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CASE REPORT

PARAQUAT POISONING: A CASE REPORT

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ABSTRACT: Paraquat {PQ}, a herbicide available as 20% solution can cause lethal effects due to production of free radicals formed by the cyclic oxidation-reduction reactions of the compound with tissues resulting in multiorgan failure. Symptoms of PQ ingestion are usually dose-dependent, and intoxication can be categorized to mild, moderate, and fulminant. Most common symptoms being vomiting (100%) followed by oral ulceration (59%), dysphagia (53%) and dyspnea (41%). Diagnosis of PQ poisoning is usually made based on circumstantial evidences. PQ levels can be estimated and is of prognostic significance. Almost always PQ causes morbidty and mortality except in few cases where dose is inadequate. Here we present a case of 25 year old patient with PQ poisoning which resulted in oral mucosal and upper gastrointestinal ulcerations which subsequently healed with antioxidants, antibiotics and local applications of povidine iodine. As there were no respiratory symptoms cyclophosphamide or steroids was not used. Patient was discharged after 1 month of hospital admission with all parameters within normal limits. . In spite of advances in medical care, prompt treatment, and supportive care, mortality still remains high mainly due to multiorgan failure.
KEYWORDS: Paraquat, Poisoning, Haemoperfusion.

INTRODUCTION: Paraquat{PQ}, (1, 1' -dimethyl-4, 4'- bipyridium dichloride) a brown or bright green corrosive liquid is most common suicidal poisoning responsible for high morbidity and mortality, available in a 20% solution form. First synthesized in 1882, is being used as a herbicide since 1955.[1] Lethal dose is 30 mg/kg, equivalent to 8-10 ml of the 20% solution sold commercially.[2,3] Its toxic effects originate from the production of free radicals formed by the cyclic oxidation-reduction reactions of the compound in tissues, main acute systemic effects being pulmonary oedema, convulsions, cardiac, renal, and hepatic failure. Despite studies and clinical practice in the last few decades, little improvement has been made in reducing the fatality of PQ poisoning, but there is a comparatively high mortality. Although widely available, incidence and reports of PQ poisoning are not common in India. We discuss a case of suicide in which an unknown amount of PQ was consumed and presented with mild-moderate form of toxicity.

CASE: A 25-year-old male, admitted with an alleged history of attempted suicidal consumption of PQ, unknown quantity, at his residence. Initially managed locally with (intravenous fluids emetics and H_2 blockers), brought to our hospital for further treatment, after 6 hours of consumption. He presented with h/o difficulty in swallowing, nausea and vomiting with negative history for loose stools, abdominal pain, breathlessness, cough, seizures, or fever.

On clinical examination, patient was conscious, oriented, pupils bilaterally equally reactive, afebrile, pulse rate of 80 beats per min regular, blood pressure was 110/70 mm. Hg, respiratory rate of 16 per min with 98% spo_2. Systemic examination was unremarkable. Gastric lavage with activated

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charcoal was performed in the emergency department. Patient had mild anaemia (Hb was 9.9gm %). Other blood investigations were within normal limits.

Investigations	On admission	After 1 week
Heamoglobin	9.9mg/dl	10.8mg/dl
RBC	4.2million/cumm	4.7million/cumm
PCV	33.1%	36%
MCV	78.4cum	76.6cum
MCH	23.5mmg	23mmg
MCHC	29.9%	30%
WBC	8, 500cells/cumm	6, 900cells/cumm
Platelet	2.91lakhs/cumm	4.1lakhs/cumm
RDW	13.7%	13.6%
Glucose	105 mg/dl	89mg/dl
Urea	68mg/dl	24mg/dl
Creatinine	1.3mg/dl	1.2mg/dl
Sodium	132m.mol/l	129m.mol/l
Potassium	4.7m.mol/l	3.6m.mol/l
Total protein	7.1g/dl	7g/dl
Albumin	3.5g/dl	3.1g/dl
Bilirubin total	0.5mg/dl	0.6mg/dl
Bilirubin –Direct	0.2mg/dl	0.3mg/dl
AST	18U/L	20U/L
ALT	32U/L	36U/L
ALP	45U/L	42U/L
HIV, HBs.Ag	Nonreactive	Nonreactive
ECG, 2D-ECHO	No abnormality detected	No abnormality detected
Chest x ray	No abnormality detected	No abnormality detected

Table 1: Table of investigations, on admission and after one week

His chest x-ray was normal on day 1 on day 6 and day 15. Upper gastrointestinal endoscopy showed small petechial haemorrhage in the body, lesser curvature and antrum of stomach. Features of duodenitis were present. Oesophagus, vocal cords, pyriform fossa was normal. He received inj. piperacillin-tazobactum 4.5g i. v tid, oral vitamin c (500 mg) qid, vitamin E (400 mg), N-acetyl cysteine, antiemetics, antacid syrup, and local applications of lignocaine, metrogyl, povidine iodine mouth wash.

