International Journal of Research in Medical Sciences
Velayudham R et al. Int J Res Med Sci. 2018 Jan;6(1):154-159
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20175712

Original Research Article

Clinical profile and the outcome of corrosive injury of GI tract

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Received: 30 November 2017
Accepted: 09 December 2017

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ABSTRACT

Background: Corrosive substances are common household substances that can be ingested either accidentally or intentionally with suicidal intent. The present study was conducted to analyse the clinical profile of 50 cases of corrosive injury of GI tract and to analyse the outcome of 50 cases of corrosive injury of GI tract.
Methods: The Cross-sectional study was conducted on 50 cases admitted in the toxicology ward in Rajiv Gandhi Government General Hospital, madras medical college, Chennai over a period of six months. Patients with history of corrosive ingestion presenting within 24 hours of ingestion subjected to Upper GI endoscopy within 24 hours of admission. Patients presenting after 24 hours, with respiratory distress, suspected perforation either radiologically clinically and normal findings in Upper GI endoscopy were excluded. The patients were serially followed and were subjected for a re-look upper GI endoscopy after 6 weeks and the findings were compared.
Results: Corrosive ingestion was more common in the age group 20-30 years and more common in males. Acid ingestion was almost twice as common as alkali ingestion. Suicidal ingestion was the most common circumstance of consumption and associated with higher grade of injury. Patients with ingestion of more than 50 ml had higher grades of injury and also were at higher risk of strictures. The spectrum of injury to the GI tract revealed esophageal injury of grade II b to be the most common finding with the duodenum being spared in majority of the cases.
Conclusions: In our study, Patients with ingestion of more than 50ml had higher grades of injury and also were at higher risk of strictures. While the lesser grade injuries (0, I, IIa) were associated with complete recovery with no sequelae, the more severe grades (IIb and IIIa) were associated with higher incidence of strictures especially the circumferential lesions. Oesophageal strictures are commonly associated with suicidal corrosive ingestion. All patients with corrosive ingestion should be subjected to early UGIE and after 6 weeks to identify stricture formation.

Keywords: Circumferential ulceration, Corrosive ingestion, Upper GI endoscopy, Upper gastrointestinal tract

INTRODUCTION

A corrosive is a substance that causes damage on contact with tissue surfaces both histologically and functionally. Corrosive substances can be 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 and a property of the corrosive known as titrable acid/alkali reserve (TAR). The circumstance of consumption of corrosive agent is usually with suicidal intent in adults. Accidental unintentional exposure can occur in children or in adults who are under the influence of alcohol or in psychiatric patients.

The primary pathology that occurs in the tissues following alkali exposure is liquefaction 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.[1] 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. The initial alkali injury hence can be transmural and if associated with perforation can lead to mediastinitis, and peritonitis.[2] Intentional ingestions generally involve larger volumes and can cause burns distally into the duodenum.[3]

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 H_2O obtained from the cells form hydronium ions (H_3O^+), 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. First is the inflammatory phase (which lasts for about 4-7days) in which there are thrombotic events in the vasculature with cell necrosis eventually leading to the destruction of the columnar epithelium of the mucosa and the submucosa. Generally, at 72-96hours after ingestion an ulcer develops after superficial mucosal necrosis and sloughing. The second phase, the high-risk time for perforation to occur, begins around 3days and lasts up to 2weeks after ingestion. Lastly, if the gastrointestinal mucosa has sustained a severe caustic-induced injury, an excessive amount of fibrous tissue may form, resulting in stricture formation 2 or more weeks after ingestion. The most common pattern being concomitant involvement of the esophagus and stomach.[4] In stomach the injuries are common in the antrum. The reason for the predilection to affect the antrum is due to the "magenstrasse" flow of liquid acids along the lesser curvature of the stomach with resultant pooling in the pylorus secondary to acid-induced pylorospasm. The relative sparing of the duodenum may be due to the pylorospasm and the alkaline pH of the duodenum, but injury does occur.[5]

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 hypo perfusion. 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.[6] Another dreaded long-term complication is the association of malignant potential in patients with strictures following alkali ingestion.[7] A step wise approach to a patient with history of corrosive ingestion.[8]

Upper endoscopy examination should be performed in the first 24 to 48hours 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.[9,10] The ideal time for performing an endoscopy in a patient who has consumed corrosive would be in the 1st 24hours following ingestion. It may be done up to 48-72hours following ingestion but should not be done between 5days and 2weeks post-ingestion as it is at this time the risk of perforation is greatest. A delay of 4 to 6hours before initial endoscopy is recommended to avoid underestimating the severity of injury.[11]

Table 1: Zargar grading system for corrosive injury to the GI tract.

