MAK HILL Publications Research Journal of Medical Sciences

Research Article
doi: 10.59218\makrjms.2023.87.92

OPEN ACCESS

Key Words
Endoscopy, GI tract, esophageal burns, safety

Corresponding Author
K. Mansoor Ali,
Department of Medical Gastroenterology, Vijayanagara Institute of Medical Sciences, Ballari, Karnataka, India

Received: 21 March 2023
Accepted: 13 April 2023
Published: 18 April 2023

Citation: K. Mansoor Ali, Halli Karibasappa and Dharma Reddy, 2023. Role of Endoscopy in Acute Corrosive Injury and to Evaluate its Safety. Res. J. Med. Sci., 17: 87-92, doi: 10.59218\makrjms.2023.87.92

Role of Endoscopy in Acute Corrosive Injury and to Evaluate its Safety

[1]K. Mansoor Ali, [2]Halli Karibasappa and [3]Dharma Reddy
[1]Department of Medical Gastroenterology, Vijayanagara Institute of Medical Sciences, Ballari, Karnataka, India
[2]Department of Medicine, Vijayanagara Institute of Medical Sciences, Ballari, Karnataka, India
[3]Department of Surgical Gastroenterology, Vijayanagara Institute of Medical Sciences, Ballari, Karnataka, India

ABSTRACT
Early endoscopy is not only safe, reliable and accurate diagnostic tool in such patients but also is of crucial importance in management and prognosis. To study the role of endoscopy in acute corrosive injury and to evaluate its safety. In this study a prospective analysis of 50 cases of corrosive acid ingestion admitted to acute medical care ward of V.I.M.S Ballari were studied. All patients were subjected to upper gastrointestinal endoscopy within 24 hrs of ingestion and severity and extent of burns determined. All patients were managed conservatively and few patients with severe degree burns in the stomach were sent for feeding jejunostomy. All surviving patients were followed up for 2 months with repeat endoscopy and barium studies in second month to look for development of strictures. 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.

INTRODUCTION

Ingestion of caustic agents can initiate a progressive and devastating injury to the esophagus and stomach. If the patient survives the acute effects of caustic ingestion, the reparative response can result in esophageal and gastric stenosis and an increased incidence of esophageal cancer. The people most susceptible to this injury are the very young, the psychotic, the suicidal and the alcoholics and hence management of these problems may be complicated.

Although, the spectrum of injury to the upper G.I. tract due to corrosive alkaline substance has been well studied, the present knowledge about the effects of acid intake is rather scanty, and owing to the fact that acid intake accounts for a mere 5% of all reported cases of corrosive ingestion in the West.

Unlike in the West, where 80% of corrosive ingestions occur as accidents in children under 5 years of age who have access to strong household cleaners, which are concentrated alkalis, in India accidental or suicidal ingestion of acids is common type of corrosive poisoning due to easy access to acids. Cheap toilet cleaners containing sulphuric or hydrochloric acid are sold across the counter and laboratory and industrial workers have easy access to acids at their places of work. Acids are also used for polishing in the gold and silver industry providing easy access to the workers there.

Contrary to the suggestion by case reports from the West, a few Indian studies on Corrosive acid ingestion have concluded that acid injury affects the esophagus and stomach equally and results in high morbidity and mortality[1,2].

The poor correlation between symptoms, physical findings and esophageal and gastric lesions, the inability to clinically predict the presence and extent of injury and the current medical and surgical management of caustic injuries make it imperative that careful diagnostic studies be performed.

A few Indian studies have evaluated the role of flexible endoscopy in the management of corrosive ingestion[1-4] and have found that early endoscopy is not only safe, reliable and accurate diagnostic tool in such patients but also is of crucial importance in management and prognosis. Since barium contrast radiography in the acute stages of the illness is not sensitive enough to delineate the severity and extent of injury the role of flexible endoscopy needs to be studied further more.

