Journal of Rawalpindi Medical College (JRMC); 2016;20(4): 254-257

Original Article

Relationship of Esophageal Strictures with Esophageal Injury after Corrosive Intake

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Abstract

Background: To study the relationship of esophageal strictures with esophageal injury after corrosive intake

Methods: In this cross-sectional study 142 patients with a history of corrosive intake and positive clinical findings were recruited. Patients underwent upper gastrointestinal endoscopy during the first 48 hours after ingestion in order to assess the extent of injury according to the grading system and after 6 weeks, endoscopy was repeated for documentation of stricture formation.

Results: Sixty two(43.7%) patients had severe esophageal injury 80 (56.3%) had mild injury. At 6 weeks, repeat endoscopy showed stricture formation among 34 (23.9% patients while 108 (76.05%) did not develop any stricture. Only 8.7% patients with mild esophageal injury developed stricture compared to 27 (43.5%) patients with severe esophageal injury (relative Risk 4.97, 95% Confidence Interval 2.32 to 10.66, p-value 0.00) and this difference was highly statistically significant.

Conclusion: Patients with high-grade esophageal injury have progressively higher frequency of stricture formation after corrosive intake.

Key Words: Corrosive poisoning, Acid,Alkali, Esophageal injury, Esophageal stricture.

Introduction

Corrosive ingestion is an important social and medical problem due to associated early and long-term complications, including bleeding, perforation, systemic complications (renal insufficiency, hepatic dysfunction, and diffuse intravascular coagulation), esophageal stricture, fistula, gastric outlet obstruction, and cancer.[1-5] Ingestion is mostly accidental in children under the age of five and intentional in adults and adolescents. [6,7,8,9]

The most common cause is ingestion of strong alkali (sodium or potassium hydroxide) contained in drain cleaners, other household cleaning products, or disc batteries. Highly concentrated acids (hydrochloric, sulfuric, and phosphoric acid) contained in toilet bowl or swimming pool cleaners, antirust compounds, or in battery fluid are less frequently ingested. Liquid household bleach (5 percent sodium hypochlorite) ingestion is frequently reported, but rarely causes severe esophageal injury.[10]

Ingestion of alkali (such as ammonia or sodium hydroxide) acutely results in a penetrating injury called liquefactive necrosis. The process of liquefactive necrosis usually lasts three to four days and is associated with vascular thrombosis and mucosal inflammation, resulting in focal or extensive sloughing and ulceration. Acid ingestion typically produces a superficial coagulation necrosis that thromboses the underlying mucosal blood vessels and consolidates the connective tissue, thereby forming a protective eschar. Because acid solutions cause pain upon contact with the oropharynx, the amount of acid ingested tends to be limited.[11]

Upper gastrointestinal endoscopy should be performed during the first 24 hours after ingestion in order to evaluate the extent of esophageal and gastric damage, establish prognosis, and guide therapy.[12] A grading system for esophageal injury to predict subsequent clinical outcome has been developed.[13] This grading may also predict prognosis.[14]

To determine the risk of stricture formation, to initiate early and effective treatment, and to prevent unnecessary malnutrition and medication use, the severity of esophageal and gastric damage should be documented.

Patients and Methods

In this cross sectional study, conducted at Gastroenterology Department, PIMS, Islamabad, from July 2013 to March 2014, 142 patients with a history of corrosive intake, were recruited.Patients with a history of corrosive intake and positive clinical findings, i.e., Hematemesis, oropharyngeal fibrous lesions, severe mucosal edema, vomiting, drooling,

oropharyngeal hyperemia and respiratory distress, and patients who underwent upper gastrointestinal endoscopy during the first 48 hours after ingestion were included. Patients with a questionable history of ingestion, if they were asymptomatic or had no oropharyngeal finding and patients in whom endoscopy was contraindicated,i.e., patients who were hemodynamically unstable, had evidence of perforation or severe respiratory distress, or exhibited severe oropharyngeal or glottis edema and necrosis, were excluded from the study. Patient were explained about the whole procedure and informed written consent was taken. Endoscopy was performed within 48 hours, in order to assess the magnitude and extent of injury according to the grading system using a modification of the method of Di Costanza e.g.,Grade-0 —Normal;Grade 1 —Mucosal edema and hyperaemia;Grade 2A —Superficial ulcers, bleeding, exudates; Grade 2B —Deep focal or circumferential ulcers;Grade 3 —Scattered small necrotic areas, and black or brown mucosa

