International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

A Case of Survival Following Intentional Paraquat Poisoning

Dr. Gopika.G[1], Dr. Saranya[2], Dr. Amal S Kuriakose[3]

[1]Pharm D Intern, Nazareth College of Pharmacy, Othera, Thiruvalla, India
[2,3]Department of General Medicine, Believers Church Medical College Hospital, Thiruvalla, India

Abstract
Paraquat is a toxic chemical that is widely used as a herbicide (plant killer), primarily for weed and grass control. Paraquat (1, r-dimethyl-4,4'-bipyridium dichloride), a brown syrupy liquid is an effective herbicide that has low chronic toxicity due to its quick inactivation upon contact with soil [1]. Paraquat poisoning can be extremely harmful and frequently fatal. It is one of the most frequently used herbicides in the agricultural sector, but cases of its misuse in India are rarely reported [1]. Most cases of agricultural and horticultural poisoning reported in the United states, but paraquat poising had the highest mortality rate, accounting 13% of all fatal cases [2]. Paraquat is classified by the WHO as a Class II, had moderate dangerous pesticide with widely use today. Paraquat is one of the pesticides most frequently used to commit suicide [3]. When paraquat poisoning occur, lung is a primary target organ and respiratory failure from lung damage is the most common cause of death. Early diagnosis and initiation of treatment are crucial. In these article we presented a case of survival from paraquat poisoning along with its clinical features, pathophysiology and management of paraquat poisoning [4].

Keywords: Paraquat, Herbicide.

1. Introduction

Paraquat is a toxic bipyridyl compound that was discovered in 1950s. For its use in India, only the formulation of 24% SL is allowed use by Central Insecticide Board and Registration Committee. There is no any Antidote found for Paraquat poisoning from till now. It is the highest selling weed killer globally. Intoxication cases are usually from suicide attempt as is the chance of accidental exposure [5]. The organs most affected where high blood flow, oxygen elevation, and energy requirements particularly in lungs, heart, kidney and liver. To reduce the toxicity of these herbicides in event of ingestion, products containing these herbicides are often co-formulated with an emetic, dye and stenching agent.

Paraquat poisoning can be classified into three levels as Mild poisoning occur by administration of 20mg per kg of body weight causes minor stomach problems, but full recovery after time. Severe poisoning caused by 20-40 mg per kg resulting in renal failure and pulmonary damage causing patient dies from breathing failure within 2-3 weeks and fulminant poisioning caused by over 40mg per kg resulting in multi organ failure and passing from hours to days.

Mechanism of paraquat poisioning shows paraquat concentrates in alveolar cells type I and II due to its resemblance to polyamines that are absorbed by the alveolar cells. Paraquat induce redox cycling leads to production of harmful reactive oxygen species which concentrate in pulmonary alveoli and nephrons and cause stress. This oxidative stress results lung damage [7]. At moderate doses, pulmonary fibrosis develops

from lung injury by rapid and excessive fibroblast proliferation. In addition, paraquat cause vacuolation in PCT results renal tubular necrosis and also hepatocelluar injury occurs secondary to mitochondria damage and endoplasmic reticulum degranulation [7]. Paraquat undergo metabolised by several enzymes systems which generate paraquat mono-cation radical [PQ+] which inside cells oxidises back to PQ2+, releasing superoxide. Thses in turn reactive with iron to form hydroxy radicals and peroxynitrite which damage cells and tissues. Renaining nitric oxide generated also react with superoxide to forming peroxynitrite which is harmful to the organs [8]. Other process including lipid peroxidation and mitochondria damage and apoptosis are involved in this process [8].

Clinical presentation reveals that paraquat concentration in lung is 10-20 times greater than plasma because of energy dependent uptake of poison by alveoli. Pulmonary fibrosis is the hallmark of paraquat poisoning [7]. Acute respiratory distress syndrome sets in after 24-48 hours of exposure. Paraquat tongue is referred as mucosa lesions of the mouth and tongue presented as ulceration with bleeding. Paraquat itself distributes in the highly perfused organs and not metabolized by the body and excreted unchanged by the kidney. Local effects include skin irritation, blistering and burns also Inhalational exposure occur due to spraying of paraquat as fine mist and inhalation of paraquat solution causes local irritation [7].

2. Case report

A 21-year old gentleman was admitted to the emergency room with no previous co morbidity had alleged history of consumption of 30ml of paraquat, at 1.30pm following a personel problem. Five hours post consumption he was rushed nearby hospital where gastric lavage was done and managed conservatively with intravenous fluids, IV Methyprednisolone and NAC infusion on the same day. 2 cylces of hemoperfusion done on next two consecutive days followed by 1 cycle of hemodialysis was done on next day. Initiate 2 doses of IV Cyclophosphamide 900mg was given. During the hospital stay, OGD Scopy done on 72 hours post ingestion showed erosions. Blood investigation showed RFT had worsening noticeably. He was subsequently referred to our hospital for further management and in view of worsening RFT.

