

Gastrointestinal System Related Clinical Features and Endoscopic Findings of Paraquat Poisoning Observed at a Tertiary Care Hospital

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ABSTRACT

Background: In South East Asia, Paraquat poisoning is a serious health issue. It is a nonselective contact herbicide (1,1'-Dimethyl-4,4'-bipyridinium dichloride). Plants are killed quickly by translocation of this herbicide into cells after contact. When it comes into touch with soil clay, it is deactivated. Due to its innate toxicity, it is extremely fatal for human. Median lethal dose is 10-15 ml of 20% paraquat solution. ARDS and pulmonary fibrosis are the leading causes of death. This cross sectional study was carried out to have a look into the occurrence of sign symptoms as tongue ulcer, nausea, vomiting, abdominal pain, diarrhoea, upper GI ulcer on endoscopy to determine their association with paraquat poisoning.

Methods: From July 2022 to June 2023, this cross-sectional study was carried out at the Department of Medicine of Rangpur Medical College Hospital, Rangpur. Total number of 60 subjects were selected after satisfactory inclusion and exclusion criteria from indoor patients' department of Medicine, Rangpur Medical College Hospital to observe presentation, in-hospital complications of paraquat poisoning and to see the relation of death to ingestion of ≥ 10 ml 20% paraquat ingestion. They were divided into two groups: **Group-A** - history of taking ≥ 10 ml 20% paraquat and **Group-B** - history of taking < 10 ml of 20% paraquat. Stomach wash was more frequently given in case of ingestion of ≥ 10 ml 20% paraquat. For statistical analysis IBM SPSS version 26.0 was used; 'Chi-square' test were performed as test for significance and $p < 0.05$ was considered as level of significance.

Results: Tongue ulcer, nausea, vomiting, abdominal pain was more common among the patient ingesting ≥ 10 ml 20% paraquat. Upper GI ulcer involving oesophagus and stomach on upper GI endoscopy occurred if an individual ingests ≥ 10 ml 20% paraquat solution.

Conclusion: Gastrointestinal manifestations and complications are prominent; upper GI ulcer involving oesophagus and stomach seen on upper GI endoscopy may occur if an individual ingests ≥ 10 ml 20% paraquat solution.

Key Words: Paraquat poisoning, Upper Gastrointestinal ulcers.

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INTRODUCTION

Weidel and Russo published the first description of paraquat in 1882. Its redox characteristics were identified in 1933 by Michaelis and Hill, who gave the substance the name, methyl viologen. Paraquat's herbicidal qualities were originally discussed in 1958, and it was first made commercially available in 1962. The principal form of paraquat is an aqueous solution with surface-active ingredients. A low strength granular formulation (also containing diquat) is offered in several countries. Paraquat is a non-selective, fast-acting contact herbicide¹. When it

comes into touch with soil clay, it is deactivated². In the 1960s, New Zealand and Ireland reported the first human exposure instances, which were brought on by accidental intake³. In many regions of Asia, including Bangladesh, paraquat, is one of the major causes of poisoning that results in death⁴. Median lethal dose (LD₅₀) in humans, which is roughly 3-5 mg/kg, 10-15 ml of a 20% solution would be sufficient⁵.

It is known that 90% of the paraquat in the body is eliminated within 12 to 24 hours following fast absorption (Pond, et al., 1993). Paraquat is sequestered inside the body and undergoes sluggish clearance,

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which may be caused by kidney and liver damage⁶. The remaining Paraquat is concentrated inside numerous cells where it undergoes redox cycling and produces reactive oxygen species. Several enzyme systems are involved in the metabolism of paraquat (NADPH-cytochrome *p*450 reductase; Xanthine oxidase; NADH: ubiquinone oxidoreductase and nitric oxide synthase)⁶.

Upon metabolism by these systems, a paraquat monocation radical (PQ⁺) is produced. PQ⁺ quickly reoxidizes to PQ₂⁺ inside the cell, producing superoxide (O₂^{•-}) in the process. In this reaction, (O₂^{•-}) serves as an electron acceptor and NADPH as an electron donor. In addition, this causes the Fenton reaction, which results in the creation of the hydroxyl free radical (HO[•]), in the presence of iron. Peroxynitrite (ONOO⁻), a very potent oxidant and nitrating intermediate, is produced when NO and O₂^{•-} combine. Nitric oxide is created enzymatically from L arginine by NO synthase, and PQ also directly or indirectly stimulates this enzymatic generation of NO⁷.

