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CASE REPORT

A Case of Mixed Herbicide Poisoning Presenting with Diverse Findings: Diagnostic Pitfalls and the Importance of Psychosocial Context

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Abstract

***Background*:** Although glyphosate-based herbicides are generally considered to have low acute toxicity to humans, commercially available formulations may contain multiple active ingredients and co-formulants that produce atypical clinical manifestations.
***Case Presentation*:** A 53-year-old woman with a history of panic disorder and kleptomania was found collapsed at home by her family just before incarceration. On arrival, she had marked bilateral mydriasis (8 mm), impaired consciousness (Glasgow Coma Scale: E1V1M1), QTc prolongation (633 ms), metabolic acidosis, and elevated lactate. Stimulant intoxication was initially considered because of mydriasis and a positive urine amphetamine screen; however, she lacked hypertension, tachycardia, diaphoresis, and agitation, and QT prolongation was not interpreted as evidence of adrenergic stimulation. Detailed history-taking and UPLC-HRMS toxicological analysis revealed ingestion of a commercial herbicide containing glyphosate isopropylamine salt, flumioxazin, and MCPA, with MCPA detected in serum. She improved with conservative treatment and was discharged on hospital day 8.
***Discussion*:** This case illustrates atypical toxicosis caused by a multi-ingredient herbicide. The presentation did not fulfill a classic sympathomimetic toxidrome; instead, mydriasis, impaired consciousness, QT prolongation, and ileus required broad differential diagnosis. Component-specific assessment suggested that glyphosate-surfactant exposure, MCPA toxicity, and uncertain co-formulant effects together contributed to the clinical picture. Quantitative pupillometry and confirmatory toxicological analysis helped avoid diagnostic anchoring.
***Conclusion*:** Commercial herbicide poisoning should not automatically be interpreted as single-agent glyphosate poisoning. Separating key clinical problems from formulation-specific toxicities may improve emergency diagnosis and management.

Keywords: Glyphosate poisoning, Toxidromes, Mydriasis, Herbicide, Diagnostic errors

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INTRODUCTION

Glyphosate-based herbicides are widely used for agricultural and household purposes and are generally considered to have low acute toxicity in humans [1]. However, intentional ingestion has been linked to severe toxicity and fatal outcomes [2]. Commercial herbicide products often contain not only glyphosate but also various other toxic compounds, including surfactants and active ingredients [3]. These co-formulants may contribute to a broad range of toxic effects, leading to atypical clinical presentations that do not fit well into traditional toxidrome categories.

In particular, symptoms such as bilateral mydriasis and altered consciousness may mimic sympathomimetic or anticholinergic poisoning, potentially causing diagnostic confusion [4]. Additionally, false-positive results in urine drug screening tests—especially for amphetamines—can mislead clinicians and delay proper management [5].

Here, we report a case of intentional ingestion of a commercial herbicide containing glyphosate isopropylamine salt, flumioxazin, and 2-methyl-4-chlorophenoxyacetic acid (MCPA) isopropylamine salt hydrate. The aim of this case report is to describe how mydriasis, impaired consciousness, and QT prolongation

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after mixed herbicide ingestion can be misinterpreted as stimulant intoxication, and to emphasize a differential diagnostic approach based on clinical discordance, product identification, quantitative pupillometry, and confirmatory toxicological analysis.

CASE PRESENTATION

A 53-year-old woman with a longstanding history of panic disorder and kleptomania had been receiving outpatient psychiatric treatment for several years. She had been sentenced to imprisonment due to repeated shoplifting episodes and was scheduled to be incarcerated five days after presentation. Her prescribed medications included levothyroxine, sertraline, alprazolam, brotizolam, clotiazepam, and fexofenadine. She had no known history of substance abuse, family discord, or suicidal behavior. On the day of admission, she was found collapsed at home by a family member, who called emergency medical services. Upon arrival, her Glasgow Coma Scale was E1V1M1, with a body temperature of 34.3°C, blood pressure of 106/78 mmHg, heart rate of 76 beats per minute, respiratory rate of 28 breaths per minute, and oxygen saturation of 98% on room air. Marked bilateral mydriasis (8 mm) with sluggish light reflexes was observed (Figure 1).

Figure 1. Quantitative pupillometry on admission
Both pupils were significantly dilated. Neurological Pupil Index (NPi) scores were lower in both eyes, especially in the left eye. Pupillary constriction speed and percentage were reduced in comparison to normal reference values.

