

Paraquat Poisoning: Study of 15 Cases in a Tertiary Level Hospital of Bangladesh

Afroz R¹, Das AR², Haque MA³, Sultana F⁴, Rahim A⁵, Islam MR⁶

Article info

Received : 15-03-2025
Accepted : 21-09-2025
No. of Tables : 04
No. of Figure : 0
No. of References : 20

Cite the Article:

Afroz R, Das AR, Haque MA, Sultana F, Rahim A, Islam MR. Paraquat Poisoning: Study of 15 Cases in a Tertiary Level Hospital of Bangladesh. Pabna Med. Col. J. 2025;4(2): 50-53.

Keywords: Diabetic foot infection, Linezolid, Amoxicillin-Clavulanic Acid, MRSA, Randomized controlled trial, Antibiotic therapy

Introduction:

Paraquat is a N,N2 -dimethyl-4,42 -bipyridinium dichloride (systematic name) is an organic compound with the chemical formula $[(C_6H_7N)_2]Cl_2$. It is classified as a viologen, a family of redox-active heterocycles of similar structure. It's used widely in our country and is a highly toxic compound.

It is first discovered in 1950 and used in agriculture by 1962. It is rapidly absorbed by the aerial plant and immediately inactivated in contact to the clay in the soil leaving minimal residue. Being cheap and effective, paraquat is unregulated used in third world countries as herbicide.

Paraquat is commercially available in Bangladesh as a bio-pesticide in 2 formulations – 20% paraquat and 20% paraquat plus 41% glyphosate combination. As Paraquat is widely available in rural Bangladesh for its agricultural use, it has grown to be the major medium of self-harm over the years.¹

Paraquat poisoning can manifest as acute and chronic toxicity. The acute effects can result in both local and systemic manifestations. Local effects include ulceration of the skin, lips, tongue, pharynx, and esophagus. Systemic effects involve multiple organ failure, including liver insufficiency, acute kidney injury, respiratory failure, and convulsions.²

The exact mechanism of toxicity is not known completely. However, Paraquat releases free oxygen radicals (Superoxide and hydrogen peroxide) near the mucus membranes, resulting in mucosal damage in different organs due to oxidative stress, which comprises lipid peroxidation, mitochondrial toxicity, oxidation of NADPH, activation of nuclear kappa B and apoptosis.³

Symptoms of Paraquat are initially vomiting, diarrhea, oropharyngeal burns, pharyngeal pseudo membranes, abdominal pain, and acute renal and hepatic insufficiency. These are followed by progressive pulmonary fibrosis and death within two to ten weeks.⁴

According to the World Health Organization (WHO), about 10,000 persons die by suicide per year in Bangladesh.⁵ It is the fourth leading cause of overall injury-related deaths in age groups of 20–39 years in Bangladesh.⁶⁻⁷

Though Paraquat poisoning is a major cause of deliberate suicidal self-harm in Bangladesh, data on clinical presentation, demographic characteristics, modes of treatments and outcomes of the patients in the Bangladeshi population are inadequate. This study highlights over this.

Methodology:

Study Design: it is an observational study of 15 cases including prospective morbidity, clinical course, treatments, and outcome of the patients presenting with paraquat poisoning.

Place of study: Medicine dept., Shaheed Suhrawardy Medical College hospital.

Evaluation Parameters: Demographic information, symptoms at the time of admission, physical examination results, and laboratory findings were thoroughly assessed.

1. Dr. Rafiya Afroz, Indoor Medical Officer, Dept. of Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka

2. Dr. Aditi Rani Das, Indoor Medical Officer, Dept. of Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka

3. Dr. Md. Ahasanul Haque, Indoor Medical Officer, Dept. of Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka

4. Dr. Farzana Sultana, Assistant Registrar, Dept. of Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka

5. Dr. Abdur Rahim, Junior Consultant, Dept. of Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka

6. Professor Dr. Mohammad Rafiqul Islam, Professor of Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka

Corresponding Author: Dr. Rafiya Afroz, Indoor Medical Officer, Shaheed Suhrawardy Medical College Hospital, Mail: ripak65@gmail.com

Results:

Among the 15 patients studied, 87 % (n=13) were men, and the median age was 23.31 year with SD 7.083. The majority of the patients were hailing from rural areas 73% (n=11). Regarding occupation of the cases, 53.3 % (n=8) were students, followed by 20 % (n=3) farmers, 6% (n=1) related to businesses, and 6% (n=1) unemployed.

