

## CASE REPORT

# Multi-Organ Failure due to Paraquat Poisoning: Case Report and Review of Literature

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### Abstract

**Background:** Paraquat is a herbicide that is used widely. Several voluntary and accidental paraquat poisoning have been reported in recent years. Gastrointestinal absorption of paraquat is rapid. Systemic effects include pulmonary injury, heart failure, acute renal failure and hepatic failure. Metabolic acidosis results from multi-organ failure. Due to these systemic effects, paraquat poisoning is associated with high rate of morbidity and mortality.

**Case presentation:** A 34-year old man has been referred from a nearby town to our educational hospital with abdominal pain, nausea, vomiting, jaundice, and decreased level of consciousness since 12 hours ago. In his history, he declared that he had drunk a glass of green colored herbicide 2 days ago as a suicidal attempt. Four hours after ingestion, he was brought to a regional hospital by family and after 24 hours of observation, he was discharged. He was symptom free during the observation. His clinical features and laboratory results on arrival to our hospital revealed multi-organ failure. Despite aggressive supportive treatment, he died at the second day of admission.

**Conclusion:** this case report emphasizes on the importance of early admission and treatment of paraquat poisonings, regardless of symptoms, to save the patient's life. Many supportive treatments must be carried out for these patients.

**Keywords:** Early Admission; Multi-Organ Failure; Paraquat Poisoning

**How to cite this article:** Reihani H, Zarmehri B. Multi-Organ Failure due to Paraquat Poisoning: Case Report and Review of Literature. *Asia Pac J Med Toxicol* 2016;5:98-100.

## INTRODUCTION

More than 300000 deaths occurs due to self-poisoning with pesticides in the Asia-pacific region annually. Paraquat, with more than 50% mortality rate is one of the major causes of death due to this kind of poisoning.(1) Paraquat is an herbicide that is used widely in agriculture. This herbicide is safe If used appropriately, however, several voluntary and accidental paraquat poisoning have been reported in recent years.(2)

Paraquat is a nonselective herbicide. It is in granules, liquid, or aerosol form and may be combined with diquat. (3) Paraquat poisoning may result in pulmonary, cardiac, renal, hepatic and adrenal injury. The high mortality rate of this herbicide is due to multi-organ failure and lack of effective treatment. Additionally, there is no effective management for this kind of poisoning in humans.(2) Because of a high level of morbidity and mortality, clinical management of paraquat poisoning is a disappointing condition. (2)The generation of reactive oxygen species is the major mechanism of paraquat toxicity.(1)

Paraquat poisoning results from ingestion or transdermal exposure. Transdermal absorption is minimal through the intact skin, so the main cause of death is due to ingestion.

Inhalation exposure to paraquat can irritate the airway mucous membrane, but rarely causes systemic injuries.(3)

In this article, we present a case of paraquat poisoning and emphasis on pitfalls in the management. Additionally, we provide a simple review of related articles.

## CASE PRESENTATION

A 34-year old man was referred from a nearby town to our educational hospital with abdominal pain, nausea, vomiting, jaundice and decreased level of consciousness since 12 hours ago. In history, he declared that he had consumed a glass of green colored herbicide 2 days ago as a suicidal attempt. Four hours later, he was brought to the regional hospital by his family. After gastric lavage, charcoal administration and 24 hours observation, he was discharged from the hospital while he was nearly asymptomatic.

Twelve hours later, nausea, vomiting, jaundice, and decrease level of consciousness had started. He was brought to the hospital again and was referred to our referral academic center.

The patient had no past medical history or drug history. On admission the patient's vital signs were Blood Pressure (110/70 mmHg), Pulse Rate (90/min), Respiratory Rate (26/min), Temperature (38.3 °C) and O<sub>2</sub> saturation (95%).

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Received 11 May 2016; Accepted 18 July 2016

But the patient was confused. His physical examination showed jaundice, pulmonary rales, normal heart sounds, diffuse abdominal tenderness without rebound or guarding, and 2+ pitting edema in lower extremities. Laboratory tests at the presentation showed renal and hepatic injury. (table 1)

According to unknown poisoning and pulmonary rales to ruling out organophosphate poisoning, the physician performed atropine challenge test that was negative. Additionally, serum and red blood cell acetylcholinesterase enzymatic activities were normal, so organophosphate poisoning was ruled out. Urine toxicological screen tests for drugs abuse were negative.

Finally, according to multi-organ failure, a probable diagnosis of paraquat poisoning suggested. Few days later, following finding a container of paraquat in the patient's home, the diagnosis was confirmed.

#### Therapeutic measures

Due to deranged renal function, renal replacement therapy with hemodialysis was performed. Following hemodialysis, the level of consciousness and oxygen saturation was decreased, so the patient was intubated. In order to minimize the adverse effect of oxygen therapy, fraction of inspired oxygen (FI O<sub>2</sub>) was adjusted to minimum level.

Despite initiation of stress ulcer prophylaxis early after admission, he revealed signs of gastrointestinal bleeding in nasogastric tube secretion. Therapeutic measures for gastrointestinal bleeding, crystalloid infusion and packed cell transfusion was performed.

The patient transferred to intensive care unit and other supportive treatments were performed.

Despite intensive care and precise vasopressive agents, the patient died at the second day of admission, due intractable hemodynamic instability.

