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Clinical Profile And Endoscopic Role In Assessing The Severity Of Corrosive Injury Of Gastrointestinal Tract- Experience From A Tertiary Care Centre In South India

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Abstract
Background*: Corrosive substances both acid and alkali are common household substances that can be ingested either accidentally or intentionally and can cause devastating injury of gastrointestinal tract causing significant morbidity and mortality when injury is severe. The present study was conducted to analyse the clinical profile and endoscopic severity of 150 cases of corrosive injury of GI tract admitted in a tertiary care referral centre in South India.*
Materials and Methods: *The Cross-sectional study was conducted with 150 cases admitted at the toxicology and Medical Gastroenterology ward in Stanley Medical College, Chennai over a period of six months. Patients with history of corrosive ingestion presenting and undergoing upper GI endoscopy within 24 - 48 hours of ingestion were included and the details were recorded in a prestructured proforma. Patients with respiratory distress or hemodynamic instability, suspected perforation either radiologically or clinically were excluded.*
Results: *Corrosive ingestion was more common in the age group 20-29 years and more common in females. Acid ingestion was more common as compared to alkali ingestion. Suicidal ingestion was the most common circumstance of consumption. The exact amount of corrosive ingestion was difficult to ascertain.Sialorrhoea was the most common symptoms. The spectrum of injury to the GI tract revealed esophageal and gastric injury of grade II b to be the most common finding with sparing of duodenum in most cases.*
Conclusion: *Corrosive acid ingestion causes burns of both the oesophagus and stomach in high percentage of patients with grade II b injury observed in the majority. Upper gastrointestinal endoscopy was found to be a safe reliable and predictable tool to identify the extent and severity of injury.*
Keywords: *Corrosive ingestion, Upper GI endoscopy, Upper gastrointestinal tract, esophageal injury,gastric injury*

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I. Introduction

Corrosive injuries of the gastrointestinal tract (GIT), are devastating and, in severe cases, are associated with significant morbidity and mortality. The nature and incidence of ingestion is different for developed and developing countries, with the incidence being higher in the lower socio-economic group. Accidental ingestion is more common in younger age group whereas in adults, suicidal intent is frequently encountered.*Ref Kishore k. et. al. Volume - 13 | Issue - 07 | July - 2023 | . PRINT ISSN No 2249 - 555X | DOI : 10.36106/ijar*

Properties of corrosive agents and pathophysiology

A corrosive agent causes destruction of the tissue with which it comes in contact and is classified typically into two types based on their pH and proton donating/accepting nature. Acids are substances which act as proton donator and alkalis are substances which act as proton acceptors. Release of thermal energy for neutralizing the corrosive on contact with tissues is responsible for the damage to the tissues. There are several factors which influence the extent of injury caused by the corrosive to the GI tract like the volume of the substance consumed, pH of the corrosive, concentration at which it was consumed, ability of the substance to penetrate tissues. Alkali ingestion is common in the Western countries whereas acid ingestion is more common in developing countries, with sulphuric acid being the most common agent (seen in 68.75% cases).The commonest sources of acids which Indian patients have easy access to are toilet cleaners(hydrochloric acid), car batteries(sulphuric acid), jewellery cleaners (hydrochloric and nitric acid in a 3:1 proportion), and certain metal cleaners (phosphoric acid). *Ref Ayushi Agarwal et. al. Br J Radiol. 2020 Jul 24;93(1114):20200528. doi: 10.1259/bjr.20200528*

The primary pathology that occurs in the tissues following alkali exposure is liquefactive necrosis. The basic mechanism is the formation of hydroxide ions from the alkali once they come in contact with the tissues. The entire process includes protein dissolution, collagen destruction, fat saponification, cell membrane emulsification, transmural thrombosis, and cell death.Vascular thrombosis occurs following the necrosis. In case of alkali ingestion, the site most commonly affected is the esophagus. The stomach is relatively spared of the damage of neutralization by endogenous HCL; with few patients having damage in the small intestine as well. Even an accidental ingestion of a small amount of concentrated alkali can result in significant injury as there is little or no immediate pain to deter an accidental ingestion.Intentional ingestions generally involve larger volumes and can cause burns distally into the duodenum. The common alkalis available are drain cleaners (30% liquid sodium hydroxide) and household cleaners (70% sodium hypochlorite).