All cases were suicidal. Family disputes (60%, n=9), followed by personal relationship issues (40 %, n=6), were reported as the primary reason for paraquat poisoning.

Table-I:

Socio-demographic & clinical characteristics of paraquat poisoning patients, n (%)

	n (%)
Gender	
Male	13 (87%)
Female	2 (13%)
Age	
<20 years	4 (26.6%)
20-40 years	9 (60%)
>40 years	2 (13.3%)
Occupation	
Student	8 (53%)
Farmer	3 (20%)
Businessman	1 (6%)
unemployed	1(6%)
Residence	
Rural	11 (73%)
Urban	4 (27%)
Reason for paraquat ingestion	
Suicidal	15 (100%)
Family disputes	9 (60%)
Personal relationship issue	6(40%)
Time interval between ingestion and hospital arrival	
>24 hours	12 (80%)
6-24 Hours	2 (13.3%)
<6 hours	1(6.66%)
Symptoms During Admission	
Shortness of breath	6(40%)
Low urine output	7(46.6%)
Mucosal ulceration	15 (100%)
Difficulty in swallowing	4(26.6%)
Abdominal pain	7 (46%)
Diarrhoea	8(53%)
Vomiting	6(40%)
Black tarry stool	9(60%)
Fever	2(13%)

Table-II:

Baseline and endline biochemical profile

Investigation parameter	Mean±SD Baseline value	Mean±SD Endline value
Serum Creatinine (mg/dl)	2.1±2.31	5.34 ±9.08
Hemoglobin (g/dl)	11.6±1.7	8.22 ±2.01
WBC count (x 1000/mm ³)	11.07±3.9	15.26 ±5.91
Platelet count (x 1000/mm ³)	231.89±13.32	135.18 ±66.19
Serum ALT (U/L)	96.52±60.13	121.52±60.13

Serum creatinine was significantly higher in endline (5.344 ±9.08) compared to baseline (2.1 ±2.31). At endline, the hemoglobin level was significantly lower in the (8.22 ±2.01g/ dl) compared to the baseline (11.6 ±1.7 g/ dl).

Table-III:

Treatment received (n=15)

Treatment / Intervention	n(%)
Methylprednisolone	15 (100%)
N-acetyl cysteine	15 (100%)
Ascorbic acid	15 (100%)
Hemodialysis	10 (66.6%)
Mechanical ventilation	3 (20%)
Gastric lavage	0 (0%)

Table-IV:

Outcome of Hospital Treatment

Outcome after treatment	n (%)
Discharge with advice from hospital	12 (80%)
In-hospital death	3 (20%)

Discussion:

Acute poisoning cases with suicidal attempt in the developing world are positively associated with male gender of young age. Though study says, in Bangladesh, women commit suicide more than men.⁸ But, In this study, 87% (n=13) were men, with a median age of 28.23 years (SD 6.032).⁷

In our study, most patients were from rural areas (73%, n=11). According to a retrospective cohort study on the epidemiology of suicide in Bangladesh, the rate of suicide was found to be 17-fold higher (95% CI 5.36-54.64) in the rural population compared to urban people.⁸

Early recognition of paraquat poisoning and providing initial first aid is the cornerstone of effective management. The plasma and urine sodium dithionate tests, a qualitative assessment are simple, bedside methods to assess paraquat toxicity. To indicate severity, a quantitative assessment can be used by measuring the paraquat concentration in plasma and urine⁹. However, as these tests are not commonly available, all patients in the study were diagnosed with paraquat poisoning on the basis of history and circumstantial evidence.

The clinical manifestations of paraquat poisoning depend upon the quantity ingested. Ingestion of large amounts of liquid concentrate (>50–100 ml of 20% ion w/v) results in fulminant organ failure like pulmonary oedema, cardiac, renal and hepatic failure and convulsions due to CNS involvement. Death results within several hours to a few days.²

Mild poisoning (<20mg/kg) causes minimal renal and pulmonary involvement with complete recovery. Moderate poisoning (20–40 mg/kg body weight) causes renal failure, hepatic impairment, hypotension/tachycardia, alveolitis, pulmonary fibrosis. In these cases, survival depends upon effective management.^{10,11}

As a corrosive agent, paraquat causes mucosal lesions of the mouth and the tongue that is called “paraquat tongue”. They begin to appear within the first few days and may become ulcerated with bleeding. Esophageal ulceration causes pain and dysphagia.¹² All of our study patients developed paraquat tongue.

