ORIGINAL RESEARCH PAPER
Volume - 10 | Issue - 01 | January - 2021 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CASE OF PARAQUAT POISONING PRESENTING WITH MULTIORGAN DYSFUNCTION

General Medicine

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ABSTRACT

- Self-poisoning with pesticides is a major public health problem in developing countries with an estimated 300 000 deaths each year.Very high case fatality (>50%) - due both to its inherent toxicity and the lack of early diagnosis and effective treatment.Though it is easily availabile, poisoning with this toxin is not common. Fatal dose of paraquat is so trivial that >10 ml poison can damage lungs permanently.
- In this context we present a fatal case of 30 yr old male patient who have consumed paraquat and presented with extensive complication of acute kidney injury, and lung injury and liver disease.

KEYWORDS

CASE REPORT:

30 years old male with no comorbidities and an alcoholic presented to medicine opd on with alleged history of paraquat ingestion of approximately 10ml , was treated in outside hospital with stomach wash and supportive treatment and referred to chettinad hospital and research institute . Patient had complaints of oral ulcers, decreased urine output ,difficulty in swallowing with history of hiccups and excessive salivation .

On examination patient was Conscious, oriented, tachypneic, GCS 15/15.E3V3M5.patient had Icterus ,but had no pallor, no cyanosis, no clubbing, no pedal edema, no generalized lymphadenopathy. Tongue excoriations were present and had pulse rate was 110 beats per min and Blood pressure was120/70 mmhg in right arm supine position and Respiratory rate was 28 / min abdominothoracic. with SpO2 was100% in room air. temperature - 102 degree Fahrenheit.

On respiratory system examination crepitations heard over both supra scapular and inter scapular region . Air entry is equal bilaterally and on examination abdomen was Soft, bowel sounds heard ,Epigastric tenderness was present with no guarding or rigidity and no organomegaly. Central nervous system examination revealed no focal neurological deficit. Cardiovascular Examination was normal

Routine investigations were sent. Complete blood count revealed raised leucocytosis with neutrophil predominance, raised urea and creatinine levels, liver function test and serum electrolytes were within normal limits and there were elevated bilirubin levels .Urine routine examination showed positive for albumin, ketone, sugar, Red blood cells and cast. PT,INR were prolonged.Radiological imaging showed ground glass opacities ,fibrotic changes were present.

At time of admission Patient presented with above mentioned complaints and admitted in ward, investigations showed features of Acute kidney injury, patient was shifted to ICU, and was taken up for one sitting of hemodialysis. Patient was put on antibiotics, thiamine supplementation, and pantoprazole infusion.Patient was started on dexamethasone and Hepatoprotective drugs were started and N-acetyl Cysteine infusion was given.

On DAY 5 Patient was shifted to ward. Patient had persistent hiccups.he was put on antispasmodics,antimotility anti oxidants, nebulization. Next day Patient developed sudden onset breathlessness, while on examination no respiratory signs were made out but a drop in oxygen saturation to 80% in room air was noticed , then patient was shifted to ICU.Patient was in ICU for 24 hrs, and improved hence was shifted back to ward.

Blood investigations	Day 1
BUN	9.1
CREATININE	6.80
URINE ALBUMIN	++
URINE SUGAR	+
URINE RBC	+
BILIRUBIN TOTAL/DIRECT	5.1/2.8
PT/INR	17.2/1.53

Patient was with oxygen supplementation maintaining a saturation of 95% in supine position and 85% in standing position.Patient was started on methylprednisolone, antibiotics, and vitals were continuously monitored.On DAY 12 .Total counts were continuously elevated.hence was planned for cyclophosphamide therapy,But patient was not willing for further management, hence discharged against advice.