Acids induce tissue injury by means of tissue protein desiccation to produce coagulation necrosis by a process in which the dissociated protons (H+) from the ingested acid, after hydration with H2O obtained from the cells form hydronium ions (H3O+), results in cellular protein desiccation, denaturation, and precipitation results in eschar formation and is usually limited to the more superficial layers of mucosal tissue as penetration into the deeper layers is impeded by the presence of the eschar.

Caustic-induced injury to the tissues can be generally characterized by three phases. *Ref. Mansoor Ali et.al. Int J Acad Med Pharm 2023; 5 (3); 1647-1651*

Phase I (within 24 -48 hours): Initially, there are mucosal erosions and ulcerations followed by small vessel thrombosis, haemorrhage and inflammation. With increasing severity, there is extensive thrombosis of the submucosal vessels that leads to necrosis of the mucosa and then transmural necrosis, which may result in perforation.

Phase II (1–2 weeks): In the first week following injury, granulation tissue begins to replace the mucosal slough. Fibroblast infiltration starts around the second week and this marks the beginning of tissue repair.

Phase III (third week to months): In this phase, there is increased fibroblastic activity and scarring which results in the formation of a stricture in due course of time. There is completion of re-epithelisation by the sixth week.

Clinical presentation in a patient who has consumed corrosive can be from being occasional asymptomatic to being extremely moribund . Pain which can be at multiple sites such as oropharyngeal pain, chest pain, epigastric or abdominal pain, burns in the oral cavity and oropharynx, Nausea, vomiting, dysphagia, refusal to swallow and drooling of secretions. The suspicion of complications includes hematemesis or melena indicates upper gastrointestinal bleeding, respiratory distress if present may be due to aspiration of contents, esophageal perforation, vocal cord injury and systemic acidemia. Rarely in patients who present late may show signs of end stage complications like shock, metabolic acidosis, DIC, and vital organ hypoperfusion. Those patients surviving a few weeks after a grade II or III injury may subsequently present with dysphagia, vomiting from stricture formation, motility abnormalities of the pharynx and esophagus, formation of aorta- and tracheoesophageal fistulas and pulmonary thrombosis. Another dreaded long-term complication is the association of malignant potential in patients with strictures following alkali ingestion. *Ref. Velayudham R et al. Int J Res Med Sci. 2018 Jan;6(1):154-159*

Upper endoscopy examination should be performed in the first 24 to 48 hours in all patients with history of corrosive ingestion permitting more precise therapeutic regimens and also for early discharge of patients with normal findings or minimal evidence of GI tract injury. The ideal time for performing an endoscopy in a patient who has consumed corrosive would be in the 1st 24 hours following ingestion. It may be done up to 48- 72hours following ingestion but should not be done between 5 days and 2 weeks post-ingestion as it is at this time the risk of perforation is greatest. A delay of 4 to 6 hours before initial endoscopy is recommended to avoid underestimating the severity of injury. *ref. Velayudham R et al. Int J Res Med Sci. 2018 Jan;6(1):154-159*

