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SPECTRUM OF GASTROINTESTINAL EVENTS AFTER PARAQUAT INGESTION: A CROSS-SECTIONAL STUDY

Gastroenterology

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ABSTRACT

Paraquat, a widely used herbicide in India despite global bans, poses significant health hazards due to its systemic toxicity and corrosive potential. Gastrointestinal (GI) injury, particularly to the esophagus and stomach, remains an underexplored consequence of ingestion. This cross-sectional study aimed to evaluate the spectrum of GI corrosive injuries in paraquat ingestion and their association with systemic complications. The study was conducted over 12 months in the Department of Medical Gastroenterology at a tertiary care hospital. A total of 72 adult patients with confirmed paraquat ingestion were consecutively enrolled. Clinical data, laboratory investigations, and upper GI endoscopy findings were systematically recorded. Esophageal and gastric injuries were graded using Zargar's classification. Systemic complications, including acute kidney injury (AKI) and acute liver injury (ALI), were also analyzed. The mean age of participants was 36.5 years, with males predominating (79.2%). Most cases (91.6%) resulted from intentional ingestion. AKI (63.9%) was the most frequent complication, followed by ALI (25.0%) and hematemesis (11.1%). Esophageal injuries were most commonly Grade II B (47.1%), while gastric injuries were predominantly Grade I (36.1%) and Grade III A (29.2%). Significant associations were observed between esophageal injury severity and duration of exposure ($p = 0.021$), hematemesis ($p = 0.001$), AKI ($p = 0.001$), and ALI ($p = 0.014$). For gastric injuries, hematemesis ($p < 0.001$) and ALI ($p = 0.019$) were significantly correlated with severity. This study, the largest to date on paraquat-related corrosive GI injury, highlights the clinical significance of systemic complications in predicting injury severity. The findings emphasize the need for early endoscopic evaluation, aggressive monitoring of high-risk patients, and stricter public health measures regulating paraquat use.

KEYWORDS

INTRODUCTION

Paraquat is a highly toxic bipyridyl herbicide introduced in the 1960s and remains widely used in India because of its affordability and agricultural effectiveness. Despite being banned in more than 30 countries, its application continues across several crops in India, often beyond approved uses and with minimal regulatory oversight. Ingestion of paraquat results in severe systemic toxicity, particularly affecting the lungs, kidneys, and liver, and no specific antidote is available. A tertiary care study from southern India reported a mortality rate of 72.7% among paraquat poisoning cases[1].

Corrosive gastrointestinal injury is a major concern following ingestion, with the esophagus and stomach being the most frequently affected. These injuries may result in strictures, perforations, chronic dysphagia, prolonged hospitalization, and increased mortality[2,3]. Most cases occur as intentional self-harm, with reported mortality rates ranging from 10% to 30% depending on severity and systemic involvement[4,5].

Herbicide-related caustic injuries, including paraquat, have been increasingly documented. Distinct toxicological features such as multiorgan dysfunction make paraquat more hazardous compared to conventional caustic agents[6]. Chen et al. demonstrated high rates of renal and hepatic complications among patients with paraquat-induced esophageal injury[7]. An Indian retrospective study similarly reported acute kidney injury in 92% of cases and acute respiratory distress syndrome in 44%, with mortality exceeding 48%[8]. Long-term consequences include chronic dysphagia, malnutrition, and increased risk of esophageal carcinoma[9].

Despite its clinical importance, published evidence on corrosive injuries due to paraquat ingestion is sparse, primarily consisting of case reports and small retrospective series[9]. Available data suggest predominant involvement of the upper gastrointestinal tract[10]. In view of ongoing widespread use in India and limited systematic data, our study aimed to classify injuries using the Zargar system and assess associations with renal, hepatic, and pulmonary complications.

MATERIALS AND METHODS

Study Design

This was a cross-sectional study.

Study Setting And Duration

This cross-sectional study was conducted in the Department of Medical Gastroenterology at a tertiary care hospital. Patients presenting to the emergency department with a history of paraquat ingestion were included. The study was carried out over 12 months, beginning from the date of Institutional Ethics Committee approval.

