

Survival from Paraquat Induced Renal and Pulmonary Toxicity - A Case Report from Central India

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How to citation this article: Dr. Archana Toppo, Dr. Rashmi Sahu, Dr. Khanda Shahbaz Yasin, “Survival from Paraquat Induced Renal and Pulmonary Toxicity - A Case Report from Central India”, IJMACR- March - April - 2022, Vol – 5, Issue - 2, P. No. 189 – 192.

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Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract: Paraquat poisoning have a very poor prognosis, and usually results in death, which may be due to its local caustic effect and shock or progressive development of multiple organ failure and pulmonary fibrosis. There is no specific clinically proven antidote or an evidence-based treatment protocol for the management of paraquat poisoning. So, we report a case of survival of a patient of paraquat induced renal and pulmonary toxicity. Considering the dire outcome, aggressive management approach and case reporting of the survivors is advocated.

Keywords: Paraquat, Hemo dialysis, Survival, Anti-oxidants

Introduction

Paraquat (N, N'-dimethyl-4, 4'-bipyridinium dichloride) is a brown liquid herbicide, although an uncommon

suicidal poison world-wide, it is a common but under-reported in Indian sub-continent and can lead to severe and often fatal toxicity.^[1] Paraquat poisoning have a very poor prognosis, and usually results in death, which may be due to its local caustic effect and shock or progressive development of multiple organ failure and pulmonary fibrosis. The prognosis is found to be uniformly poor world-wide, including those who treat aggressively with multimodal therapies.^[2] The pathophysiology is due to its ability to undergo redox-cycling and subsequent generation of reactive oxygen species (ROS).^[3] Apart from being secreted in urine, paraquat is actively taken up against a concentration gradient into the type II pneumocytes which could be considered a 'toxic effect' compartment. Elimination from this compartment is slower than from the other organs.^[4] There is no specific

clinically proven antidote or an evidence-based treatment protocol for the management of paraquat poisoning. So, we report a case of survival of a patient of paraquat induced renal and pulmonary toxicity. The basic theory followed was, early digestive decontamination and hemodialysis followed by antioxidant therapy.^[5]

Case Report

A 26-year-old male presented in medicine casualty of Dr. Bhimrao Ambedkar Memorial Hospital, Raipur with alleged history of ingestion of around 100 ml of paraquat (24%) 18 hours before the presentation. He complained of burning sensation in throat and multiple episodes of vomiting. There was no history of any prior psychological disorder, addictive behaviour or any suicidal attempts in past. Testimony was taken by the patient validating the type and quantity of poison consumed. He was managed conservatively with intravenous fluids, pantoprazole, ondansetron and gastric lavage was done with 10 pints of clear normal saline. On admission, all the vital parameters were normal, blood pressure was 120/80 mmHg, oxygen saturation was 97% at room air, and pulse rate was 80 per minute. He was conscious, oriented to time place and person, on auscultation, bilateral lung fields were clear with no added sound, and on cardiovascular examination S1 and S2 were normal, with no murmur or gallop. Abdomen was soft and non-tender with no organomegaly and normal bowel sounds.

He was admitted and soon started on following treatment, intravenous N- Acetylcysteine loading dose 150mg/kg over 1 hour followed by 50 mg/kg over 4 hrs and later 100 mg/kg over 16 hrs, intravenous Vitamin C 1000 mg 8 hourly, Intravenous Dexamethasone 8 mg 8 hourly, Capsule Vitamin E 400mg 12 hourly, Capsule

Vitamin D 60,000 U. On day of admission, his complete blood cell count, kidney and liver function tests and chest x ray were normal. During hospital stay, he started complaining of oral ulceration and decreased mouth opening, and developed erosions over lips and erythematous plaque over tongue and posterior pharyngeal wall suggestive of development of paraquat tongue. On day 3 of admission his urine output was decreased to 600ml/24 hour, associated with derangement in kidney function test, and hence early hemodialysis was initiated and total 5 cycles of hemodialysis were done during his hospital stay. On day 14 repeat kidney functioning showed good improvement. (Table 1) On 7th day of presentation, patient also complained of difficulty in breathing, but respiratory examination and chest x-ray were normal. However, tablet nintedanib was soon started considering the risk of pulmonary fibrosis. High resolution computed tomography of thorax was done on day 24, which showed multiple bilateral fibrotic band and multiple small cysts extending all over lung parenchyma. (Figure 2-B)

He was discharged in a stable condition with prescribed anti-fibrotic agent and was on regular follow up. He was doing well and leading a normal life on follow up after 3 months.