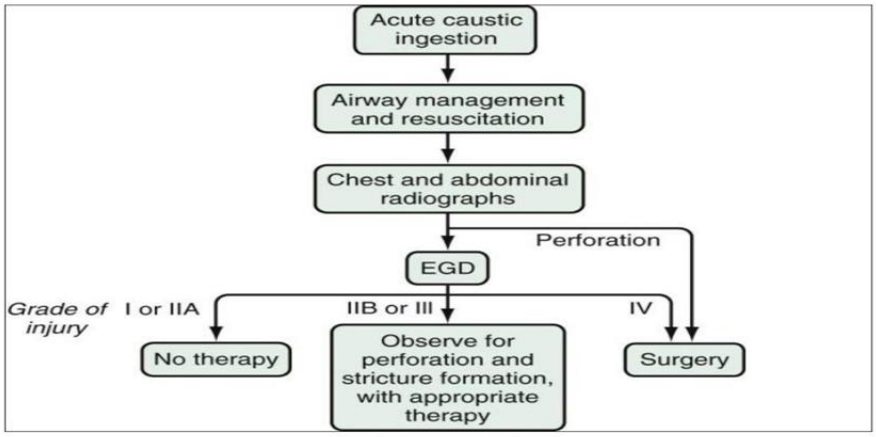


Figure 1: Step wise approach to corrosive ingestion

II. Materials And Methods

The Cross-sectional study was conducted on 150 cases admitted at the toxicology and medical gastroenterology ward in Stanley Medical College, Chennai over a period of six months. Selection criteria for cases in detail

Inclusion criteria

Patients age >15yrs, Patients with history of corrosive ingestion presenting and undergoing upper GI endoscopy within 24 - 48 hours of ingestion.

Exclusion criteria

- Patients with age less than 15 yrs of age
- Patients with respiratory distress or hemodynamic instability
- Patients with suspected perforation either radiologically or clinically
- Patients who do not give informed consent.

All patients who were admitted with history of corrosive ingestion underwent thorough history taking and detailed clinical examination after initial stabilization of airway, breathing and circulation. The parameters taken into consideration were history regarding amount consumed, type of corrosive, duration since consumption, symptomatology, physical signs, upper GI endoscopy findings and they were correlated with outcome. Laboratory investigations including complete blood counts, renal and liver function tests were done in all patients. Chest and abdomen x-rays were taken to rule out perforation. Within next 24 - 48 hours under local xylocaine anaesthesia esophagogastroduodenoscopy using a flexible Olympus GIF TYPEQ150 video endoscope was done in all patients to assess the location, extent and severity of injury to upper GI tract. The injury was graded according to classification by Zargar and collegues into 0,I,IIA,IIB,IIIA,IIIB,IV.

All patients at admission were kept nil per oral,iv fluids and iv pantoprazole. After initial endoscopy, patients with grade I and IIA injury were allowed oral fluids and eventually discharged. Patients with grade IIB injury and above were kept nil per oral for initial 2 weeks and subsequently allowed oral liquids if tolerated.

Table 1: Zargar classification and its corresponding endoscopic description:

Grade	Description
0	Normal mucosa
I	Edema and erythema of mucosa
IIA	Haemorrhage, erosions, blisters, superficial ulcers
IIB	Circumferential lesions
IIIA	Focal deep gray or brownish black ulcers
IIIB	Extensive deep gray or brownish black ulcers
IV	Perforation

Statistical analysis

Mean age, standard deviation is calculated and pearson chi-square test used to analyse the variable and comparision and predicting the outcome. Data`s were analysed using Statistical package-SPSS software version 22. The significance of difference in mean between the groups was calculated by Fisher exact test. Variables were considered to be significant if $P<0.05$.

III. Results

One hundred and fifty patients with a definite history of corrosive acid ingestion were included in the present study. There were 82(54.67%) women and 68(45.33%) men. Their ages ranged from 16 years to 57 years with mean age of 36.50 ± 20.5years.

Table 2: Age and sex distribution:

Age(years)	Men	Women	Total	Percentage
10-19	8	12	20	13.33
20-29	40	55	95	63.33
30-39	13	8	21	14.00
40-49	6	5	11	7.33
50-59	1	2	3	2.00
Total	68	82	150	100

Table 3: Cause of ingestion:

Circumstances	Male	Female	Total	Percentage
Suicidal	45	60	105	70
Accidental	23	22	45	30
Total	68	82	150	100

In our study it was observed that most common circumstances of ingestion was suicidal (105/150) i.e 70% as compared to accidental ingestion (45/150) i.e 30%. Majority of them were females.

