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Case Report

Anuric Acute Kidney Injury Following Accidental Ingestion of a Suspected Diquat Herbicide: A Case Report

Laura A. Vicenzi Souza[1]*, Bruno Zanardo Faggion[2], Carolina Huedepohl Marques Vieira[3], Rafaella dos Anjos Neves[2], Mayara Mayer Alves[2], Bruno Coelho Siniscalchi[4], Luiz Henrique Barros Santos Pontelli[4], Maria Fernanda Barros Pontelli[4] and Letícia Buque Santos[5]

[1]Critical Care Medicine, Polydoro Ernani de São Thiago University Hospital, Florianópolis, Brazil

[2]General Surgery, Polydoro Ernani de São Thiago University Hospital, Florianópolis, Brazil

[3]Resident at Santa Catarina School of Public Health, Santa Catarina School of Public Health, Florianópolis, Brazil

[4]General Practice, Independent Physician, Florianópolis, Brazil

[5]Medical Student, University of the Region of Joinville (UNIVILLE), Joinville, Brazil

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***Corresponding author:** Laura A. Vicenzi Souza, Critical Care Medicine, Polydoro Ernani de São Thiago University Hospital, Florianópolis, Brazil

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ABSTRACT

Bipyridyl herbicide poisoning can be associated with severe acute kidney injury despite initial hemodynamic stability. A 54-year-old man with hypertension sought medical attention one day after accidentally ingesting a small sip of herbicide while attempting to take medication for pre-existing diarrhea. He presented with oliguria and developed anuria on the first hospital day. A qualitative urinary colorimetric assay reported paraquat/diquat as detected, although the available result did not distinguish the individual compound. Diquat poisoning was clinically suspected. Serum creatinine increased from 1.1 to 6.0 mg/dL. The patient remained initially alert, hemodynamically stable without vasoactive support, and breathing spontaneously on room air. Anuria persisted despite approximately 1,000 mL of intravenous crystalloid, and hemodialysis was initiated. Hemodialysis was required for approximately six weeks, after which kidney function improved sufficiently to discontinue treatment. Serum creatinine after recovery was 1.8 mg/dL. This case illustrates severe anuric kidney injury following suspected bipyridyl herbicide poisoning, with subsequent dialysis independence. Initial clinical stability should not obscure the significance of declining urine output. Concurrent gastrointestinal fluid losses may have contributed to kidney injury, and neither the specific toxic compound nor the relative contribution of individual mechanisms could be definitively established.

Keywords: Acute kidney injury; Anuria; Bipyridyl herbicides; Diquat; Herbicide poisoning

Introduction

Diquat is a nonselective bipyridyl herbicide associated with potentially severe systemic toxicity. Its toxic effects involve intracellular redox cycling and oxidative cellular injury, with manifestations that may include gastrointestinal symptoms, acute kidney injury (AKI), neurological complications, and multiorgan dysfunction. The kidneys are an important target of toxicity, and management remains largely supportive because no specific antidote has established clinical efficacy[1,2].

Clinical and histopathological reports describe acute tubular injury in patients with diquat-associated AKI, although the severity and clinical course vary[3,4]. Accurate identification of the ingested compound is important when interpreting suspected exposure, particularly when the available toxicological report does not distinguish diquat from paraquat.

We report anuric AKI requiring approximately six weeks of hemodialysis after accidental ingestion of a herbicide suspected to contain diquat. The case illustrates severe renal dysfunction despite initial hemodynamic stability, followed by recovery sufficient to discontinue dialysis.

Case Presentation

A 54-year-old man with a history of hypertension sought medical attention one day after accidentally ingesting a herbicide. According to the patient, he had intended to take medication for diarrhea but instead ingested a small sip of the product. Watery diarrhea, with multiple daily episodes, had begun before ingestion and had been present for approximately two days at presentation. He was already oliguric when he sought care and subsequently developed anuria on the first hospital day.