Grade	Visible appearance	Clinical significance
0	History positive, no symptoms and visible damage	Able to take fluids immediately
I	Edema, hyperemia, loss of normal mucosal pattern, no trans mucosal injury	Temporary dysphagia, able to swallow liquids in 0-2days, no long-term sequelae
II a	Transmural injury, friability, blistering, exudates, hemorrhage, scattered superficial ulceration	Scarring, no stenosis, no long-term sequelae
II b	2a plus deep discrete ulceration and/or circumferential ulceration	Small risk of perforation, scarring may result in later stenosis (75%)
III a	Scattered deep ulceration with necrosis of tissue	Risk of perforation, High risk of later stenosis (70-100%)
III b	Extensive necrotic tissue	High risk of perforation and death (65%), high risk of stenosis

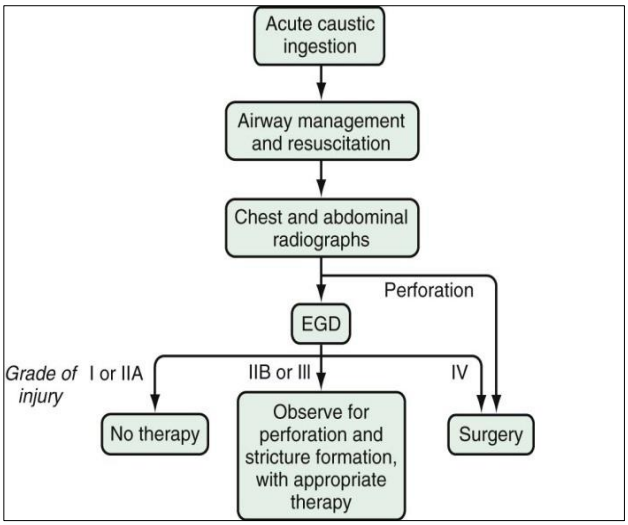

Figure 1: Step wise approach to corrosive ingestion.

METHODS

The Cross-sectional study was conducted on 50 cases admitted at the toxicology ward in Rajiv Gandhi Government General Hospital, Madras Medical College, and Chennai over a period of six months. Selection criteria for cases

Inclusion criteria

Patients age >12yrs, Patients with history of corrosive ingestion presenting within 24 hours of ingestion, Upper GI endoscopy done in patients within 24hours of admission.

Exclusion criteria

Patients presenting after 24hours of corrosive ingestion, Patients with respiratory distress, Patients with suspected perforation either radiologically clinically or endoscopically (grade III b injury), Patients with normal findings in Upper GI endoscopy (no evidence of initial injury)

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. An arterial pH below 7.22 is suggestive of DIC and metabolic acidosis indicating severe nature of corrosive injury.[12] Patients were kept Nil per oral and subjected to Upper GI endoscopy within 24hours of admission. The findings were noted, and patients were managed accordingly (oral feeds within 24hours for normal and grade I-II a injuries and NG tube placement for grade II b-III a injuries and feeding jejunostomy for duodenal injuries). The patients were serially followed and were subjected for a re-look upper GI endoscopy after 6weeks and the findings were compared, and the outcome was graded into 2 categories. Category I-normal endoscopy study. Category II-stricture esophagus or stricture antri or pylori.

Statistical analysis

Statistical analysis was carried out for 50 patients with history of corrosive ingestion after categorizing each variable-age sex, type of corrosive consumed, duration since consumption, amount consumed, circumstance of consumption, symptomatology, presence of physical signs and upper GI endoscopy findings. Data's were analysed using Statistical package-SPSS software version 11.5. The significance of difference between the proportions was indicated by the Chi square (x2) statistic. 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

50 cases with history of corrosive ingestion and with positive findings on endoscopy formed the study group. In these patients age wise distribution, sex wise distribution, circumstances of poisoning (suicidal/accidental), agent of exposure (acid/alkali), symptomatology, physical findings and endoscopy findings were analysed.

