Not all herbicides are Paraquat: pitfalls of class-based management in 2,4-D poisoning

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Dear Editor,

In developing countries, poisoning with agrochemicals remains an important public health problem [1]. Clinicians often follow "class-wide" management protocols in acute agricultural poisonings, similar to organophosphate (OP) toxicity. Because OPs commonly produce a cholinergic toxidrome, clinicians can initiate antidote therapy purely based on clinical presentation before knowing the exact compound [2].

Unlike OPs, herbicides lack a universal toxidrome or shared antidote. Therefore, applying a class-based approach to herbicide poisoning may result in delayed diagnosis and inappropriate treatment.

Acute liquid herbicide poisonings in agricultural settings are frequently assumed to be paraquat due to its historical prevalence and notorious mortality rate [3]. Managing an unconfirmed herbicide exposure as "presumptive paraquat" creates delay in appropriate treatment when the actual culprit is 2,4-dichlorophenoxyacetic acid (2,4-D). Although both may initially present with gastrointestinal symptoms, mucosal irritation, altered sensorium, renal dysfunction or metabolic acidosis, their management differs substantially [4].

Paraquat toxicity primarily results from redox cycling with the generation of reactive oxygen species, leading to diffuse alveolitis and progressive pulmonary fibrosis. Treatment involves early gastrointestinal decontamination, when appropriate, with activated charcoal or Fuller's earth. Early hemoperfusion, antioxidant, and immunosuppressive therapies have been used with variable evidence and unnecessary hyperoxia should be avoided because excessive oxygen may worsen oxidative lung injury [3,4].

In contrast, 2,4-D poisoning is characterized by mitochondrial dysfunction, skeletal muscle toxicity, rhabdomyolysis, neurological manifestations, cardiovascular instability, and acute kidney injury. As a weak acid, 2,4-D may offer a therapeutic advantage through urinary alkalinization, which may enhance its renal elimination by ion trapping and reducing tubular reabsorption [1,2]. This intervention has no established role in paraquat poisoning as renal clearance is ineffective once tubular necrosis sets in [3]. Early recognition of 2,4-D poisoning is therefore essential because delayed diagnosis may postpone this potentially beneficial intervention.

Similarly, glyphosate formulations differ from both paraquat and 2,4-D, with toxicity caused not only by glyphosate itself but also by surfactants such as polyoxyethyleneamine (POEA). Management is mainly supportive, and prognosis may vary according to the formulation involved [5], further emphasizing that herbicides should not be considered a homogeneous toxicological class. Key differences among major herbicides are summarized in Table 1.

Recognition of the specific herbicide ingested should therefore be a priority. Unexplained severe metabolic acidosis along with neuromuscular symptoms (hypotonia, fasciculations, and altered sensorium) should raise suspicion for chlorophenoxy compounds [1]. Identify the actual product whenever possible – through history, container/label examination, poison-information resources, and toxicology consultation – rather than assuming that all herbicides require paraquat-style management.

What is new?

This letter highlights the important management pitfalls in 2,4-D poisoning and importance of compound-specific management in acute herbicide poisonings rather than managing herbicides as a homogeneous class. Also highlighting the contrasting features and distinct management strategies of common herbicides, particularly highlighting the early identification of the specific herbicide compound ingested so that interventions such as urinary alkalinization can be implemented timely.

Table 1. *Toxicological and management differences among major herbicide classes.*

CLINICAL PARAMETER	PARAQUAT (BIPYRIDYL)	2,4-D (CHLOROPHENOXY)	GLYPHOSATE FORMULATIONS
Key distinguishing features	Mucosal ulceration ("Paraquat tongue"), early acute kidney injury (AKI), progressive pulmonary fibrosis	Severe metabolic acidosis, neuromuscular signs, rhabdomyolysis	Shock, dysrhythmias, metabolic acidosis, gastrointestinal burns
Principal supportive management and selected elimination strategies	Gastrointestinal decontamination, avoid unnecessary oxygen, early hemoperfusion in selected cases	Supportive care, urinary alkalinization in significant poisoning may enhance elimination, dialysis when indicated	Supportive care, dialysis when indicated

*Sources: Table compiled by the author based on Refs. 1–5.

List of abbreviations

2,4-D 2,4-Dichlorophenoxyacetic acid
OP Organophosphate

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