The patient was transferred by the Mobile Emergency Medical Service (Serviço de Atendimento Móvel de Urgência, SAMU) to a tertiary referral hospital. The regional Toxicological Information and Assistance Center (Centro de Informação e Assistência Toxicológica, CIATox) was contacted during the initial assessment, and diquat poisoning was suspected.

Urinary toxicological testing yielded a positive qualitative colorimetric screening result for paraquat/diquat, without differentiation between the compounds or quantification. A urinary immunochromatographic drug screen performed on the same date detected benzodiazepines, consistent with the reported diazepam use; all other substances included in the panel were not detected. The individual test results, reported detection limits, and interpretations are summarized in **(Table 1)**.

Table 1: Urinary toxicological test results and interpretation.

Test	Method	Result	Reference result or detection limit	Interpretation
Paraquat/diquat	Qualitative colorimetric assay	Detected	Reference result: not detected; detection limit not reported	Positive combined screening result for paraquat/diquat. The report does not distinguish between the two compounds or provide a quantitative concentration.
Methamphetamine	Immunochromatography	Not detected	300 ng/mL	Not detected at the assay's stated detection limit.
Amphetamine	Immunochromatography	Not detected	300 ng/mL	Not detected at the assay's stated detection limit.
Cannabinoids (marijuana)	Immunochromatography	Not detected	50 ng/mL	Not detected at the assay's stated detection limit.
Morphine/opioids	Immunochromatography	Not detected	300 ng/mL	Not detected at the assay's stated detection limit.
Cocaine/benzoylecgonine	Immunochromatography	Not detected	150 ng/mL	Not detected at the assay's stated detection limit.
Benzodiazepines	Immunochromatography	Detected	300 ng/mL	Consistent with the reported diazepam use; the screening result does not identify the individual benzodiazepine.
Tricyclic antidepressants	Immunochromatography	Not detected	1,000 ng/mL	Not detected at the assay's stated detection limit.
Barbiturates	Immunochromatography	Not detected	200 ng/mL	Not detected at the assay's stated detection limit.
Phencyclidine	Immunochromatography	Not detected	25 ng/mL	Not detected at the assay's stated detection limit.
3,4-Methylenedioxymethamphetamine (ecstasy)	Immunochromatography	Not detected	500 ng/mL	Not detected at the assay's stated detection limit.
Methadone	Immunochromatography	Not detected	300 ng/mL	Not detected at the assay's stated detection limit.
Propoxyphene	Immunochromatography	Not detected	300 ng/mL	Not detected at the assay's stated detection limit.

All tests were performed on urine specimens and reported qualitatively. Detection limits are reproduced from the laboratory report and do not represent measured urinary concentrations. "Not detected" indicates a negative screening result at the stated detection limit and does not establish the complete absence of the substance. The combined paraquat/diquat result does not independently confirm diquat exposure.

On admission, the patient was alert, hemodynamically stable without vasoactive support, and breathing spontaneously on room air. During hospitalization, serum creatinine increased from 1.1 to 6.0 mg/dL in association with anuria (Reference range between 0.7 and 1.3 mg/dL). The precise interval between these measurements was not specified.

During the first 24 hours in the intensive care unit, he remained hemodynamically stable and reported nausea. Anuria persisted despite intravenous administration of approximately 1,000 mL of crystalloid.

Hemodialysis was initiated because of persistent anuria in the setting of worsening kidney function.

The patient required hemodialysis for approximately six weeks. Kidney function subsequently improved sufficiently to discontinue dialysis, and serum creatinine after recovery was 1.8 mg/dL. No further hemodialysis sessions were required. The principal clinical events and available laboratory findings are summarized in **(Table 2)**.

Table 2: Clinical course and available laboratory findings.

