CASE REPORT

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Rare Survival from Paraquat Poisoning: 'A Case Report'

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Abstract

Paraquat is a highly toxic herbicide which is most commonly used in agriculture to kill weeds. Paraquat (bipyridyl) is a corrosive liquid which is green in colour with pungent smell. This is a case report of a 26-year-old male, who was admitted in SVBP Hospital emergency with history of alleged intake of paraquat poison. On admission, the patient was in severe respiratory distress. Due to prompt and aggressive management, the patient improved and got discharged. Although there is no proper management protocol and antidote available for managing paraquat poisoning, proper strategies and effective management of ARDS, along with early intervention to prevent complications is the key to patient survival .

Key words: Paraquat poisoning, ARDS.

Introduction

Paraquat, whose chemical composition is 1,1-dimethyl-4, 4 bipyridyl dichloride, is commonly used herbicide in agriculture because it is rapidly inactivated in soil. It prevents photosynthesis in plants by interfering with ETC (electron transport chain). It is green coloured liquid which often is fatal on ingestion. When paraquat is taken orally , it causes release of hydrogen and *superoxide* anions. These anions cause lipid damage in the cell membrane and lead to pulmonary fibrosis, hepatotoxicity, and nephrotoxicity[4]. Paraquat exposure has a high case fatality rate of up to 68 - 74%[1]. It presents with a wide variety of clinical symptoms including nausea, vomiting, dyspnoea, oral ulcers (characteristically called as paraquat tongue, which are mucosal lesions in the oral cavity)[2]. Additional symptoms like abdominal pain, diarrhoea, altered mental status and malaena has been reported in some cases[2].

The primary organs affected by paraquat poisoning are the lungs and kidneys. Its chemical resemblance to polyamines facilitates its uptake by the alveolar cells. Additionally, the kidneys actively secrete paraquat, resulting in its accumulation within the proximal tubular epithelial cells. When paraquat accumulates in the pulmonary alveoli and nephrons, it induces redox cycling and generates harmful reactive oxygen species. This oxidative stress surpasses the cellular defense mechanisms, leading to pulmonary injury characterised by alveolitis and fibrosis, which arises from the proliferation and differentiation of fibroblasts[1]. It causes pulmonary fibrosis, hepatic toxicity and nephrotoxicity by free radical damage. There is currently no antidote available and only supportive management is provided.

Case Summary

A 26 years male with an alleged history of parquat ingestion presented to us with chief complaints of pain abdomen, breathlessness and vomiting. Patient was referred from district hospital, Amroha, 4 hrs after the ingestion of approximately 30 to 40 mL of a 20% paraquat solution as suicidal attempt. On examination, patient had tachypnoea and was using his accessory respiratory muscles. His vitals on presentation were: BP - 158/96 mmHg, PR - 98 bpm, SpO2 - 88% on room air. His random blood sugar was 111 mg/dL. On local examination, oral ulcers were observed with characteristic paraquat tongue (Fig. 2). On respiratory system examination, crepitations were present more on the right side of chest in the infra-axillary and mammary area, and on left side only basal crepitations were present. On abdominal examination, mild epigastric tenderness was present. Patient also had decreased urine output of 500 mL/24 hr. On investigations, ABG analysis showed mild hypoxaemia with PaO_2 of 81 mmHg, PH 7.4, and HCO_3 - 22.9 meq/L with serum lactate - 1.1 meq/L (Fig. 3). The bicarbonate PaO_2/FiO_2 ratio on presentation was approximately 180, indicating moderate ARDS. The ROX index calculation was also suggestive of respiratory compromise. Aspartate transaminase (AST) and alanine transaminase (ALT) levels were 20 and 36 U/l, respectively; serum creatinine and blood urea levels were 2.4 mg/dL and 144 mg/dL, respectively. Chest X-ray (PA view) on the day of admission showed bilateral basal haziness and some fibrotic changes more on right side with heterogenous opacity over right lower zone (Fig. 1). High resolution CT scan of the chest was done, which revealed bilateral ground-glass opacities and consolidation.