## DISCUSSION

Paraquat poisoning is usually a disappointing medical challenge for emergency physicians. Paraquat concentrates in the lung, so the pulmonary concentration of paraquat is higher than the plasma (6 to 10 times higher). Dinis-Oliveira published a paper in 2008 "Paraquat poisonings: mechanisms

of lung toxicity, clinical features, and treatment" states that the pulmonary effects of paraquat can be explained by the participation of the polyamine transport system abundantly expressed in the membrane of alveolar cells type I, II, and Clara cells. (2)

Pulmonary injury due to paraquat poisoning has two phases. In the initial phase, some destructive events like loss of alveolar cells, pulmonary infiltration and lung hemorrhage happen. The second phase is characterized by lung fibrosis.

It should be noted that paraquat and oxygen have a synergistic pulmonary destructive effect. Additionally, some myocardial injuries and adrenal necrosis may occur after paraquat poisoning.(3)

Gastrointestinal absorption of paraquat is rapid and plasma concentration of paraquat peaks about 1 to 2 hours after oral ingestion. After this phase, paraquat concentrates mostly in lungs and kidneys. An oral dose of 10 to 20 ml from the 20% solution may be lethal in adults. This dose for children is about 4 to 5 ml. (3)

Other injuries associated with paraquat poisoning are cutaneous irritation and ulceration, corneal injury in eye exposure, and some signs and symptoms in upper respiratory tract exposure like epistaxis and mucosal injury. Inhalation exposure may cause dyspnea, pulmonary edema and hemoptysis, which may persist for several weeks.

Gastrointestinal symptoms like nausea, vomiting, dysphagia, abdominal pain and hypovolemia due to GI fluid loss may occur.(3) In our patient, gastrointestinal bleeding occurred 3 days after ingestion.

Other systemic effects include heart failure, acute renal failure and hepatic failure. Metabolic acidosis results from multisystem failure. Manifestations of renal and hepatic failure usually develop two to five days later. Refractory hypoxemia due to pulmonary fibrosis usually manifests after 5 days from ingestion.(3) This may explain normal oxygen saturation in our patient at early few days after intoxication.

Paraquat qualitative and quantitative assay in blood or urine may be diagnostic.(1, 4, 5) There are some nomograms for survival prediction based on paraquat plasma level.(1)

For determination of severity and prognosis of acute paraquat poisoning, we can use the initial arterial lactate level and 12-hour lactate clearance.(6)

Other practical tools for toxicity monitoring are chest radiographs, serial pulmonary function tests and alveolar-arterial gradient.(3)

Patient's clothes should be removed and the skin should be decontaminated by water and mild detergent. Any conjunctival exposure should be irrigated by copious amounts of saline or water.

Exposure to paraquat is an emergency condition and all the patients, even asymptomatic patients, should be hospitalized. This may be the most important pitfall in clinical management of our patient. Similar to other toxicities, early decontamination is crucial to prevent more absorption.

The treatment of paraquat poisoning is mostly supportive. Since oxygen can form superoxide radical, administration of supplemental oxygen should be withheld unless the patient is hypoxic.(3)

Correction of fluid and electrolytic abnormality and

**Table 1.** Laboratory result after hospitalization

<b>Count blood cells</b>	<b>Urine analysis</b>
White Blood Cells:12500 /ml	blood: +++
hemoglobin: 11.7 G/DL	White Blood Cells: 8-10 /ml
Platelet Count: 208000 /ml	red blood cells: many
<b>Biochemistry</b>	<b>Arterial Blood Gas</b>
Total Bilirubin: 6.7 mg/dl	PH: 7.33
Direct Bilirubin: 6 mg/dl	PCO <sub>2</sub> : 37 mmHg
Creatine phosphokinase: 4508 IU/L	HCO <sub>3</sub> : 19 mEq/L
Aspartate transaminase: 220 IU/L	BE: -4
Alanine transaminase: 183 IU/L	
Creatinine: 13.6 mg/dl	
Blood urea nitrogen: 97mg/dl	
Alkaline phosphatase: 387 IU/L	
Na: 139 mmol/L	
K: 5.8 mmol/L	

maintaining urinary output is crucial to prevent pre-renal azotemia. Maintaining urinary output and renal function assist poison excretion and preventing toxic accumulation.(3)

A single dose of activated charcoal is recommended. Charcoal hemoperfusion has some benefits and should be started as soon as possible and continued for six hours but its efficacy is not approved.(1) According to a study in 2000, charcoal hemoperfusion could not decrease the paraquat mortality.(7)

High doses of cyclophosphamide and dexamethasone have been used for treatment of severe paraquat poisoning, but one study has been proved more benefits with pulses of cyclophosphamide and methylprednisolone.(8)

According to the results of some Randomized Clinical Trails, treatment with glucocorticoid plus cyclophosphamide in addition to standard treatment, have some benefits in patient with paraquat-induced pulmonary fibrosis.(9) It should be noted due to gastrointestinal bleeding, we could not administer any of these therapeutic agents for our patient.

### CONCLUSION

Paraquat poisoning should be kept in mind in any case of multisystem failure due to poisoning. According to the life threatening effects of paraquat poisoning, all cases of paraquat intoxication, regardless of symptoms, must be hospitalized. Many supportive measures and treatments must be carried out for these patients to save the patient's life.

### ACKNOWLEDGEMENT

This study was supported by a grant from the Vice Chancellor for Research of the Mashhad University of Medical Sciences.

**Competing interest:** None to be declared.

**Funding and Support:** None.

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