The role of corticosteroids in the acute phase of the illness has been the subject of intense debate. Some studies have recommended that early steroid therapy may be useful in preventing strictures in patients with severe injury. Kochhar *et al.*[6] and Gumaste and Dave[7] believe that steroids fail to reduce the incidence for stricture formation and that moreover they may obscure evidence of peritonitis and mediastinitis and hence should not be used. Most of these studies have been on alkaline injuries and their role in acid injuries need not be evaluated further.

Endoscopic dilation of strictures has recently emerged as a powerful tool for the Management of corrosive acid strictures and serves to reduce the morbidity and mortality associated with surgery for this condition. This needs to be assessed further, before definitive recommendations can be made[7.8].

Until more conclusive diagnostic and treatment recommendations can be made, corrosive acid injury continues to be a serious condition resulting in high morbidity and mortality.

MATERIALS AND METHODS

This study was conducted among 50 patients who were admitted to Acute Medical Care Ward of Vijayanagara Institute of Medical Sciences, Ballari with the history of corrosive acid ingestion were studied prospectively during a period of 12 months from June 2018 to June 2019.

Inclusion criteria: All patients with history of corrosive acid ingestion presenting within 24 hrs of consumption.

Exclusion criteria: Those who presented beyond 24 hrs of consumption. The initial history was directed towards ascertaining details regarding the corrosive consumed i.e., type of acid, concentration, amount and weather suicidal, accidental or otherwise.

Subsequently detailed history to evaluate the presence of oropharyngeal, esophageal and gastric symptoms. After routine serum chemistry and blood grouping each patient was subjected to plain radiographs of chest and abdomen for evidence of perforation. Within next 24 hrs under local xylocaine anesthesia esophagogastroduodenoscopy using a flexible Olympus GIF TYPEQ150 video endoscope was attempted in all patients to assess the location, extent and severity of injury to upper GI tract. The injury was graded according to modified 6 point classification by Zargar and colleagues into I, IIA, IIB, IIIA, IIIB. Endoscopy was possible in all patients

All patients at admission were kept nil by mouth, intravenous antibiotics, intravenous fluids and intravenous pantocid. 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 given nil oral for initial 2 weeks and subsequently allowed oral liquids if tolerated.

Upper gastrointestinal endoscopy was repeated at the end of 4 weeks in all patients to assess degree of healing. These patients were also subjected to barium

studies (barium swallow) during early follow up period of up to 2 months to look for development of complications.

Statistical analysis: Mean age, standard deviation is calculated and Pearson chi-square test used to analyze the variables and comparison and predicting the outcome. Data's were analyzed using Statistical package-SPSS software version 11.5. The significance of difference in mean between the groups was calculated by Fisher exact test. Variables were considered to be significant if $p<0.05$.

RESULTS

Out of fifty patients with a definite history of corrosive acid ingestion, There were 22(44%) women and 28(56%) men. Their ages ranged from 19 years to 69 years with mean age of 31.10±11.17years. In all 50 patients the injury was suicidal while there were no accidental cases of injury and suicidal attempt in all cases were first time

All patients had consumed acid in liquid form. However, the nature of the acid consumed differed. The commonest corrosive acid ingested was Hydrochloric acid (n = 48) followed by sulphuric acid (n = 1), phenyl (n = 1).

The exact volume ingested was difficult to ascertain in each case but ranged apparently from 15-100 mL. All patients presented between 12 hrs to 24 hrs after ingestion with a mean interval of 14.27 hrs.

Epigastric pain and vomiting occurred in 50 (100%) and 44(88%) of patients respectively. Burning sensation in the oral cavity was seen in 40 (80%) and hematemesis in 46 (92%) patients. The other symptoms noted were sialorrhoea 30 (60%), odynophagia 44 (88%), dysphagia 40 (80%) and heart burn 25 (50%)

All the symptoms were maximal in Grade IIB and beyond. Burns of one or more of the following-lips, tongue, buccal mucosa, soft palate, hard palate or pharynx were seen in 40 (80) patients. Epigastric tenderness was seen in 44 (88%) patients, while 2 had diffuse abdominal tenderness and guarding 4 showed normal abdomen.