DISCUSSION: Symptoms of PQ ingestion are usually dose-dependent, and intoxication can be categorized to mild, moderate, and fulminant. PQ≤20 mg/kg causing mild intoxication, usually present with minor gastrointestinal problems like transient vomiting, diarrhea, and oropharyngeal burns. Usually complete recovery is possible. Doses between >20 mg/kg and <50 mg/kg cause moderate intoxication. While ≥50 mg/kg of the poison have a fulminant course, causing multi organ

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failure, may lead to death within 3 days.[3,4,5] Sandhu et al., 2003 mentioned frequency of common symptoms for PQ poisoning as vomiting (100%) followed by oral ulceration (59%), dysphagia (53%) and dyspnea (41%).[6] Gastrointestinal toxicity is universal in those ingesting PQ concentrate. Mucosal lesions of the mouth and the tongue ('Paraquat tongue'),[7] that appear within the first few days and may become ulcerated with bleeding are of little prognostic significance as they occur even in those who spit PQ out without swallowing (the products commonly contain stenching and bittering agents). Mucosal lesions in the pharynx, oesophagus and stomach are also very common and much more sinister. These may result in perforation, mediastinitis and/or pneumomediastinum. In patients who survive longer, fibrotic changes in the alveoli result in gas exchange interference in the lungs and may progress to ARDS. Renal tubular necrosis and hepatic necrosis may occur. It has been found that plasma concentration of >1.6 pg/ml 12 h after ingestion is universally fatal.[8] Diagnosis of PQ poisoning is usually made based on circumstantial evidences. It is always important to identify ingested amount of substance as specifically as possible, unfortunately unavailable in our patient. In our patient, the clinical history, presentation, and documentation of PQ consumption endorses the diagnosis positive as urinary and serum levels could not be estimated because of lack of laboratories performing PQ levels.

In spite of advances in medical care, prompt treatment, and supportive care, mortality still remains high, in such patients, mainly due to multiorgan failure. High dose ingestion or severe PQ poisoning has a poor prognosis. Conventional treatment includes nasogastric tube fixation, gastric lavage with normal saline, charcoal-sorbitol or fuller's earth lavage, forced alkalinized diuresis and hemodialysis or hemoperfusion. In contrast, the use of oxygen can enhance the toxicity by providing more electron acceptors. If at all needed, oxygen should be given in lower concentrations to the hypoxic patients.

Hemoperfusion with activated charcoal is effective if initiated within 4 hours of PQ intoxication. As our patient was admitted 6 hours after consumption, hemoperfusion was not performed. As seen in the present case, PQ ingestion resulted in an inflammation of the tongue, oral mucosa and throat, corrosive injury to the gastrointestinal tract. The patient complained of burning and ulceration of the throat, tongue and esophagus initially but after a week all symptoms resolved except for oral ulcerations. At present, there is no specific antidote to PQ poisoning. Therefore, it is recommended that the crucial focus should be on preventive measures. In case of oral exposure, aggressive decontamination should be instituted to prevent further absorption.

CONCLUSION: The available data suggest rare incidence, high mortality & Morbidity, and very few survivors, following PQ poisoning. We report our experience of acute PQ poisoning with oral, mucosal and upper GI ulceration. The unexplained combination of gastrointestinal symptoms, acute renal injury, and respiratory failure must lead to suspicion of PQ toxicity, even in the absence of ingestion history. If patient presents early, the therapeutic interventions with hemo perfusion and dialysis may be recommended to prevent pulmonary and multi-organ failure. Both, urine and serum concentrations of samples at known time intervals, post-ingestion, are to be determined, not only for assessing severity of the intoxication, but also to predict and improve the survival chances.

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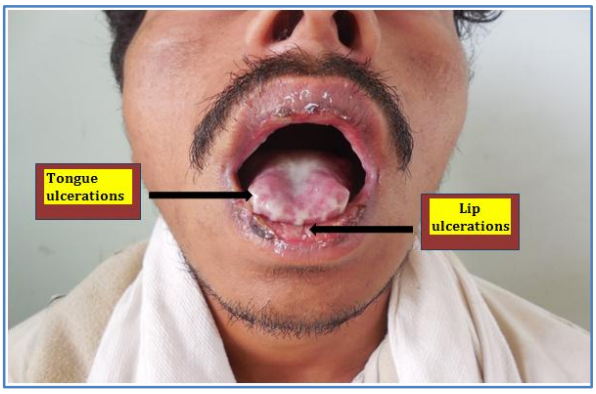

Fig. 1: Ulcerations over tongue and lips due to paraquat

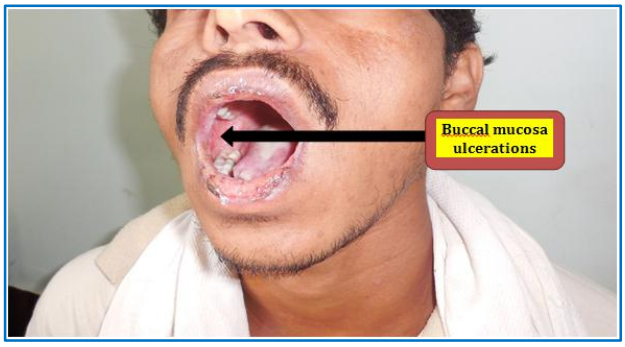

Fig. 2: Ulcerations of buccal mucosa due to paraquat poisoning

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