Fig 1:Tongue ulcers

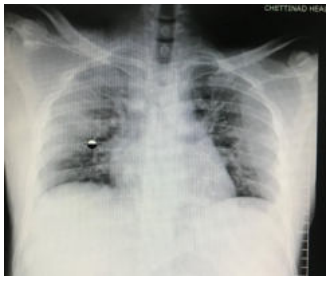

Fig 2 Chest x ray p/a view Expiratory film,rotated to tight B/L Diffuse alveolar infiltrates

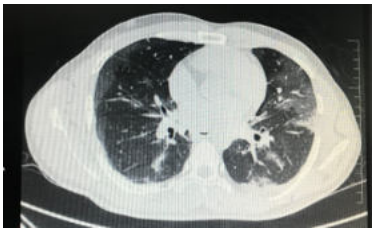

FIG 3 HRCT CHEST- Fibrotic changes with surrounding ground glassing haziness in bilateral lung fields predominantly involving upper lobes peripheral segment of bilateral lower lobes

DISCUSSION

PARAQUAT POISIONING[1]

Paraquat toxicity is most severe in the lungs and leads to an acute alveolitis.

Further effects include diffuse alveolar collapse, vascular congestion and adherence of activated platelets and polymorphonuclear leucocytes to the vascular endothelium.

The primary target of toxicity in the lung is the alveolar epithelium. During the acute 'destructive phase' both type I and type II pneumocytes demonstrate swelling, vacuolation and disruption of mitochondria and the endoplasmic reticulum. Sloughing of the alveoli is associated with pulmonary oedema

This initial phase is followed by a proliferative phase where the alveolar space is filled with mononuclear profibroblasts which mature into fibroblasts within days to weeks. This stage is followed by lung fibrosis.

Kidneys exposed to paraquat demonstrate development of large vacuolation in proximal convoluted tubules which leads to necrosis. Congestion and hepatocellular injury associated with rough and smooth endoplasmic reticulum degranulation and mitochondrial damage occur in the liver. These changes can be observed within a few hours to days.

MECHANISM OF TOXICITY[3]

Main mechanism include Generation of free radicals and oxidative stress -Redox-cycling and subsequent generation of reactive oxygen species (ROS).

Paraquat is metabolized by several enzyme systems - paraquat mono-cation radical, INTRA CELLULARLY and Generation of highly reactive oxygen and nitrite species results severe toxicity in the lungs.

Other mechanisms include Lipid peroxidation, Mitochondrial toxicity, Oxidation of NADPH And activation of nuclear factor kappa B (NF-κB) And which may lead to Apoptosis

CLINICAL MANIFESTSTIONS [4,5]

Mild - <20 mg of paraquat ion/kg body weight – symptoms of GIT – recover.

Moderate to severe – 20-40 mg paraquat ion/kg – renal, pulmonary – majority of them – death – delayed by 2-3 weeks.

Acute fulminant poisoning - >40 mg paraquat ion/kg – multi organ – death within hours or few days Renal failure develops quite rapidly, and creatinine and/or cystatin-C concentrations can be monitored over the first day.The pulmonary lesion has two phases-an acute alveolitis over 1–3 days followed by a secondary fibrosis.

The patient typically develops increasing signs of respiratory involvement over 3–7 days and ultimately dies of severe anoxia due to rapidly progressive fibrosis up to 5 weeks later.

TREATMENT[6]

1) Dexamethasone -There was a significant reduction of paraquat accumulation in the lung tissue and an increase of faecal excretion of paraquat.Ameliorate the histological and biochemical changes.
2) Moderate to severe paraquat poisoning treated with 1 g of cyclophosphamide daily for 2 days and 1 g of methylprednisolone daily for 3 days.
3) Vitamin E 200–4000 mg &Vitamin C has Ability to donate an electron to free radicals thereby neutralizing.
4) N-acetylcysteine (NAC) -Replenishes cysteine which is rate-limiting in the synthesis of glutathione, a critical antioxidant defence.Deferoxamine (DFO) Iron is an important contributor to the generation of ROS through the Fenton reaction &Salicylic acid have shown improvement

CONCLUSION

There are no widely accepted guidelines on treatment of patients with paraquat self-poisoning .Because small amount of paraquat poison leads to fatal outcome. Very high case fatality (>50%) - due both to its inherent toxicity and the lack of any effective treatment. There is no specific antidote for this poison. Treatment remains mainly supportive in nature, Immediate and adequate interventions play an important role in prognosis after ingestion of paraquat.

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