IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 4 Ser. 8 (April. 2019), PP 06-09
www.iosrjournals.org

Successful Treatment of Paraquat Poisoning In a Tertiary Care Hospital of Eastern Part of India: A Therapeutic Challenge

Raja Bhattacharya[1], Tapabrata Das[2], Angshuman Roy[2], Sanjib Nandy[2], Kajal Kora[2], Sampurna Chowdhury[2]

[1]*Assistant Professor, Department of Medicine, Medical college Kolkata, 88 college street,Kolkata- 700073, West Bengal, India*

[2]*Post Graduate Trainee, Department of Medicine, Medical college Kolkata, 88 college streetKolkata- 700073, West Bengal, India*

Corresponding Author: Raja Bhattacharya

Date of Submission: 27-03-2019 Date of acceptance: 11-04-2019

I. Introduction

Paraquat (1, 1-dimethyl-4, 4-bipyridium dichloride)ingestion is a major cause of fatal poisoning in manyparts of Asia and Pacific nations.1 Commercialpreparations of paraquat are normally sold in the form ofliquid concentrate with a concentration ranging from 20%to 42%. Besides being supplied in the form of a singleactive ingredient, there are products in the marketcontaining paraquat in combination with other herbicidessuch as sodium chlorate and 2, 4-dimethylamine. Deathin paraquat poisoning is either due to significant lunginjury, acute kidney injury or multi organ failure.2 Thecommonest mode of poisoning with paraquat is oralintake of poison. Paraquat interferes with the intracellularelectron transfer systems, thus inhibiting the reduction ofNADP to NADPH (Figure 1). This will then result in theaccumulation of superoxide radical which causesdestruction of lipid cell membranes.3 Toxicologicalanalysis of plasma and urine samples is used to establishthe diagnosis. Mortality rate of paraquat poisoning isdirectly related to plasma and urine paraquatconcentrations. Paraquat is mainly eliminated by kidneyand acute kidney injury is the complication of it.Ingestion of small quantities can cause severely damageto lung and kidney.4

As there is no specific antidote for forparaquate,the main treatmentar present is conservative medical management.Poison removal through digestive and circulatory system is the most frequently usef treatment strategy.The extracorporeal elimination including haemoperfusion(HP), Haemodialysis(HD),Hemofiltration(HF),Plasma exchange,and continuous vevo venous haemofiltration(CVVH) of which HP is more efficient in the clearance of plasma paraquate.HP was first used in 1960,initially with uncoated columns and later with charcoal.

II. Materials And Method

PATIENT 1

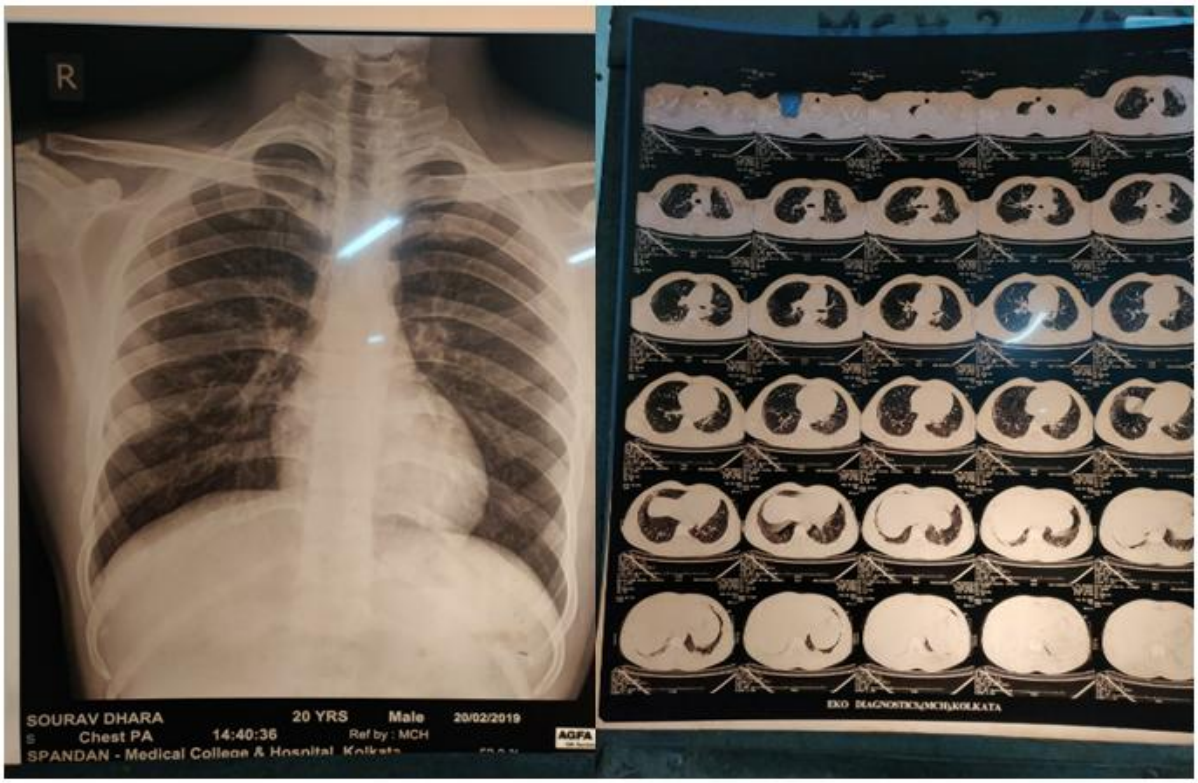
A 19 year old male SAMIR DHAR presented with history of ingestion of 15ml of undiluted paraquat48 hours prior to presentation.