Ethical Considerations

Ethical clearance was obtained prior to commencement. Written informed consent was obtained, and confidentiality was maintained. The study adhered to ethical guidelines for human research.

Inclusion And Exclusion Criteria

Eligible participants were adults (≥18 years) with a documented history of paraquat ingestion. Exclusion criteria included patients <18 years, pregnant women, those requiring ventilatory support, and individuals who did not provide consent.

Sample Size And Sampling Technique

Seventy-two patients were recruited using purposive sampling. All eligible individuals presenting during the study period were consecutively enrolled to ensure complete case capture.

Study Procedure

After consent, demographic details, intent of ingestion (accidental or intentional), and duration of exposure (DOE) were recorded. Clinical evaluation emphasized nausea, vomiting, oral burns, dysphagia, and

respiratory distress. Investigations included complete blood count, renal and liver function tests, arterial blood gas analysis, and chest radiography. Upper gastrointestinal endoscopy was performed by a senior gastroenterologist using a flexible endoscope. Management was individualized according to clinical severity, and outcomes were systematically documented. Data were compiled into a structured database.

Definitions of Clinical Events

- **Acute Kidney Injury (AKI):** Rise in serum creatinine ≥0.3 mg/dL within 48 hours, or ≥1.5 times baseline within 7 days, or urine output ≤0.5 mL/kg/h for 6 hours.
- **Acute Liver Injury (ALI):** ALT ≥70 U/L or total bilirubin >3.0 mg/dL.
- **Hematemesis:** Vomiting of fresh or altered blood.
- **Upper GI Injury:** Classified according to Zargar's grading system.

Variables And Statistical Analysis

Independent variables were age, sex, DOE, hematemesis, AKI, and ALI. Outcome variables were severity of esophageal and gastric injuries. Continuous variables were summarized as mean ± standard deviation; categorical data as frequencies and percentages. Associations were tested using appropriate statistics, with significance set at $p<0.05$.

RESULTS

A total of 72 patients were enrolled, with a mean age of 36.5 ± 14.04 years. The majority (54.2%) were between 30–60 years, followed by 17–30 years (40.3%), while only 5.6% were older than 60. Males predominated (79.2%), and intentional ingestion was observed in 91.6%. The most frequently reported durations of exposure were 24 hours (37.5%) and 28 hours (33.3%).

Systemic complications were common. Acute Kidney Injury (AKI) occurred in 63.9% of patients, Acute Liver Injury (ALI) in 25.0%, and hematemesis in 11.1%.

Esophageal Injury

Endoscopic evaluation showed Grade II B as the most frequent esophageal lesion (47.1%), followed by Grade II A (25.7%) and Grade I (15.7%). Severe esophageal injury (Grade III A) was seen in 2.9% of cases.

Gastric Injury

In the stomach, Grade I lesions were most frequent (36.1%), followed by Grade III A (29.2%) and Grade 0 (25.0%).

Associations

Analysis of demographic and clinical parameters revealed that severity of esophageal injury was significantly associated with duration of exposure ($p = 0.021$), hematemesis ($p = 0.001$), AKI ($p = 0.001$), and ALI ($p = 0.014$). No significant associations were found for age ($p = 0.542$) or sex ($p = 0.256$).

For gastric injury, hematemesis ($p < 0.001$) and ALI ($p = 0.019$) were significantly associated with higher severity, while age ($p = 0.119$), sex ($p = 0.905$), duration of exposure ($p = 0.319$), and AKI ($p = 0.731$) were not.

DISCUSSION

Paraquat, chemically known as 1-methyl-4-(1-methylpyridin-1-ium-4-yl)pyridin-1-ium[12], is stable in acidic environments but unstable in alkaline media, where hydrolysis may contribute to corrosive injury[13]. It reacts with strong acids, bases, and oxidizing agents and is highly corrosive to metals. Following ingestion, about 20% is absorbed in the gastrointestinal tract, mainly in the small intestine. Once absorbed, it is rapidly distributed to highly perfused organs, including the lungs, kidneys, liver, and muscles. Importantly, paraquat is not metabolized and is eliminated unchanged in the urine. In minor exposures, most of the absorbed dose is excreted within 12–24 hours[1].