All patients were admitted to the hospital and mean duration of stay in hospital was 5±3 days. Upper gastrointestinal endoscopy, to assess the extent and severity of burns was possible in 50 patients. Severe esophageal burns (IIB or more) were found in 43 patients (86%), while 7 had mild to moderate burns (grade I and IIA). Severe gastric burns (IIB or more) were seen in 34 patients (68%).3 patients(6 %) had mild burns (I) and 2 patients had moderate burns (IIA) and 11 (22%) patients had a normal study. The endoscope could not be passed into the duodenum due to pylorospasm in 34 patients. 16patients had a normal duodenal mucosa (Table 1).

Isolated involvement of the esophagus was seen in 20 (40%) patients while 3 (6%) patients had isolated involvement of the stomach. About 38 Patients (76%) had burns of both esophagus and stomach simultaneously. About 21 patients (42%) had burns of a similar degree in both esophagus and stomach, while the others had difference of one or two grade. 10 patients with no oral burns were seen to have upper G.I. burns on endoscopy as follows: Thus, all the 10 patients had upper G.I. burns, with 8 having severe injury, either in the stomach or esophagus or both. A normal oral cavity therefore does not exclude upper gastrointestinal injury.

Thus 76% of patients (n = 38) with odynophagia and 80% of patients (n = 40) having dysphagia, had severe esophageal injury (IIB or more). Epigastric pain was present in all the patients and 84% of patients with hematemesis have grade IIB and more injury. About 86% of patients with epigastric pain were having injury grade 2B or more 80% of patients with vomiting had injury of grade 2B or more (Table 2).

In patients found to have only epigastric tenderness 43 (88%) and 32 (64%) were found to have severe esophageal and gastric inury respectively (grade IIB or more). Diffuse tenderness was present in 2 patients with grade III Gastric injury. (Table 3).

There were 4 patients in this category with a maximum of up to grade IIA injury in either esophagus or stomach. These patients were kept on intravenous fluids and noting was given by mouth. Intravenous proton pump inhibitor (40 mg I.V. BD) and ampicilline

Table 1: Simultaneous comparison of esophageal and gastric burns

	Gastric burns					
Esophageal burns	0	I	IIA	IIB	IIA	IIIB
0	-	-	-	1	1	1
I	-	1	-	-	-	-
IIA	3	1	1	-	1	-
IIB	8	1	-	17	4	-
IIIA	-	-	-	1	6	-
IIIB	-	-	-	-	-	4

Table 2: Correlation of symptoms with severity of burns

Symptoms		I.	2A	2B	3A	3B
Oropharyngeal burns	P	3	2	27	4	4
	A	2	0	7	0	0
Salivation	A	2	0	11	2	1
	P	3	2	24	2	3
Odynopohagia	A	2	0	5	0	0
	P	3	2	30	4	4
Dysphagia	A	5	0	5	0	0
	P	0	2	30	4	4
Heart burn	A	1	0	3	0	0
	P	5	2	31	4	4
Epigastric pain	P	5	2	35	4	4
Vomitings	A	2	1	0	0	0
	P	4	1	32	4	4
Heamatemesis	A	3	0	1	0	0
	P	2	2	34	4	4

P: Present, A: Absent

Table 3: Correlation of abdominal signs with severity of injury

Esophageal burns (Grade)	Normal (n = 5)	Epigastric tenderness (n = 43)	Diffuse tenderness (n=2)
0	-	-	-
I	5	-	-
IIA	-	2	-
IIB	-	35	-
IIIA	-	4	-
IIIB	-	4	-
Gastric burns			
0	-	4	-
I	-	3	-
IIA	-	2	-
IIB	-	27	-
IIIA	-	5	1
IIIB	-	-	1

Table 4: Esophageal Injury

	Initial esophageal injury		
No. of patients	Grade	initial site	Barium Swallow
5	I	lower 1/3rd	Normal
2	IIA	Lower 1/3rd	Normal
35	IIB	Diffuse	Stricture at lower 2/3
4	IIIA	3 Diffuse and1 IN lower 1/3rd	Stricture at lower 2/3
4	IIIB	Diffuse	Stricture at lower 2/3

(500 mg I.V.) every 6th hrs was also given during the first 48-72 hrs. They were gradually put on oral feed, starting with liquids and discharged subsequently.