Generation of highly reactive oxygen and nitrite species results in toxicity. So, damage occurs to the mitochondria in numerous cell types⁸, oxidation of the NADPH, which results in an increase in oxidative stress susceptibility⁹, generation of inflammatory cytokines as a result of NF-κB activation¹⁰. All these processes eventually result in cellular death. Toxicity is particularly severe in the lungs as paraquat is taken up against the concentration gradient in to the lung⁶.

Pneumonitis and lung fibrosis are caused by the active concentration of paraquat in lung tissue¹¹. Additionally, paraquat harms the liver, kidney, and gastrointestinal systems¹². In humans, ARDS and pulmonary fibrosis are the leading causes of death¹³.

There is no known specific antidote, hence the fatality rate is extremely high. Early arrival, quick decontamination, and the implementation of resuscitative procedures are essential for management. Further absorption can be reduced using Fuller's earth and activated charcoal. Contraindications apply to gastric lavage. Hemodialysis and hemoperfusion have a low chance of altering the clinical trajectory.

Weak evidence supports the use of immunosuppression with dexamethasone, cyclophosphamide, and methylprednisolone. It might be advantageous to employ antioxidants like acetylcysteine and salicylate to scavenge free radical⁶.

This study was carried out with an aim to observe tongue ulcer, nausea, vomiting, abdominal pain,

diarrhoea and upper GI ulcer in endoscopy to determine their association with paraquat ingestion specially at higher doses (≥ 10 ml of 20% solution).

MATERIALS & METHODS

This cross-sectional study was carried out at the Department of Medicine of Rangpur Medical College Hospital, Rangpur from July 2022 to June 2023. A total of 60 patients were included in this study by purposive sampling technique. Minimum age was 18 years. They were divided into two group, **Group-A**, 30 patients with a history of taking ≥ 10 ml 20% paraquat and **Group-B**, 30 patients with a history of taking < 10 ml of 20% paraquat. Patient who had history of multiple drugs poisoning and paraquat was not properly ingested or thrown away just after taking into the oral cavity were excluded from the study. Several clinical and upper GI endoscopy variables were studied.

Data were coded, edited and entered into computer and were analyzed by using SPSS program. Data presented on categorical scale were expressed as frequency and corresponding percentages and were compared between groups using Chi-square (χ^2) test and p value < 0.05 was taken as significant. Ethical consideration was achieved by taking an informed written consent after briefing about objectives of the study. Quality was assured through avoidance of missed data, filling of code, regular entry of data and careful data analysis.

RESULTS

Female to Male ratio was 1.22:1.

In Group A (≥ 10 ml of 20% paraquat) 26 cases expired out of 30 and in Group B (< 10 ml of 20% Paraquat) 1 case expired out of 30.

In this study patients with tongue ulcer were 30(100%), 18(60%) in Group-A and Group-B respectively, and the difference in the development of tongue ulcer (paraquat tongue) was significant ($p < 0.05$).

Patients with stomach wash needed were 28(93.334%), 6(20%) in Group-A and Group-B respectively. The need for stomach was statistically significant ($p < 0.05$) between the groups.

The symptom of nausea, vomiting and abdominal pain all were significantly different between Group-A and Group-B ($p < 0.05$ for all symptoms) (Table I).

Table I: Comparison of clinical and endoscopic parameter related to gastrointestinal system between two groups in study patients.

Variable	Group-A (n=30) N (%)	Group-B (n=30) N (%)	p value
Oral ulcer			
Present	30(100)	18(60)	^a 0.000 ^s
Absent	0(0)	12(40)	
Stomach wash			
Given	28(93.33)	6(20)	^a 0.000 ^s
Not given	2(6.668)	24(80)	
Nausea			
Present	25(83.334)	13(56.668)	^a 0.000 ^s
Absent	5(16.667)	17(43.333)	
Vomiting			
Present	26(86.668)	13(43.334)	^a 0.000 ^s
Absent	4(13.334)	17(56.667)	
Abdominal pain			
Present	24(80)	13(43.2)	^a 0.003 ^s
Absent	6(20)	17(56.667)	
Diarrhoea			
Present	2(6.668)	0(0)	^b 0.492 ^{ns}
Absent	28(93.334)	30(100)	
Upper GI ulcer on endoscopy			
Present	*26(86.67)	12(40)	^a 0.000 ^s
Absent	4(13.334)	18(60)	

(*4 patients refused to do endoscopy)

