

Paraquat Poisoning (Lethal Herbicide): A Rare Case Report

Dr. Gunja Jain¹, Dr. Mayank Gupta², Dr. Sudhir Mehta³, Dr. Laxmikant Goyal⁴,
Dr. Prabhav Bhansaly⁵, Dr. Ajeet Singh⁶

^{1,4}Assistant Professor, Department of Medicine, S.M.S Medical College, Jaipur(Rajasthan) India

²Senior Resident, Department of Medicine, S.M.S Medical College, Jaipur(Rajasthan) India

³Senior Professor, Department of Medicine, S.M.S Medical College, Jaipur(Rajasthan) India

^{5,6}Junior Resident, Department of Medicine, S.M.S Medical College, Jaipur(Rajasthan)India

Abstract— Paraquat, chemically 1, 1' dimethyl-4-4'-bipyridinium, is a green colored herbicide. It is one of the most lethal herbicide and is hazardous in very small quantities. More than 93% of deaths by paraquat are suicidal in nature. Poisoning by paraquat is commoner in developing countries because of agriculture based economy. The presentation of paraquat poisoning varies from case to case and depends on the amount consumed. As the compound is highly lethal, an early suspicion & management is must to prevent complications. Present case is of paraquat poisoning with full blown early picture of mucosal ulceration, pneumonitis and acute kidney injury which later on complicated in esophageal perforation, pneumothorax and pneumomediastinum.

Keywords: Paraquat, AKI, Pneumomediastinum, Pneumonitis.

I. INTRODUCTION

Paraquat is a green colored, liquid corrosive and have aromatic odour. It is bipyridal herbicide, chemically 1, 1' dimethyl-4-4'-bipyridinium, and is a contact poison. At present it is the second highest selling herbicide globally, available in 20% solution and needs dilution before use¹. Most of the adult cases of poisoning are suicidal in nature. It acts by inhibiting reduction of NADP to NADPH during photosynthesis. It leads to production of reactive oxygen species which in turn results in destruction of lipid membranes of organelles.

There is scarcity of case reporting on paraquat in India². So this rare case of adult accidental paraquat poisoning with acute and late complications was decided to report.

II. METHODOLOGY

A case of Paraquat poisoning attended at Department of Medicine, SMS Medical College, Jaipur (Rajasthan) India. It is one of the most lethal herbicide and is hazardous in very small quantities. This case is very rare, so case study was done thoroughly and case report was prepared to publish.

III. CASE REPORT

A young male of 24 years, farmer by occupation had an accidental history of paraquat intake and was admitted in Medicine department of SMS hospital, Jaipur. He was admitted 38 days after consuming paraquat with late and dreaded complications.

Before this the patient was admitted in a local hospital for the same. After consuming paraquat he had severe vomiting around 15-20 times a day followed by reduced urine output, development of mouth ulcers and sore throat for which he was hospitalized immediately at a local hospital. Laboratory reports of that time revealed occurrence of acute kidney injury (AKI) with serum creatinine-4.1mg/dl on 3rd day of consumption. He also developed cough and shortness of breath on third day of admission which

progressed rapidly. Discharge summary from peripheral hospital revealed bilateral crepitations and reduced oxygen saturation at that time with chest x-ray PA view suggestive of bilateral pneumonitis.

He was managed conservatively with i.v. methyl prednisolone, Vit E, Vit C and moist oxygen to maintain Spo₂ of 88-90%.

After a few days, patient became hypoxic and USG pleural space was suggestive of right pneumothorax. Right sided intercostal drainage tube (ICD) was placed. Patient showed clinical improvement and chest x-ray revealed lung expansion but after a few days, he was referred to SMS tertiary hospital (38 days after ingestion of paraquat) with complaints of neck edema & puffiness of face. Chest X-Ray PA view on admission was suggestive of pneumomediastinum due to esophageal perforation and subcutaneous emphysema.

ICD tube was re-positioned and feeding jejunostomy was done for enteral nutrition. ICD tube was taken out after 7 days of hospitalization with evidence of resolved subcutaneous emphysema.

This patient developed oral ulcers, acute kidney injury (AKI) and pneumonitis initially, which was conservatively managed and improved. Later on he developed complications like esophageal perforation and pneumothorax.

He was discharged afterwards with stable vitals and saturation of about 94% (off oxygen). Patient complained of persistent cough with breathlessness on exertion on next follow up. Physical examination revealed bilateral crepitations. Imaging in the form of High Resolution Computed Tomography thorax (HRCT) was done suggestive of fibrosing alveolitis.

IV. DISCUSSION

Paraquat is a bipyridal herbicide, caustic in nature, the mucosal injury can be very severe after ingestion of solution.³ If taken orally, it is sequestered in lungs against gradient and releases superoxide ions and free radicals leading to disruption of lung architecture. In life threatening cases of paraquat toxicity, histopathological examination ranges from pulmonary edema, hemorrhage to extensive fibrosis.⁴

Renal failure results from direct toxic effects, hemodynamic changes and sepsis.⁵ Preservation of renal function is essential to reduce serum levels of toxin and thereby reduced accumulation in lung³.