About 36 patients had grade IIB injury or more either in the esophagus or stomach or both. All the patients were given intravenous fluids, intravenous proton pump inhibitor (40 mg I.V. BD) and intravenous ampicillin (500 mg I.V.) every 6th hourly and nothing was given by mouth. Steroids were not given to any patients. Overall no mortality was noted. Among the survivors (n = 16) 32% of patients were subjected to feeding gastrostomy. No endoscopy related complications were encountered in any patients. No emergency surgery was undertaken. 43 of 50 (86)% patients came for follow up with >grade IIB esophageal injury had developed stricture while the remaining 7 (14%) had healed to one grade lower. About 33 of 50 (66%) endoscopically evaluated patients on follow up UGIE with gastric injury > grade IIBand beyond had developed strictures and 17 patients with milder grade burns had healed without sequele.

Barium study between 4-8th weeks: Out of 50 patients followed up with contrast radiography of the upper G.I. tract, to look for development of strictures. All patients with >grade IIB esophageal burns developed strictures (100%). While none of patients with IIA injury had developed stricture. All esophageal strictures occurred in the lower 2/3rd irrespective of the initial extent of injury All Patients with esophageal strictures were subjected to endoscopic dilatation. All strictures in the stomach lead to pyloric stenosis.. All patients with pyloric stenosis were referred for surgery. There was statistically significant association between initial higher grade of injury and development of strictures (Table 4).

All the 33 patients evaluated with grade IIB injury and beyond in the stomach had developed strictures , all in pyloric antrum leading to pyloric stenosis. About 30 of 50 evaluated patients seen to have developed esophageal and gastric stricture simultaneously. No Perforation or Fistula encountered in any patient. By the end of 8th weeks, 43 patients had developed Esophageal stricture and 33 patients developed Gastric strictures. Most common immediate complication was bleeding and short term complication was stricture in both esophageal and gastric burns. No perforation or fistula was noted no mortality was noted. All esophageal strictures developed in lower 2/3 irrespective to initial site of involvement (Table 5).

Table 5: Gastric injury

	Initial gastric injury		
Number	Grade	Site	Barium meal study
3	I	Lesser curvature	Normal
2	IIA	Pylorus	Normal
26	IIB	Diffuse	Pyloric stenosis
6	IIIA	Diffuse	Pyloric stenosis
1	IIIB	Diffuse	Pyloric stenosis

DISCUSSIONS

Oropharyngeal burns were seen in 40 patients (80%) in the present study. However, their absence did not rule out underlying upper G.I. injury in any of the patients.

Symptoms and physical examination were unreliable in determining the severity or extent of injury. Presence of epigastric tenderness was associated with severe burns to the esophagus in 47% and stomach in 58%. However, a normal abdomen did not rule out upper G.I. injury (10%). The availability of flexible endoscopes has made the assessment of upper G.I. burns very accurate, safe, reliable and practicable. The extent, severity and site of burns can accurately be determined and hence endoscopy is advocated as the investigation of choice for evaluating corrosive burns.

In the present day study, no complication attributable to early endoscopy were encountered. Endoscopy was also useful in deciding treatment, prognosis and in assessing healing of burns. The burns were graded according to modified classification given by Zargar *et al*.[1] All the patients with minor burns recovered without any sequelae, where as those with higher burns needed nutritional support and developed both acute and chronic complications in the form of strictures .There was no incidence of gastric perforation in the present study among grade III patients. Zargar *et al*.[1] reported 4 out of 17 patients reported gastric perforation (23.5%).