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Complete haemogram showed mild leuocytosis with TLC - 11,980 cells/dL, haemoglobin of 12.6 g/L and platelet count of 154,000 per cumm. Patient was shifted to ICU, gastric lavage was done with normal saline and activated charcoal (1 gm/kg). As there was no dermal exposure, hence dermal decontamination was not required. For management of respiratory distress, patient was managed conservatively on intermittent Bi PAP along with methylprednisolone 500 mg iv, tab. cyclophosphamide and antioxidant (NAC and vitamin E 800 mg TDS vitamin C - 500 mg BD, glutathione 50 mg BD). Immunosuppression therapy along with antioxidants were given in accordance with Dinis-Oliveira recommendations. Nephrology consultation was taken in view of acute kidney injury, along with strict monitoring of urine output. Haemodialysis was not initiated due to improving renal functions. Patient was closely monitored with hourly blood pressure and oxygen saturation monitoring, daily kidney function tests, and serum electrolytes. ABG analysis was done every 4 hourly. Patient showed gradual improvement with better renal and hepatic profile, improved oxygenation with saturation of 93% on minimum oxygen requirement (1 L/min oxygen) by nasal prongs. On day 2, ABG revealed mild partially compensated metabolic acidosis with some hypoxaemia PH - 7.32, PaO_2 - 98 mmHg, HCO_3 - 21. Serial renal function tests showed improvement and ABG also reflected improved

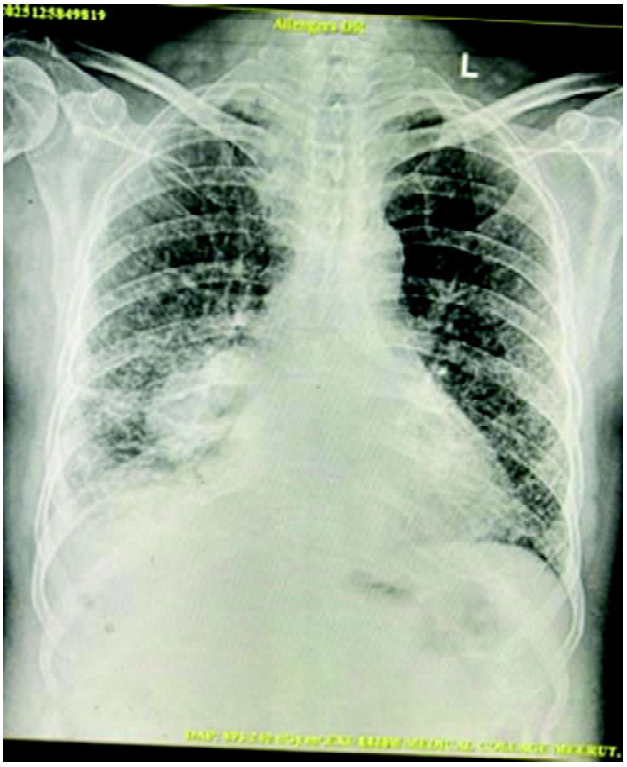

Fig. 1: *Chest X-ray showing bilateral basal haziness and heterogenous opacity over right lower zone.*

