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NAVIGATING PARAQUAT POISONING: A CASE REPORT UNRAVELLING THE COMPLEX JOURNEY FROM ACUTE KIDNEY INJURY AND PULMONARY COLLAPSE TO RECOVERY

Internal Medicine

Dr. Swarnim Varshney Resident-General medicine, GMERS Valsad
Dr. Vidhi Mashru JR-General Medicine, GMERS Valsad
Dr. Abhishek Patel AP-General medicine, GMERS Valsad
Dr Yugam Oza* Intern doctor, M P Shah Medical College. *Corresponding Author

ABSTRACT

Introduction Paraquat poisoning holds on to its diabolical status with different outcomes to different trials of management. Life threatening complication occur involving pulmonary and renal system occasionally setting into sepsis. **Purpose:** To report our observations and experience of managing this patient who came with deliberate self harm with paraquat which was complicated by pulmonary and renal toxicity.Patient was managed using immunosuppressive, hemodialysis and anti fibrotic therapy **Method** Descriptive study of a patient admitted to ICU of tertiary care hospital of south Gujarat with diagnosis of Paraquat poisoning was performed with follow up of 1 week **Conclusion** Paraquat poisoning is uncommon or rather unexplored term in field of medicine in India with a lot of under reporting. It remains to hold high mortality There is a definite role of immunosuppressant in patient with moderate to severe poisoning

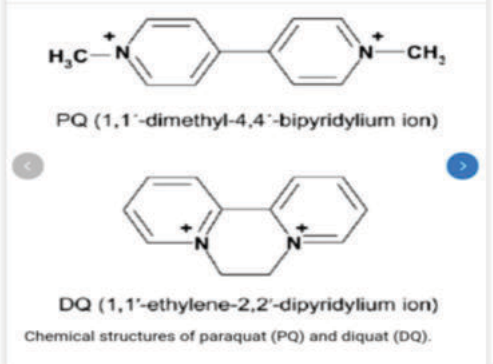
KEYWORDS

INTRODUCTION

[1]Paraquat , or N,N′-dimethyl-4,4′-bipyridinium dichloride (systematic name), also known as methyl viologen, is an organic compound with the chemical formula [(C6H7N)2]Cl2. It is classified as a viologen, a family of redox-active heterocycles of similar structure.This salt is one of the most widely used herbicides
Appearance: Yellow
Odor: ammonia like

[1]Paraquat is toxic to humans by the oral route and moderately toxic through the skin.Pure paraquat, when ingested, is highly toxic to mammals, including humans, causing severe inflammation and potentially leading to acute respiratory distress syndrome (ARDS), and death.

[2]PQ mainly accumulates in the lung (pulmonary concentrations can be 6 to 10 times higher than those in the plasma), where it is retained even when blood levels start to decrease. The pulmonary effects can be explained by the participation of the polyamine transport system abundantly expressed in the membrane of alveolar cells type I, II, and Clara cells.



Chemical structures of paraquat (PQ) and diquat (DQ).

BACKGROUND

Consumption of PQ > 40 mg/kg causes acute multiorgan failure with death within the first 2 days, while < 20 mg/kg of PQ causes mild symptoms and most survive.

Paraquat of 20–40 mg/kg causes severe mucosal damage followed by multiorgan failure. The few, who survive, die within 2–4 weeks due to lung fibrosis. The pulmonary alveolar cells selectively accumulate polyamines that are required for cellular functions. Being structurally similar to polyamines, PQ gets accumulated in these cells, causing selective delayed lung injury.

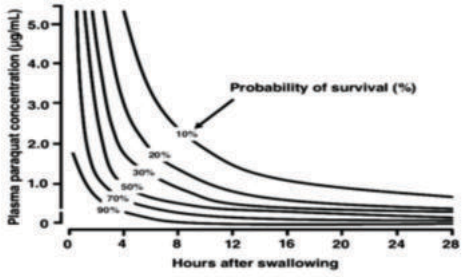
[5]Paraquat Poisoning: A Retrospective Study of 55 Patients From a Tertiary Care Center in Southern India,showed a very high in-hospital mortality rate of 72.7%. The amount of PQ consumed and the presence of lung injury had correlation with mortality rate, while the use of steroids/ Cyclophosphamide did not lead to reduced mortality rate.

We in this study are checking whether the trends of efficacy of Cyclophosphamide is reciprocated in our hospital setting with a patient of different topography(south Gujarat)

According to [6]Experience with paraquat poisoning in a respiratory intensive care unit in North India Paraquat poisoning is an uncommon entity in India, and is associated with a high mortality rate. There is a potential role for immunosuppressive therapy in patients with moderate to severe poisoning.

As per data provided by this study, we check trial of steroids and anti fibrotic agent in recovery of our patient in ICU care

[7]There is a evidence of patients receiving prolonged treatment having better lung function 2 to 3 months after PQ poisoning



Nomogram showing relation between plasma paraquat concentrations (µg/ml), time after ingestion, and probability of survival. Adapted from Hart et al. (1984).

Case Report

A 29 year old male patient presented to our emergency department with alleged history of suicidal poisoning of paraquat at his residency at around 8pm consuming around 20 mg of compound diluted in tea around 20 ml under influence of alcohol followed by 2 episode of vomiting and then presented after around 2 hr.

