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Case Report

Paraquat poisoning-a dreadful and lethal poisoning: a case report of two cases from East Godavari, Andhra Pradesh, India

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ABSTRACT

Paraquat, a bipyridyl compound is used as a contact herbicide. It is a highly lethal compound with high mortality rates in cases of intentional ingestion for suicidal purpose. Although widely used in East Godavari District of Andhra Pradesh, India, cases of poisoning with this herbicide have been rarely reported from this region. This compound affects the lungs (leading to pulmonary fibrosis) and the kidney (leading to Acute Kidney Injury) the most. Ingestion of small quantities can cause death and lack of a specific antidote and specific treatment guidelines makes it a dreadful and lethal poison. We report two cases of suicidal ingestion of Paraquat which presented to the emergency department of KIMS and RF hospital, Amalapuram, East Godavari District, Andhra Pradesh, India.

Keywords: Paraquat, Poisoning, Lethal, East Godavari district

INTRODUCTION

Paraquat (1, 1'- dimethyl -4, 4'- bipyridinium) a nonselective contact herbicide is used for weed control and as preharvest defoliant.[1,2] Paraquat is one of the most toxic pesticides available, with ingestion of as little as 10 to 20 mL of the 20% wt/vol solution is sufficient to cause death. Overall, the mortality varies between 50% and 90% in various case series, but in cases of intentional self-poisoning with concentrated formulations, mortality approaches 100%.[1]

In the present study two cases of lethal paraquat poisoning was reported which was presented to the emergency department of KIMS and RF, Amalapuram, East Godavari District in the state of Andhra Pradesh, India which is a tertiary care teaching hospital in this region.

Paraquat is a rapidly-acting herbicide. It is rapidly absorbed from the gastrointestinal tract upon oral consumption. A peak level is reached at 60–90 min following ingestion regardless of the plasma paraquat levels.[2,3] Absorption through intact skin is minimal, and inhalation exposure does not occur, as paraquat has no appreciable vapour pressure.[4] Fatalities usually result from ingestion but have been reported after intramuscular injection, after vaginal and percutaneous exposure, and rarely after inhalation.[2]

On absorption, independently of the route of exposure, paraquat accumulates in the lung and the kidney, and these two organs are the most susceptible to paraquat induced injury. Only a small fraction of paraquat is metabolized, and the greater part is excreted unchanged in the urine. In humans the estimated lethal dose of 20% paraquat is 10-20 mL for adults and 4-5 mL for children.[2] In India, most of the concentrates of paraquat are available as 10-20% solutions, and therefore 10 ml of a 20% solution can contain about 2 grams of paraquat. Common brands available in India include Weedol, Gramoxone and Uniquat.[5]

Paraquat induces intracellular toxicity by the generation of reactive oxygen species that non-specifically damage the lipid membrane of cells, inducing cellular toxicity and death.[1] Generation of highly reactive oxygen and nitrite species results in toxicity in most organs and is particularly severe in the lungs as paraquat is taken up against a concentration gradient in to the lung.[6] Systemic effects of paraquat are renal and hepatic failure, pulmonary oedema and fibrosis, cardiac failure, shock, convulsions, and multiorgan failure.[7]

Clinical features are dependent upon the doses consumed and can be categorised into Typical form, Hyperacute form and Subacute form.[5]

Typical form (ingestion of 30 to 50 mg/kg of paraquat)

Typical form of poisoning passes through 3 phases. The initial phase is characterised by pain in the mouth, oesophagus, and stomach due to corrosion, vomiting, diarrhoea, dysphagia, and aphonia. There may be gastric perforation/GI haemorrhage. Second Phase begins after 2 to 5 days and is characterised by renal and hepatic toxicity, i.e. renal tubulopathy and centrilobular hepatic necrosis respectively. Third Phase begins after 5 days and is characterised by pulmonary fibrosis which leads to progressive respiratory failure.

Hyperacute form (ingestion of more than 50 mg/kg of paraquat)

Hyperacute form there is rapid development of cardiogenic shock ending in death in 3 to 4 days. Renal and hepatic lesions are also common.

Subacute form (ingestion of less than 30 mg/kg of paraquat)

The Subacute form is characterised only by gastrointestinal manifestations.

Diagnosis is mainly through history and clinical features. Specific assays are not urgent and essential as their results don't influence the management. Liver, renal and electrolyte studies, CBC, arterial blood gas, and chest radiography are useful.[2] Treatment is mostly supportive. There is no specific antidote for paraquat.[8] Immediate and aggressive GI decontamination in the form of gastric lavage preceded by activated charcoal is probably the only effective treatment that may affect the outcome significantly after paraquat ingestion.[2]