Parameter	Day 1	Day 3	Day 5	Day 14
Urea	37	58	95	49
Creatinine (mg/dl)	0.7	1.8	3.4	1.4
Urine output ml	1500	750	100	1350 ml

Table 1: Trend of renal function\



Figure 1: Tongue on Day 3

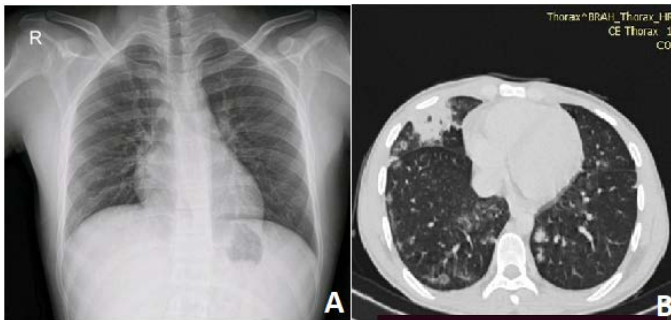


Figure 2: A. Chest X-ray on day 1, B. HRCT Thorax on day 24

Discussion

Paraquat causes both local and systemic toxicity. Although paraquat has local corrosive effect, but considering the risk benefit analysis, gastric lavage has been suggested in acute ingestion along with adsorbents like charcoal to prevent the absorption of the poison.[6] In absence of any evidence-based protocol, the management is basically supportive, and considering the poor outcome, no possible trial be left unfolded. As nothing could further worsen the poor outcome.

Vitamin E causes membrane stabilization of poly unsaturated fatty acids and reactive oxygen species scavenging, also inhibits the activation of NF- κ B (nuclear factor kappa-light-chain-enhancer of activated

B cells). N-acetyl cysteine replenishes cysteine, a rate limiting substrate for synthesis of the antioxidant glutathione, and reduces paraquat-induced apoptosis and inflammatory response [7] he common late complications among survivors were renal failure, esophageal erosions, esophagitis, and strictures he common late complications among survivors were renal failure, esophageal erosions, esophagitis, and strictures. The common late complications among survivors were renal failure, esophageal erosions, esophagitis, and strictures. he common late complications among survivors were renal failure, esophageal erosions, esophagitis, and strictures. The common late complications among survivors include, renal failure, esophageal erosions and strictures and pulmonary fibrosis,

Conclusion

In our patient with history of paraquat ingestion, early institution of hemodialysis, antioxidants, and steroids was given, along with other supportive measures. Early diagnosis, followed by early gastro-intestinal decontamination and early initiation of Hemodialysis associated with supportive treatment is the current approach used. Considering the dire outcome, aggressive management approach and case reporting of the survivors is advocated. More evidence-based studies are needed to formulate the treatment plan and adjusting dose combination and duration of therapy.

References

1. Jeyaratnam J. Acute pesticide poisoning: a major global health problem. World Health Stat Q. 1990; 43:139–44.
2. Janeela, MA sisha & John, Ajoy & Mishra, Ajay & Iyadurai, Ramya. (2017). Paraquat poisoning: Case

report of a survivor. *Journal of Family Medicine and Primary Care*. 6. 672. 10.4103/2249-4863.222042

3. Adam A, Smith LL, Cohen GM. An assessment of the role of redox cycling in mediating the toxicity of paraquat and nitrofurantoin. *Environ Health Perspect*. 1990; 85:113–7.

4. Gaudreault P, Karl PI, Friedman PA. Paraquat and putrescine uptake by lung slices of fatal and new-born rats. *Drug Me tab Dispose*. 1984; 12:550–2.

5. Lheureux P, Leduc D, Vanbinst R, Askenasi R. Survival in a Case of Massive Paraquat Ingestion. *Chest* [Internet]. 1995 Jan 1 [cited 2022 Apr 1];107(1):285–9.

6. Jiang YF, Kang J, Huang PP, Yao JX, Wang ZH, Jiang L, Wang J, Qiao L, Zhu BL, Sun H, Zhang JS. Evaluation of gastric lavage efficiency and utility using a rapid quantitative method in a swine paraquat poisoning model. *World J Emerg Med*. 2020;11(3): 174-181. Doi: 10. 5847/ wjem.j. 1920-8642. 2020.03.008. PMID: 32351651; PMCID: PMC7183924.

7. Yeh ST, Guo HR, Su YS, Lin HJ, Hou CC, Chen HM, Chang MC, Wang YJ. Protective effects of N-acetyl cysteine treatment post-acute paraquat intoxication in rats and in human lung epithelial cells. *Toxicology*. 2006; 223:181–90