Diagnosis of paraquat poisoning is made by circumstantial evidences; it is wise to have an idea of ingested amount of poison to predict survival. Urinary paraquat levels of <1 mg/l in 24 hours of poisoning has higher chances of survival.^{6, 7, 8} As this present patient reported to us after 38 days of poisoning, serum and urinary paraquat levels were not done.

Diagnosis was confirmed by clinical history, presentation of patient and documentation of poison as paraquat. Treatment of paraquat poisoning involves immediate induced emesis, gastric lavage by fuller's earth or activated charcoal. Supplemental oxygen should not be given until Po₂ <70 mm Hg, as oxygen inhalation may lead to free radical generation and more damage to lungs.⁹ Hemoperfusion with activated charcoal reduces paraquat levels. It is effective only if done in less than 4 hours of ingestion as peak concentration in lungs reaches in 5-7 hours.⁵ Hemodialysis is supportive, but it doesn't increase the clearance because of rapid distribution of paraquat.⁵ Immunotherapy with cyclophosphamide and methylprednisolone helps by preventing ongoing inflammation.¹⁰ Antioxidant treatment in the form of controlled hypoxia, Vit C, Vit E, N-acetyl cysteine (NAC), desferrioxamine and nitrous oxide although used but showed no benefit.^{11,12}

In present patient steroid, Vit C, Vit E, NAC and i.v. antibiotics were given along with insertion of ICD because of pneumothorax. Feeding jejunostomy was done for enteral nutrition.

This present case is probably one of the few first cases reported in Rajasthan.

V. CONCLUSION

Paraquat poisoning leads to dreaded life threatening complications and has high mortality. Early presentation of patient within 6-8 hours of ingestion and lesser quantity of ingestion (20mg/kg) has better survival rates. No antidote is available till date. Early diagnosis and rigorous management is essential to save lives. Combination of supportive measures, immune suppression and antioxidant medication is the key to the management of paraquat poisoning.

CONFLICT OF INTEREST

None declared till now.

REFERENCES

- [1] Arts J, Schuit G, Schipper A, Kleij van der B. A case report of paraquat poisoning. *Eur J Hosp Pharm.* 2006; 12:22-4.
- [2] S. Singh, S. Yadav, N. Sharma, P. Malhotra, and P. Bambery. Fatal 2,4-D(ethyl ester) ingestion. *Journal of Association of physicians of India.* 2003; 51:609-610
- [3] Dinis-Oliveira RJ, Duarte JA, Sanchez-Navarro A, Remiao F, Bastos ML, Carvalho F. Paraquat poisonings: Mechanism of lung toxicity, clinical features, and treatment. *Crit Rev Toxicol.* 2008;38:13-71.(PubMed)
- [4] Blake D K, Gallegher R T, Wollen B H. Improved methods for the analysis of paraquat in biological fluids. *Chromatographia.* 2002; 55(Suppl):S183-S5.
- [5] J.S. Sandhu, A. Dhiman, R. Mahajan, and P. Sandhu. Outcome of paraquat poisoning –a five year study. *Indian journal of Nephrology.* 2003; 13:64-68
- [6] Soloukides A, Moutzouris DA, Kassimatis T, Metaxatos G, Hadjiconstantinou V. A fatal case of paraquat poisoning following minimal dermal exposure. *Ren Fail.* 2007; 29:375-7.(PubMed).
- [7] Sandhu JS, Dhiman A, Mahajan R, Sandhu P. Outcome of paraquat poisoning- a five year study. *Indian j Nephrol.* 2003;13:64-8.
- [8] Botella de Maglia J, Belenguer Tarin JE. Paraquat poisoning: A study of 29 cases of and evaluation of the effectiveness of the Caribbean scheme. *Med Clin (Barc)* 2000; 115:530-3. (PubMed).
- [9] K.R. Olson. *Poisoning in Current Medical Diagnosis Treatment.* Eds., p.1663, McGraw Hill, New York, NY, USA, 46th edition, 2007.
- [10] S. Singh, P. Bambery, D. Chaudhary, G. Makharia, N. Kakkar, and D. Singh. Fatal paraquat poisoning: report of two cases. *Journal of Association of physicians of India.* 1999;47(8)831-832
- [11] Z. E. Suntres. Role of antioxidants in paraquat toxicity. *Toxicology.* 2002; 180(1):65-77
- [12] M. Eddleston, M. F. Wilks, and N. A. Buckley. Prospects for treatment of paraquat-induced lung fibrosis with immunosuppressive drugs and the need for better prediction of outcome: a systematic review. *QJM.* 2003;96(11):809-824.