He complained of difficulty in deglutition,decreased urine output, and occasional vomiting .

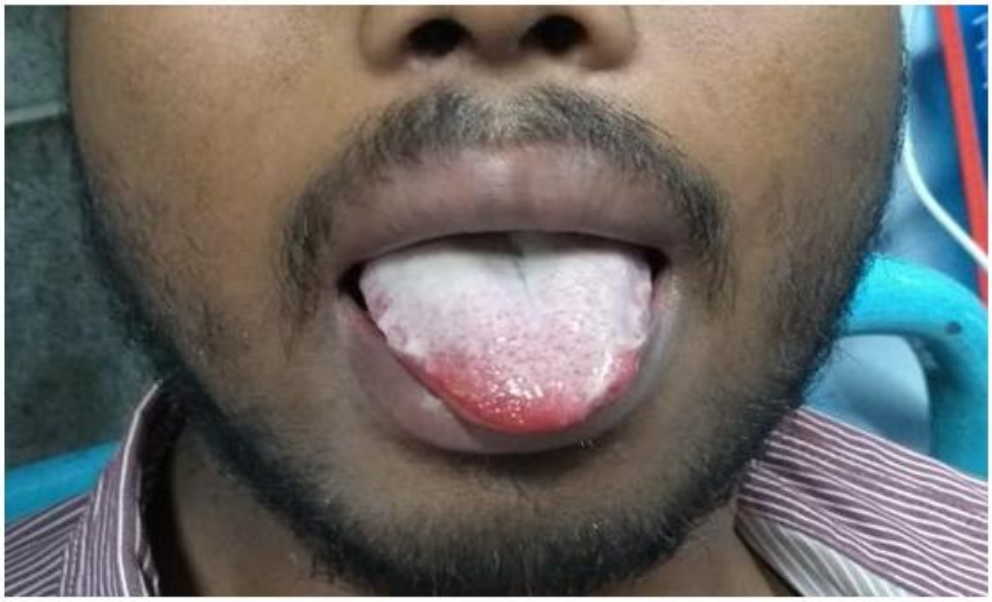
On examination he had a green coated tongue which had unhealthy mucosa and facial puffiness.

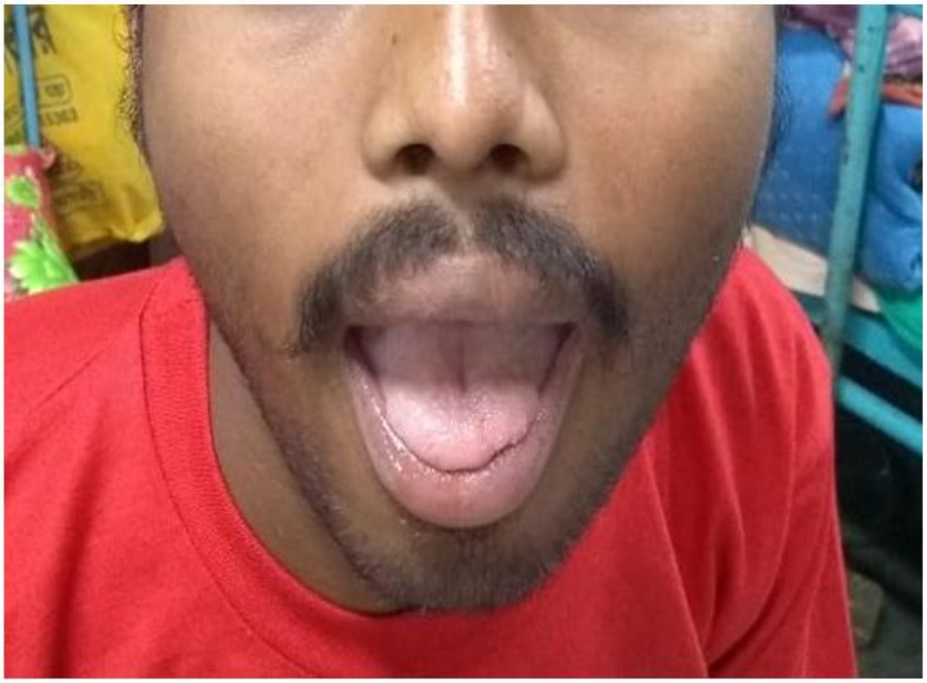
There was bilateral decreased air entry in both the lungs,along with decreased urine output (<300ml) /day.Haematological parameters showed urea 146 and creatinine 9.6 with increased Crp86.9.

