Case Report: Paraquat Poisoning Associated Multiple Organ Dysfunction Syndrome

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ABSTRACT
An 18-year female presented with an alleged history of the conception of liquid paraquet of an unknown quantity on 3rd August 2021. On examination, the patient was unconscious and disoriented. She was further diagnosed with Multiple Organ Dysfunction Syndrome. She had no pallor, icterus, cyanosis, clubbing, pedal adeno, and no lymphadenopathy. Paraquet is basically a toxic herbicide. It is a brown colored syrup liquid. It has chronic toxicity because of its rapid deactivation in contact with soil. After the ingestion, the herbicide is liable for further causative lesions. A very low dose or as little as one mouthful is very severe and dangerous. Although, Herbicides poisoning cases are very uncommon in India.

Key words: Paraquat poisoning, Herbicide, Multiple Organ Dysfunction Syndrome.

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INTRODUCTION

Paraquat is a very toxic herbicide used to kill weed insects on plants[1,2] In developing countries pesticides/herbicides consumption is very serious issue and major public problem. Paraquat ingestion has very serious harmful effects which can lead to often fatal toxicity.[3] Although, Herbicides poisoning cases are very uncommon in India. We are discussing a fatal suicidal case of fulminant paraquat poisoning characterized with Multiple Organ Dysfunction Syndrome. Severe paraquat poisoning is characterized by multiple-organ failure, involving predominantly the lungs, kidneys, and liver.[4] The lung is a major target organ in paraquat poisoning, and respiratory failure from lung injury is the most widely recognized cause of death. Early diagnosis and starting appropriate treatment as soon as possible are very important in such cases.[5,6]

CASE PRESENTATION

An 18-year-old female was admitted in the emergency ward presented with alleged history of consumption of liquid paraquet of unknown quantity at her residence on 3rd August, 2021. Initially managed at other hospital and came to our hospital for further management. She experienced nausea and vomiting sensation soon after the ingestion. She had sore throat and epigastric pain. On examination patient was unconscious and disoriented. She had no pallor, icterus, cyanosis, clubbing, pedal adeno and no lymph adenopathy. She had decreased output of and pain in opening her mouth. She had no history of vomiting, loose stools, stomach ache, seizures, or fever. She was diagnosed with acute kidney injury on dialysis. There was hepatopathy and urosepsis. Clinical examination revealed, the Pulse was recorded as 106/min, blood pressure (BP) was noted to be 120/70 mm/hg, with the respiratory rate was noted to be 22 per min. ECG readings showed faster heartbeat usual than normal rhythm which refers to Sinus tachycardia and also considered left ventricular hypertrophy (Figure 1). The Chest radiography was not clear and showed indefinite infiltrates (Figure 2). She had no difficulty in breathing, also there were no sounds found on the examination of respiratory system. Pupils were bilaterally equal and receptive to light. Performed Gastric lavage and treated with charcoal in the emergency department. In the mobile intensive care unit (MICU), she got IV fluids and an antiemetic as a supportive measure. Patient was treated with frequent doses of sorbitol and charcoal and also intravenous fluids and analgesics for the epigastric pain control.

In the next following 24 hr, she experienced increased epigastric pain, severe dysphagia, and shortness of breath. Blood and urine cultures were sterile. Other blood investigations including thyroid and liver functions were normal. Urine tests were normal. Ultrasound of the abdomen showed bilateral changes in the kidneys. Initial labs abg – severe lactic acidosis; WBC are 33,800, creatinine – 1.2. Inj. Meropenem IV, Infusion sodabirab and other supportive medication was started in view of no urine output. Nephrologist consultation was taken and dialysis was done, 2 cycles. SGOT – 51; SGPT: 20; ECG indicated sinus tachycardia. For hypotension inotropes and vasopressors where started guarded prognosis explained to family. On day-2 of ICU stay patient sensorium worsened with bradycardic arrest, emergency intubation was done and CRP initiated according to ACLS protocol, despite all efforts patient could not be revived and declared dead at 3:30 am on 05/08/2021.

DISCUSSION

After paraquat consuming, the poison is separated in lungs and releases superoxide anions and hydrogen which causes lipid damage in cell membrane and oxidant free radical damage which results in hepatic/nephrotoxicity.[3,7,8] Paraquat toxicity can show local and systemic effects. In this case, consumption of paraquat resulted in inflammation of the tongue, oral mucosa and throat, Acute kidney injury on dialysis, hepatopathy, and urosepsis.[9,10] Identification of paraquat poisoning in the urine confirmed the diagnosis.[11,12] As there is no proper proven

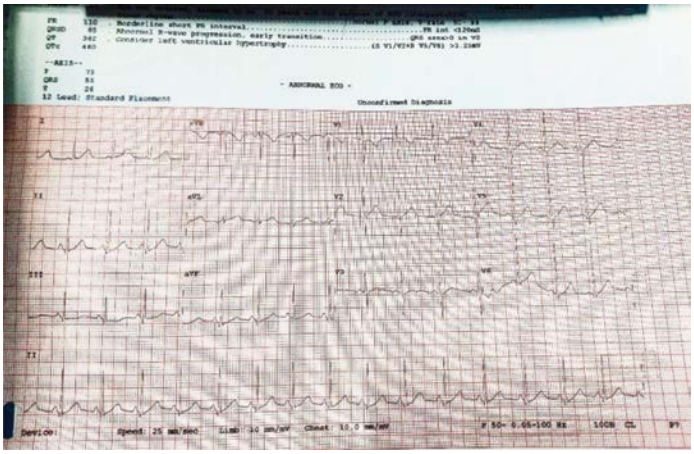

Figure 1: Sinus rhythm- normal P axis, V-rate 50-99. Borderline short PR interval, Abnormal R-wave progression, early transition and also considered left ventricular hypertrophy.

Figure 2: Chest X-ray posteroanterior (PA) view showing bilateral patchy consolidation involving left lower lobe.

antidote clinically for paraquat poisoning, supportive treatment is given to avoid free radical damage to lungs with pulse therapy using steroids, elimination of paraquat from circulation by doing hemodialysis, sodabirab is given to control the urosepsis i.e., lower the acid levels in urine. Regardless of the way that there have been isolated case reports of survivors (due to very low dose or effective and early treatment), an ingestion of a huge amount paraquat poisoning has a very poor cure rate.[3,13]

At this point, there is no specific fix to paraquat poisoning. In this way, it is suggested that the focus should be on preventive ideas and in exposure events, when it has been ingested, the foundation of forceful and strict decontamination to prevent further absorption.[11,12,14,15]

CONCLUSION

Management decisions are influenced by two opposing philosophies. The first recognizes that the outcome is bleak and that no treatments are likely to be effective, and aims to provide minimal low-risk interventions while managing to keep patients comfortable. The second acknowledges that the outcome is bleak however no treatment is highly probable to be much worse than disorder. This group specializes in HP/HD, immunosuppression, and a wide range of other treatments. We would recommend anyone who sees a high number of paraquat poisonings to use a coherent strategy for a large number of patients, measure the paraquat concentration and report their findings.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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