Literature on gastrointestinal corrosive injury due to paraquat ingestion is limited, with only a handful of reports between 1978 and 1993 and one study in 2013 addressing this issue[14-18]. Given the ongoing widespread availability of paraquat in India, our series is particularly relevant and may represent the largest systematic evaluation of its corrosive effects.

In our cohort, the most frequent esophageal injury was Zargar Grade II B (47.1%), followed by Grade II A (25.7%), with severe Grade III A injury in only 2.9%. Gastric injuries were most often Grade I (36.1%) and Grade III A (29.2%). These findings differ slightly from Chen et al., who reported Grade II A as the predominant esophageal injury[7]. Zargar et al. previously demonstrated that Grades II and III are strongly associated with strictures and long-term dysphagia[5,9].

We found no significant association between age or gender and injury severity. This contrasts with Hoffman et al., who reported that younger patients often sustain more severe injuries due to accidental ingestion[19]. Similarly, Kluger et al. suggested that gender-related differences might reflect the type of corrosive substance ingested, with males more frequently exposed to industrial agents and developing more severe damage[20]. Our findings suggest that clinical parameters may have a greater impact than demographic variables.

Significant associations were observed between esophageal injury severity and duration of exposure ($p=0.021$), hematemesis ($p=0.001$), AKI ($p=0.001$), and ALI ($p=0.014$). Gastric injury severity was linked to hematemesis ($p<0.001$) and ALI ($p=0.019$). These correlations are consistent with Ryu et al., who emphasized exposure duration as a predictor of upper GI damage[3]. Hematemesis, a marker of mucosal ulceration, has also been linked with advanced injury. Similarly, Rafeey et al. reported systemic complications, including AKI and ALI, as common in severe caustic ingestion[21].

The relatively low proportion of Grade III A esophageal injuries in our series may reflect differences in substance concentration and ingestion practices, a variation also described by Hall et al.[20]

Our results underscore the importance of early endoscopic evaluation and close monitoring of patients with systemic complications. These findings align with Kluger et al., who advocated for early stratification to guide management[18]. Beyond clinical practice, preventive strategies including stricter regulation of paraquat availability and public education are vital to reduce morbidity and mortality.

Strengths And Limitations

A key strength of this study is the use of a standardized endoscopic grading system, allowing direct comparison with published literature. Inclusion of systemic complications such as AKI and ALI provided a broader assessment of paraquat's clinical impact. Importantly, this represents the largest reported series on paraquat-induced corrosive injury, adding valuable data to scarce existing evidence.

Limitations include the relatively small sample size, which may restrict generalizability. Exclusion of patients requiring ventilatory support could underestimate the true spectrum of severe injuries. The cross-sectional design also limited evaluation of long-term outcomes such as strictures or malignancy. Future multicenter, prospective studies with extended follow-up are required.

CONCLUSION

This study highlights that paraquat ingestion predominantly affects middle-aged males, usually through intentional exposure. Esophageal injury was most often Zargar Grade II B, while gastric injury was commonly Grade I, though severe Grade III A lesions were also observed. Duration of exposure, hematemesis, AKI, and ALI were significantly associated with injury severity. These findings underscore the need for early risk identification, close monitoring, and stronger public health regulation of paraquat use in India.