In this study, major organ involvement included acute kidney injury (80%), acute respiratory distress syndrome (32%), hepatic impairment (20%), and septic shock (13%). Initial presentation with acute kidney injury and late development of pulmonary fibrosis is observed in several case reports in acute paraquat poisoning in Bangladesh.¹³

In majority of our study patients, initial presentation was AKI evidenced by raised creatinine, n=12 (80%). Among these, 10 patients needed hemodialysis.

AKI is common following paraquat ingestion, and certain clinically useful variables such as acute hepatitis, the time to hospital arrival, Severity Index and Plasma Paraquat (SIPP) score and PaO₂ at admission are powerful predictors of AKI.¹⁴

In our study, 6(32%) patients developed lung involvement in the form of mild respiratory distress to consolidation on imaging, among them 3 patients need mechanical ventilation due to ARDS, and all of them died subsequently. As the hypoxemic strategy will reduce the production of ROS, so routine use of supplemental oxygen is not recommended in these patients with mild-to-moderate hypoxia. The development of type 2 respiratory failure may be an early predictor of ARDS in patients with worse outcome.²

But, following lung fibrosis, a decrease in arterial O₂ saturation and tissue hypoxia occurs when the patients need admitted to intensive care units (ICU), where they are placed under mechanical ventilation.¹⁵

Regarding management, there is no effective antidote, only supportive management depending upon organ involvement. We do not recommend gastric lavage as its use is contraindicated in caustic injury.²

Paraquat toxicity is primarily due to oxidative stress. So Immunosuppression is widely practiced as a treatment of paraquat self-poisoning, that inhibits acute inflammatory response, ultimately slowing the processes of lung fibrosis and reduces death. The most widely studied regimen uses cyclophosphamide, MESNA, methylprednisolone and dexamethasone.¹⁵ In our study, we used 1g of methylprednisolone for 5 days from very first day of admission in hospital for all patients along with antioxidants like N-acetylcysteine, ascorbic acid. A cohort study shows that methylprednisolone-treated patients had better survival (P = 0.04). Though, no strong evidence supports the use of vitamin C or N-acetylcysteine.¹⁶

A cross-sectional study shows, in-hospital fatality rate was found to be 21.8%. Several other studies have reported paraquat poisoning mortality rates ranging from 33.0% to 91.7%.¹⁷

Multivariate analysis revealed that the amount of paraquat ingested and clinical presentations, particularly pulmonary and cardiovascular system disorders, were significantly associated with mortality.¹⁸

The majority of the population in rural Bangladesh are dependent on agriculture. Due to wide availability of paraquat pesticides and lack of urgent primary care in rural areas has led to an increased rate of case fatality in acute paraquat poisoning.

As study shows removing Highly hazardous pesticides(HHPs) from agriculture can rapidly reduce suicides without any apparent effect on agricultural output.¹⁹For same reason, paraquat was banned in Korea in 2012.²⁰So it is high time to rethink about control of paraquat as pesticides.

Conclusion:

In conclusion, this study of paraquat poisoning cases in Bangladesh provides valuable insights into the clinical profile, treatment outcomes, and management approaches. High mortality even with multiple treatment approaches emphasizes the need to restrict the use of paraquat poisoning and look for safer alternative pesticides for agricultural use.

Recommendation:

Paraquat has a long history of existence. However, its agricultural and commercial success is deemed naughty by its fatal outcome, even with mechanical ventilation or hemodialysis support when used as a method of deliberate self-poisoning. So, it is high time to restrict its access, storage, use, or ban it and shift to safer alternative herbicides for agricultural purposes.

There is male predominance of suicidal tendency in this study. We should address it, give emphasis to find out causes and solution in psychosocial aspects.