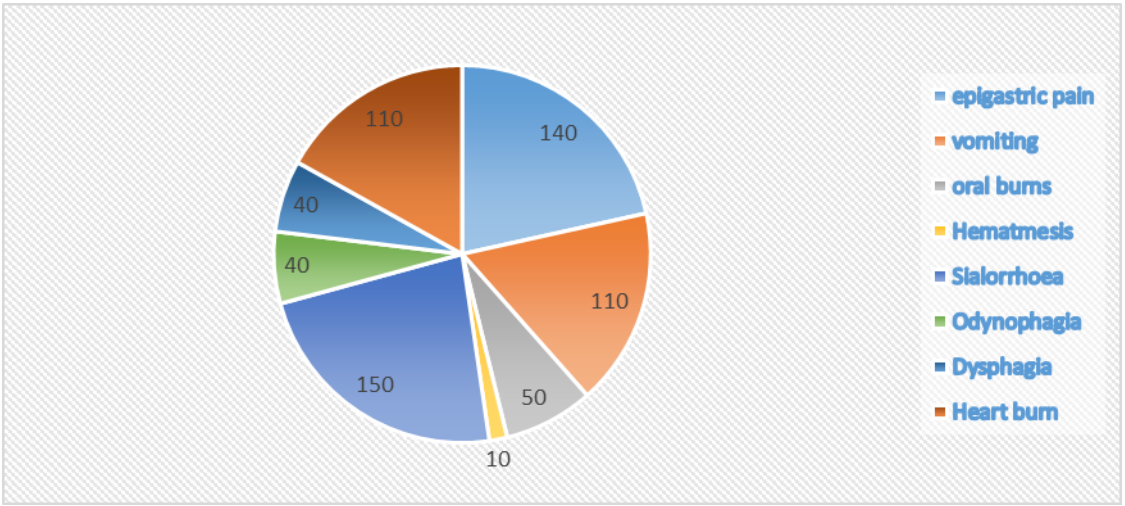
Nature and amount ingested:

Majority of patients had consumed acid(n=90) in liquid form as compared to alkali(n=60). However, the nature of the acid consumed differed. The commonest corrosive acid ingested was Hydrochloric acid(n=48) as opposed by findings by Lakshmi et.al.[6] where sulphuric acid was the most common acid consumed,followed by phenyl (n=30) , sulphuric acid (n=12). Commonest alkali was Sodium hypochlorite (n=35) followed by sodium hydroxide(n=15), ammonia (n=10).

The exact volume ingested was difficult to ascertain in each case but ranged apparently from 15ml to 100ml. The concentration of the corrosive ingested by the patients was unknown.

Table 4: Symptoms at admission:

Symptoms	Number	Percentage
Epigastric pain	140	93.33
Vomiting	110	73.33
Oral burns	50	33.33
Haematemesis	10	6.67
Sialorrhoea	150	100
Odynophagia	40	26.67
Dysphagia	40	26.67
Heart burn	110	73.33



Interval before Admission to the Hospital

All patients presented between 6 hours to 20 hours after ingestion with a mean interval of 13 ±7 hours and underwent endoscopy within 4 hours to 8 hours (mean 6 ± 2 hours) since admission.Sialorrhoea, epigastric pain and vomiting occurred in 150(100%) , 140(93.33%) and 110 (73.33%) of patients respectively. Burning sensation in the oral cavity was seen in 50 (33.33%) and heamatemesis in 10(6.67%) patients. The other symptoms noted were odynophagia 40(26.67%), dysphagia 40(26.67%) and heart burn 110(73.33%). All the symptoms were maximal in Grade IIB & beyond.