During emergency department observation, her consciousness gradually improved to a level of GCS E3V4M6. At this point, an abdominal exam revealed significant tenderness around the umbilicus. Head computed tomography (CT) showed no evidence of acute cerebrovascular events. Abdominal and pelvic CT scans demonstrated diffuse fluid retention in the small intestine, suggestive of paralytic ileus (Figure 2). Electrocardiography showed a significantly prolonged corrected QT interval (QTc 633 ms). Laboratory findings included leukocytosis (WBC 19,400/μL), elevated lactate (4.87 mmol/L), and

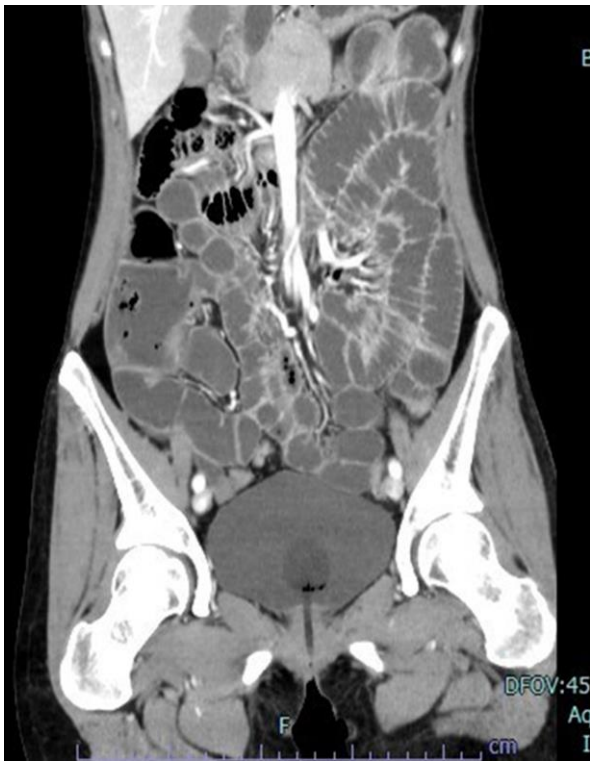

Figure 2. Contrast-enhanced abdominal computed tomography
Significant fluid retention and dilation of the small intestine were observed. A small amount of ascites was also present, but no specific site of intestinal obstruction was identified.

metabolic acidosis (pH 7.269, base excess −7.5 mmol/L). Urinalysis showed 2+ protein, 2+ glucose, and a trace of hematuria (Figure 3). A urine drug screening test returned a positive result for amphetamines, raising initial suspicion for a sympathomimetic overdose. However, the absence of hypertension, tachycardia, or agitation prompted reconsideration, and the result was later confirmed to be a false positive. Given the atypical toxidrome—including altered consciousness, mydriasis, QT prolongation, and metabolic acidosis—acute poisoning was suspected. Gastric lavage and activated charcoal administration were performed as initial decontamination. After regaining consciousness, the patient disclosed that she had ingested approximately 250 mL of a commercially available herbicide in a suicide attempt. The product contained glyphosate isopropylamine salt, flumioxazin, and MCPA isopropylamine salt hydrate. Ultra-Performance Liquid Chromatography–High-Resolution Mass Spectrometry (UPLC-HRMS) confirmed the presence of MCPA in her blood, supporting the diagnosis. On hospital day 2, urinary output was maintained, and acidosis improved with fluid therapy alone. Psychiatric consultation revealed marked depressive symptoms, including profound guilt and suicidal ideation. The patient was diagnosed with a depressive episode. The psychiatric team noted that her long-standing

kleptomania, legal stress, and social isolation contributed significantly to her suicidal behavior. On hospital day 5, esophagogastroduodenoscopy was performed to evaluate potential mucosal injury from herbicide ingestion, prompted by the presence of dark-colored vomitus. The findings included erosive esophagitis and chronic gastritis, with no evidence of corrosive injury or active bleeding. Oral intake

Parameter	Result	Reference range	Unit
Hematology			
WBC	19,400	3,300–9,000	/μL
Hb	16.4	13.5–17.5	g/dL
Platelet	310,000	140,000–340,000	/μL
Biochemistry			
BUN	16.6	8.0–20.0	mg/dL
Creatinine	0.89	0.47–0.79	mg/dL
AST	37	10–40	IU/L
ALT	14	5–45	IU/L
Lactate	4.87	<1.3	mmol/L
Sodium	146	137–147	mEq/L
Potassium	4.1	3.5–5.0	mEq/L
Glucose	151	70–109	mg/dL
CRP	0.04	<0.3	mg/dL
Venous blood gas analysis			
pH	7.269	7.35–7.45	–
pCO_2	42.7	35–45	mmHg
HCO_3^-	19.1	23–28	mmol/L
BE	−7.5	−2.2–+1.2	mmol/L
Anion gap	32.3	10–14	mEq/L
Urinalysis			
Protein	2+	Negative	–
Glucose	2+	Negative	–
Occult blood	±	Negative	–
pH	6.0	5.0–8.0	–
Specific gravity	1.019	1.010–1.025	–

Figure 3. Laboratory results upon admission
BUN: blood urea nitrogen, AST: aspartate aminotransferase, ALT: alanine aminotransferase, CRP: C-reactive protein, WBC: white blood cell, Hb: hemoglobin, BE: base excess

was resumed following endoscopy. Her cognitive function and activities of daily living remained intact. She was discharged home on day 8 in stable condition, with continued psychiatric outpatient follow-up arranged through her usual clinic.