Table 1: Zargar's Grading Of Corrosive Injuries

Grade	Description
0	No injuries
I	Edema and hyperaemia
IIa	Superficial ulceration, erosions, friability, blisters, exudates, hemorrhages, membranes, whitish.
IIb	Grade IIa + discrete or circumferential deep ulcerations
IIIa	Small, scattered areas of multiple ulceration and areas of necrosis with blackish-brown or greyish discoloration.
IIIb	Extensive necrosis

Table 2. Distribution Of Demographic And Clinical Characteristics

Variable	Frequency (n)	Percentage (%)

Age in years		
17-30	29	40.28
30-60	39	54.17
>60	4	5.56
Sex		
Female	15	20.83
Male	57	79.17
Duration Of Exposure (hours)		
<8	7	9.72
8-12	14	19.44
12-24	27	37.5
24-48	24	33.33
Hematemesis	8	11.11
Acute Kidney Injury	46	63.89
Acute Liver Injury	18	25.0

Table 3: Esophageal And Gastric Injury

Zargar Classification	Frequency (n)	Percentage (%)
Esophageal injury		
Grade 0	8	11.11
Grade I	11	15.27
Grade II A	18	25.71
Grade II B	33	45.83
Grade III A	2	2.77
Gastric injury		
Grade 0	18	25.0
Grade I	26	36.11
Grade II A	7	9.72
Grade III A	21	29.17

Table 4: Association Of Demographic And Clinical Features With Corrosive Esophageal Injury

Variable	Grade of Esophageal injury					χ^2	P-value
	0 n (%)	1 n (%)	2A n (%)	2B n (%)	3A n (%)		
Age in years							
17-30	3 (10.34)	7 (24.14)	6 (20.69)	13 (44.83)	0 (0.00)	6.95	0.542
30-60	3 (8.11)	3 (8.11)	10 (27.03)	19 (51.35)	2 (5.41)		
>60	0 (0.00)	1 (25.00)	2 (50.00)	1 (25.00)	0 (0.00)		
Sex							
Female	1 (7.14)	0 (0.00)	6 (42.86)	7 (50.00)	0 (0.00)	5.32	0.256
Male	5 (8.93)	11 (19.64)	12 (21.43)	26 (46.43)	2 (3.57)		
DOE (hrs)							
<8	2 (28.57)	1 (14.29)	1 (14.29)	3 (42.86)	0 (0.00)	23.85	0.021
8-12	4 (16.00)	5 (20.00)	9 (36.00)	5 (20.00)	2 (8.00)		
12-24	0 (0.00)	5 (20.83)	5 (20.83)	14 (58.33)	0 (0.00)		
24-48	0 (0.00)	0 (0.00)	3 (21.43)	11 (78.57)	0 (0.00)		
Hematemesis	0 (0.00)	0 (0.00)	2 (25.00)	4 (50.00)	2 (25.00)	17.71	0.001
AKI	6 (25.00)	6 (25.00)	6 (25.00)	6 (25.00)	0 (0.00)	18.35	0.001
ALI	6 (11.54)	11 (21.15)	12 (23.08)	23 (44.23)	2 (11.11)	12.57	0.014

Table 5. Association Of Demographic And Clinical Features With Corrosive Gastric Injury

Variable	Grade of Gastric injury				χ^2	P-value
	0 n (%)	1 n (%)	2A n (%)	3A n (%)		
Age in years						
17-30	6 (20.69)	12 (41.38)	1 (3.45)	10 (34.48)	10.1	0.119
30-60	11 (28.21)	13 (33.33)	4 (10.26)	11 (28.21)		
>60	1 (25.00)	1 (25.00)	2 (50.00)	0 (0.00)		
Sex						
Female	3 (20.00)	6 (40.00)	2 (13.33)	4 (26.67)	0.56	0.905
Male	15 (26.32)	20 (35.09)	5 (8.77)	17 (29.82)		
DOE (hrs)						
<8	2 (28.57)	1 (14.29)	1 (14.29)	3 (42.86)	10.4	0.319
8-12	4 (14.81)	14 (51.85)	4 (14.81)	5 (18.52)		
12-24	6 (25.00)	8 (33.33)	1 (4.17)	9 (37.50)		
24-48	6 (42.86)	3 (21.43)	1 (7.14)	4 (28.57)		
Hematemesis	2 (25.0)	0 (0)	4 (50.0)	2 (25.0)	18.3	<0.001
AKI	10 (21.7)	16 (34.8)	5 (10.9)	15 (32.6)	1.29	0.731
ALI	2 (11.1)	6 (33.3)	5 (27.8)	5 (27.8)	0.966	0.019

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