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Qualitative Detection of Paraquat Poisoning Using Sodium Dithionite Test: A Case Series

[1]Dr. Sarvabhavi Kumavat, [2]Dr. Pramod Ingale, [3]Dr. Pooja Rai

Corresponding Author: Dr. Sarvabhavi Kumavat

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Abstract

Paraquat poisoning is associated with high morbidity and mortality due to its severe multi-organ toxicity and lack of a specific antidote. Early diagnosis plays a crucial role in patient management and prognosis. The sodium dithionite test is a simple bedside qualitative test used for the detection of paraquat cations in urine. We report a case series of three patients with suspected paraquat poisoning, highlighting the diagnostic utility of the sodium dithionite test and the importance of timing in sample collection. Among the three cases, only one showed a positive urine dithionite test, correlating with early testing within 48 hours of ingestion. This case series emphasizes the role of timely urine dithionite testing as a rapid, cost-effective diagnostic tool in acute paraquat poisoning.

Keywords: Diarrhoea, Haemoptysis Kidney, Plasma

Introduction

Paraquat (N,N′-dimethyl-4,4′-dipyridylium dichloride), also known as methyl viologen, is a widely used non-selective herbicide with extremely high human toxicity [1,2]. Despite regulatory restrictions in several countries, paraquat poisoning continues to be reported, particularly in developing nations, with mortality rates ranging from 50–90% [3,4]. Diagnosis is often challenging due to nonspecific early symptoms and lack of rapid confirmatory tests in peripheral healthcare settings. The sodium dithionite test provides a simple bedside method for detecting paraquat cations in urine and plasma. This case series describes three patients with suspected paraquat poisoning and demonstrates the impact of timing on urine dithionite test results.

Case Series

Case 1

A 17-year-old female presented with a history of accidental ingestion of an unknown poison two days prior to admission. She was referred from a district hospital for further evaluation. On examination, the patient had vomiting and abdominal discomfort. Laboratory investigations revealed a rising serum creatinine level, suggestive of early renal involvement. Urine was sent for toxicological analysis with a

provisional diagnosis of organophosphorus poisoning versus paraquat poisoning.

Case 2

A 56-year-old male was brought to the emergency department with unconsciousness and breathlessness of 1–2 hours duration. He had multiple episodes of vomiting and abdominal pain for the preceding 2-3 days. Relatives reported accidental ingestion of paraquat dichloride 24% (Dhanuka Ozone®) six days earlier. The patient required intubation and was shifted to the MICU. Serum creatinine was 8.2 mg/dL, and he was initiated on hemodialysis for acute renal failure. A urine sample was sent for confirmation of paraquat poisoning.

Case 3

A 21-year-old female was brought with complaints of breathlessness, throat pain, abdominal pain, and multiple episodes of vomiting since two days. There was a history of intentional ingestion of paraquat poison six days prior. She was admitted to the MICU, and a urine sample was received for toxicological confirmation.

Materials and Methods

Sodium Dithionite Test

Principle: Sodium dithionite reduces paraquat cations to a blue-coloured radical ion in an alkaline medium, indicating the presence of paraquat [5–7].

Procedure: Freshly prepared 1% sodium dithionite in 1 N sodium hydroxide (250 mg sodium dithionite + 1 g NaOH in 25 mL distilled water) was added to urine in a 1:2 ratio (5 mL reagent + 10 mL urine). The reaction mixture was allowed to stand for 2–3 minutes, and colour change was observed.

Results

Case	Time since ingestion	Dithionite Test Result	Interpretation
Case 1	2 days	Blue colour	Paraquat present
Case 2	6 days	No colour change	Paraquat absent
Case 3	6 days	No colour change	Paraquat absent

Discussion

Paraquat is rapidly absorbed following ingestion, with peak plasma concentrations occurring within one hour, followed by rapid redistribution to tissues, particularly lungs and kidneys [8,9]. More than 90% of absorbed paraquat is excreted unchanged in urine within the first 24 hours[10]. The sodium dithionite test has a detection limit of approximately 1 mg/L in urine and is most useful when performed within 24–48 hours of exposure[6,11].