DISCUSSION

This patient presented with impaired consciousness, marked bilateral mydriasis, QT prolongation, metabolic acidosis, and ileus after ingesting a commercial herbicide formulation. These findings should be interpreted as separate diagnostic problems rather than as a single sympathomimetic toxidrome. Bilateral mydriasis with impaired consciousness suggests a broad differential diagnosis, including intracranial lesions, hypoxic-ischemic injury, anticholinergic poisoning, sympathomimetic poisoning, serotonergic toxicity, sedative or hypnotic co-exposure, and mixed toxic ingestion [6-10]. QT prolongation is also non-specific and may occur with various toxicological and non-toxicological conditions, including antiarrhythmic agents, psychotropic medications, electrolyte disturbances, hypothermia, and metabolic derangements. Therefore, in this case, QTc prolongation was considered a finding requiring cardiac monitoring and broad toxicological evaluation, rather than evidence of adrenergic stimulation.

The initial consideration of sympathomimetic poisoning was understandable because the patient had marked mydriasis and a positive urine amphetamine screen. However, the overall clinical picture was inconsistent with a classic sympathomimetic toxidrome. She had no hypertension, tachycardia, diaphoresis, hyperthermia, agitation, or hyperreflexia; instead, she was centrally depressed and nearly comatose. The positive urine immunoassay was therefore interpreted as a possible false positive rather than definitive evidence of amphetamine poisoning [5]. This discrepancy emphasizes the need to reassess an initial diagnostic impression when toxidrome pattern recognition and rapid screening tests are inconsistent with vital signs and mental status.

The clinical manifestations should also be considered according to the individual components of the herbicide formulation. Glyphosate-containing products may cause gastrointestinal irritation, mucosal injury, metabolic acidosis, hypotension, renal dysfunction, respiratory failure, and death after large intentional ingestions; surfactants and other co-formulants may substantially contribute to toxicity [1-4]. MCPA is a chlorophenoxy herbicide. In a prospective case series of MCPA self-poisoning, most patients had mild toxicity, but severe manifestations including coma, rhabdomyolysis, renal dysfunction, cardiorespiratory arrest, and death were reported [11]. These reported features are compatible with the impaired consciousness, metabolic acidosis, and need for supportive care observed in the present case. In contrast, human data on acute flumioxazin ingestion are limited; therefore, its contribution remains uncertain. Taken together, we interpret this case as mixed herbicide toxicity rather than isolated glyphosate poisoning or a true sympathomimetic toxidrome.

Quantitative pupillometry was useful because it objectively documented severe bilateral mydriasis and impaired light reflexes, findings that are difficult to assess reproducibly by manual examination. Previous reports suggest that automated pupillometry can support the assessment of poisoning and critical illness [12-15]. In this case, pupillometry did not identify the causative agent by itself, but it helped define the neuro-autonomic abnormality and supported reconsideration of the differential diagnosis when the vital signs did not fit stimulant poisoning.

This case has several limitations. Serum glyphosate, flumioxazin, surfactant concentrations, and serial MCPA concentrations were not measured; therefore, the relative contribution of each compound cannot be determined. In addition, the positive urine amphetamine screen could not

be attributed to a specific cross-reacting substance. Nevertheless, the confirmed ingestion history, product information, detection of MCPA by UPLC-HRMS, and clinical course provide credible evidence supporting mixed herbicide poisoning.

This case underscores the risk of relying solely on a single toxidrome label or a rapid urine drug screening result. In commercial herbicide poisoning, clinicians should first analyze the differential diagnosis of key clinical findings such as mydriasis, impaired consciousness, and QT prolongation, and then consider the expected toxicity of each formulation component. Product identification, systematic evaluation of altered mental status, objective pupillary assessment, and confirmatory toxicological testing may improve diagnostic accuracy in atypical herbicide poisoning.

CONCLUSION

This case describes mixed commercial herbicide poisoning presenting with impaired consciousness, marked bilateral mydriasis, QT prolongation, metabolic acidosis, and ileus. Although stimulant poisoning was initially considered, the absence of typical sympathomimetic features and the detection of MCPA supported mixed herbicide toxicity rather than a classic sympathomimetic toxidrome. Commercial herbicide poisoning should be approached by separating the differential diagnosis of key clinical findings from the expected toxicity of each formulation component. Careful product identification, avoidance of anchoring on rapid urine drug screening results, quantitative pupillometry, and confirmatory toxicological analysis may improve diagnosis and management.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Declaration

Use of Artificial Intelligence: ChatGPT (OpenAI, GPT-5) was used to assist with English language editing and phrasing. The authors reviewed and verified all outputs, and no AI tools were used to generate data, analysis, or interpretations.

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