Group-A: Ingestion of ≥ 10 ml of 20% paraquat

Group-B: ingestion of < 10 ml of 20% paraquat

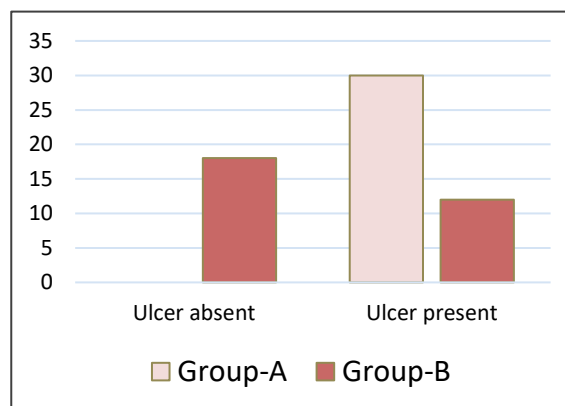
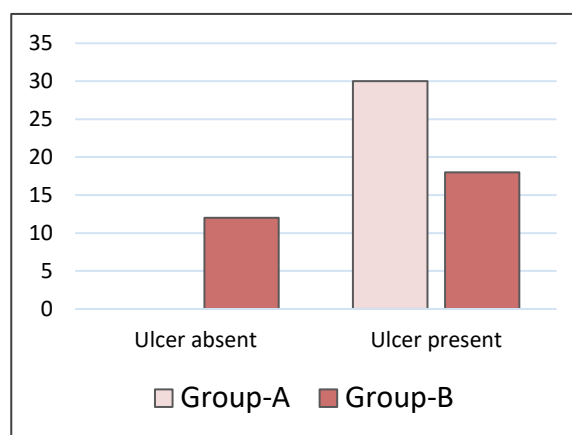
s=significant ns=not significant

^aP value reached from chi square test

^bP value reached from Fisher exact test

Diarrhoea was a symptom in 2(6.668%) cases of Group-A, where there was no such case in Group-B. ($p > 0.05$)

On endoscopy of upper GIT, 26(86.667%) of Group-A revealed upper GI ulcer whereas 12(40%) in Group-B revealed so ($p < 0.05$), and 4 patients from Group-A refused to undergo endoscopy. (Table I)

**Figure 1:** Bar chart showing upper GI ulcer due to ingestion of paraquat in between two groups.**Figure 2:** Bar chart showing incidence of tongue ulcer due to paraquat ingestion in between two groups.

DISCUSSION

In this current study patients with nausea were 25(83.4%), 13(43.4%) in Group-A and Group-B respectively. The development of nausea was statistically significant ($p < 0.05$) between the both groups in chi square test. which is similar to Delirrad et al, (2015) where it shows 53.7% cases of nausea among study population. It might have happened due to gastro-intestinal irritation¹⁴.

Vomiting was a symptom in 26(86.668%), 13(43.4%) of Group-A and Group-B respectively. The development of vomiting was statistically significant ($p < 0.05$) between the both groups in chi square test. which is similar to Delirrad et al, (2015) where it shows p value is 0.004

which was statistically significant. It might have happened due to gastro-intestinal irritation¹⁴.

Patients with abdominal pain was 24(80%), 13(43.2%) in Group-A and Group-B respectively. The development of abdominal pain was statistically significant ($p < 0.05$) between the both groups in chi square test. which is similar to Delirrad et al, (2015) where it shows 36.6% epigastric pain. It might also have happened due to gastro-intestinal irritation and direct mucosal damage by paraquat due to its toxic effect¹⁴.

In this current study patients with diarrhoea were 2(6.668%), 0(0%) in Group-A and Group-B respectively. The development of diarrhoea was not statistically significant ($p > 0.05$) between the both groups in chi square test. which is not similar to Peterson et al (2013) where the book tells us that diarrhoea is common among paraquat poisoning patients¹⁵.

Upper GI ulcer revealed on endoscopy was 26(86.667%), 12(40%) in Group-A and Group-B respectively; (here 4 patients from group A, has refused to undergo endoscopy of upper GIT). The development of upper GI ulcer was statistically significant ($p < 0.05$) between the both groups in chi square test. which was similar to Chen et al. (2013) where p value was 0.005. It might have happened due to gastro-intestinal irritation and direct mucosal damage by paraquat due to its toxic effect¹⁶.

Conclusion

This study was undertaken to find out presentation and in hospital complication of paraquat poisoning to ingestion of ≥ 10 ml 20% paraquat ingestion. Where stomach wash was more frequently given in case of ingestion of ≥ 10 ml 20% paraquat. Tongue ulcer, nausea, vomiting, abdominal pain among the patient ingesting ≥ 10 ml 20% paraquat. Upper GI ulcer involving oesophagus and stomach on upper GI endoscopy can occur if an individual ingests ≥ 10 ml 20% paraquat solution.

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