Clinical phase	Findings and management
Before ingestion	Watery diarrhea with multiple daily episodes; symptoms had been present for approximately two days by presentation.
Day before seeking care	Accidental ingestion of a small sip of herbicide while attempting to take medication for diarrhea.
Initial presentation	Oliguria; preserved consciousness, hemodynamic stability, and spontaneous breathing on room air.
Toxicological assessment	Qualitative urinary colorimetric assay: paraquat/diquat detected. Urinary immunochromatographic drug screening: benzodiazepines detected, with reported diazepam use.
First hospital day	Development of anuria.
During hospitalization	Serum creatinine increased from 1.1 to 6.0 mg/dL; exact measurement times were not specified.
First 24 hours in intensive care	Persistent anuria despite approximately 1,000 mL of intravenous crystalloid; nausea without hemodynamic instability.
Renal support	Hemodialysis initiated for persistent anuric AKI and continued for approximately six weeks.
Renal recovery	Hemodialysis discontinued; serum creatinine of 1.8 mg/dL, with no further dialysis sessions required.

The timeline distinguishes the first hospital day from the first 24 hours in intensive care because their exact temporal correspondence was not specified. The toxicological result was qualitative and did not separately identify paraquat or diquat. AKI: acute kidney injury.

Discussion

The principal feature of this case is anuric AKI requiring prolonged but temporary hemodialysis after accidental herbicide ingestion, despite initial hemodynamic stability and preserved consciousness. The transition from oliguria at presentation to anuria on the first hospital day provides an important marker of clinical progression. Subsequent dialysis independence demonstrates meaningful renal recovery, although the creatinine concentration of 1.8 mg/dL remained above the earlier reported value of 1.1 mg/dL.

The exposure history and positive urinary toxicological assay support suspected bipyridyl herbicide poisoning. However, the report explicitly grouped paraquat and diquat within a single qualitative result. Although diquat was clinically suspected, the available laboratory documentation does not establish which compound was detected. Identification of the product and clarification of the assay's analytical characteristics would strengthen compound-specific attribution. Published reports emphasize the value of integrating the exposure history with toxicological identification when evaluating suspected diquat poisoning[5].

Direct tubular toxicity is a plausible contributor to this patient's renal dysfunction. Diquat-associated oxidative injury is described in toxicological studies, and the renal biopsy reported by Xia et al. demonstrated acute tubular injury and lipid degeneration with limited interstitial inflammation[1,3]. Clinical and pathological observations by Zeng et al. provide further evidence of renal injury following diquat poisoning[4]. In the present case, the absence of renal histopathology prevents confirmation of the underlying lesion; toxic tubular injury therefore remains a clinical interpretation.

The chronology of the gastrointestinal symptoms is particularly relevant. Diarrhea preceded ingestion, as the patient was attempting to treat an existing symptom when the accidental exposure occurred. Accordingly, the diarrhea cannot be attributed solely to the herbicide. Gastrointestinal fluid losses may have contributed to reduced renal perfusion and could have acted together with toxic injury. Hemodynamic stability at presentation does not establish normal intravascular volume, and persistent anuria after approximately 1,000 mL of crystalloid does not independently confirm adequate volume replacement or exclude a hemodynamic component.

Other potential contributors include urinary tract obstruction, medication-associated kidney injury, and pigment-associated injury. Rhabdomyolysis accompanying diquat poisoning has been reported[6], but no creatine kinase result was available for the present case. Similarly, the absence of detailed urinary sediment findings, renal imaging results, and a complete medication history limits the exclusion of alternative or concurrent mechanisms. These possibilities should be recognized without suggesting that they were demonstrated in this patient.

Hemodialysis was initiated for persistent anuria and worsening renal function. KDIGO guidance recommends that the decision to initiate renal replacement therapy incorporate the clinical context, conditions potentially modified by treatment, and laboratory trends rather than an isolated creatinine threshold[7]. The available information establishes the treating team's indication of anuric AKI but does not provide potassium, acid-base status, fluid balance, or other findings at dialysis initiation. Consequently, additional indications cannot be inferred, and the case should not be interpreted as establishing a universal creatinine threshold or a fixed fluid-response criterion for dialysis.

Renal support must also be distinguished from extracorporeal toxin removal. Earlier reports described hemoperfusion in severe diquat poisoning[8], whereas Jones and Vale found no clinical evidence that hemodialysis or hemoperfusion prevented organ failure or death through toxin elimination[2]. In this patient, hemodialysis provided support during persistent renal dysfunction. Without quantitative toxin measurements, serial concentrations, or treatment-specific clearance data, its contribution to herbicide elimination cannot be determined.