An open airway is maintained and ventilation assisted if needed. Fluid and electrolyte imbalance are corrected. Excessive oxygen administration is avoided, as oxygen is the substrate from which dipyridyls create harmful free radical species which will increase the injury.[2] Antioxidants like N-Acetyl Cysteine can be given, it has shown to reduce paraquat induced apoptosis and inflammatory response in human lung cultures.[9,10] In severe hypoxemia least possible amount of oxygen is given so that oxygen saturation is maintained above 60%.[2] Pain due to corrosive injury is treated with adequate doses of analgesics or opioids. Haemodialysis and forced dieresis do not enhance elimination, although renal failure may necessitate haemodialysis.[11,12] The combination of corticosteroids and cyclophosphamide has shown promise in reducing paraquat mortality, but a large randomised controlled trial is required to affirm the role of immunosuppression in paraquat poisoning.[6]

CASE REPORT

Case 1

A 25 year old female was admitted to the emergency department of our hospital with an alleged history of attempted suicide in which 10-15ml of liquid form of paraquat mixed in a soft drink was consumed at her residence 2 days back in the early morning hours when she was alone. Patient was admitted to a local hospital immediately by the parents with complaints of 20 episodes of loose watery stools and 8 episodes of vomiting.

History of consumption of the poison was concealed by the patient on the day of poisoning and none of their relatives were aware of the act. She was treated as a case of acute gastroenteritis at the local hospital and administered i.v. fluids, antiemetics, antibiotics and pre/probiotics and was kept under observation.

On the 3rd day of alleged poisoning she complained of burning sensation in the throat and mild discomfort in breathing and decreased urination. On repeated enquiry by the local physician, in the evening she revealed that she had consumed some herbicide kept in her home. Thereafter her relatives shifted her from the local hospital to our emergency department. Meanwhile poison container was recovered and compound was identified as paraquat.

Upon admission patient was having grade 4 shortness of breath and difficulty in swallowing liquids. On examination patient was conscious and coherent, pupils were normal in size and reacting to light, oral cavity showed extensive mucosal erosions over tongue, hard palate, oral mucosa without any active bleeding, had a pulse rate of 94/min, blood pressure of 120/80 mmHg, respiratory rate of 28/min and was afebrile. On auscultation of the lungs bilateral coarse crepitations were heard at the bases with normal vesicular breath sounds elsewhere. Abdomen was soft. Cardiovascular and neurologic examination revealed no abnormality. ECG was normal.

Gastric lavage was done and i.v. fluids, antibiotics, methylprednisolone, antiemetics, pantoprazole, N-acetylcysteine were administered to the patient and was admitted into intensive care unit for further care and

evaluation. At the time of admission patient had following blood parameters-serum creatinine 8.5, urea 180mg/dl, Alanine Transaminase (ALT) 668,Aspartate Transaminase (AST) 740, Alkaline Phosphate (ALP) 213, Total bilirubin 2.9 (direct 1.9,indirect 1.0), arterial blood gas analysis

(ABG) showed mild metabolic acidosis pH = 7.312 and pO2 of 57.5 mmHg, initial chest x ray showed bilateral lower lobe haziness (Figure 1). O_2 inhalation was given to maintain saturations above 80 mmHg. Immediate tracheostomy was done and mechanical ventilation was provided through the tracheostomy tube. ABG post intubation showed pH=7.047, pO2=35.6 mmHg and repeat X ray was consistent with ARDS (Figure 2).

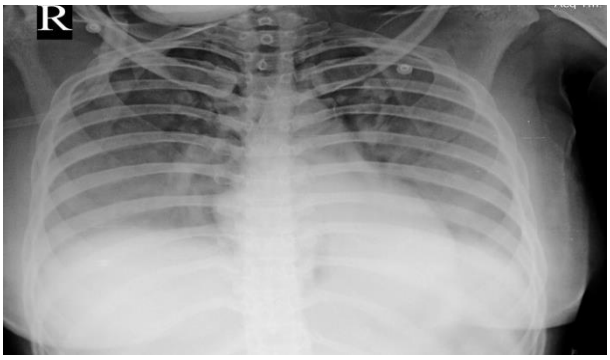

Figure 1: Initial chest x ray showing bilateral lower lobe haziness.

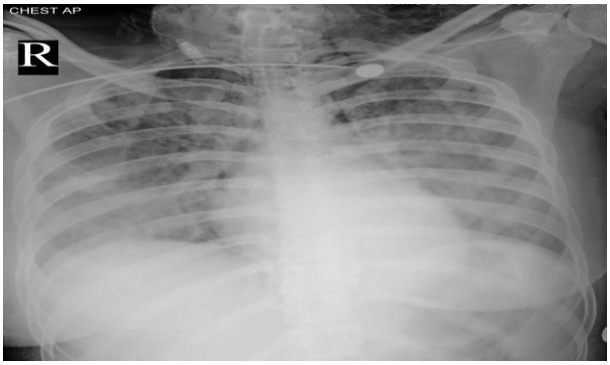

Figure 2: Chest x ray after intubation.

Routine urine examination revealed granular casts. Patient developed shock 14 hours post admission for which inotropic support was given. As patient had oliguria since time of admission kidney function test were repeated and serum creatinine was found to be 10.4.Hemodialysis could not be performed as patient was in shock. Patient finally succumbed to her illness 26hrs post admission.