Oropharyngeal Burns:

Burns of one or more of the following – lips, tongue, buccal mucosa, soft palate, hard palate or pharynx were seen in 50(33.33%) patients.

Abdominal Signs

Epigastric tenderness was seen in 140(93.33%) patients, while 6 had diffuse abdominal tenderness and guarding and 4 showed normal abdomen. All patients were admitted to the hospital and mean duration of stay in hospital was 6±3 days.

Endoscopy Findings:

Upper gastrointestinal endoscopy, to assess the extent and severity of burns was possible in all 150 patients.

Table 5: Esophageal injury:

Grade	Number	Percentage
0	22	14.67
I	30	20
IIA	18	12
IIB	63	42
IIIA	10	6.67
IIIB	7	4.67

Severe esophageal burns (IIB or more) were found in 80 patients (53.33%), while 48 patients (32%) had mild to moderate burns (grade I and IIA) and 22 patients (14.67%) had normal mucosa.

Table 6: Gastric injury:

Grade	Number	Percentage
0	9	6
I	45	30
IIA	10	6.67
IIB	67	44.67
IIIA	11	7.33
IIIB	8	5.33

Severe gastric burns (IIB or more) were found in 86 patients (57.33%), while 55 patients (36.67%) had mild to moderate burns (grade I and IIA) and 9 patients (6%) had normal mucosa (grade 0).

Duodenal injury: The endoscope could not be passed into the duodenum due to pylorospasm in 28 patients. 84 patients had a normal (grade 0) duodenal mucosa whereas 38 patients had mild to moderate burn (grade I and IIA).

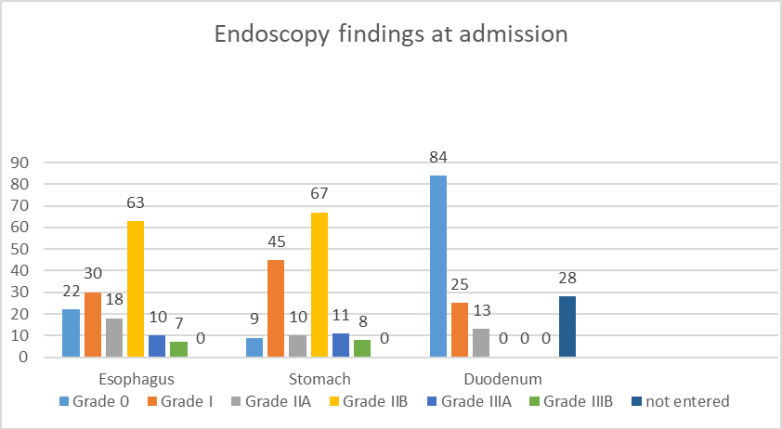


Table 7: Simultaneous comparison of esophageal and gastric injury:

Esophageal injury		Gastric injury				
	0	I	IIA	IIB	IIIA	IIIB
0	-	-	-	3	3	3
I	-	3	-	-	-	-
IIA	9	3	3	-	3	-
IIB	24	3	-	51	12	-
IIIA	-	-	-	3	18	-
IIIB	-	-	-	-	-	12

Isolated involvement of the esophagus was seen in 60(40%) patients while 9(6%) patients had isolated involvement of the stomach.

114Patients (76%) had burns of both esophagus and stomach simultaneously.

63 patients (42%) had burns of a similar degree in both esophagus and stomach, while the others had difference of one or two grade.

No mortality was noted. 18 patients (12%) were subjected to either feeding gastrostomy or feeding jejunostomy due to severe burn. No endoscopy related complications were encountered in any patients. No emergency surgery was undertaken.