The renal outcome was favorable in terms of dialysis independence after approximately six weeks. Nevertheless, the creatinine concentration of 1.8 mg/dL does not demonstrate a return to the earlier reported concentration of 1.1 mg/dL. Because the timing and pre-exposure significance of the latter value are uncertain, the extent of residual renal impairment cannot be precisely characterized. The available follow-up also does not permit a diagnosis of chronic kidney disease. Further assessment after AKI is important to document the trajectory of recovery[7].

Published outcomes following diquat poisoning are heterogeneous. Tao et al. described three fatal cases[9], and Fan, et al. reported severe poisoning with multiorgan failure[10]. These

reports establish the potential severity of exposure but do not provide directly comparable prognostic estimates for this patient, whose ingested dose and product concentration were not established. His account of a small sip should therefore be reported as a subjective exposure estimate rather than interpreted as evidence of a low absorbed dose.

The principal limitations are uncertain compound identification, an unquantified exposure, incomplete laboratory characterization, unavailable hemodialysis session details, and limited follow-up information. Despite these limitations, the case documents progression from oliguria to anuria, the need for approximately six weeks of hemodialysis, and subsequent recovery sufficient to discontinue treatment. Its practical message is that initial clinical stability should not obscure progressive kidney injury after suspected herbicide exposure. Serial assessment of urine output and renal function, evaluation of reversible contributors, and individualized renal support remain central to management[7].

Conclusions

This case describes severe anuric acute kidney injury following accidental ingestion of a herbicide suspected to contain diquat. Oliguria at presentation progressed to anuria on the first hospital day despite initial hemodynamic stability. Approximately six weeks of hemodialysis were required before renal recovery permitted treatment discontinuation, with a subsequent serum creatinine concentration of 1.8 mg/dL.

The clinical course underscores the importance of monitoring renal function and urine output after suspected bipyridyl herbicide exposure, even when other initial clinical findings are reassuring. Pre-existing gastrointestinal losses may contribute to kidney injury and should be considered alongside potential direct nephrotoxicity. Dialysis independence represents an important recovery outcome, but it should not be equated with complete normalization of renal function or proof of effective extracorporeal toxin removal.

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design

Laura A. Vicenzi Souza[1], Bruno Zanardo Faggion[2], Carolina Huedepohl Marques Vieira[3], Rafaela dos Anjos Neves[2], Mayara Mayer Alves[2], Bruno Coelho Siniscalchi[4], Luiz Henrique Barros Santos Pontelli[4], Maria Fernanda Barros Pontelli[4], Letícia Buque Santos[5]

Acquisition, analysis, or interpretation of data

Laura A. Vicenzi Souza[1], Bruno Zanardo Faggion[2], Carolina Huedepohl Marques Vieira[3], Rafaela dos Anjos Neves[2], Mayara Mayer Alves[2], Bruno Coelho Siniscalchi[4], Luiz Henrique Barros Santos Pontelli[4], Maria Fernanda Barros Pontelli[4], Letícia Buque Santos[5]

Drafting of the manuscript

Laura A. Vicenzi Souza[1], Bruno Zanardo Faggion[2], Carolina Huedepohl Marques Vieira[3], Rafaela dos Anjos Neves[2], Mayara Mayer Alves[2], Bruno Coelho Siniscalchi[4], Luiz Henrique Barros Santos Pontelli[4], Maria Fernanda Barros Pontelli[4], Letícia Buque Santos[5]

Critical review of the manuscript for important intellectual content

Laura A. Vicenzi Souza[1], Bruno Zanardo Faggion[2], Carolina Huedepohl Marques Vieira[3], Rafaela dos Anjos Neves[2], Mayara Mayer Alves[2], Bruno Coelho Siniscalchi[4], Luiz Henrique Barros Santos Pontelli[4], Maria Fernanda Barros Pontelli[4], Letícia Buque Santos[5]

Supervision

Laura A. Vicenzi Souza

Disclosures

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