The upper GI endoscopy findings were compared with the final outcome. The agent(acid/alkali) exposed to and the circumstances of poisoning were compared with the final outcome. Other independent variables were entered into the comparison model and appropriate statistical was made.

The mean age among the patients was 32.88±12.74. The youngest age was 15years and the oldest was 67years. There was no significant difference in the outcome among different age groups (p value >0.05).

Table 2: Frequencies of the circumstances of consumption.

Circumstance	N (%)	I (%)	II (%)
Suicidal	38 (76)	21 (55.2)	17 (44.8)
Accidental	8 (16)	8 (100)	0
Accidental (alcohol influence)	4 (8)	2 (50)	2 (50)

In our study though majority of strictures occurred in the suicidal consumption group, the difference was not statistically significant (p value-0.053). Mean duration for performing the endoscopy was 14.06±3.48hours. The

least duration was 8hours and the maximum duration was 20hours.

Table 3: Amount consumed.

Amount consumed	N (%)	I (%)	II (%)
Not known	6 (12)	4 (12.9)	2 (10.5)
< 50ml	26 (5)	20 (64.5)	6 (37.6)
> 50ml	18 (36)	7 (22.6)	11 (57.9)

Among the 50 patients in our study group, the incidence of strictures was more in the group of patients who had consumed more than 50ml and the difference was statistically significant (p value-0.037).

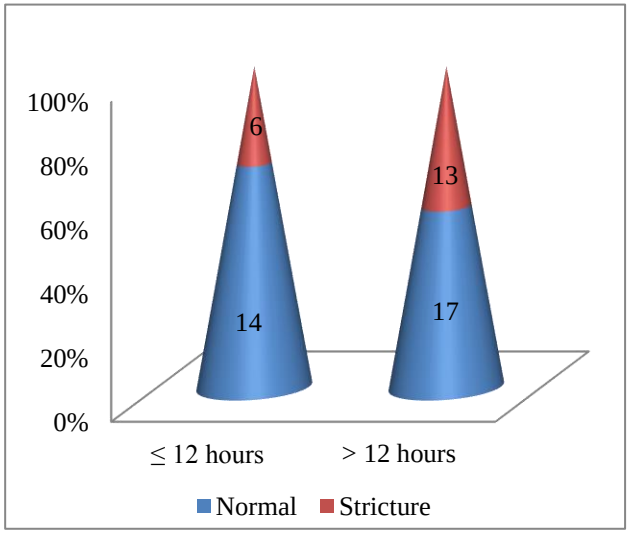

Figure 2: Duration since consumption versus outcome.

The incidence of strictures was higher in the patients with normal physical examination than those with oropharyngeal burns and the difference was statistically significant (p value-0.043). Hence oropharyngeal burns were not a reliable predictor of injury and long-term outcome.

The most common symptoms among the symptomatic group was chest pain (78%) and vomiting (12%) followed by dysphagia (8%) and hematemesis (2%).

Table 4: Analysis of physical signs versus outcome.

Physical signs	N (%)	I (%)	II (%)
Normal physical examination	45 (90)	30	15
Oropharyngeal burns	5 (10)	1	4

The incidence of strictures was higher in patients with Grade IIb and IIIa injuries especially with circumferential injuries and the difference was statistically significant (p value < 0.05). The patients with grade I and IIa injuries had no incidence of strictures.

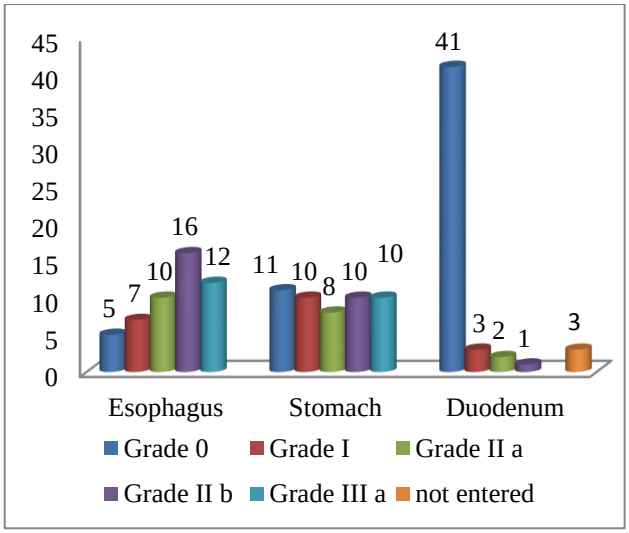

Figure 3: Endoscopy findings at admission.

