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Paraquat Poisoning – A Brief Overview

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ABSTRACT

Paraquat poisoning in the modern era has become a major medical health problem, due to its high case fatality rate. The toxicological importance of this herbicide is due to its inherent toxicity and lack of specific antidote . It is one of the commonly used agents responsible for death among the reported pesticide poisonings in south east Asian countries.

The high mortality rates associated has led to the withdrawal of this herbicide from the market by the European Union in 2007, but in developing countries like India it is widely marketed due to its low cost and high efficacy . Although several studies have been done globally, there are no extensive published human trials. In Asian pacific regions where pesticides are commonly used for suicidal or homicidal ingestion, it has become a subject of concern and there is a growing need to focus on prevention due lack of specific antidote .

This article endeavors to revisit the profile of paraquat (PQ) and to investigate the impacts of this chemical on human health with a future direction to create awareness about this highly toxic nonselective herbicide and to consider it as a part of national strategy to lower suicidal mortality rates.

KEYWORDS: Paraquat (PQ), Herbicide ban, Redox Cycling(Rox), Cyclophosphamide (CTX), Acute Kidney Injury (AKI)

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MAIN TEXT

Toxicology Letters:

Paraquat (PQ; 1,1-dimethyl -4,4'– bipyridinium dichloride)is an organic heterocyclic herbicide used in agricultural settings. It is a highly toxic contact herbicide available primarily as liquid preparation and is commonly marketed by trade names: Gramoxone (10-20% w/v), Finish , All Quit and also known as Weedol which is commonly used for horticultural purposes .[1,3]

The most likely mechanism of action of paraquat is by its ability to undergo redox-cycling (Rox) and subsequent generation of reactive oxygen species (ROS) by interfering with intracellular electron transfer system inhibiting the reduction of NADP to NADPH during photosynthesis , generating a paraquat mono-cation radical ($PQ.^{+}$), which further gets reoxidized producing super oxide and peroxynitrite leading to cellular injury by oxidative stress, inflammatory response and autophagy.[3]

Type II pneumocytes actively absorb the chemical against the concentration gradient, causing the herbicide primarily to concentrate in the lung tissue , leading to irreversible and progressive damage with death occurring few days to weeks after injury and is typically considered as a third 'toxic effect' compartment. The elimination half-life varies from 6 h to 4 days and is largely eliminated unchanged in urine .[2,3]

The clinical manifestations vary as per the amounts of liquid concentrate ingested , fulminant organ failure leading to digestive tract involvement , acute kidney injury (AKI), liver injury, pulmonary edema, respiratory failure, convulsions leading to hypoxia , shock , metabolic acidosis and death from multi organ failure results in few hours to days.[5,6]

Prolonged contact and higher concentration of the chemical hastens the toxicity.The basic work up include serum and urine paraquat assay , sodium dithionite test, metabolic panel, CT chest and Endoscopy.[6]

Complications following poisoning include pulmonary hemorrhage, refractory shock, multi system inflammatory syndrome finally leading to death from respiratory failure and pulmonary fibrosis.[6,7]

There are no widely accepted guidelines for treatment of paraquat (PQ) self poisoning.

There are two treatment strategies outlined for PQ poisoning .The first predicts the outcome to be dire and states that no treatment would benefit the most and as per the second no treatments are likely to be worse than the disease per se and advice to give supportive treatment. The available treatment options include: Activated charcoal and Fullers earth to minimize absorption from the gut, gastric lavage is not routinely practiced. Antioxidants such as acetylcysteine, salicylate, Vitamin C and D are used for free radical scavenging and anti inflammatory actions. Glucocorticoids like methylprednisolone, dexamethasone and cyclophosphamide (CTX) are used to treat immunosuppression. Hemodialysis and hemoperfusion are used as elimination methods in case of AKI and metabolic acidosis.

