ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 08 | August - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

"THE SIP TO DEATH"- A CASE REPORT OF PARAQUAT POISONING

Anaesthesiology

Dr. Parvathy R S* MD General Medicine, DrNB Resident in Critical Care Medicine, Ananthapuri Hospital & Research Institute Thiruvananthapuram, Kerala, India *Corresponding Author

Dr. Anilkumar Asokan MD (Anaes), DA, DNB (Anaes), MNAMS HOD - Department of Critical Care, Ananthapuri Hospital & Research Institute Thiruvananthapuram, Kerala, India

Dr. Karthika Asokan MD Anaesthesia, Fellowship in Critical Care, Consultant, Department of Critical Care, Ananthapuri Hospital & Research Institute Thiruvananthapuram, Kerala, India

Dr. Hari Krishnan Somasekaran MD (Anaes), DA(Anaes), FNB(Critical Care) Senior Consultant, Department of Critical Care, Ananthapuri Hospital & Research Institute Thiruvananthapuram, Kerala, India

ABSTRACT

Paraquat ingestion is a leading cause of fatal poisoning in many parts of Asia, the Pacific nations, and the Americas [1]. Poisoning by pesticides is a major problem in low-income and middle-income countries. Paraquat is a toxic bipyridylium herbicide and its ingestion in a small amount can be fatal with toxic effects on the kidney, liver, lungs, gastrointestinal tract, and other organs(2). No specific antidote is available for this poisoning, and there is a dearth of evidence-based recommendations for the treatment (3). Here we present a patient who was admitted with paraquat poisoning, he had lethal effects of poisoning and met with death.

KEYWORDS

Paraquat Acute Kidney Injury Mucosal Erosion Acute Lung Injury

INTRODUCTION

Paraquat is a nonselective herbicide that is readily available in India. Since it is relatively inexpensive, it is broadly utilized in developing countries. Paraquat is safe to use in agriculture, however, dermal or spray exposure commonly causes minimal, localized injury in humans [4]. Nevertheless, accidental or deliberate ingestion has an extremely high case-fatality rate [5]. After ingestion, paraquat is absorbed and focussed inside many cells where it undergoes redox cycling. Over a period of hours to days, it can lead to multi-organ failure. It will mainly affect organs with high blood flow, oxygen tension, and energy requirements, mainly the lungs, heart, kidneys, and liver [6]. Excessive case fatality of paraquat is due to both its inherent toxicity and the lack of any effective treatment. There is no much data or guidelines regarding the management of this toxic compound poisoning. Ingestion of huge amounts of liquid concentrate (>50–100 ml of 20% ion w/v) results in fulminant organ failure: pulmonary oedema, cardiac, renal & liver failure and convulsions due to CNS involvement.

Case Report

A 68-year-old man, known case of systemic hypertension was admitted to our facility as alleged history of ingestion of poison Paraquat (PARAQUAT DICHLORIDE 24%) 150 ml mixed in juice on previous night. After that, he had multiple episodes of vomiting and green coloured loose stools. Gastric decontamination done from local hospital. On admission, he was afebrile (temperature: 98°F), his pulse rate was 104 beats /minute, blood pressure was 170/90 mm Hg and peripheral oxygen saturation was 98% on room air. Examination revealed ulcerated, tongue and oral mucosa with erosions. Baseline renal function values were normal, serial monitoring showed worsening renal function and had decreased urine output. Serial ABG showed metabolic acidosis. Haemodialysis was started for the management of oliguric acute kidney injury.

Table.1.laboratory Investigation Report After Admission

Investigations	Day1 (Morning)	Day 1 (evening)	Day 2
Blood urea nitrogen	31	47	47
S Creatinine	0.8	1.5	2.7
Bilirubin (Total/Direct)	0.6/0.1	1.9/0.5	3/0.6
SGOT	54	284	332
SGPT	41	102	155
PT	11.6	18	34.6
INR	0.84	1.5	2.87
S Lactate	3.9	4.6	6

Table-2 Serial ABG Analysis After Admission

DATE & TIME	DAY 1, 5am	DAY 1 12 pm	DAY 1 8 pm	DAY 2 6 am	DAY 2 12 pm
Ph	7.36	7.15	7.29	7.13	7.01
Pco2	28	25	30.5	40.9	38.3
Po2	87	80	119	88	73
HCO3	15.9	13.9	14.6	24.6	12.5
Base excess	-8.6	-10.4	-11	-4.6	-10.6

Initial radiography of the chest did not reveal anything unusual. He was put on injection N-acetyl-cysteine, Inj dexamethasone. He was managed conservatively. Towards evening patient had desaturation and was tachypneic (respiratory rate 30 breaths /minute) with associated bi-basal crackles. Radiography of the chest revealed bilateral minimal infiltrates in the lower zone. The patient was put on mechanical ventilation in view of progressive hypoxemia. Serial Arterial blood gas analysis showed metabolic acidosis. Later he developed hypotension, echo study done was normal, started on inotropic support. Coagulation parameters worsened on the next day. Liver function test showed features of transaminitis.However, he died within 2 days despite our best effort. Our patient had acute kidney injury, metabolic acidosis, pulmonary manifestations and oral mucosal erosions.