References:

- List of Registered Agricultural Pesticides, Bio Pesticides Approved up to 81st Pesticide Technical Advisory Committee (PTAC) Meeting (2020), Department of agriculture Extension, Bangladesh. 2020;137-144.
- Gawarammana, I. B., & Buckley, N. A. (2011). Medical management of paraquat ingestion. *British journal of clinical pharmacology*, 72(5), 745-757.
- Yasaka T, Ohya I, Matsumoto J, Shiramizu T, Sasaguri Y. Acceleration of lipid peroxidation in human paraquat poisoning. *Arch Intern Med*. 1981;141:1169-71
- Delirrad, M., Majidi, M., & Boushehri, B. (2015). Clinical features and prognosis of paraquat poisoning: a review of 41 cases. *International journal of clinical and experimental medicine*, 8(5), 8122-8128.
- World Health Organization. (2014). Preventing suicide: A global imperative. World Health Organization, 12-14
- Shahnaz A, Bagley C, Simkhada P, Kadri S. Suicidal behaviour in Bangladesh: A scoping literature review and a proposed public health prevention model. *Open Journal of Social Sciences*. 2017 Jul3;5(7):254-82.
- Khan, F. B. S. ., Rahman, A. S. M. M. ., Haque, M. Z. ., & Rahman, M. K. . (2023). Outcome of paraquat poisoning patients admitted in a tertiary hospital in Bangladesh. *TAJ: Journal of Teachers Association*, 36(2), 35-46
- Chan, L. F., Chin, S. J., Loo, T. H., Panirselvam, R. R., Chang, S. S., Chang, H. Y., Mokhzani, A. R., Rahman, F. H. A., Utyasheva, L., & Eddleston, M. (2023). Pesticide poisoning and the early impact of a national Paraquat ban. *BMC psychiatry*, 23(1), 472
- Shanbhag V, Sukumar CA, Shastry AB. Paraquat: The Poison Potion. *Indian Journal of Critical Care Medicine*. 2019 Dec;23(S4):0-0.
- Sabzghabae AM, Eizadi-Mood N, Montazeri K, Yaraghi A, Golabi M. Fatality in paraquat poisoning. *Singapore Med. J*. 2010;51:496-500.
- Shanbhag V, Sukumar CA, Shastry AB. Paraquat: The Poison Potion. *Indian Journal of Critical Care Medicine*. 2019 Dec;23(S4):0-0.
- Lock EA, Paraquat Wilks MF, In: *Handbook of pesticide toxicology*, 3rd ed., Krieger RI San Diego: Academic Press; 2010.
- Isha, I. T., Alam, Z. H. M. N., Shaha, B. K., Bari, M. S., Bari, M. Z. J., & Chowdhury, F. R. (2018). Paraquat induced acute kidney injury and lung fibrosis: a case report from Bangladesh. *BMC research notes*, 11(1), 344.
- Weng, C. H., Chen, H. H., Hu, C. C et al (2017). Predictors of acute kidney injury after paraquat intoxication. *Oncotarget*, 8(31), 51345-51354
- Delirrad M, Majidi M, Boushehri B. Clinical features and prognosis of paraquat poisoning: a review of 41 cases. *Int. J. Clin. Exp. Med*. 2015; 8:8122-8128.
- Chen C-K, Yeh Y-T, Mégarbane B, et al. A novel flowchart to predict mortality in paraquat poisoning. *Basic Clin Pharmacol Toxicol*. 2021; 129(6): 496-503.
- Liu H-L, Chen W-L, Yang M-C, Lin H-M, Chou C-C, Chang C-F, et al. Prediction of early mortality in patients with paraquat intoxication. *J Acute Med*. 2013;3(1):6-10.
- Tajai, P., & Kornjirakasemsan, A. (2023). Predicting mortality in paraquat poisoning through clinical findings, with a focus on pulmonary and cardiovascular system disorders. *Journal of Pharmaceutical Policy and Practice*, 16(1).
- Chowdhury FR, Dewan G, Verma VR, Knipe DW, Isha IT, Faiz MA, et al. Bans of WHO Class I Pesticides in Bangladesh—suicide prevention without hampering agricultural output. *International Journal of Epidemiology*. 2018 Feb 1;47(1):175-84.
- Kim J-W, Kim D-S. Paraquat: toxicology and impacts of its ban on human health and agriculture. *Weed Science*. 2020;68(3):208-213. doi:10.1017/wsc.2019.70.