IV. Discussion

Corrosive burns of the upper gastrointestinal tract due to acid ingestion are common in India.Our study included 150 patients with history of corrosive ingestion and with endoscopic evidence of corrosive injury. They underwent detailed history elicitation and thorough physical examination and were subjected to Upper GI endoscopy within 24 to 48 hours of consumption. A study by Chuan-Mei Chen et., al showed that females are more likely to consume caustics.In our study corrosive ingestion was more common in age group of 20- 29 years and more common in females. Chaitanya Mittal et.al. study had similar results that young & middle-aged females are vulnerable to the consumption of corrosive poison.But Immaneni et.al. showed a high incidence of male corrosive injury around 67%. In the present study involving a total of 80 patients all had easy access to acids, as they are commonly used as toilet bowel cleansers in Indian households and are available freely and cheaply in the market.

Hashmi et.al. observed that acid poisonings are more common than alkali & majority of patients consume bathroom cleaner as a caustic agent.In the present study, hydrochloric acid was the commonest agent ingested (n=48). This is a parameter where Indian data differs strikingly from western data where alkali consumption is more common than acid ingestion. The reason is attributed to the easy availability of acids when compared to alkalis. The mean duration since consumption when the patient was subjected to upper GI endoscopy was 6 ± 2 hours since admission.

Uday Shankar Baluni et., al in their study had suicidal intent as the cause for corrosive consumption. But one study conducted by Thomas et al. showed varying results such as that caustic ingestion was accidental in 62 of 78 patients In the present study suicidal intent was the commonest cause for ingestion i.e. 105 out of 150 patients (70%).

The degree of mucosal damage depends on the nature of the agent, its amount and concentration, as well as the amount of food in the stomach during ingestion. The clinical presentation of corrosive ingestion varies greatly; the initial presentation usually does not give adequate information about the severity of the damage. The oral lesion is seen in 11 out of 58 cases in a prospective study conducted in a tertiary care center, but more than 50% of patients had a severe injury. Another study by Uday Shankar Baluni et al. showed that 80.5% (n=33) of cases had oral lesions.In our study the degree of injury could not be correlated with the type of acid as other factors like exact amount ingested and concentration were difficult to ascertain.

Contrary to the general belief in the western literature that the esophagus is spared in acid ingestion, the present study found the esophagus was involved in 128 patients (85.33%). Zargar et al. reported esophageal burns in 87.7% of their patients, while Dilawari et al reported esophageal involvement in 13 out of 15 patients (87%).In our study 80 out of 150 patients (53.33%) had severe esophageal injury and esophagus was diffusely involved in 95 patients (63.33%) whereas rest had involvement of mid and distal esophagus. Our study showed acute gastric injury in 94 % of the patients (n=141) where severe gastric burns (IIB or more) in 86 patients (57.33%). The early use of endoscopy was crucial in determining the high incidence of esophageal and gastric involvement in corrosive ingestion.

In the present study, sialorrhoea (100%), epigastric pain (93.33%) were the commonest symptoms encountered. This was contary to that of Dilawari et al which showed above symptoms were present in 54% and 94% respectively. However, in the study by Zargar et al odynophagia (73%) and dysphagia (56%) were the commonest symptoms encountered. Where as in the present study, both were seen in 26.67% and 26.67%

respectively.Rakesh Kochhar et. al in their retrospective study concluded that Nasoenteral tube feeding is as effective as Jejunostomy feeding in maintaining in patients with severe corrosive injury. In our study 18 patients (12%) were subjected to either feeding gastrostomy or feeding jejunostomy for 4-6 weeks due to severe burn.

V. Conclusion

Corrosive ingestion causes burns of both the oesophagus and stomach in high percentage of patients (85.33% and 94% in present study). Symptoms and signs were unreliable in predicting the severity and extent of injury.Upper gastrointestinal endoscopy was found to be a safe reliable and predictable tool to identify the extent and severity of injury.It was useful in deciding treatment and assessing prognosis and predicting the outcome according to grade of initial injury.

Footnotes
Financial support: Nil
Conflict of interest: None
Limitation of study: Follow up of the cases not done

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