In the present case series, the test was positive only in Case 1, where urine was tested within two days of ingestion. In Cases 2 and 3, the six-day delay likely resulted in complete urinary elimination of paraquat, leading to false-negative results despite confirmed ingestion. This highlights that a negative dithionite test does not exclude paraquat poisoning if there is a significant delay between ingestion and testing.

Absorption, distribution and excretion –

Absorption is mainly through GI tract and via inhalation; absorption via skin or eye contact is minimal. Only 5 to 10 % of the ingested does is absorbed, and rest is excreted in the faeces. After absorption it is distributed to all the organs, but highest concentrations are found in the kidneys and lungs, followed by muscles from which paraquat can redistribute in the circulation as plasma concentration decreases. More than 90% of the absorbed paraquat is excreted unchanged in the urine within first 24 hrs.

Action - Paraquat undergoes a NADPH depended reduction to form Paraquat free radicals, which reacts with molecular oxygen to form superoxide free radicals and hydroxyl radicals, disturbing cellular function, structure and cell death.

Sign & Symptoms – Doses of less than 1.5 gm produce transient vomiting and diarrhoea. Locally irritation and inflammation of skin, nails, cornea, conjunctivae and nasal mucosa are produced. Oropharyngeal corrosion and ulceration, oliguric and non- oliguric renal failure, cough, dyspnoea, haemoptysis, Pulmonary edema and fibrosis, centrilobular hepatic necrosis and cholestasis, hypovolemic shock and arrhythmia, convulsion cerebral edema, adrenal insufficiency also do occur. Death occurs due to multi organ failure or corrosive effect on G.I. tract.

Diagnosis – Paraquat poisoning can be diagnosed by detection of paraquat in body fluid (e.g. serum, plasma, urine) by simple and bed sided Na- Dithionite test. The plasma level peaks early, within 1 hrs post Paraquat ingestion followed by rapid and steep decline due to its rapid redistribution from circulation to other compartments.

In urine sample Na- Dithionite should be added with alkaline medium (sodium bicarbonate or sodium hydroxide) in 1:2 ratio (Reagent to Urine ratio). The lowest detection level by dithionite test in urine is 1 mg/L. First Urine sample should be collected possibly by foleys catheter emptying bladder fully. This urine sample represent the average blood paraquat levels during the previous several hrs. The result of second urine sample collected after first urine indicate the current blood paraquat level. An observation of higher levels in the first urine sample than the second urine sample can be interpreted as decreasing serum paraquat levels from the initial levels prior to patient hospitalization. Urine dithionate test may becomes negative after 48 hrs of consumption as most of the paraquat is excreted via kidney in 24-48 hrs, It can also be detected by HPLC with lowest detection level of 0.01 mg/L. HRCT can be used to detect paraquat induced lung injury. The initial pathology of the lung is an inflammation of the alveoli presenting as ground glass opacity (GGO) signals on HRCT imagining. A GGO area >50% of the total lung volume is usually fatal, but all surviving patients have a GGO area <20% at 7 days post Paraquat ingestion.

Conclusion

- The sodium dithionite test is a rapid, inexpensive, and effective bedside screening test for paraquat poisoning when performed early.
- A Negative dithionite test does not exclude paraquat poisoning if there is a significant delay between ingestion and testing.
- Timely urine sampling is critical for accurate detection. Clinicians should interpret negative results cautiously in delayed presentations as sodium dithionite test may be negative after excretion of >90% paraquat via kidney in 24-48 hrs
- Paraquat poisoning is associated with very high mortality rates due to the high toxicity of the compound and lack of effective treatment. Clinician should consider history, clinical findings and supportive investigations for diagnosis and management.

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