

Paraquat poisoning: A case report

Aliasghar Manouchehri¹  

¹Assistant Professor, Department of Internal Medicine, Shahid Beheshti Hospital, Babol University of Medical Sciences, Babol, Iran. Email: drmanouchehri@yahoo.com Corresponding Author, Dr. Aliasghar Manouchehri; Assistant Professor, Department of Internal Medicine, Shahid Beheshti Hospital, Babol University of Medical Sciences, Babol, Iran. Email: drmanouchehri@yahoo.com

Article Info

Article type:
Case report

Article History:

Received: 02 Oct 2022

Received in revised form:
09 Nov 2022

Accepted: 11 Nov 2022

Published online: 31 May
2023

Keywords:

Paraquat, Poisoning, WHO, Toxicity, Toxicology

Abstract

Objective: According to world health organization (WHO), paraquat is categorized as moderately hazardous, but its ingestion is associated with high toxicity and mortality and there is no specific antidote for paraquat poisoning.

Case report: A 75-year-old female was admitted to the emergency room with an alleged history of ingestion of 1 glass of paraquat (liquid form). Examination of oral cavity showed tongue and mucosal erosion. Cardiovascular, chest, and CNS examinations were normal. Gastric lavage was performed and she received IV fluids and an antiemetic as a supportive measure. Intravenous steroids and N-Acetylcysteine and proton pump inhibitors were added to the treatment. Input/output charting and vital monitoring was done. Her initial chest X ray, ECG, and abdominal ultrasonography were normal. Initially, her CBC, Electrolytes, liver function tests, and kidney function tests were within the normal ranges. During the hospital stay, blood creatinine increased to 2.2 mg% and kidney function tests deteriorated gradually. The patient developed AKI and was supported with haemodialysis. Patient's condition improved over period of time, she started accepting orally and urine output was adequate. She was then discharged with stable vital signs and was asked to follow-up in the outpatient department. Early diagnosis, aggressive decontamination and supportive care should be established in paraquat poisoning.

Conclusion: Since there is no known antidote for it, further absorption must be prevented in order to manage paraquat poisoning successfully.

Introduction

Paraquat (1, r-dimethyl-4,4-bipyridium dichloride), is a widely used liquid herbicide which rapidly deactivates on contact with soil. According to world health organization (WHO), paraquat is categorized as moderately hazardous, but its ingestion is associated with high toxicity and mortality and there is no specific antidote for paraquat poisoning. Fatality of paraquat poisoning is between 60 and 80%. Paraquat poisoning leads to multisystem organ failure including lung, liver, kidneys and

cardiovascular system with the most severe damage to the lungs. Although there is an extensive access to paraquat, leading to high risk of exposure among agriculture workers, reports of this herbicide ingestion and poisoning are rare in Iran. Here, we report a case of acute Paraquat poisoning in which paraquat was orally consumed [1-5].

Case report

A 75-year-old female was admitted to the emergency room with an alleged history of ingestion of 1 glass of paraquat (liquid form). She had 2 episodes of vomiting and presented with complaints of discomfort in throat and epigastric pain. There was no history of loose stools, loss of consciousness, difficulty in breathing, seizures or fever. She had no medical or surgical history. On clinical examination. The pulse rate was 84 beats per min, regular. Blood pressure 125/84, with respiratory rate of 18.

Examination of oral cavity showed tongue and mucosal erosion. Cardiovascular, chest and CNS examinations were normal. Gastric lavage was performed and she received IV fluids and an antiemetic as a supportive measure. Intravenous steroids and N-Acetylcysteine and proton pump inhibitors were added to the treatment. Input/output charting and vital monitoring was done. Her initial chest X ray, ECG and abdominal ultrasonography were normal. Initially, her CBC, Electrolytes, liver function tests and kidney function tests were within the normal ranges. During the hospital stay, blood creatinine increased to 2.2 mg% and kidney function tests deteriorated gradually. The patient developed AKI and was supported with hemodialysis. Patient's condition improved over period of time, she started accepting orally and urine output was adequate. She was then discharged with stable vital signs and was asked to follow-up in the outpatient department.

Discussion

Toxicity of Paraquat is through redox cycling, leading to hydroxyl radical formation which destructs cell membranes and results in hepato\nephrotoxicity and pulmonary fibrosis. Ingestion of more than 30 mL of paraquat 20%-24% (w/v) causes death. In cases of oral consumption, paraquat causes oromucosal injuries and systemic toxicity. Early manifestations include gastrointestinal irritation, abdominal pain, nausea, vomiting and diarrhea [1, 9,14]. Parquet, being excreted mainly by kidneys, [15] is highly concentrated in lungs, leading to pulmonary congestion, oedema, hemorrhage and fibrosis through superoxide radical generation which interacts with

unsaturated lipids of cell membranes and causes extensive cellular necrosis and pulmonary damage. Respiratory failure is the major cause of death. Other features are elevated serum aminotransferase, alveolitis, metabolic acidosis, acute kidney injury, mitochondrial toxicity and hypotension [3, 6, 8]. There is no known antidote to paraquat poisoning and patients should receive supportive care and close observation. Gastrointestinal decontamination including activated charcoal and Fuller's earth is recommended for all patients as soon as possible. Extracorporeal removal techniques, in particular hemoperfusion and hemodialysis, if commenced within 4 hours of ingestion, decrease paraquat levels.

Other routine resuscitations including intravenous fluid administration and electrolyte abnormality correction is needed. Urine output quantification, daily routine laboratory tests and analgesia for pain control should be considered as ongoing management [2, 11, 16].

