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Case Report

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Inflammatory Esophagogastritis and Acute Kidney Injury After Intentional Ingestion of Paraquat

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Abstract

Paraquat a rapidly acting, nonselective herbicide is a leading cause of fatal poisoning in many parts of Asia. This compound is very notorious to cause rapid development of renal, liver, and respiratory failure with very high mortality due to lack of specific antidote and evidence-based treatment. We describe a clinical presentation of inflammatory esophagogastritis and acute kidney injury (AKI) in young adult male after intentional ingestion of paraquat. Dosage of paraquat is 200 ml. The patient has physical problems with swallowing, sore throat, nausea and vomiting, upper abdominal pain, and hematemesis. Intravenous fluid, immunosuppressant, and antioxidant was given to reduce the effect of toxins on target organs.

Keywords: Inflammatory Esophagogastritis, Acute Kidney Injury, Paraquat

Introduction

Paraquat is a bipyridilium herbicide, a corrosive weed killer available as a 20% solution for use in agriculture. This chemical is highly toxic to humans especially when ingested and can cause multiple organ failure with high mortality [1]. Paraquat poisoning commonly causes local irritative effects on the tongue, oropharynx, and esophagus in mild cases and may affect multiple organs especially the kidney, lungs, liver, and heart in moderate to severe cases [2]. The high mortality from paraquat is due to its toxicity and ineffective treatment. The absence of guidelines for the management of patients with paraquat ingestion is one of obstacles in management. The treatment given is supportive therapy combined with corticosteroids, antioxidant, hemoperfusion and Hemodialysis [3]. We report an uncommon case of intentional ingestion of paraquat with manifestations of dysphagia and abdominal pain due to inflammatory esophagogastritis and acute kidney injury.

Case Description

19-year-old male presented to the emergency department with an alleged history of consumption of around 200 ml of 24% paraquat 3 days prior to admission 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. On examination, he was conscious, oriented; his vitals were normal and systemic examination revealed multiple erosions and superficial ulcers on the tongue. Floor of the ulcers were covered with yellowish necrotic debris. Center of the tongue was relatively spared (Figure 1).

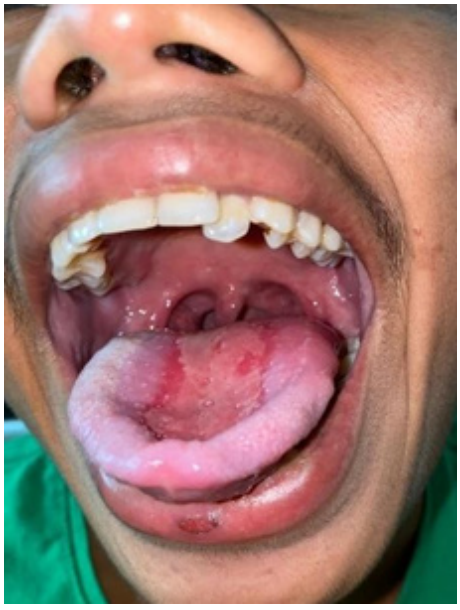

Figure 1: Multiple erosions and superficial ulcers over tongue covered with yellowish necrotic debris on 4th day after oral ingestion of paraquat.

Laboratory investigations shows leukocytosis, elevated levels of serum urea, and creatinine. Chest CT (computed tomography) (figure 2), electrocardiography (ECG) (figure 3), urine routine, and electrolytes were normal. Subsequent day's investigations are given in (table 1).

On follow up, his urea and creatinine levels increased gradually till the ninth day, and then gradually decreased after hydration over next three days.

Table 1: Laboratory investigations of the patient

Parameters	Day-1	Day-3	Day-5	Day-9	Day-11
WBC (cells/mm^3)	16,150	13,530	-	10,500	9,500
Hemoglobin (g%)	14.0	15.6	-	13.0	13.4
Platelets (cells/mm3)	221,000	220,000	-	243,000	165,000
Neutrophils %	86,7	75,6	-	80,9	78,4
Lymphocytes %	10,5	9,6	-	8,4	11,4
Monocytes %	2,8	9,8	-	10,4	3,4
Blood urea (mg/dL)	-	77	236	165	56
Serum creatinine (mg/dL)	-	4,33	7,05	2,57	1,65
SGOT (IU/L)	-	27	11	-	-
SGPT (IU?L)	-	19	13	-	-
Blood glucose (mg/dL)	-	89	-	-	-
Albumin (g/dL)	-	3,8	-	3,8	-
Sodium (mmol/L)	-	135	143	151	145
Potassium (mmol/L)	-	4,0	4,6	4,5	4,6
Chloride (mmol/L)	-	100	113	123	118
Urine routine	Normal	-	-	2-4 pus cell; protein trace	-
WBC: White blood cell, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase					