Chest xray showed reticulate pattern in the basal area suggestive of early fibrosis



HRCT thorax also suggested similar changes of fibrosis (early).
As the patient was detoriorating rapidly he was ginen IV Methylprednisolone with N-Acetylcysteine,followed by CHATCOALHAEMOPERFUSION(5CYCLES).
Following which the patient was given a single dose of iv CYCLOPHOSPHAMIDE
The patient responded well and the renal profile and pulmonary status improved considerably.





ORAL MUCOSAL LESION(BEFORE AND AFTER)

The patient was given oral vitamin E and vitamin c as an antioxidant and for better mucosal healing zinc supplement was given,and the results were remarkable.

PATIENT 2

A 17 year old female presented with history of ingestion of paraquat approximately 100ml,3days before admission.Initiallygastric lavage was done at a local hospital,and then she was referred at medical college kolkata for management of furthur complications.

Her urea creatinine was deranged (180,8.5), chest xray showed presence of early fibrosis..As charcoal hemoperfusion was not available back then,she was put on hemodialysis.After 9 cycles of hemodialysis her kidney functions normalised.

Along with it she was also given long term dexamethasone,and n-acetylcysteine, which showed good results..

Inhalation with n acetylcysteime and ambroxol was done which improved the pulmonary functions considerably.

III. Discussion

Paraquat poisoning is of toxicological importance insouth India as it is widely used as an herbicide. Thefatality rate of paraquat poisoning in our hospital wasfound to be 100%. In hospital cases, fatality rate rangesbetween 35-62% around the world.5,6 Also majority of thepatients did not reach the hospital in the golden hour dueto varied reasons. This shows that in India, we still havemuch to do to reduce the mortality associated withparaquat poisoning.

The median age of paraquat poisoned patients was foundto be 28.5 years in our study which was comparable to astudy done by Kanchan et al, where the mean age wasfound to be about 30 years.7 The reason for suicidal intentwith paraquat poison among the youth may be due to itseasy availability and its wide use as an herbicide. In ourstudy, paraquat poisoning was more common in malesthan females probably reflecting the easier accessibilityto the farm working population.Treatment of paraquat poisoning is largely supportive andaimed at removing paraquat from the site of absorption.Increasing its excretion from blood and preventingpulmonary damage is the major target of management.Hospitalization is required as soon as possible in all casesof suspected paraquat poisoning.2 Paraquat accumulatesselectively in the lung tissues. Lung injury, which ismediated through lipid peroxidation, is exacerbated bythe administration of oxygen therapy. Thus,supplementation of oxygen should be withheld untilunless pO2 is less than 70 mm of Hg.8 All the paraquatpoisoned patients received treatment with 1g/kg ofactivated charcoal through the nasogastric tube followinggastric lavage with normal saline in the emergency triage.Paraquat can be removed by haemodialysis andhaemoperfusion but, although the clearance values arehigh for paraquat compounds, the effective quantitiesrecovered are insignificant. Prevention of death is mostunlikely.9 According to Cavalli et al, the survival rate inpatients without active treatment was only 13% in nonfatal dose ingestion of paraquat poison and

it increased tomore than 50% with active treatment modality likehaemoperfusion, in patients with fatal doseconsumption.10Combined therapy with haemoperfusion and ContinuousVenoVenousHaemofiltration (CVVH) increased thesurvival duration in patients with acute paraquatpoisoning.11 CVVH as a standalone therapy was alsofound to be beneficial in reducing the mortalityrecently.12 In a study done by Hsu et al., it was found thatearlyhaemoperfusion (within 6 hours) improved thesurvival outcomes in paraquat poisoned patients.13Some studies found that haemoperfusion was not usefulwhich might have been due to potentially lethalconcentration of paraquat getting accumulated in highlyvascular tissues of the vital organs and pneumocytesbefore the initiation of haemoperfusion.14According to Raghavendra et al, patients who receivedearly haemoperfusion (< 6 hours) were more likely tobenefit compared to those who received late.