However, while Zargar *et al*.[1] encountered esophageal perforation in 14.2% of their patients (2 out of 14), No esophageal perforation was encountered in our present study. There was no mortality in this study, However Zargar *et al*.[1] quoted 12.2% mortality in their study, however 18.7% mortality rate was seen in study by Dilawari *et al*.[2].

The use of corticosteroids, antibiotics and early bouginage to prevent stricture formation in patients with severe injury is subject to debate with conflicting results. Lahoti and broor state that at present no therapy has been proven to be effective for prevention of stricture formation. Dilawari *et al*.[2] had used antibiotics and corticosteroids in 13 of 15 patients (86%) (four with grade 2 and nine grade 3) thus it was felt unlikely that steroids helped in corrosive injury and hence suggested that complications depend on degree of burns rather than use of steroids.

In present study steroids were not used in any patient though antibiotic and ppi was used in all. In the present study all 43 patients with >grade IIB and above esophageal burns available for follow-up had developed esophageal stricture by 2 months. While Zargar *et al*.[1] state that patients with IIB injury and beyond will eventually develop stricture. This was probably due to longer follow up in their study (mean 20.2 months, range 2-32 months. Compared to only 2-6 months in our study.

Significantly all esophageal strictures encountered in present study involved the lower 2/3 of esophagus irrespective initial site of burn. In the present study gastric stricture occurred in 33 of 50 (66%)of patients with grade IIB and beyond at follow up of 2 months as compared to 33% of patients in study by Dilawari *et al*.[2].

Thus if endoscopy done early in the course within 24 hrs following caustic ingestion to classify mucosal injury subsequent to caustic ingestion useful to determine the severity of the injury. And thus helpful in predicting outcomes A 6 point grading system rather than 5 point system is useful for predicting immediate and long term complications and guiding appropriate therapy.

CONCLUSION

Corrosive acid ingestion causes burns of both the Oesophagus and Stomach in high percentage of patients (100 and 78% in present study). Symptoms and signs were unreliable in predicting the severity and extent of injury. In short term follow up of 2 months all survivors with grade IIB esophageal and gastric injury developed stricture and was statistically significant.

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.

REFERENCES

1. Zargar, S.A., R. Kochhar, B. Nagi, S. Mehta and S.K. Mehta, 1989. Ingestion of corrosive acids. spectrum of injury to upper gastrointestinal tract and natural history. Gastroenterol., 97: 702-707.

2. Dilawari, J.B., S. Singh, P.N. Rao and B.S. Anand, 1984. Corrosive acid ingestion in man: A clinical and endoscopic study. Gut, 25: 183-187.
3. Lahoti, D. and S.L. Broor, 1993. Corrosive injury to the upper gastrointestinal tract. Indian J. Gastroenterol, 12: 135-141.
4. Zargar, S.A., R. Kochhar, S. Mehta and S.K. Mehta, 1991. The role of fiberoptic endoscopy in the management of corrosive ingestion and modified endoscopic classification of burns. Gastrointestinal Endoscopy, 37: 165-169.
5. Anderson, K.D., T.M. Rouse and J.G. Randolph, 1990. A controlled trial of corticosteroids in children with corrosive injury of the esophagus. New Engl. J. Med., 323: 637-640.
6. Kochhar, R., J.D. Ray, P.V.J. Sriram, S. Kumar and K. Singh, 1999. Intralesional steroids augment the effects of endoscopic dilation in corrosive esophageal strictures. Gastrointestinal Endoscopy, 49: 509-513.
7. Gumaste, V.V. and P.B. Dave, 1992. Ingestion of corrosive substances by adults. Am. J. Gastroenterol., 87: 1-5.
8. Ramasamy, K. and V.V. Gumaste, 2003. Corrosive ingestion in adults. J. Clin. Gastroenterol., 37: 119-124.