Inflammatory Esophagogastritis and Acute Kidney Injury After Intentional Ingestion of Paraquat

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Short Report

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Abstract

We describe a clinical presentation of Inflammatory esophagogastritis and acute kidney injury in young adult male after intentional ingestion of paraquat. The high number of deaths from paraquat is due to its toxicity and ineffective treatment. Intravenous fluid, immunosuppressant, and antioxidant was given to reduce the effect of toxins on target organs.

1. Introduction

Paraquat is a bipyridilium herbicide, a corrosive weed killer available as a 20% solution for use in agriculture. The high number of deaths from paraquat is due to its toxicity and ineffective treatment. Paraquat ingestion causes conditions such as gastritis, inflammation of the mouth, to severe complications such as acute kidney injury, acute hepatitis, pneumonia, pulmonary fibrosis, to the occurrence of multiple organ failure that causes death. The absence of guidelines for the management of patients with paraquat ingestion is one of obstacles in management.

2. Case Presentation

A 19-year-old male presented to the emergency department with an alleged history of consumption of around 200 ml of 24% paraquat 12 hours earlier in an apparent suicide attempt. Before admission to our hospital, the patient was taken to the local hospital for gastric lavage. On admission, the patient had a burning sensation in the throat accompanied by nausea, vomiting with blood, and abdominal cramps but had no chest distress and dyspnea or palpitation. However, 4 hours after admission, chest tightness, shortness of breath, and dyspnea were observed. The routine investigations revealed leukocytosis, elevated levels of serum urea, and creatinine. On follow up, his urea and creatinine levels increased gradually till the ninth day, and then gradually decreased after hydration over next seven days. There was no pulmonary involvement. Clinical management was done by Department of Internal Medicine with intravenous fluid, N-acetyl cysteine, methylprednisolone, and lansoprazole. The tongue lesions started with redness and gradually progressed to painful erosions and ulcers. Examination of tongue revealed multiple erosions and superficial ulcers, few of which were coalesced. Floor of the ulcers were covered with yellowish necrotic debris. Center of the tongue was relatively spared (Fig. 1).

CT Thorax show normal. Esophagogastroduodenoscopy (Fig. 2) revealed pharyngeal mucosa hyperemia, hyperemic epiglottis, arytenoid, and ventricular folds with active inflammation and profuse secretions. Esophageal mucosa looks edematous, easily eroded and bleeds especially in the proximal esophagus. The gastric mucosa appears edematous and hyperemic with active inflammation in the fundus and corpus.

The patient was followed for 1 week up to present day. Sore throat has reduced, the patient is able to eat soft consistency foods. There was normal on kidney function control.

3. Discussion

Paraquat is a toxic herbicide which is easily available so it is often used as a self-poisoning.[1] After being absorbed rapidly, paraquat is concentrated in several cells. Clinical symptoms that appear in general are intracellular effects. Paraquat is actively concentrated in lung tissue which will cause pneumonitis and pulmonary fibrosis. Paraquat also causes gastrointestinal, renal, and hepatic injury.[2] Paraquat can inhibit the reduction of nicotinamide adenine dinucleotide phosphate (NADP) to nicotinamide adenine dinucleotide phosphate hydrogen (NADPH), which results in overproduction of reactive oxygen and nitrite species that destroy fat cell membranes.[3] Paraquat causes tissue death through the production of free radicals that ultimately lead to cell death.[4]

Paraquat intoxication manifests as mild symptoms such as gastritis, inflammation of the mouth, and gastrointestinal symptoms that can improve completely. Moderate-severe symptoms are acute renal impairment, and in some severe cases acute hepatitis, pneumonia, pulmonary fibrosis, which often cause death in the first 2–3 weeks, and in severe degrees, multiple organ failure and cardiac arrest may result in death.[5] The clinical manifestations of paraquat intoxication depend on the amount consumed. Consumption of large amounts of fluid concentrations (> 50–100 ml of 20% w/v ions) causes fulminant organ failure such as pulmonary edema, multiorgan failure, and seizures due to central nervous system involvement.[6]

The patient complains of vomiting after ingestion the herbicide with a composition of paraquat dichloride 288 grams/liter or equivalent to paraquat ion 209 grams/liter as much as 500cc. The patient entered the ER at local Hospital with fully consciousness, tachycardia and tachypnea but the patient's oxygen saturation was still normal, which was different from most cases found where hypoxia, shock, and metabolic acidosis occurred in patients after taking paraquat. The amount of urine of the patient seems to be reduced with laboratory results getting an increase in serum creatinine which indicates the occurrence of acute kidney injury. Consumption in small amounts causes toxicity to two target organs, namely the kidneys and lungs within 2–6 days.[3]

