Case Report

Herbicide poisoning: A diagnostic challenge

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Abstract

Despite widespread availability, reports of herbicide poisoning from India are not common. Diagnosis is often difficult in the absence of proper history, non-specific clinical features and lack of diagnostic tests. A case of Paraquat poisoning is reported where diagnosis could be established only after the recovery of the patient. The literature is reviewed.

Keywords: Herbicide, Paraquat, 2,4-D

Access this article online

Website: www.ijccm.org

DOI: 10.4103/0972-5229.94437

Quick Response Code:

Introduction

Despite the widespread availability, reports of herbicide poisoning with suicidal intention is scarce in the Indian literature. One reason for this underreporting could be the inability to differentiate them from other more commonly used compounds like anticholinergic pesticides. In the absence of specific clinical features and diagnostic tests, the diagnosis is completely based upon a reliable clinical history.

We report a case of Paraquat ingestion with suicidal intention where the final diagnosis could be made only after the complete recovery of the patient.

Case Report

A previously healthy, 24-year-old farmer was brought to the emergency department 1 h after ingestion of an unknown quantity of a liquid. The mother of the patient described the liquid as possibly WEEDAR, a herbicide containing 2,4 D as the active ingredient. Following the ingestion, he had several episodes of vomiting. Relatives denied any episode of seizure. On examination, he was drowsy but arousable, febrile (102°F) with heart rate 120/min, regular, BP 110/76 mmHg, respiratory rate 20/min and oxygen saturation (while breathing room air) 95%. His oral mucosa was congested and edematous. Pupils were bilateral 2 mm and reacting to light. Both lung fields were clear on auscultation. Rest of the systemic examination was unremarkable.

Gastric lavage was performed and repeated doses of charcoal were given in the emergency department and he was admitted to the intensive care unit (ICU) for observation and further evaluation. In the ICU, he received IV fluid and antiemetic (Ondansetron) as supportive measure. Initial complete blood count, electrolytes, liver and renal function tests, arterial blood gas, serum amylase and choline esterase levels were within normal limit. ECG revealed sinus tachycardia and 2-D echocardiography showed normal cardiac chambers with LVEFof 60%. His initial chest X-ray showed right lower and mid zone infiltrate [Figure 1].

Nine hours after admission, the patient became severely hypoxic, SpO_2 84% while on 60% Venturi Mask. ABG showed PaO_2 of 46 mmHg with mild respiratory alkalosis. Rapid sequence intubation was performed and he was placed on mechanical ventilation. Fresh and altered blood was aspirated through the Ryle's tube. A probable diagnosis of pulmonary aspiration was made

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and he was started on Pipercillin-Tazobactam and Clindamycin empirically. Pantoprazole infusion was started. On the following day, he continued to remain febrile. UpperGI endoscopy was performed revealing diffuse erythema and superficial ulcerations in the esophagus and multiple linear ulcerations of fundus and body of stomach [Figure 2]. Blood investigations on day 2 were unremarkable except mild rise in BUN and serum creatinine (26 and 1.5 mg/dl). His arterial blood gas values on day 2 were pH 7.42, $PaCO_2$ 32 mmHg and PaO_2 62 mmHg (50% FIO_2 and PEEP 5 cm H_2O). There was no fresh change in the chest X-ray. Bronchoscopy performed on the same day revealed uniformly blood-stained bronchoalveolar lavage fluid. On day 3, he remained febrile and drowsy. His FIO_2 requirement decreased to 40%. This time he had no further GI bleeding. On subsequent days in the ICU, he showed progressive improvement, became afebrile on day 4 and azotemia improved from day 3 onward. On day 6, he was conscious, obeying command, and had a PaO_2/FIO_2 ratio >300 with normal blood chemistry. He was extubated later on during the day. Subsequently, he revealed that the liquid he had ingested was not WEEDAR but GRAMOXONE 24% that contains Paraquat. The next day, he was transferred to the floor and 3 days later he was discharged following psychiatry consultation.

Discussion

The present case posed a number of diagnostic challenges to the critical care team. Firstly, there was considerable confusion regarding the identity of the liquid ingested. 2,4-D was sprayed in the field on the day of ingestion, so initially it was suspected as the ingested substance. Secondly, the initial clinical features were non-specific. Initial symptoms of nausea, vomiting, drowsiness or mucosal burns are common to both Paraquat and 2,4-D. Some of these symptoms may also mimic anticholinergic poisoning.[1,2] Subsequent development of renal failure and hypoxia are also well known with 2,4-D.[1]

Paraquat (1,1′-dimethyl-4,4′-bipyridium) dichloride is a non-selective contact herbicide, widely used in many countries since the 1960s. It is highly toxic to humans at a dose of 3–6 g. Thus, even a small sip is considered to be fatal for adults. The first case of Paraquat poisoning from India was reported by Singh *et al*.[3] The toxicity of Paraquat is through formation of superoxide anions during the "redox cycling process," which then leads to the formation of other more toxic reactive oxygen species such as hydrogen peroxide and hydroxyl radical in the presence of NADPH and cytochrome P450 reductase. Hydroxyl radical is a potent oxidant and can induce lipid peroxidation, which causes cell membrane damage and cell death.[4,5] The toxicity of Paraquat may be enhanced by concomitant ingestion of alcohol.[4]

