



Research Article

PARAQUAT INDUCED ACUTE RENAL FAILURE: A CASE REPORT

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ABSTRACT

Paraquat is most commonly and widely used herbicide. It is highly toxic in nature and with high dose it can cause liver, renal and respiratory failure. We hereby report a case of 20-year-old male with a history of paraquat consumption moreover, he is an alcoholic, smoker and works in a carrot farm which contributes to the easy availability of the poison. The victim had the complaints of stomach pain, throat pain, burning sensation and loss of appetite. The patient was admitted in the Emergency unit and underwent haemodialysis. Laboratory investigations like CBC, RFT and LFT were performed in which serum creatinine and blood urea levels were elevated. In addition to this the patient was provided with a psychological counselling session. The patient developed post paraquat poisoning complication of acute renal failure after getting discharged from a hospital and was admitted to secondary care hospital ICU unit and managed symptomatically for the complaints of generalised fatigue and burning sensation in the oesophagus. Early diagnosis and appropriate management of paraquat poison can reduce the mortality as even small amount of paraquat can lead to fatal outcomes. In present scenario there is no specific antidote to treat paraquat poisoning; hence there is a crucial need to focus on the prevention and management measures. The occurrence of poisoning cases is more among younger population (between 21-30 years) and it may be due to the high stress, depression and economic status. In addition to this, lack of psychological support and ease availability majorly contributes to self-harm.

Keywords: Paraquat, poison, complication, acute renal failure, management

INTRODUCTION

Paraquat is a highly toxic bipyridyl herbicide which is available in a bright green liquid with a pungent smell¹. It is widely used in agriculture as a majorly used herbicide which is highly toxic to humans and animals². Paraquat herbicidal properties were first discovered in 1950s and it got first marketed in 1962¹. Globally paraquat is a second highest-selling weed-killer and it is available in 20% solution¹. The lethal/toxic dose is very less 35mg/kg that is around 10ml-15ml which is highly toxic. It affects many organs and can lead to cardiac, hepatic and renal failure. It is highly toxic in nature and if it is ingested it can lead to acute respiratory distress syndrome (ARDS).³

Poisoning is a major problem worldwide which leads to many complications and in severe cases it can lead to death. The World Health Organization (WHO) has reported that globally more than 200,000 people die of pesticide poisoning. Lifestyle changes as well as changing social frameworks are the major reasons for the increased incidence of poisoning cases⁴. As India is highly categorised into an agro-based country, where most of the agricultural chemicals such as pesticides and herbicides were available with limited regulation, which are used as poisons for committing self-harm. Apart from this, easy availability, low-cost and unrestricted sale of pesticides is the other key reasons for self-harm⁴. Paraquat poisoning has been categorised into three categories such as mild, severe and fulminant⁵.

Mild poisoning: It ranges from 20mg/kg in which patient generally faces the minor gastrointestinal symptoms.

Severe poisoning: It ranges from 20-40mg/kg in which patient develops lung, kidney injury along with pulmonary fibrosis and pulmonary edema.

Fulminant poisoning: It ranges from 40mg/kg in which patient develops the multi-organ failure.

Very few studies on paraquat poisoning are done globally and in India. As there is no specific antidote available at present to cure paraquat poisoning so there is a need to focus on management procedures. This study reports a case of post-paraquat poisoning complication acute renal failure.

CASE REPORT

Informed Consent Form

Informed consent was obtained from the patient for publishing the case. A copy of informed consent form is available with the author for future proceeding.

A 20-year-old male patient was brought to the hospital causality for the alleged consumption of paraquat poison at home around 6 pm. The quantity of poison consumed by patient was 5-8 ml. Patient had a complaint of stomach pain, throat pain, burning sensation and loss of appetite. Patient was admitted to ICU for the

decontamination procedure and he was treated for 1 day as there was increase in Blood urea (130mg/dl) and serum creatinine (3.5mg/dl). Patient condition was getting deteriorate and he was referred to tertiary care hospital for the further management.