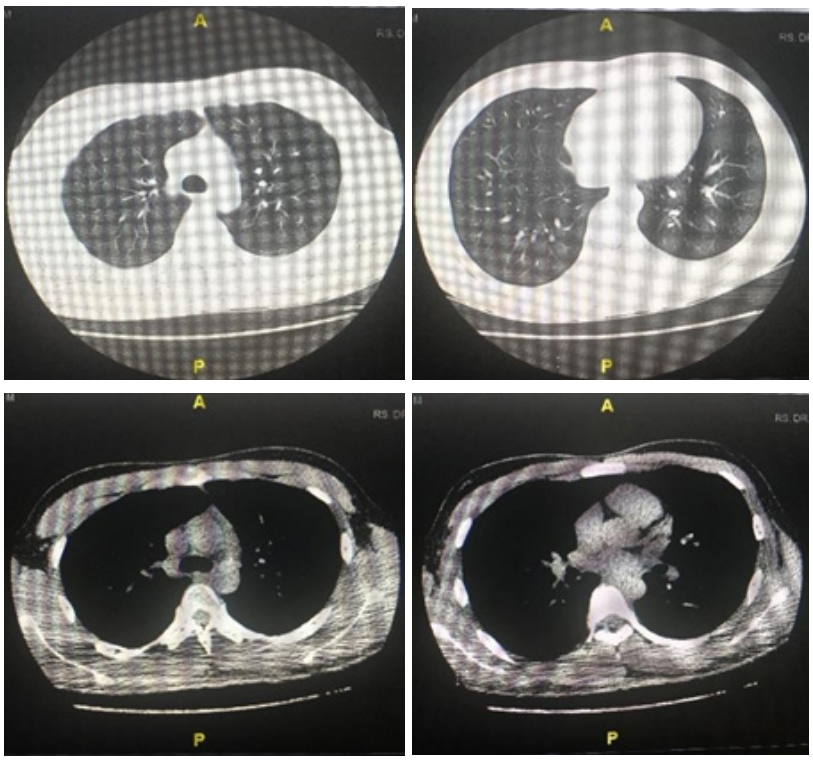

Figure 2: Chest CT of the patient.

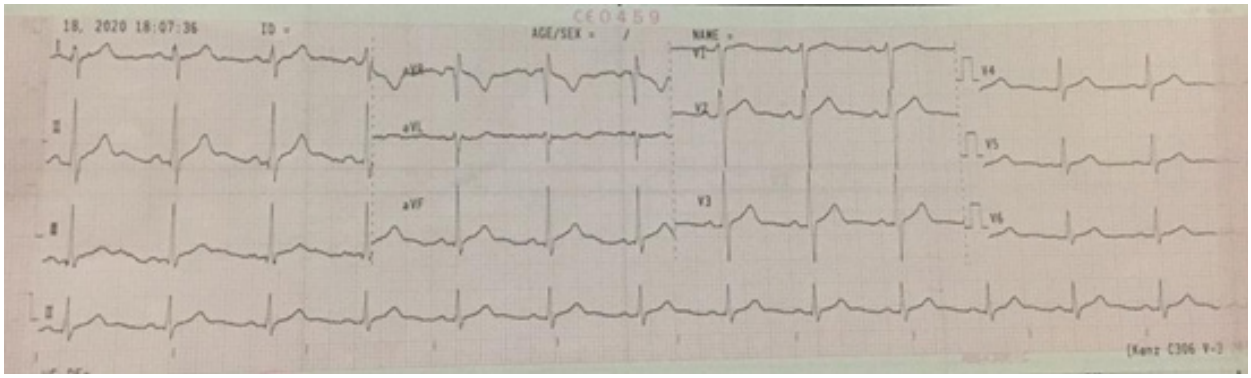

Figure 3: Electrocardiography of the patient

Clinical management was done by Department of Internal Medicine with intravenous fluid, injection ceftriaxone 2 g OD, injection methylprednisolone 62.5 mg TID, injection lansoprazole 30 mg BID, N-acetyl cysteine (NAC) 300 mg TID and other symptomatic treatment. On day-3, patient underwent esophagogastroduodenoscopy (EGD) to diagnosed the cause of dysphagia. Esophagogastroduodenoscopy (Figure 4) 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.