IV. Conclusion

Early diagnosis and aggressive management of paraquatpoisoning is necessary. Even a less than fatal dose ofparaquat poison can lead to fatal outcomes as there is nospecific antidote available. Treatment remains supportive in nature.

Hence there is more scope for furthur research; to find the perfect combination to combat this fatal toxin ,to which thousands of people are losing their lives.

References

[1]. Gunnell D, Eddleston M, Philips MR, Konradsen F. The global distribution of fatal pesticide selfpoisoning: systematic review. BMC Public Health. 2007;7:357.
[2]. Sandhu JS, Dhiman A, Mahajan R, Sandhu P. Outcome of paraquat poisoning - a five-year study. Indian J Nephrol. 2003;13:64-68.
[3]. Suntres ZE. Role of antioxidants in paraquat toxicity. Toxicol. 2002;180(1):65-77.
[4]. Saravu K, Sekhar S, Pai A, Barkur AS, Rajesh V, Earla JR. Paraquat - A deadly poison: Report of a case and review. Indian J Crit Care Med. 2013;17:182-4.
[5]. Sabzghabaee AM, Eizadi-Mood N, Montazeri K, Yaraghi A, Golabi M. Fatality in paraquat poisoning. Singapore Med J. 2010;51(6):496-500.
[6]. Lee EY, Hwang KY, Yang JO, Hong SY. Predictors of survival after acute paraquat poisoning. ToxicolInd Health. 2002;18:201-06.
[7]. Kanchan T, Bakkannavar SM, Acharya PR. Paraquat poisoning: analysis of an uncommon cause of fatal foisoning from Manipal, South India. Toxicol. Int. 2015;22(1):30-4.
[8]. Olson KR. Poisoning. In: McPhee SJ, Papadakis MA, Tierney LM. ed. Current Medical Diagnosis Treatment. 46th ed. New York: McGraw Hill; 2007;1663.
[9]. Holubek WJ, Hoffman RS, Goldfarb DS, Nelson LS. Use of haemodialysis and haemoperfusion in poisoned patients. Kidney Int. 2008;74:1327-34.
[10]. Cavalli RD, Fletcher K. An effective treatment for paraquat poisoning. In: Biochemical mechanisms of paraquat toxicity. Autor AP(Ed.). New York:Academic Press. 1977;213-34.
[11]. Gao Y, Zhang X, Yang Y, Li W. Early haemoperfusion with continuous venovenous hemofiltration improves survival of acute paraquat poisoned patients. J Int Med Res. 2015;43(1):26-32.
[12]. Koo JR, Kim JC, Yoon JW. Failure of continuous venovenoushaemofiltration of preventing death in paraquat poisoning. Am J Kidney Dis. 2002;39(1):55- 99.
[13]. Hsu CW, Lin JL, Lin-Tan DT, Chen KH, Yen TH, Wu MS, et al. Early hemoperfusion may improve survival of severely paraquat-poisoned patients. PLoS One. 2012;7(10):e48397.
[14]. Tominack RL, Pond SM. Herbicides. In: Goldfrank LR, Howland MA, Flomenbaum NE, et al. Goldfrank's Toxicologic Emergencies. 7th ed. New York: McGraw-Hill; 2002;1393-410.
[15]. Rao R, Bhat R, Pathadka S, Chenji SK, Dsouza S. Golden Hours in Severe Paraquat Poisoning-The Role of Early Haemoperfusion Therapy. J ClinDiag Res. JCDR. 2017;11(2):OC06.
[16]. Singh S, Bambery P, Chaudhry D, Makharia G, Kakkar N, Singh D. Fatal paraquat poisoning: report of two cases. J AssocPhys Ind. 1999;47(8):831-32.
[17]. Agarwal R, Srinivas R, Aggarwal AN, Gupta D. Immunosuppressive therapy in lung injury due to paraquat poisoning: a meta-analysis. Singapore Med J. 2007;48:1000-05.
[18]. Eddleston M, Wilks MF, Buckley NA. Prospects for treatment of paraquat induced lung fibrosis with immunosuppressive drugs and the need for better prediction of outcome: a systemic review. QJM.

Raja Bhattacharya. "Successful Treatment of Paraquat Poisoning In a Tertiary Care Hospital of Eastern Part of India: A Therapeutic Challenge." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 4, 2019, pp 06-09.