Injection co-amoxiclav and ranitidine was routinely administered to all patients before EGD. EGD was performed in all patients under local anesthesia by a fibreoptic Pentax LH-150PC (Japan) endoscope. Intravenous antibiotics and H2 (histamin-2) receptor blocker were discontinued in patients with grade-0 and grade-1 injuries. Patients with grade-0 and grade-1 esophageal injury without severe gastric injury were fed orally and discharged after endoscopy. Gastric decompression and medical treatment which included steroid, intravenous antibiotics (co-amoxiclav and metronidazol) and H2 receptor blocker were given in patients with Grade 2 and 3 injuries or severe gastric injury. All patients who had grade-2 or 3 esophageal injuries without severe gastric injury were fed via nasogastric tube after endoscopy. Enteral nutrition was not started and total parenteral nutrition (TPN) was given in patients who had severe gastric injury which is characterized with mucosal necrosis till 2 weeks. A repeat endoscopy was performed after 6 weeks for documentation of stricture formation.

Results

The mean age of patients was 25.09± 8.11 years. The gender distribution revealed a female preponderance, with 21 (14.8%) males and 121 (85.2%) females.In 19 (13.4%) patients no esophageal injury was detected on endoscopy. In the remaining 123 patients some degree of esophageal damage was identified. Grade 1 esophageal injury (43%) was commonest (Table 1). This endoscopy was done within 48 hours of corrosive intake. Grade 2a, 2b, and 3 were defined as severe lesions. Hence 62 (43.7%) patients had severe esophageal injury (Table 2). Among 19 patients with grade 0 esophageal injury none developed a stricture. Among 61 patients with grade 1 esophageal injury 7 (11.47%) developed a stricture. Among 41 patients with grade 2a esophageal injury 12 (29.26%) developed a stricture. Among 15 patients with grade 2b esophageal injury 10 (66.67%) developed a stricture. Among 6 patients with grade 3 esophageal injury, 5 (83.3%) developed a stricture (Table 3). Hence, the greater the grade of esophageal injury the greater was the development of a stricture on follow up endoscopy. This difference was statistically significant; p= 0.00. i.e p<0.05.Among 62 patients with severe esophageal injury, 27 (43.54%) developed an esophageal stricture as opposed to 7 out of 73 (8.75%) patients with mild esophageal injury

Table 1: Grades of esophageal inury

Grade	No(%)
Grade 0	61(43)
Grade 1	41 (28.9)
Grade 2a	15 (10.6)
Grade 2 b	15(10.6)
Grade 3	6(4.2)

Table 2: Esophageal stricture among different grades of esophageal injury

		Esophageal stricture	
		Yes	No
Grade of esophageal injury	Grade 0	0	19
	Grade 1	7	54
	Grade 2a	12	29
	Grade 2b	10	5
	Grade 3	5	1
Total		34	108

Table 3: Comparison Of Stricture Formation In Mild And Severe Injury

Esophageal injury	Stricture formation		Total No(%)
	Stricture Formed No(%)	Nno stricture No(%)	
Mild injury (Grade 0 & 1)	7 (8.75%)	73 (91.25%)	80 (100%)
Severe injury (Grade 2a, 2b & 3)	27 (43.5%	35 (56.4%)	62 (100%)
Total	34(23.6%)	108(80.5%)	144(100%)

developed an esophageal stricture. This difference was statistically significant; p= 0.00. When proportion of patients with development of stricture were compared among those with mild esophageal injury(grade 0&1) with those having severe esophageal injury(grade 2a,2b and 3) a highly statistically significant difference was observed with a chi statistics of 23.32 and p-value 0.00. When risk of development of stricture formation was compared in both mild and severe groups, a relative risk of 4.97(95%) and confidence interval of 2.32-10.66 was observed which showed that patients with severe esophageal injury are almost five times more at risk of development of stricture formation as compared to those with mild esophageal injury

