboratory test to guide clinical management. These tests should include a complete blood count, renal function tests, liver function tests, serum electrolyte levels, arterial blood gas analysis, toxicology screening for herbicide serum levels, creatine phosphokinase levels, urinalysis, random blood glucose measurement, and stool examination [6]. Analytical quantification of the herbicide can be achieved through acid extraction followed by gas chromatography with electron capture detection [23]. However, owing to resource constraints, we were unable to perform arterial blood gas analysis to evaluate potential acidosis, measure serum levels of 2,4-D, or assess creatine phosphokinase levels, which may be elevated as a result of toxin-induced rhabdomyolysis.

The management of 2,4-D poisoning is primarily supportive, as no specific antidote is available. Immediate supportive measures should include airway management and, if necessary, ventilatory support. Decontamination procedures, such as the administration of activated charcoal and sorbitol, as well as gastric lavage following significant ingestion, should be performed under appropriate circumstances [24]. Routine resuscitation with crystalloid fluids is critical, and vasopressors may be required if hypotension persists despite fluid resuscitation. It is also essential to address electrolyte imbalances and acidosis; urinary alkalinization has been shown to be beneficial in severe cases of 2,4-D poisoning by facilitating the ionization of phenoxy acids, thereby reducing renal reabsorption and enhancing elimination [25].

Clinicians must remain vigilant for the complication of colonic perforation, particularly in patients exhibiting signs of peritonitis, such as severe abdominal pain, fever, nausea, vomiting, and an inability to pass stool or gas.

Imaging modalities, including plain abdominal X-ray, ultrasound, and computed tomography (CT) scan are essential for detecting and assessing the extent of perforation. Upon confirmation of colonic perforation, immediate surgical intervention is necessary. An aggressive surgical approach is generally recommended for patients with large bowel perforation, as mortality and morbidity are closely associated with the severity of intraperitoneal infection [26]. The management of colonic perforation can be guided by the severity of peritonitis, which can be evaluated via the Hinchey classification, the Mannheim peritonitis index (MPI), and the Acute Physiology and Chronic Health Evaluation (APACHE) II score [27]. Surgical options may include primary resection and anastomosis, the Hartmann procedure, or simple colostomy. In this particular case, total colectomy with ileostomy was performed due to multiple perforations extending from the cecal base to the distal sigmoid colon, along with extensive contamination and necrotic colonic tissue. Following recovery, the ileostomy was reversed, and the distal part of the ileum was connected to the rectum. Despite the rarity of this case, ileorectal anastomosis is also indicated for other causes of spontaneous colonic perforation, such as familial adenomatous polyposis, Crohn's disease, functional bowel disorders, ulcerative colitis, and colonic carcinoma [28].

Conclusion

2,4-Dichlorophenoxyacetic acid is a herbicide known for its high toxicity, and this case underscores the importance of promptly identifying and managing complications related to herbicide poisoning. Healthcare providers should be particularly vigilant for gastrointestinal perforations in patients who exhibit severe abdominal symptoms after ingesting 2,4-D. Increasing public awareness regarding the risks associated with herbicides and creating guidelines for their safe application to help prevent these occurrences are crucial.

Patient perspective

As a 19-year-old farmer, I made the regrettable decision to ingest 2,4-D herbicide following a heated argument with my father. The immediate symptoms were severe: vomiting, abdominal pain, and loss of consciousness. My family found me and rushed me to a local health center, and subsequently to a larger hospital. I regained consciousness in the hospital after several days, with machines attached to me. I recall beginning to experience abdominal discomfort, which escalated into severe pain within days, necessitating a nerve-wracking trip to the operating room. For approximately eight months, I had to use a fecal diversion due to my abdominal condition, but eventually, it was reversed, providing me relief from this traumatic experience. This incident was life-changing and taught me the importance of seeking help during difficult times. I hope my story raises awareness about the dangers of herbicide poisoning and encourages others to seek support before resorting to drastic actions.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12245-025-00875-7.

Supplementary Material 1

Supplementary Material 2

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Author contributions

TBA conceptualized, wrote original draft of the manuscript, reviewed, edited, and visualized the final manuscript. DIA managed the patient, conceptualized, wrote original draft of the case presentation, assisted in manuscript revision and data curation. DGB reviewed and edited the manuscript, and supervised. EGW assisted in manuscript revision and data curation. All authors read and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The patient has provided written informed consent for the publication of their case details and any related images. Approval for this case report was not required to secure from the Ethics Committee of the institution.

Consent for publication

Written informed consent was obtained from the patient for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Competing interests

The authors declare no competing interests.

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