As the lung damage is induced by peroxidation biochemical mechanisms, anti-oxidant treatments seem to be potentially promising. Therefore a number of the potential treatments such as controlled hypoxia, vitamins C and E, superoxide dismutase, N-acetylcysteine, deferoxamine and nitrous oxide have been under study and are proven to be effective. Even thigh, none of the potential antidotes are specific to paraquat poisoning [4, 16]. Cyclophosphamide and corticosteroid suppress inflammatory mechanisms and so they are beneficial in preventing pulmonary fibrosis [10, 12].

Paraquat poisoning prognosis depends on the individual vulnerability and the amount of paraquat ingested. Initial arterial blood gas analysis, complete blood count, renal and pancreatic function tests and plasma paraquat concentration have high survival predictive values [5, 7, 13].

At present, since there is no proven antidote, such case reports may lead to more interest in randomized studies which will help to improve treatment and management protocols.

Conclusion

Early diagnosis, aggressive decontamination and supportive care should be established in paraquat poisoning. Since there is no known antidote for it, further absorption must be prevented in order to manage paraquat poisoning successfully.

Conflicts of Interest

The authors hereby declare no conflicts of interest.

Authors` Contribution

All authors contributed in the experiments, analysis and preparation of this manuscript.

Funding/Support

Not Applicable

References

1. Bhalla A. 2, 4-D (ethyl ester) poisoning: experience at a tertiary care centre in northern India. *Emergency Med J* 2008; 25(1):30-32. doi: [10.1136/emj.2007.050047](https://doi.org/10.1136/emj.2007.050047)
2. Esfahani M. Effects of pre-harvest application of parquat on grain moisture reduction, grain yield and quality of rapeseed (*Brassica napus* L.) cultivars. *Caspian Journal of Environmental Sciences*, 2012; 10(1): 75-82.
3. Manouchehri A, Ghareghani S, Shamaei S, Nilechi M, Bossaghzadeh F. A review on Aluminum phosphide (Rice Tablets) Poisoning; From Exposure to the Applicable and New Strategies of Clinical Management. *Advancements in Life Sciences*. 2021; 31; 8(4):326-32.
4. Mazhari, M., Ferguson, J. Bacterial responses to environmental herbicide pollutants (glyphosate and paraquat). *Caspian Journal of Environmental Sciences*, 2018; 16(1): 35-43. doi: [10.22124/cjes.2018.2780](https://doi.org/10.22124/cjes.2018.2780)
5. Hong SY, Yang DH, Hwang KY. Associations between laboratory parameters and outcome of paraquat poisoning. *Toxicol lett* 2000; 118(1-2): 53-59. doi: [10.1016/s0378-4274\(00\)00264-2](https://doi.org/10.1016/s0378-4274(00)00264-2)
6. Houze, P., et al., Toxicokinetics of paraquat in humans. *Human & Experim Toxicol* 1990; 9(1): 5-12. doi: [10.1177/096032719000900103](https://doi.org/10.1177/096032719000900103)
7. Ikebuchi J. Toxicological index of paraquat: a new strategy for assessment of severity of paraquat poisoning in 128 patients. *Forensic Sci Int* 1993; 59(2): 85-87. doi: [10.1016/0379-0738\(93\)90147-3](https://doi.org/10.1016/0379-0738(93)90147-3)
8. Jha VK, Kannapur AS, Hiremath R. Fatal paraquat poisoning: A case report and review of literature. *Curr Med Issues* 2020; 18(2): 142. doi: [10.4103/2230-8229.122023](https://doi.org/10.4103/2230-8229.122023)
9. Khosya S, Gothwal S. Two cases of paraquat poisoning from Kota, Rajasthan, INDIA. *Case Reports Critical Care* 2012; 2. doi: [10.1155/2012/652146](https://doi.org/10.1155/2012/652146)
10. Lin JL, Wei MC, Liu YC. Pulse therapy with cyclophosphamide and methylprednisolone in patients with moderate to severe paraquat poisoning: a preliminary report. *Thorax* 1996; 51(7): 661-663. doi: [10.1136/thx.51.7.661](https://doi.org/10.1136/thx.51.7.661)
11. Meredith T, Vale J. Treatment of paraquat poisoning in man: methods to prevent absorption. *Human Toxicol* 1987; 6(1): 49-55. doi: [10.1177/096032718700600108](https://doi.org/10.1177/096032718700600108)
12. Newstead C. Cyclophosphamide treatment of paraquat poisoning. *Thorax* 1996; 51(7): 659. doi: [10.1136/thx.51.7.659](https://doi.org/10.1136/thx.51.7.659)
13. Scherrmann J. Prognostic value of plasma and urine paraquat concentration. *Human Toxicol* 1987; 6(1): 91-93. doi: [10.1177/096032718700600116](https://doi.org/10.1177/096032718700600116)
14. Singh, S., et al., Fatal 2, 4-D (ethyl ester) ingestion. *JOURNAL- ASSOCIATION OF PHYSICIANS OF INDIA*, 2003. 51: 609-610. PMID: [15266931](https://pubmed.ncbi.nlm.nih.gov/15266931/)
15. Sittipunt C. Paraquat poisoning. *Respiratory care*. 2005. 50(3): 383-385. PMID: [15779152](https://pubmed.ncbi.nlm.nih.gov/15779152/)

16. Suntres, Z.E., Role of antioxidants in paraquat toxicity. *Toxicology*. 2002.180(1): 65-77. doi: [10.1016/s0300-483x\(02\)00382-7](https://doi.org/10.1016/s0300-483x(02)00382-7)