Upon arrival at our center patient exihibited progressive worsening of RFT and severe erosions in GI tract. On examination, his vitals were stable and he was conscious and oriented. His chest was clear without crepitations or wheeze and other systemic examinations revealed normal. Patient exhibited no vomiting, loose stools, abdominal pain, hematemesis, malena, decrease urine output, shortness of breath, chest pain was not observed.

On evaluation, his kidney function tests were deranged with creatinine was 3.54mg/dl and urea 181.6 mg/dl. Rest of the blood investigations were showed hyponatremia with Sodium level 132mmo/L and hypokalemia with potassium level 3.12mmo/L, Remaining RFT showed deranged bilirubin level with [Bilirubin total 1.07 and Direct 0.24]. His Chest X-ray was normal. ABG results came back rather abnormally. IV potassium correction was administered and oral potassium supplements were initiated due to hypokalemia. He was managed with oral antioxidant supplements and IV steroids. There is a suspicion of pulmonary fibrosis Tab.Nintedanib 100mg BD started. USG KUB showed bilateral mild diffuse increase in renal cortical echo pattern along with faint internal echoes within urinary bladder. Nephrology consultation was sought out and they prescribed Syrup Potkor 10ml TID [stop if K greater than 3.8] and continue supportive management and advice there is no need of dialysis anymore. Psychiatry consultation was done in view of stressors and family issues revaling subconscious anxiety and low self -esteem so

they counselled ongoing psychotherapy sessions on OPD basis post discharge. On third day of hospital stay his renal function becomes to reduce with Urea 171.5mg/L and Creatinine 2.99mg/L.
On fifth day of the hospital stay, patients had complaints of cough and whitish nasal discharge so advice from pulmonology department were subsequently seek. Pulmonology considered he is at high risk for lung injury so close monitoring for saturation and development of lung fibrosis was needed and HRCT chest was advised. Antioxidants like EVION C BD, VITAMIN C 500 BD and MUCINAC 600 TID. HRCT Thorax showed multifocal patchy predominant peripheral ground glass to consolidatory opacities in bilateral lungs and trace pleural effusion with cardio and spleenomegaly. Pulmonology consultation was done again with HRCT test and steroids Inj Dexamethasone 8mg TID were considered as per literature findings. On 10^{th} day of hospital stay, blood investigation showed elevated SGPT and SGOT with 131U/L and 54 U/L. USG abdomen showed Bilateral increase renal cortical echogenicity with cortico medullary differentiation with mild hepatomegaly and minimal pleural effusion. He was discharged with stable condition and was advised to be on regular follow-up. At the end of the hospital stay the blood investigation becomes normal and he was hemodynamically stable. Patient reported back after a month basis with complaints of papules over face manifesting as monomorphic and comedonal acne and Benzoyl peroxide gel was prescribed.

3. Discussion

Paraquat intoxication is a common cause of fatal herbicide intoxication often resulting from various routes of exposure. Unintentional and intentional oral ingestion, skin or respiratory tract exposure has also occurred. However parenteral route has rarely reported [9]. Commercial paraquat formulations usually contain 20%-42% w/w concentration, and are used in agriculture as water soluble dichloride salts. Paraquat can be found in preparations that combine with other herbicides like sodium chlorate and 2,4-Dimethylamine[10].Oral ingestion causes erosions of tongue and oral mucosa damages GI tract with poison concentration <20mg/L .According to the previous case reports suggst complications related to paraquat poisoning occur with consumption of 20-50mg were death occur within 2 weeks.In our case, patient take 30ml of poison according to patient history,our patient presented 5days after consumption so serum and urine paraquat levels could not be measured. Diagnosis was based on history, clinical investigations, imaging and documentation of poison consumption.
Comparing to the study conducted by Deirra M et.al, most of their paraquat intoxicated patients were from the age group of 21 to 30 years old and the majority of the exposures were intentional, mainly from deliberate self-harm [11]. Management of paraquat poisoning is mainly supportive because no antidote exists or seems nowadays. Gastric lavage should be initiated early enough to prevent the absorption of the poison and antioxidants like Vitamins C, E and N-acetylcysteine have been used. Hemoperfusion decreases paraquat levels quite rapidly and supportive hemodialysis treats patients developing tubular necrosis [12]. A recent cochrane meta-analysis concluded that patients who received glucocorticoids with cyclophosphamide in addition to standard care had a lower risk of death at final follow-up than those receiving standard care only (risk ratio 0.72; 95% confidence interval 0.59 - 0.89). Hemoperfusion decreases paraquat levels according to some case reports but evidence supporting survival benefits in humans remains inadequate. Hemodialysis should be reserved for patients with acute renal failure. Combination of cyclophosphamide and corticosteroids had half benefits in moderate to severe cases and also few cases are reported of lung transplantation after paraquat poisoning [4]. In our case, patient received gastric lavage at nearby hospital after poison administration, then they proceeded quickly to