In patients taking paraquat > 20 ml of the 20% preparation it causes high mortality due to organ failure and cardiogenic shock and 1–4 days, whereas in smaller amounts (10–20 ml) initiates pulmonary fibrosis and renal impairment leading to death within a week. a few weeks. The half-life of paraquat is very long, so mortality is high in patients with renal impairment.[7] Renal impairment occurs rapidly, creatinine and/or cystatin-C levels can be monitored on the first day to detect renal impairment and predict patient prognosis.[2]

Gastrointestinal toxicity is common with paraquat intoxication. Ingestion of paraquat or vomiting without swallowing will produce a typical tongue appearance.[1] Lesions of the oral mucosa and tongue ('paraquat tongue') appear in the first few days and may become ulcerated with bleeding. Mucosal lesions of the pharynx, esophagus, and stomach are common and can worsen and cause perforation, mediastinitis, and pneumomediastinum.[6] The patient experienced dysphagia after consuming paraquat where on physical

examination found hyperemia of the tongue and on esophagogastroduodenoscopy hyperemic lesions of the pharyngeal mucosa, epiglottis, arytenoids, edema and hyperemia of the esophageal mucosa to the corpus gastric. The risk of esophageal and gastric perforation can occur so that further observation and appropriate management are needed.

The main goals of therapy for patients with paraquat intoxication are to rapidly remove toxins from the gastrointestinal tract (prevent absorption), increase the output and excretion of toxins from the blood (diuresis), and prevent lung damage with anti-inflammatory and antioxidant agents.[5] The principle of management is resuscitation (assessment and management of the airway, respiration, and circulation).[2] Airway obstruction can occur due to mucosal toxicity and vomiting. Tachypnea and hypoxia due to metabolic acidosis, aspiration, and/or acute alveolitis require blood gas analysis and chest X-ray to confirm the diagnosis.

Patients with hypovolemia are given fluid boluses (15–20 ml/kg over 5–30 minutes) and can be repeated if necessary. Acute renal injury often occurs in the first 24 hours, so close monitoring of fluid balance is required.[6] The patient was admitted to the hospital with complaints of vomiting accompanied by tachycardia and tachypnea while the patient's oxygen saturation was within normal limits. The patient was then resuscitated with 1500 cc of saline and measure fluid balance using urinary catheter but the patient refused. A chest X-ray was performed to rule out aspiration pneumonia, but the results were normal. Examination of urine dithionite test and plasma paraquat cannot be done.

The patient was given intravenous fluids and gastric lavage as soon as the patient arrived at the ER, monitoring of consciousness, vital signs, urine production, laboratory tests as an indication of multiple organ failure. Gastric lavage action with activated charcoal is recommended in patients taking paraquat within the first hour. Paraquat is a toxic substance with no known antidote, the use of a single dose of activated charcoal or fuller's earth is recommended in patients with threatened airway obstruction.[6] The patient had severe oral ulcers which cause the patient avoid to eating and drinking. The patient's oral intake gradually improved along with the improvement of the patient's ulcers and gastrointestinal mucosal inflammation. In patients who consume large amounts of paraquat, there is an association with the extent of gastric lesions.[8]

Kidney is the main organ in the excretion of paraquat, through glomerular filtration and active secretion, so that the kidney is the organ that contains the highest concentration of paraquat. Paraquat is toxic to proximal renal tubule cells through the generation of reactive oxygen species, which causes lipid peroxidation in cell membranes, loss of cell membrane integrity, and cell death.[9] In the control of renal function, the patient's urea and creatinine levels were increased, so hemodialysis was considered if corticosteroids and N-acetyl cysteine is not responding. Elimination of paraquat can be done by hemodialysis and hemofiltration. The problem is that paraquat is quickly out of circulation and the time of initiation has an impact on the deposition of paraquat levels in the lungs.[2]

Based on data from several studies, hemodialysis after 2–4 hours is not only to eliminate paraquat from the plasma compartment but also to reduce paraquat in the airways so that it can change the patient's prognosis. Hemodialysis is performed in patients with symptomatic acute renal impairment.[6] Hemodialysis reduces plasma overload of paraquat but does not reduce the effect of toxins on target organs.[2] After being given adequate management and controlling for serum creatinine, improvement was found so that the patient no longer required hemodialysis.