Case 2

A 21 year old male was brought in an unconscious state to our emergency department with an alleged history of consumption of 100ml of paraquat poison 8 hours ago. At presentation patient was unconscious with a Glasgow coma scale of 3/15, pupils were mid dilated sluggishly reacting to light and conjunctival chemosis was present. Pulse and blood pressure were not recordable, breathing was shallow at a rate of 30/min and regular, heart sounds were muffled and diffuse crepitations were heard throughout the lung fields. Gastric lavage was done with administration of other supportive measures like i.v. fluids and inotropes. ECG showed sinus tachycardia.

Patient had severe hypoxia, ABG analysis showed pO2= 41.6mmhg, pH= 7.197, so mechanical ventilatory support was given. Blood parameters were as following-Serum creatinine 2.2mg/dl, AST 306 IU/L, ALT 339IU/L, ALP 136 IU/L, Total count 26000 with 87% neutrophils. Patient developed sudden cardiopulmonary arrest 2 hours post admission and died eventually although prompt treatment was instituted.

DISCUSSION

First case presented with a typical form of paraquat poisoning. She consumed 10-15ml of the compound and was seen by us on 3rd day after consumption of poison. She had features suggestive of renal and hepatic toxicity which is seen in the second phase (2-5 days) of typical form of poisoning.[5] Patient also had signs of pulmonary toxicity at the time of presentation which progressed rapidly to ARDS needing mechanical ventilation. Later on she developed shock and succumbed inspite of aggressive supportive management. A typical form of poisoning would pose a diagnostic challenge to any treating physician if history is inaccurate, as was evident in our 1st case. Paraquat poisoning may mimic acute gastroenteritis or an organophosphorous poisoning and the absence of any specific treatment guidelines or antidote further complicates the treatment.[8] Second case was a hyperacute form of paraquat poisoning, who had consumed around 100ml of paraquat and had developed features suggestive of hepatic and renal involvement within 8 hours of consumption and succumbed to shock within 2 hours of admission inspite of aggressive supportive management.[5]

It's important to distinguish this form of paraquat poisoning from conditions like septic shock or anaphylaxis from history, for which specific treatment guidelines are available. Hyperacute poisoning of paraquat is a rapidly progressive and fatal condition where the management is mainly supportive. Thus both typical and hyperacute forms of paraquat poisoning are lethal as seen in our cases.

CONCLUSION

Paraquat poisoning is a rare case of poisoning in East Godavari district of Andhra Pradesh, India. Diagnosis is difficult if history is not clear and the progression of disease is very fast leading to multiorgan failure, high clinical suspicion and prompt decontamination is

necessary. Even after administration of aggressive treatment, mortality remains high as evidenced in our cases thus making paraquat a dreadful and lethal poison for humans.

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REFERENCES

1. Roberts DM. Bipyridyl Compounds, Paraquat, and Diquat in Goldfrank's Toxicologic Emergencies, Lewis S. Nelson, Neal A. Lewin, Mary Ann Howland, Robert S. eds, McGraw Hill, NewYork, NY, USA. 2011;1502.
2. Geller RJ. Paraquat, Diquat in Poisoning and Drug Overdose. Olson KR, Anderson IB, Benowitz NL, Blanc PD, Clark RF, eds, 6th edition, McGraw Hill, New York, NY, USA. 2012;321-3.
3. Kang MS, Gil HW, Yang JO, Lee EY, Hong SY. Comparison between kidney and hemoperfusion for paraquat elimination. J Korean Med Sci. 2009;24:S156-60.
4. Costa LG. Toxic Effects of Pesticides in Casarett and Doull's Toxicology- The Basic Science of Poisons, Curtis D. Klaassen, ed 8th Edition, McGraw Hill, NewYork, NY, USA, 2013;961.
5. Pillay VV. "Pesticides" in Modern Medical Toxicology, V V Pillay,ed, 4th Edition, Jaypee Brothers, New Delhi, India. 2013;398-400.
6. Agarwal R, Srinivas R, Aggarwal AN, Gupta D. Immunosuppressive therapy in lung injury due to paraquat poisoning: a metaanalysis. Singapore Med J. 2007;48(11):1000-5.
7. Vale JA, Meredith TJ, Buckley BM. Paraquat poisoning: Clinical features and immediate general management. Hum Toxicol. 1987;6:41-7.
8. Khosya S, Gothwal S. Two Cases of Paraquat Poisoning from Kota, Rajasthan, India. Case Rep Crit Care. 2012.
9. Cappelletti G, Maggioni MG, Maci R. Apoptosis in human lung epithelial cells: triggering by paraquat and modulation by antioxidants. Cell Biol Int. 1998;22:671-8.
10. Yeh ST, Guo HR, Su YS, Lin HJ, Hou CC, Chen HM, et al. Protective effects of Nacetylcysteine treatment post-acute paraquat intoxication in rats and in human lung epithelial cells. Toxicology. 2006;223:181-90.
11. Wong OF, Fung HT, Kam CW. Case series of paraquat poisoning in Tuen Mun Hospital. Hong Kong J Emerg Med. 2006;13:155-60.
12. Pavan M. Acute kidney injury following Paraquat poisoning in India, Iran J Kidney Dis. 2013;7(1):64-6.

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