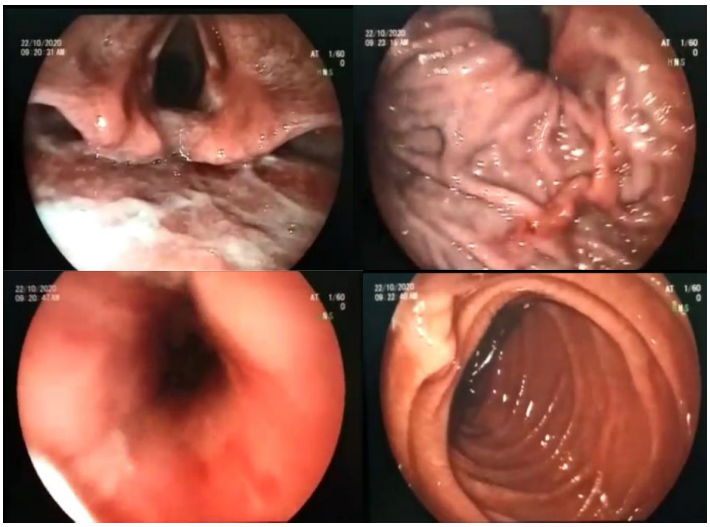

Figure 4: Esophagogastroduodenoscopy shows edematous and easily eroded of esophageal mucosa especially in the proximal esophagus and active inflammation in the gastric fundus and corpus.

At the same day, patient was consulted to nephrologist with AKI and given adequate rehydration therapy, injection NAC 5 g TID, and education to avoid nephrotoxic drugs. The urine output was monitored and planned for kidney function test 48 hours later. On day-5, his blood urea and creatinine levels gradually decreased. On day-9, his condition was improved. Complaints of dysphagia was reduced; the patient is able to eat soft consistency foods. The patient was followed for 3 days after discharged. There was normal on kidney function control.

Discussion

Paraquat is a toxic herbicide which is easily available so it is often used as a self-poisoning [4]. 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 [5]. 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 [6]. Paraquat causes tissue death through the production of free radicals that ultimately lead to cell death [7].

Paraquat poisoning commonly causes local irritative effects on the tongue, oropharynx, and esophagus in mild cases and may affect multiple organs especially the kidney, lungs, liver, and heart in moderate to severe cases [2]. The clinical manifestations of paraquat poisoning 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 [3].

The patient complains of dysphagia and 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 200 ml. 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 ingestion of paraquat. The urine output of the patient seems to be reduced with laboratory results getting an increase in serum creatinine which indicates the occurrence of acute kidney injury.

Gastrointestinal toxicity is common with paraquat poisoning. Ingestion of paraquat or vomiting without swallowing will produce a typical tongue appearance [4]. 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 [3]. 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. 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 [8]. 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 [5].

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 [2]. The principle of management is resuscitation (assessment and management of the airway, respiration, and circulation). 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 [5].

The patient was given intravenous fluids and gastric lavage as soon as the patient arrived at the ER, monitoring of consciousness, vital signs, urine output, 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 [3].

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 [5]. 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. Study by Iyyadurai et al found that the administration of high doses of vitamin C and antioxidants reduces 1 in 10 mortalities [5]. Another study by Wang et al reported that ambroxol can also act as an antioxidant and anti-inflammatory as a treatment for acute lung injury in cases of paraquat intoxication [9]. The study found that combination therapy with high-dose ambroxol increased PaO_2 (WMD 13.73 mmHg, 95% CI; 8.68-1879) and PaO_2/FiO_2 (WMD 38.81 mmHg, 95%CI 29.85-47.76) [9]. The patient was also given NAC 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 [7]. Since there is no evidence-based standard protocols, and lack of specific antidote makes the management of paraquat poisoning difficult [1].

Conclusion

Paraquat is a toxic herbicide which is easily available so it is often used as a self-poisoning. Paraquat poisoning commonly causes local irritative effects on the tongue, oropharynx, and esophagus in mild cases and may affect multiple organs especially the kidney, lungs, liver, and heart in moderate to severe cases. 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.

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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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Data availability
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.

Conflict of Interest
The authors declare no conflict 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.

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