Immunosuppressants are often used in the management of paraquat intoxication. This is based on the occurrence of an acute inflammatory response to paraquat intoxication which causes pulmonary fibrosis so that by stopping the inflammatory response it can inhibit the fibrosis process and lead to death. The use of glucocorticoid agents such as methylprednisolone and chemotherapeutic agents such as cyclophosphamide is widely used. A study comparing the administration of dexamethasone, methylprednisolone and cyclophosphamide showed better mortality than standard therapy, but this study still needs further study due to many limitations.[2] Patients since the first day of treatment have been given methylprednisolone 62.5mg intravenous every 8 hour as immunosuppressive with the aim for suppressed the inflammatory response.

Administration of antioxidants is one of the treatments given to patients with paraquat intoxication. N-Acetyl cysteine is a glutathione generation that reduces paraquat-induced apoptosis and an in vitro inflammatory marker in lung culture. NAC suppresses the production of malondialdehyde and superoxide and increases glutathione levels in all tissues. Several studies have also shown that the administration of high doses of vitamin C and antioxidants reduces 1 in 10 mortalities.[2] Another study stated that ambroxol can also act as an antioxidant and anti-inflammatory as a treatment for acute lung injury in cases of paraquat intoxication. The study stated that combination therapy with high-dose ambroxol increased PaO_2 (WMD 13.73 mmHg, 95% CI; 8.68–1879) and PaO2/FiO2 (WMD 38.81 mmHg, 95%CI 29.85–47.76).[10] The patient was also given NAC (Rasfar®) as an antioxidant intravenous during treatment.

The mortality of paraquat intoxicated patients depends on the amount of paraquat in the blood. Poor outcome is usually associated with blood plasma levels greater than 0.2 mg/ml within 24 hours of ingestion and 0.1 mg/ml within 48 hours. Laboratory tests include urine tests detecting paraquat concentrations of 1 mg/ml or more, gas chromatography (1 mcg/ml) and radioimmunoassay (< 0.1 mcg/ml).[11]

4. Conclusions

The high number of deaths from paraquat is due to its toxicity and ineffective treatment. The main goals of therapy for patients with paraquat intoxication are to rapidly remove toxins from the gastrointestinal tract, increase the output and excretion of toxins from the blood, and prevent lung damage with anti-inflammatory and antioxidant agents.

Declarations

Funding

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Availability of data and material

No new data were created or analyzed in this study. Data sharing is not applicable in this article.

Ethics approval and consent to participate

Approval number 535/UN4.6.4.5.31/PP36/2021 of the Ethics Committee of Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia. Written informed consent has been obtained from the patient to publish this paper.

Competing of Interest

The authors declare no competing of interest.

Consent for publication

I, the undersigned, give my consent for the publication of identifiable details, which can include photograph(s) and/or videos and/or case history and/or details within the text to be published in the above Journal and Article.

Authors' contribution

FJG, RH, AMLP and HR were the principal investigators of the study and drafted the manuscript; FJG and RH collected and analyzed the data; RH, AMLP, and HR contributed to the concept and design of case report; AMLP and HR revisited the manuscript and critically evaluated the intellectual contents. All authors participated in the final draft preparation, manuscript revision, and critical evaluation of the intellectual contents. All authors have read and approved the content of the manuscript and confirmed the accuracy or integrity of any part of the work.