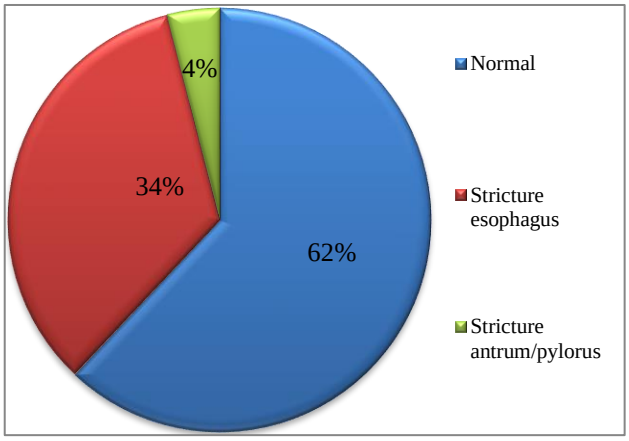

Figure 4: Follow up endoscopy findings.

Analysis of follow up endoscopy findings in stomach

The 17 patients who had stricture esophagus in the follow up endoscopy were not taken into consideration for assessing the recovery of stomach findings as the endoscope was not passed beyond the strictured part in the esophagus. The remaining patients were analysed for assessing the outcome of stomach injuries. The incidence of stricture was equal in grade IIb and IIIa injuries, but the difference was not statistically significant (p value >0.05).

Among the 50 patients in the study group those with grade 0, I and IIa were started on oral feeds and none of them had stricture in the re-look endoscopy. The patients with grade IIb and IIIa were subjected to nasogastric tube placement under fluoroscopic guidance. Two patients had to undergo feeding jejunostomy. The incidence of stricture was higher in the patients with higher grade of injuries despite the placement of Naso gastric tube and the difference was statistically significant (p value <0.05).

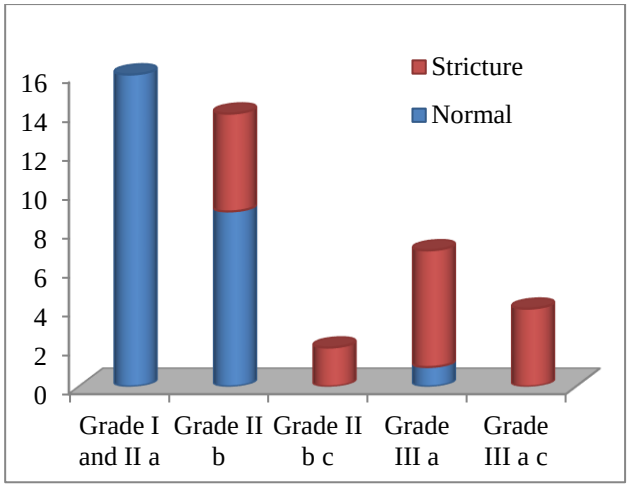

Figure 5: Analysis of treatment versus outcome.

DISCUSSION

Poisoning with corrosive substances is a very common occurrence in our country due to the readily available nature of these compounds in our household products. Ingestion of these substances causes a wide variety of damage to the GI tract both acute (perforation, hemorrhage, etc.) and delayed (stricture, carcinoma).

In our study, observations were made in patients with history of corrosive consumption with respect to the history of consumption, symptoms, signs and findings in initial upper GI endoscopy and they were compared with the final outcome. The following inferences were drawn.

Age distribution

The mean age in our study was 32.88±12.74. The youngest age was 15years and the oldest was 67years. There was no significant difference in the outcome among different age groups. The male: female ratio was approximately 2.5:1. The higher incidence of corrosive ingestion in males was slightly high but in concordance with other studies where the percentages observed were 66.6% (Gupta and Gupta) and 55.0% (Ananthakrishnan et al). The difference in outcome among males and females was not statistically significant.