On presentation patient was vitally stable and was confused with GCS of 13/15. Pt was given charcoal infused gastric wash .He was started on iv Methyl Prednisolone and N-Acetylcystein. He was started on Cyclophosphamide 500mg od and injection vitamin C . On day 3 patient developed breathlessness, hypertension and AKI with

creatinine of 5 with u/o of 300 ml per day

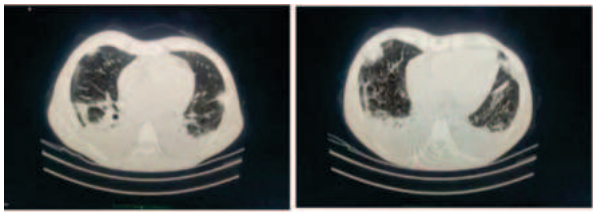
Hemodialysis cycles were initiated on veno-venous dialysis after securing double lumen central line along side with diuretic and iv fluid and antihypertensive.

On day 6 Mesna was given. On day 8 ,Methyl Prednisolone was tapered and Cyclophosphamide was held. On day 11 HRCT chest revealed Bilateral gross pleural effusion with passive atelectasis and fibrotic changes. On day 16 patient developed pancytopenia with Hemoglobin of 5.1,total leucocyte count <200 and patient count 56000.His antibiotic regimen was shifted accordingly to prophylactically ward off sepsis in view of leucocytopenia On day 20 antihypertensive were stopped and patient was shifted on oral steroids

These parameter showed improving trends after day 19,and he was put back on Ceftriaxone and Metronidazole on day 21 Patient was given 1 pint packed cell volume on 19 and 23rd day of admission

Patient was discharged with stable hematological parameter and vitals after 28 days on oral Prednisolone and oral Cefexime and Pantoprazole and After 1 week follow-up, double lumen catheter was removed as LFT RFT and CBC parameter were within normal limit

Patient was followed 1 month later and reported to OPD with normal hematological and imaging report



HRCT slices of patient on day 11 showing high level of fibrosis and atelactasis

DISCUSSION

[11]Prognosis of patient with accidental/suicidal poisoning of paraquat or similar congener containing compound depends upon following factors
A large amount of ingested volume,
Vomiting,
Loss of consciousness,
Need to ICU admission on entrance,
Leukocytosis,
Initial appearance of respiratory, hepatic or renal failure,
Development of sever hypotension or cardiogenic shock and need the infusion of vasopressor agents.

Patient generally present with URTI symptoms of burning of mouth, pain in swallowing,nausea,vomiting,abdominal pain. Patient may also complain of burning sensation on skin depending upon duration and magnitude of exposure These symptoms can be attributed to the solvent or compound itself

Over a period of hours to day these processes lead to multi organ failure. The organs most affected are those with high blood flow, oxygen tension and energy requirement, in particular the lungs,heart,kidneys and liver. Brain is uncommonly affected as paraquat doesn't cross blood brain barrier, although paraquat has been detected in CSF

[3]KINETIC:Upon ingestion, paraquat is rapidly but incompletely absorbed. It is rapidly distributed to lung, liver, kidney and muscle. Paraquat has a volume of distribution of 1.2–1.6 l/kg. Within 12–24 h after ingestion 90% of the absorbed paraquat is rapidly excreted unchanged in urine Bioavailability appears to increase non linear with increasing doses, perhaps due to gut and liver toxicity. After a few hours, renal clearance declines rapidly in severe poisoning. Thus, the small proportion of paraquat that distributes into the deeper compartments is only slowly eliminated by the kidneys over many days to weeks . The initial elimination half-life is around 6 h but this is around 4 days after the first day . Paraquat is actively taken up against a marked concentration gradient into the type II pneumocyte which could be considered a third 'toxic effect' compartment. Elimination from this compartment is even slower than from the other deep compartments.

[8]TGF-β1 regulates the expression of target gene CTGF to exhibit its pro-fibrogenic effects by activating TGF-β1/Smad signaling pathway in PQ-induced pulmonary fibrosis.

[10]The highest mortality rate was in patients with respiratory distress, followed by oral ulceration and excess salivation. In all PQ-poisoned patients, the dose of greater than approximately 2250 mg predicted death with 86.2% specificity and 75.7% sensitivity.

Symptomatology of paraquat poisoning differ from patient to patient but following have good possibility of occurrence hence a patient should be screened for following at presentation

1. **Ulceration** inflammation and/or necrosis of upper respiratory as well as alimentary tract
2. **dehydration** precipitated by vomiting and odynophagia
3. Evidence of **hypoxia** and air hunger as indicated by tachypnea and pulse oximetry
4. Heart rate and blood pressure aberration due to refractory hypotension in setting of **MODS or sepsis**. On contrary **renal hypertension** in response to systemic toxicity leading to renal fibrosis
5. Adventitious sound, crackles or evidence of diminished air entry as the outcome of toxicity progress further due to volume loss **atelectasis,pulmonary hypertension or pneumonitis**
6. Tender abdomen due to acute pancreatitis, hepatitis or bladder insult either directly or as a part of adverse reaction to treatment administered
7. **Skin ulceration** or signs of dermatitis

[4]The efficacy and greater availability of activated charcoal and magnesium citrate make these materials the treatment of choice for gastrointestinal decontamination in oral paraquat poisoning.

[9]Repeated pulses of CP and MP, rather than high doses of CP and DEX, may result in a lower mortality rate in patients with severe PQ poisoning.

[12]Haemodialysis could be considered in patients who have developed symptomatic acute renal failure. However, such patients have a very poor prognosis in terms of their lung injury, so this is unlikely to change outcome

Investigation

Routine **hematological panel** with **arterial ABGA**, **toxicology screen**, **urine and blood level of paraquat**, **serum lactate** with **chest imaging** to look out for pulmonary complication

CONCLUSION:

From the observation made by us during the course of stay of patient followed by regular follow-up at 1 week 3 week and 1 month,We can conclude that patient clinical profile has significant improvement, which can be attributed to to early decontamination and early use of immunosuppressive and antifibrotic therapy.

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