Discussion

In present study 62(43.7%) patients had severe esophageal injury . Patients with high grade esophageal injury had higher frequency of stricture formation , after corrosives intake.In a study by Lucky et al,[15] 30 patients who were admitted with a history of corrosive ingestion over a five year period (2005 to 2010) were included. The age range of the patients was 2-47 years old with a mean of 23.9 ± 13.4 years. Among adults there was a high incidence in the third decade while in children there was a high incidence among the under five. The most common type of corrosive ingested was caustic soda in 40% of cases. The commonest complication was esophageal stricture (40%). Fifty percent of cases were due to suicide attempt, while 9 (30%) cases were accidental ingestion of which all were children. This study revealed a male to female ratio of 4:1. The commonest clinical presentation was odynophagia (30%) while mortality accounted for 10% 0f cases. These results differ from the results of our study where acid ingestion was much more frequent. Acid injuries appear to be more common in developing countries where hydrochloric acid and sulfuric acid are easily accessible in toilet cleaners. Children under five years of age constitute the highest risk group among children in many studies but this was not observed in our study since children < 12 years of age are referred to children hospital PIMS. This study further revealed that >50% of the patients' reason for ingesting corrosives was suicidal. Besides, high family stress, marital conflicts, deaths in the family, mental illness, and physical illness have been identified as predisposing factors associated with ingestion of corrosives .These findings agreed with the reports of other researchers. Children under five years of age have well developed skills to locate and drink liquids, but are unable to discriminate edible liquids from toxic ones.

In our study esophageal stricture developed in 23.9% patients. Esophageal strictures of short segment type were evident in 40% of cases in a Nigerian study.[16]Evidence from other studies has also shown that stricture formation following the ingestion of corrosive could be as high as 63%.[17] Sometimes, the unpleasant taste of some corrosives particularly acids can cause choking, sputtering, and vomiting which may cause chemical epiglottitis and pneumonitis.Studies comparing the degree of corrosive injury with the probability of stricture formation are scarce. In the study by Zargar et al, 71.4% of patients with grade 2b injury and all survivors (100%) with grade 3 injury developed esophageal or gastric cicatrization or both. [18] The degree of injury correlates directly with stricture formation and mortality. Over 80% of patients with grade 3 burns go on to stricture formation while one-third of those with grade 2 burns may stenose. Stricture formation is extremely rare in injuries of the first degree. Mortality is also more common in grade 3 injury.

In another study by Chen et al, 32 children with esophageal injury due to ingestion of caustic material were included. [19] Sixteen had low grade injury and 16 had high grade esophageal injury. Most patients with low-grade esophageal injury healed without sequelae, except for 1 (6.25%) who developed esophageal stricture. Ten patients (62.5%) with high-grade esophageal injury developed esophageal stricture. These results were similar to our results where 43.54% patients with severe esophageal injury developed an esophageal stricture as opposed to 8.75% with mild injury.

Some authors have shown that mucosal injury to the oesophagus is more serious and grades 3 and 4 injuries are more frequently seen in patients who attempted suicide as compared with accidental ingestion.[20] Suicidal corrosive injuries are more often associated with marked oral, oropharyngeal and proximal oesophageal injuries because of hesitant sipping of the fluid whereas accidental injuries are usually associated with ingestion of larger volumes which are gulped down fast and are associated with a higher proportion of gastric injuries.

Conclusion

Patients with severe esophageal injury are almost five times more at risk of developing stricture formation as compared to those having mild esophageal injury.

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