At tertiary care hospital, patient was treated with 5 cycles of haemodialysis, Inj. Dexamethasone 8mg IVBD, Tab. Propanol 40mg $\frac{1}{2}$ -0- $\frac{1}{2}$ BD, Tab. Ranitidine 150 mg PO BD, Tab. Dulcolax 5mg PO HS along with multivitamins and calcium carbonate. Patient developed the symptoms of post paraquat poison after getting discharge from a hospital and was admitted to secondary care hospital ICU unit for the complaints of generalised fatigue, burning sensation and basal crepts found to be positive. His vitals reports are: BP: 130/90 mmHg, PR: 98 beats/minute, RR: 22 per minute, Temp: 98.4 Fahrenheit, CVS, RS: NAD P/A: Soft

Patient is a known smoker and alcoholic though he stopped last month. He works in a carrot farm which reveals the reason for ease availability of herbicide. His laboratory investigations were done on day 1 is presented in Table 1.

At the time of admission, patient was given with Intravenous fluids RL and DNS 1 pint each followed by Inj. Rantac 50 mg IVBD. Around 8 pm patient's general condition was not fair BP:

120/70 mmHg, PR: 88/min with a complaint of giddiness and 2 episodes of diarrhoea. And he was given with 2 pint of RL and 1 pint of NS, ORS powder and Inj. Ciprofloxacin 200mg IVBD and CBG: 82 mgs%. Next day on examination patient was conscious, afebrile, P/A- soft, CVS/RS: NAD, Temp- 98.4' F, SPO₂- 98% in room air, BP: 150/110 mmHg (9:00 am). Blood urea: 29 mg/dL, Serum creatinine: 0.7 mg/dL. Patient was given with IVF- RL 2 pint and DNS- 1 pint followed by Tab. B-Complex and Inj. Ciprofloxacin 200mg IVBD. And he was asked for observation. His vitals were recorded as Temp: 98.4'F, PR: 28/min, RR- 22/min. (4 pm) His ECG reports doesn't reveals any significant harm to patients CVS (Fig 1.)

On Day 3 patient was observed as conscious, oriented, afebrile, CVS/RS: NAD, Temp- 98.4' F, SPO₂: 96% in room air, BP: 100/70 mmHg and his vitals were recorded with RR: 82/min, PR: 20/min. He was treated with IVF NS 2 pint and Cap. Omeprazole 20mg BD. On day 4 patient's general condition was found to be fair with a complaint of generalised body ache and he was given with Tab. Diclofenac 50mg BD and Tab. Ranitidine 150mg BD. On the last patient got discharged and was asked to get review after 15 days with further reports of Blood urea, CBC, LFT and Serum creatinine.

Table 1: Laboratory investigation detail at the day of admission

Investigation	Patient observed value	Normal values
Hb(Hemoglobin)	15.9 g/dL	14-18g/dL
TC (Total count)	17.2x10 ³ cells/mm ³	3.2-9.8x10 ³ cells/mm ³
Polymorphs	81%	54-62%
Lymphocytes	11%	25-33%
Monocytes	08%	3-7%
Platelet Count	457x10 ³ cells/mm ³	130-400x10 ³ cells/mm ³
RBC's(Red blood cells)	5.57x10 ⁶ /mm ³	3.5-5.0x10 ⁶ /mm ³
Hct (Hematocrit)	44.4%	39-49%
MCV(Mean cell volume)	79.7fL	76-100fL
MCH(Mean cell hemoglobin)	28.5pg/cell	27-33pg/cell
MCHC(Mean cell hemoglobin concentration)	35.8g/dL	33-37g/dL
RBS(Random blood sugar)	69 mg/dL	<200mg/dL
Sodium	132mEq/L	135-147mEq/L
Potassium	4.8mEq/L	3.5-5.0mEq/L
Blood urea	48mg/dL	20-40mg/dL
Sr.cr. (Serum creatinine)	1.3mg/dL	0.6-1.2mg/dL
AST(Aspartate aminotransferase)	10 U/L	0-35U/L
ALT(Alanine aminotransferase)	18 U/L	0-35U/L
ALP(Alkaline phosphates)	175 U/L	30-120U/L
Bilirubin	Total- 0.8 mg/dL	0.1-1mg/dL
	Direct- 0.4 mg/dL	0-0.2mg/dL
	Indirect- 0.4 mg/dL	0.1-0.8mg/dL
Deposits	Pus cells(PC)- 2-3 hpf	1-2 hpf
	Epithelial cells- 2-3 hpf	1-2 hpf

