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Paraquat Poisoning and Acute Kidney Injury

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ABSTRACT
N, N2-dimethyl-4, 42-bipyridinium dichloride (paraquat) is a widely used synthetic, non selective contact herbicide. Ingestion of toxic doses of paraquat can be fatal with life-threatening effects on the lungs, gastrointestinal tract, kidney, liver, heart and other organs. Till date, there is no specific antidote. Although it is a very common herbicide, there are very few cases reported from India and awareness among people needs to be widened.
Keywords- Paraquat , herbicide , Acute kidney injury, Nephrotoxic .

I. INTRODUCTION

Paraquat has been widely used in much of the developing world, however thousands of individuals succumb due to paraquat intoxication every year in the developing world.
Paraquat is a highly toxic compound and the fatality rate of paraquat is between 60% and 80% (1) because of lack of a specific antidote.

A paraquat dose of 30 mg/kg may be fatal, which is equivalent to 8–10 mL of the 20% solution sold commercially (2).

Paraquat has been shown to cause significant damage to organs, including the lung, liver, myocardium and kidneys, with the highest concentration of paraquat found in the lungs (3).

The prognosis of patients with multiple organ failure caused by fulminant poisoning (>40 mg/paraquat ion per kg of body weight) is extremely dangerous and affected individuals may succumb within hours to a few days following ingestion (4,5)

Aim
The study aims to analyze the incidence of Acute kidney injury in patients of paraquat poisoning.

Objectives
To understand mechanism of Acute kidney injury in paraquat poisoning.
To identify association of Acute kidney injury with mortality in patients of paraquat poisoning.

Method
•It is a prospective study carried out at tertiary care hospital in Nashik between the time period of Jan 2021 to Oct 2022
•A probable case of paraquat poisoning was diagnosed and during the hospital stay,patients were classified into AKI and Non AKI patients.

Case Description
A previously healthy 22 years old male was referred to the emergency department at MVP,Nashik with A/H/O consumption of liquid herbicide(Paraquat) around 200ml.
He experienced nausea,sweating,uneasiness after ingestion.He was ill in appearance but was arousal and able to speak.On Examination
Afebrile
HR- 90bpm
BP-110/70mmHg
SpO2- 99% on RA.
RR- 18/min
His oral mucosa was congested and edematous.
Pupils were b/l 2mm and reacting to light.
Both lung fields were clear on auscultation.
There were no neurological symptoms,organomegaly,tachycardia and tachypnea.
In the emergency he was given gastric lavage.He received intravenous fluids and antiemetics as supportive measure.
Initial complete blood count,electrolytes,liver function tests,arterial blood gas were within normal limits.
His initial CXR was normal.Blood cultures and urine cultures were sterile.

The kidney functions seriously damaged within two days of ingestion.Because of clinical signs and the continued increase in blood urea and creatinine,hemodialysis was initiated.

However,the kidney functions gradually recovered 12 days later completely.
The dialysis was started on day 3 and after 7 cycles of hemodialysis kidney functions recovered.
Although KFT were improved,patient died from delayed pulmonary fibrosis and hypoxemia.The main laboratory test results shown in the table

II. DISCUSSION

We observed 5 patients of paraquat poisoning between Jan 2021 to Oct 2022.
4 patients out of 5 had developed AKI and required dialysis.
The mortality rate was higher for AKI than non AKI patients.
The AKI HD group demonstrated increased risk of ARDS.

The Mechanism whereby paraquat causes AKI is not fully understood,however it is known that this compound accumulates within renal tubular cells,leading to cycles of reduction and oxidation,generating reactive oxygen species and ultimately damaging the proximal tubules.

As the kidney is the main organ responsible for paraquat excretion,the resultant kidney injury may reduce the elimination of paraquat and increase its toxicity to other organs.

Acute liver failure is frequently accompanied by concurrent AKI as a result of renal hypoperfusion,the nephrotoxicity of drug itself or sepsis and systemic inflammation.Although renal damage is reversible,death may still occur from delayed pulmonary fibrosis and hypoxemia.

The exact mechanism of toxicity caused by PQ is not known yet. Following PQ poisoning, the lungs are the main target organs, and the redox reaction occurs after the uptake of PQ in the lungs, which interferes with mitochondrial electron transfer, generates a large number of oxygen free radicals, and induces lipid peroxidation injury.In the alveolar epithelium, absorbed paraquat concentrations can be up to 10 to 20 times the serum paraquat levels.(6) Numerous studies found that outcome is related to the plasma concentration of PQ.(7-11)

III. CONCLUSION

In summary, AKI was common(80%) following paraquat ingestion and certain variables such as acute hepatitis,the time to hospital arrival,higher AKIN score were powerful predictors of AKI.
In addition the mortality rate tends to be higher in AKI patients.
And also AKI HD groups demonstrated higher risk of ARDS.
As this study included small population of patients,further studies are warranted to confirm our results.

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