Type of corrosive distribution

Ingestion of acids (64%) was more common than alkali ingestion (36%). 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.[13] The mean duration since consumption when the patient was subjected to upper GI endoscopy was 14.06±3.48hours. The incidence of strictures was higher in the patients who underwent endoscopy later than 12hours, but the difference was not statistically significant.

In our study suicidal consumption was the most common cause for corrosive ingestion accounting for 76% of cases. Accidental consumption under the influence of alcohol formed 8% of the cases. The outcome was not statistically significant different between the patients with suicidal and accidental consumption especially due to the persons with accidental consumption under the influence of alcohol. The severity of injury was also high in the patients with accidental consumption under the influence of alcohol. In other patients with accidental consumption, those who had ingested amount >50ml had higher grading of injury. Occurrence of oropharyngeal burns was higher in the patients with suicidal intention possibly due to the hesitant sipping.[14]

The mean duration since consumption when the patient was subjected to upper GI endoscopy was 14.06±3.48 hours. The incidence of strictures was higher in the patients who underwent endoscopy later than 12hours, but the difference was not statistically significant. Symptomatology at presentation: In our study the most common clinical features among the symptomatic group were chest pain and dysphagia.

Physical signs at presentation

Oropharyngeal burns were present in only 10% of the patients at the time of presentation. The incidence of significant esophageal and gastric injury as well as stricture was higher in the patients with normal physical examination and the difference was statistically significant. This correlates with studies which have the shown the poor predictive nature of oropharyngheal in predicting the occurrence of esophageal or gastric injury following corrosive ingestion.[15]

The most common pattern observed in the esophagus was IIb (32%) whereas in the stomach, the most common pattern was grade 0 injury (22%) followed by grade IIb and IIIa (20% each). The duodenum was spared in majority of the cases (82%) but could not be entered in 6% cases due to extensive gastric injury. The once observed dictum that acid spares the esophagus and affects the stomach was observed only in 5 patients with acid ingestion in our study.

Outcome of ingestion

The follow up endoscopy revealed that grade 0, I and IIa injuries healed without sequelae while strictures in 19 patients (38%) in patients with grade IIb and IIIa. 17 patients had esophageal strictures in whom further endoscopy was not done. In the remaining patients 2 patients had pyloric strictures. The incidence of esophageal strictures was higher in patients with grade IIb and IIIa injuries (65%) especially with circumferential injuries. This difference was statistically significant. The placement of NG tube was also associated with the development of strictures in majority of patients hence was not protective in preventing strictures but are

nevertheless indicated for maintaining nutrition in the acute stages. 4% patients required feeding jejunostomy for nutrition.

CONCLUSION

Our study included 50 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 24hours of consumption. Corrosive ingestion was more common in the age group 20-30years and more common in males. Acid ingestion was almost twice as common as alkali ingestion. Suicidal ingestion was the most common circumstance of consumption and associated with higher grade of injury though accidental intake under the influence of alcohol also had the risk of higher grades of injury and long-term sequelae. Patients with ingestion of more than 50ml had higher grades of injury and also were at higher risk of strictures. Chest pain and dysphagia were the most common symptoms among the symptomatic patients. Oropharyngeal burns were present only in 10% of patients. Symptoms and physical signs were not reliable in predicting the outcome of injury both acute and long term.

The spectrum of injury to the GI tract revealed esophageal injury of grade II b to be the most common finding with the duodenum being spared in majority of the cases. While the lesser grade injuries (0, I, IIa) were associated with complete recovery with no sequelae, the more severe grades (IIb and IIIa) were associated with higher incidence of strictures especially the circumferential lesions. In our study only one patient with grade IIIa and 9 patients with grade IIb injuries recovered completely. All patients with circumferential lesions went onto develop strictures. Hence the extent of initial GI tract injury at endoscopy had the most significant correlation with the development of strictures later. The placement of NG tube was not associated with decrease in the occurrence of strictures. Two patients with very severe lesions underwent feeding jejunostomy.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Velayudham R, Senthilkumar S, Karthikeyan RK. Clinical profile and the outcome of corrosive injury of GI tract. Int J Res Med Sci 2018;6:154-9.