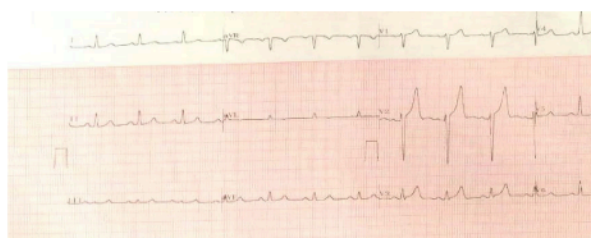


Fig 1: ECG pattern of the self-harm patient at the time of admission

DISCUSSION

Self-poisoning remains a significant problem in various parts of India. Paraquat poison has been associated with morbidity and mortality⁶. In humans the toxicity mechanism is not clear⁷.

Inhalation is a most common route of poisoning which can results in local irritation. Paraquat does not actively metabolise in the body it is highly distributed to organs such as kidneys, lungs, liver.

At initial stage treatment consists mainly of supportive care that includes nasogastric intubation, gastric lavage, and fluid administration⁸. Management focuses on prevention of further damage to organs and absorption of poison. Haemodialysis or hemoperfusion is used as supportive treatment for the patients who develop kidney failure to prolong the life span⁹. At high doses dexamethasone and cyclophosphamide can be used for the supportive treatment with the 75% survival rate¹⁰. N-acetyl cysteine (NAC) has also been used in the treatment, further it delays the inflammation¹¹.

The Acute Dialysis Quality Initiative (ADQI) group for the study of AKI a consensual RIFLE (Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease) classification for AKI definition emerged, which was published in Critical Care¹². In this case report due to the increase in the elevated Blood urea, Serum creatinine and low creatinine clearance values revealed Paraquat induced acute renal failure (ARF) after calculating the creatinine clearance which was found to be 72ml/min by applying Cockcroft- Gault equation.

Acute ARF in this patient can be managed by treating any life threatening conditions, contributing to further renal insult, and if unsuccessful providing support by renal replacement anticipating reversal of renal insufficiency. Supportive care can be given to the patient which includes electrolyte management and psychological support. Our patient works in a carrot farm and due to its ease availability the patient used it as a measure of self-harm. Patient should be counselled about the importance of life and also to avoid further occupational hazards by usage of masks which in turn reduces inhalational exposure. There is no significant antidote to paraquat hence the management for this makes it difficult. The prognosis of paraquat poison is highly associated with the amount of paraquat absorption.

CONCLUSION

Early diagnosis and appropriate management of paraquat poison can reduce the mortality as small amount of paraquat can lead to fatal outcome. Moreover there is no specific antidote so a crucial need is require for the management of paraquat poison. Financial burden, family disharmony, stress and depression commonly observed in high-altitude inhabitants could be the reasons for the increased rate of poisoning. Hence, more number of psychological programmes should be conducted to create awareness about the value of life among the public.

ABBREVIATIONS

1. NS- Normal saline
2. RL- Ringer lactate
3. DNS- Dextrose normal saline
4. CVS- Cardiovascular system
5. RS- Respiratory system
6. BD- Bis die (twice daily)
7. BP- Blood pressure
8. PR- Pulse rate
9. RR- Respiratory rate
10. SPO₂- Periphery capillary oxygen saturation

11. P/A- Per abdomen
12. NAD- No abnormalities detected
13. IVF- Intravenous fluids
14. ORS- Oral rehydration salt
15. PO- Per oral
16. ECG- Electrocardiogram
17. CBC- Complete blood count
18. RFT- Renal function tests
19. LFT- Liver function tests
20. CBG- Capillary blood glucose

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