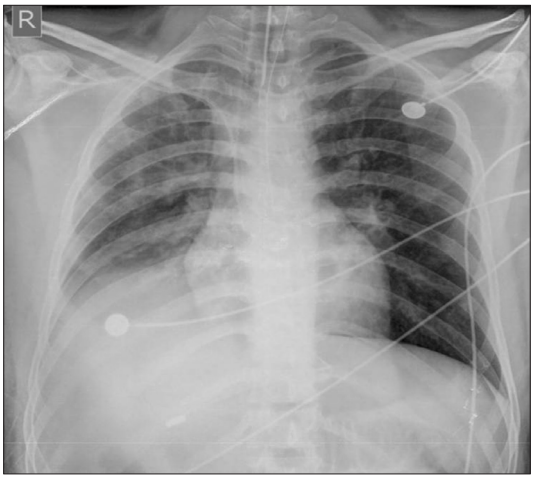

Figure 1: Chest X-ray on day 1

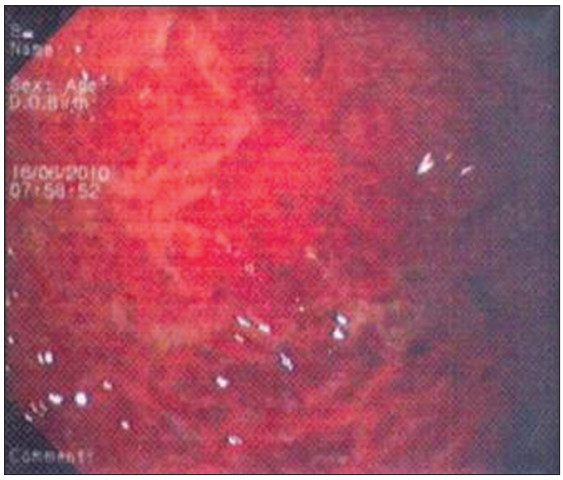

Figure 2: Endoscopy findings on day 2

Poisoning with Paraquat leads to both local and systemic effects. In an Indian series of 17 patients, the most common symptoms were vomiting (100%), followed by altered sensorium (59%), oral ulceration or dysphagia (53%), dyspnea (41%) or loose stools (24%).[5] Systemic effects of Paraquat are renal and hepatic failure, pulmonary edema and fibrosis, cardiac failure, shock, convulsions and multiorgan failure. Involvement of lung in the form of diffuse alveolitis and subsequent pulmonary fibrosis is the hallmark of Paraquat poisoning. Acute respiratory distress syndrome because of Paraquat usually appears 24–48 h after ingestion.[3] In our patient, the cause of hypoxia was aspiration of blood, substantiated by uniformly blood-mixed bronchoalveolar lavage fluid. Cause of renal failure is multifactorial – hypovolemia, circulatory failure, septicemia and direct toxicity related to redox cycling.[5]

The diagnosis of Paraquat poisoning is usually straightforward from the reliable history of exposure. But, it can be difficult when the history is unclear, as in our patient. The urine dithionite test can quickly confirm the presence of Paraquat in urine. Paraquat reacts with dithionite to produce blue color. It is also useful to estimate the level of exposure – colorless or light blue color of urine indicates mild poisoning, whereas navy or dark blue color indicates moderate to severe poisoning.[4] However, the test may be false-negative in the presence of renal dysfunction. Urine or plasma Paraquat levels can also confirm the diagnosis. Plasma Paraquat concentration–time data have been used to predict outcome and are well validated.[6] Unfortunately of these tests are widely available in India.

The treatment of Paraquat poisoning is supportive. The initial management focuses on prevention of further absorption by use of adsorbents like activated charcoal and Fuller's earth and gastrointestinal decontamination. Hemoperfusion using activated charcoal has been shown to decrease Paraquat level, but data to support survival benefit in humans is insufficient.[5,7] It is only effective if initiated within 4 h of ingestion, as peak Paraquat concentration in the lung is achieved in 5–7 h.[5] Hemodialysis is used as a support of acute renal failure, but it does not increase clearance of the substance as it is rapidly distributed to the lungs and other organs.[5] Immunosuppression with combination of cyclophosphamide and methylprednisolone was shown to be beneficial in moderate to severe cases by prevention of ongoing inflammation and pulmonary fibrosis.[8]

In conclusion, as there is no specific antidote available for Paraquat poisoning. It is important to establish the diagnosis early and to pursue aggressive decontamination and prevention of further absorption. Increased awareness of the clinician and availability of the laboratory diagnostic methods will definitely help in successful management of Paraquat poisoning.

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How to cite this article: Ghosh S, Singh A, Dewan H, Walia G, Bansal A. Herbicide poisoning: A diagnostic challenge. Indian J Crit Care Med 2012;16:52-4.

Source of Support: Nil, **Conflict of Interest:** None declared.