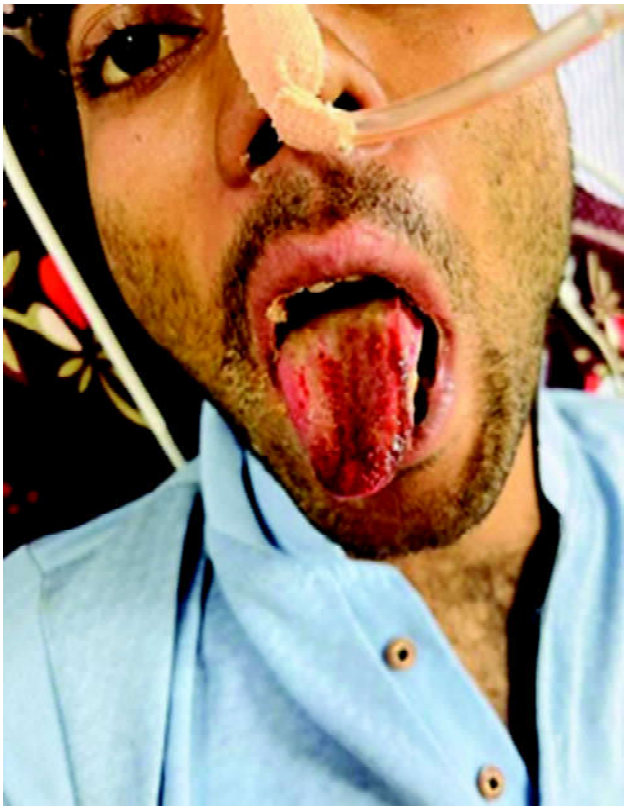

Fig. 2: *Paraquat Tongue.*

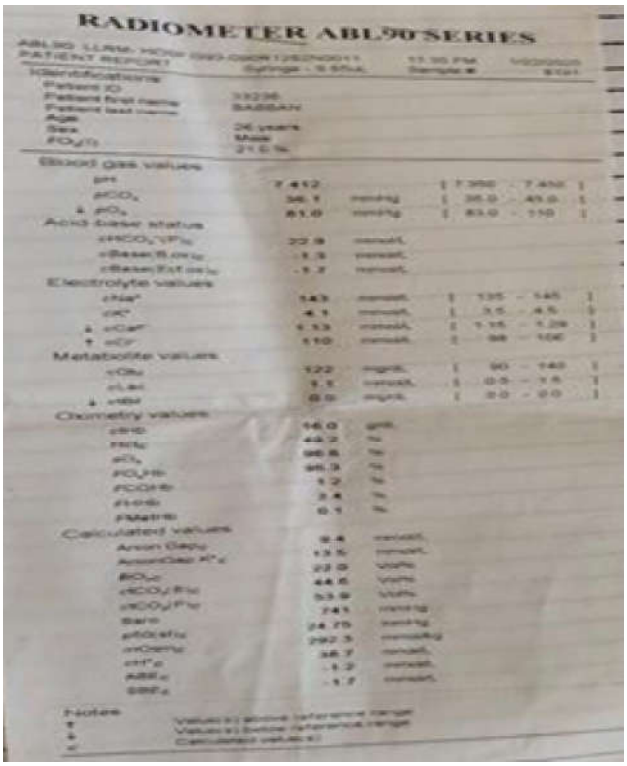

Fig. 3: *ABG report.*

oxygenation status with improvements in lactate levels. He maintained oxygen saturation on intermittent oxygen support of 1 - 2 L/min on nasal prongs. Patient continued to show further clinical improvement and was discharged on day 5.

Discussion

Paraquat when taken orally is highly toxic therefore, it has been classified as "restricted use herbicide". GI decontamination is most effective when done within one hour of exposure, but our patient was a referred case, and he reported to us after 4 hours. Even though there is less benefit of GI decontamination after 3 to 4 hours, we decided to do gastric lavage with charcoal as it was not clear from the referral whether it was done at the primary health centre. There was no dermal exposure in our case so dermal decontamination was not required. Paraquat causes release of super oxide anions which cause lipid damage of the cell membranes in the lungs in the form of pulmonary congestion and fibrosis. In our case also, pulmonary involvement was present in the form of breathlessness, increased respiratory rate, crepitations in mammary and infra-axillary areas with consolidation on chest X-ray, as paraquat has high affinity for alveolar type 1 and type 2 cells. Its concentration in lungs is 10 - 20 times greater than in plasma[5]. Excessive oxygen supplementation should be avoided in paraquat poisoning, as it can increase reactive oxygen species (ROS) production. Therefore, we should provide minimal required oxygen therapy to avoid further free radical injury. Paraquat causes local toxicity in the tongue, oral mucosa like a corrosive injury which was also present in our case (Fig. 2). Renal tubular necrosis due to free radical damage can cause nephrotoxity, though in our case there was only minimal involvement of kidneys in the form of decreased urine output which was improved on conservative management[6]. The best predictor of survival after ingestion is calculated from the time of ingestion and the start of treatment, and also the concentration of paraquat in plasma. The index is calculated by multiplying the time from parquat ingestion to the start of treatment.

Conclusion

Paraquat is highly toxic poison which predominantly affects the vital organs. As there is no specific antidote available, so supportive care focusing on prevention of free radical injury and airway protection are of paramount importance. Paraquat is banned in Europe, so we also recommend strict monitoring in India, to limit its misuse. Timely intervention and awareness can prevent many deaths.

Limitations

There was certain limitation in our report like paraquat levels in plasma and urine were not measured, as this test is not available in our institute.

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