DISCUSSION

Poisoning by paraquat compounds is an important medical problem as it is associated with a very high mortality (case fatality rate of 50%–70%) and morbidity (7). Paraquat is a toxic bipiridyl compound, and it is lethal even in very small amounts (15–20 mL of 20% w/v). The mode of poisoning is usually suicidal or unintended. Paraquat poisoning should be suspected in cases of corrosive oral and oesophageal ulcerations in conjunction with features of hepatic, renal or pulmonary involvement (2).Paraquat concentrated within the cells undergoes redox cycling and cause cellular damage through lipid peroxidation, mitochondrial dysfunction, necrosis and apoptosis and triggers a pronounced secondary inflammatory reaction. Ingestion of smaller quantities mainly cause toxicity in the two key target organs (kidneys and lungs). Pulmonary lesion has two phases: an acute alveolitis over 1–3 days accompanied by a secondary fibrosis. The affected patient usually develops increasing signs of respiratory involvement over 3–7 days and in the end, dies of extreme anoxia because of rapidly progressive fibrosis up to 5 weeks later. Renal failure develops very rapidly, and creatinine and/or cystatin-C concentrations should be monitored from the first day to detect this group and it will predict long-term outcome (8, 9). Diagnostic tests consists of urine and serum paraquat levels, arterial blood gas analysis, arterial lactate, renal function test, chest imaging. Serum creatinine concentrations correlates with survival, increase greater than 4.3

μmol/L per hour (>0.049 mg/dL per hour) over six hours is associated with demise [10]. Our patient had mortality within 2 days of paraquat poisoning. No precise antidote is available for this poisoning, and there is dearth of evidence-based recommendation for the treatment (6). Treatment is mainly supportive. Gastric decontamination with activated charcoal is suggested in those presenting within 1–2 hours of ingestion; but gastric lavage should not be done due to the corrosive nature of paraquat. Oxygen therapy should not be administered except if there is confirmed hypoxia, as it is able to exacerbate the oxygen-mediated cellular harm. Treatment with hemoperfusion should be initiated within four hours of ingestion (11, 12). Extracorporeal treatment plans, such as intermittent haemodialysis or hemofiltration, are effective in enhancing removal (13). In acute paraquat poisoning, dexamethasone 8 mg intravenously (IV) every 8 hours for the first 72 hours is usually given. Immunosuppressive agent such as cyclophosphamide, along with antioxidants such as N-acetyl-cysteine and ascorbic acid, are under investigation, but its definite role is still unproven.

CONCLUSION

Paraquat is a rapidly acting herbicides and a leading cause of deadly poisoning. The outcome relies upon the severity of poisoning and time taken to avail medical help. The overdue complications encompass oropharyngeal ulcerations, oesophageal erosions, renal failure and pulmonary fibrosis. No antidote is available, so management is only supportive care.

Conflict of Interest: Nil

REFERENCES

1. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. BMC Public Health 2007; 7:357.
2. Somu B, Halkur Shankar S, Baitha U, et al. Paraquat poisoning. QJM 2020;113:752
3. Gawarammana IB, Buckley NA. Medical management of paraquat ingestion. Br J Clin Pharmacol 2011; 72:745–57.
4. Lock EA, Wilks MF. Paraquat. In: Handbook of Pesticide Toxicology, 3rd e d, Krieger RI (Ed), Academic Press, and San Diego 2010.
5. Senarathna L, Eddleston M, Wilks MF, et al. Prediction of outcome after paraquat poisoning by measurement of the plasma paraquat concentration. QJM 2009; 102:251.
6. Gawarmmana IB Buckley NA Medical management of Paraquat ingestion BrJ.Clin Pharmacology 2011:72:745
7. Sukumar CA, Shanbhag V, Shastry AB. Paraquat: the poison potion. Indian J Crit Care Med 2019;23:S263–6
8. Scherrmann JM, Houze P, Bismuth C, Bourdon R. Prognostic value of plasma and urine paraquat concentration. Hum Toxicol 1987; 6: 91–3.
9. Roberts DM, Wilks MF, Roberts MS, Swaminathan R, Mohamed F, Dawson AH, Buckley NA. Changes in the concentrations of creatinine, cystatin C and NGAL in patients with acute paraquat self-poisoning. Toxicol Lett 2011; 202: 69–74.
10. Roberts DM, Wilks MF, Roberts MS, et al. Changes in the concentrations of creatinine, cystatin C and NGAL in patients with acute paraquat self-poisoning. Toxicol Lett 2011; 202:6
11. Rao R, Bhat R, Pathadka S, et al. Golden Hours in Severe Paraquat Poisoning-The Role of Early Haemoperfusion Therapy. J Clin Diagn Res 2017; 11:OC06.
12. Wang HR, Pan J, Shang AD, Lu YQ. Time-dependent haemoperfusion after acute paraquat poisoning. Sci Rep 2017; 7:2239.
13. Park S, Lee S, Park S, et al. Concurrent Hemoperfusion and Hemodialysis in Patients with Acute Pesticide Intoxication. Blood Purif 2016; 42:329.