extracorporeal therapy and was cyclophosphamide and steroids initiated. In view of AKI they preferred to our hospital and we initiated supportive management with antioxidants till he returned to normal. Similar to the study conducted by Tajai P et.al follow the same treatment as our study [13] whereas Aacha barma et.al presented a 45-year old female patient alleged ingested paraquat with suicidal intent where despite aggressive dialysis, immunosuppressants and antioxidants being administered her condition didn't improve significantly. These case study highlights there is lack of treatments options for paraquat poisoning [14]. Outcome of poisoning depends on severity and availability of medical help quickly. In our patient, he takes solution of highrisk quantity but he survive because of rapid medical intervention subsequently being discharged with outpatient psychology counselling.

4. Conclusion

We are publishing this case because reports of poisioning from paraquat an extensively used herbicide remain remarkably uncommon despite its widespread availablity. Important to know the exact causative agent of poisoning so right management can be undertaken effectively with specific treatments. In our case report, patient had aware about what he consumed so that physicians can taken the appropriate decision about the management. As there is no clinically proven antidote for paraquat poisoning. Measuring both urine and serum concentration levels various time intervals after ingestion help assess severity and predict survival rate accurately. Although there have been isolated case reports of survivors due to smallness of dose and delayed interventions. So it is crucial to focus on preventive measures and in case of exposure to prevent further complications induce death.

5. Abbrevations

OPD- Outpatient Department
PCT- Proximal Convoluted Tubule
AKI- Acute Kidney Injury
RFT- Renal Function Test
WHO- World Health Organization
OGD- Oesophago Gastro Duodenoscopy
USG- Ultra sound Sonography
NAC- N-acetylcysteine
ABG- Arterial Blood Gas

References

References within Main Content of the Research Paper

1. 1.Raghu K, Mahesh V, Sasidhar P, Reddy PR, Venkataramaniah V, Agrawal A. Paraquat poisoning: A case report and review of literature. Journal of family and community medicine. 2013 Sep 1;20(3):198-200.
2. Klein-Schwartz W, Smith GS. Agricultural and horticultural chemical poisonings: mortality and morbidity in the United States. Annals of emergency medicine. 1997 Feb 1;29(2):232-8.
3. Gupta N, Chugh A, Kanwar BS, Lamba B. A case report of paraquat poisoning. Journal, Indian Academy of Clinical Medicine. 2018;19(3):210-1.
4. Sittipunt C. Paraquat poisoning. Respiratory care. 2005 Mar 1;50(3):383-5.

5. Kavitha Saravu KS, Sonal Sekhar SS, Ananth Pai AP, Barkur AS, Rajesh V, Earla JR. Paraquat-a deadly poison: report of a case and review.
6. Agarwal R, Srinivas R, Aggarwal AN, Gupta D. Experience with paraquat poisoning in a respiratory intensive care unit in North India. Singapore Med J. 2006 Dec 1;47(12):1033-7.
7. Sukumar CA, Shanbhag V, Shastry AB. Paraquat: The poison potion. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine. 2019 Dec;23(Suppl 4):S263.
8. Gawarammana IB, Buckley NA. Medical management of paraquat ingestion. British journal of clinical pharmacology. 2011 Nov;72(5):745-57.
9. Chen CW, Wu YH, Chien SC, Lin JC. A survival case of intravenous paraquat intoxication: A case report. Medicine. 2018 Jul 1;97(30):e11669.
10. Kumar H, Singh VB, Meena BL, Gaur S, Singla R. Paraquat poisoning: a case report. Journal of Clinical and Diagnostic Research: JCDR. 2016 Feb 1;10(2):OD10.
11. Delirrad M, Majidi M, Boushehri B. Clinical features and prognosis of paraquat poisoning: a review of 41 cases. International journal of clinical and experimental medicine. 2015 May 15;8(5):8122.
12. Janeela MA, Oommen A, Misra AK, Ramya I. Paraquat poisoning: Case report of a survivor. Journal of family medicine and primary care. 2017 Jul 1;6(3):672-3.
13. Tajai P, Kornjirakasemsan A. Predicting mortality in paraquat poisoning through clinical findings, with a focus on pulmonary and cardiovascular system disorders. Journal of Pharmaceutical Policy and Practice. 2023 Dec 31;16(1):123.
14. Barma A, Poudel A, Rawal G, Karn M, Kc U, Ojha L, Bajgai D, Mandal M. Fatal paraquat poisoning: a case report and literature review on rapid deterioration and therapeutic challenges. Annals of Medicine and Surgery. 2025 Apr 1;87(4):2421-5.