For acute poisoning it is important to limit oxygen therapy unless the Pao_2 falls below 50 as it may lead to generation of reactive oxygen species and irreversible oxidative damage. [7]

Ghaffari et al 2011 and Lin N. C et al 2003 states that glucocorticoids and CTX could be effective to treat PQ poisoning. Zerin et al states that steroids decrease the leukocyte aggregation and improve the respiratory function.[7,8]

The present study is undertaken to evaluate the outcome–(mortality) in cases of paraquat poisoning in emergency department in a region where management guidelines still remain in substandard level, with a future direction to bring down the case fatality rates.

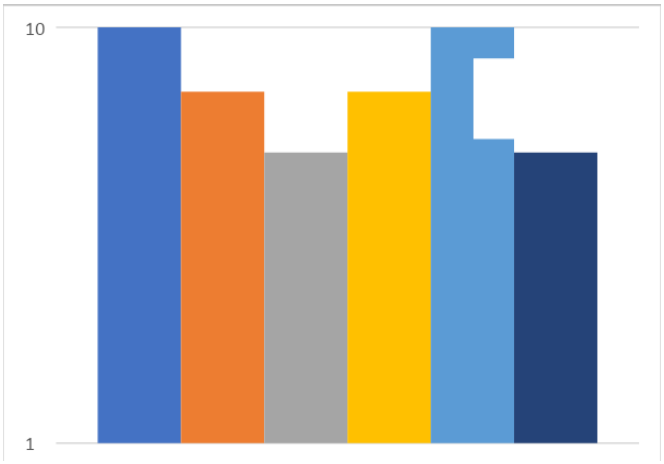
DISCUSSION

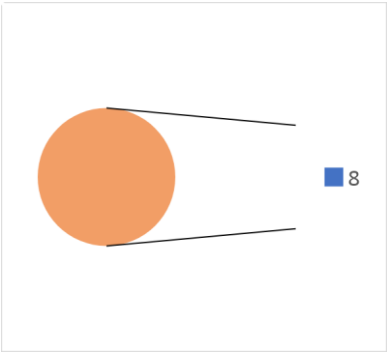
In the present pilot study , the clinical profile of 12 adult patients (18 years and older)presenting to emergency following paraquat ingestion was studied , majority of them were males -(66.67%) , total mortality rate was around –(83%) ,two patients were eliminated from the study due to insufficient data as death occurred within 2hrs of presentation to the hospital.

Among the non survival group majority of them - 70% had acute kidney injury 60%- suffered from shock ,80% had respiratory failure ultimately leading to death .

Few patients also suffered from hepatic injury- 60%. Almost all patients had oral ulceration and gastro intestinal tract impairment. It was found that there was an association between mortality and the amount of poison ingested, as the survival group history is consistent with minimal intake of poison. The present analysis results although are in agreement with the previous studies, being done on limited study subjects, further research is contemplated to validate the results.

Vale et al -1987 and Xu et al.,- 2011 stated that an amount over 20 mg of PQ ion/kg body weight results in fatal respiratory system involvement by inflammation leading to pulmonary fibrosis and death.[3]





Chinta et al., 2008;Yang et al.,2009 suggest that PQ has a complex mechanism of action with multiple targets inducing endoplasmic reticulum ER stress ,Bonilla et al., (2006) studied about PQ-induced oxidative stress and stated that minocycline would prevent it.

Jones et al.., (1999) reported that out of 375 patients, 49 had renal toxicity and median time from ingestion to death in the 241 deaths reported was 270 hours. Sabzghabaee et al-2010 states that in hospital fatality rate with PQ poisoning is high. [5] Yamaguchi Index and the Severity Index of Paraquat poisoning (SIPP) are calculated and used for the prognosis prediction in PQ poisoning . They have good reliability and are equally predictive in in acute intoxications .[8,10] Future research with RCT`s with an aim to study the mechanism of immune suppression, would be a restorative attempt for management of PQ poisoning.

CONCLUSION

As there is no specific antidote available for paraquat intoxication, "Prevention is thought to be better than cure."

- Hence forth imposing a ban on liberal marketing of this herbicide and restricting usage of this weedicide would bring down the suicidal mortality rates.
- Creating wide spread awareness with a strategy to limit contact exposure especially to the people in the agricultural sector would aid in the prevention of herbicidal intoxications.

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