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Figures

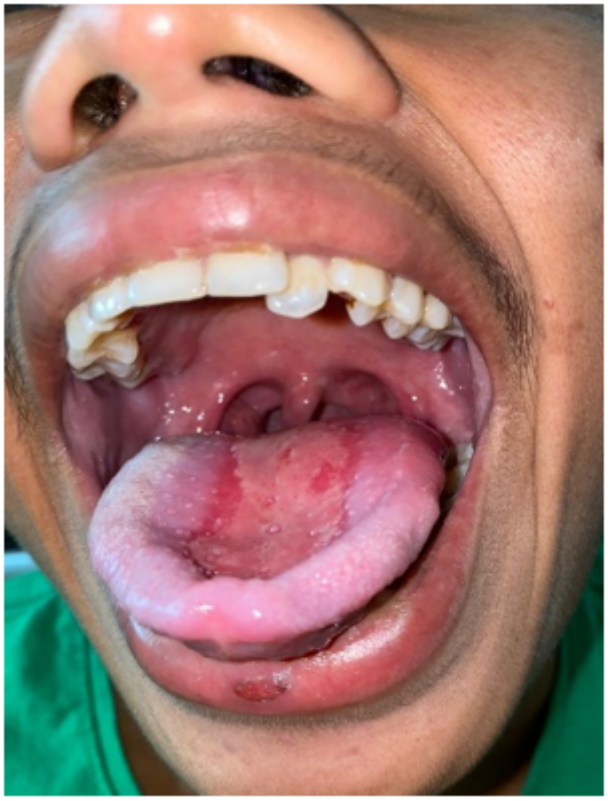

Figure 1

Multiple erosions and superficial ulcers over tongue covered with yellowish necrotic debris on 6th day after oral ingestion of paraquat.

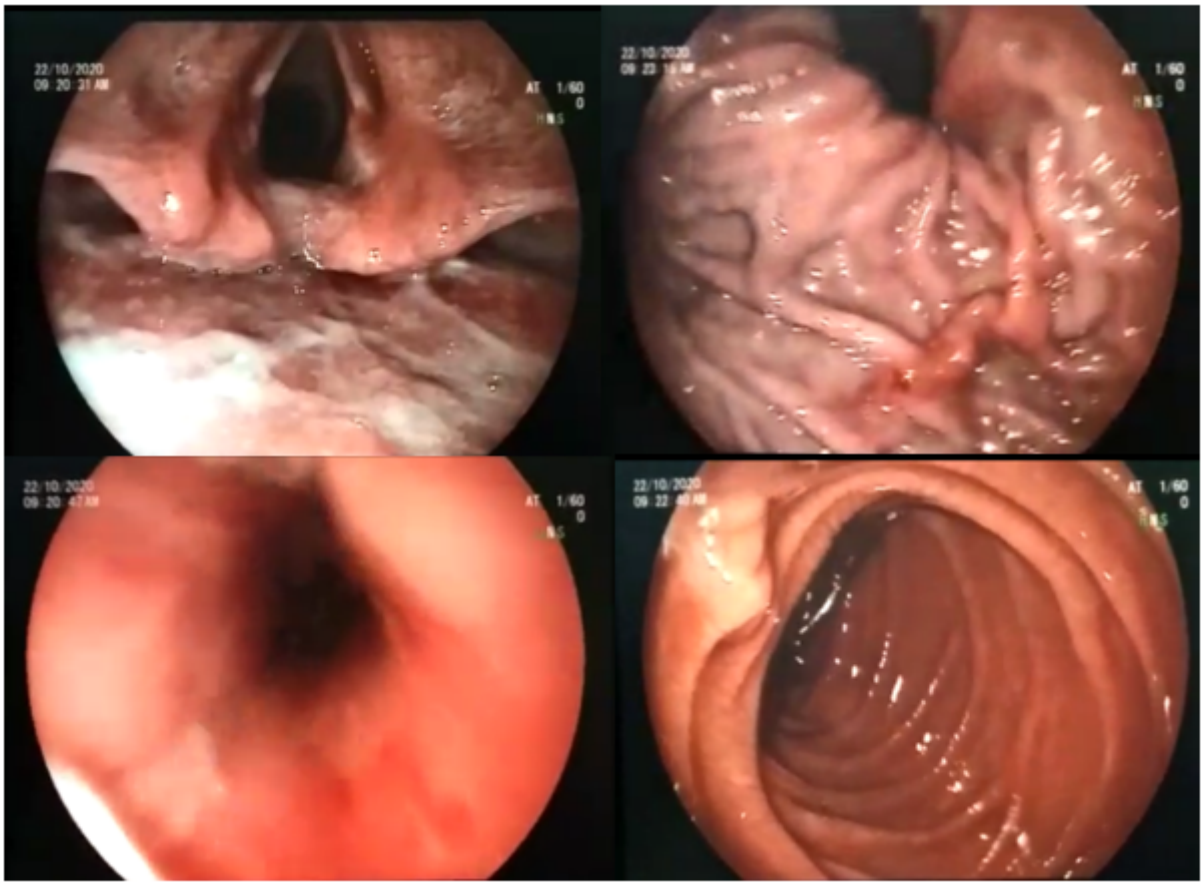

Figure 2

Esophagogastroduodenoscopy shows edematous and easily eroded of esophageal mucosa especially in the proximal esophagus and active inflammation in the gastric fundus and corpus.