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Corrosive Injury Causing Stricture Of Upper, Middle, Lower Esophagus And Pylorus- A Prospective Study In Tertiary Care Centre

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Abstract

Ingestion of corrosive substances induces an extensive spectrum of injuries to the gastrointestinal tract which include extensive necrosis and perforation of the esophagus and stomach. Out of all the long-term complications stricture formation is most commonly seen. Among corrosive consumption Alkali ingestions classically affect the esophagus more, whereas acid ingestion causes severe gastric injury. The esophagogastroduodenoscopy (EGD) is considered the gold standard investigation to assess the extent, depth of injury, and to plan appropriate therapeutic intervention. We studied 25 patients with corrosive injury causing stricture of upper, middle and lower esophagus and pylorus. EGD was done to all corrosive agents consumption patient within 24 hours of ingestion and also after 2 months to assess the mucosal injuries. In our study all 25 patients had esophageal stricture at presentation out of which 5 (20%) patients required repeated pneumatic dilation after 2 months. Hence, we noted that early endoscopy is essential in the management of patients with corrosive injury as it helps to directly assess mucosal healing and predicting the outcome which might require further intervention.

Keywords: NIL Introduction

Ingestion of corrosive substances induces an extensive spectrum of injuries to the gastrointestinal tract which include extensive necrosis and perforation of the esophagus and stomach. Among corrosive consumption Alkali ingestions classically affect the esophagus more, whereas acid ingestion causes severe gastric injury.

Tracheal and laryngeal injury can also be caused by acid or alkali aspiration which can occur during ingestion. In contrast with children in whom ingestion is usually accidental, corrosive ingestion in adults is usually intentional hence amount ingested will be larger leading to more severe esophageal and gastric damage.(1) complications there can be life threatening complications associated with corrosive injuries which are massive hemorrhage due to gastric erosion, tracheobronchial fistula, aortoenteric fistula or gastrocolic fistula, perforation usually involving stomach and stricture seen in around 10% of the patients.(2).

Out of all the long-term complications stricture formation is most commonly seen. Around 90% of patients with third -degree burns and 15%-30% of second-degree burns develop stricture.(3) The esophagogastroduodenoscopy (EGD) is considered the gold standard investigation to assess the extent, depth of injury, and to plan most appropriate therapeutic intervention. (1)

Aim & Objectives

To study the corrosive injury patients causing stricture of upper, middle and lower esophagus and pylorus.

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Materials & Methods

Study Design - prospective study.

Study Setting – Study is conducted in MGM Medical college, Navi Mumbai, which is a tertiary care center.

Study Period - May 2019- August 2021

Sample Size - 25. Patients who meet the inclusion criteria was selected.

Inclusion Criteria – All patients with all age groups with history of acid and alkali consumptions and who gave consent for study during study period were included

Exclusion Criteria – nil

Study Procedure- First we do Upper GI Endoscopy of all corrosive agents consumption patient within 24 hours of ingestion, then we will assess the mucosal injuries of esophagus and stomach, after that we will do needle jejunostomy for feeding and nutritional requirements.

After 1.5 to 2 months, we will reassess and do UGI Endoscopy. If stricture present then do pneumatic dilatation depending on site of Stricture.

Results

In our study out of 25 patients 15 (60%) were females, 10 (40%) were males, 80 pc were younger age group that is less than 30 years.

Out of 25 patients 50% had history of harpic poisoning, 20% with phenol poisoning, 10% sulphuric acid and 20% other poisoning.

Out of 25 patients, 23 patients had esophageal stricture, 2 had pyloric stricture and 2 had both esophageal and pyloric stricture. Further out of 25, 4 (16%) patients had stricture at cricopharynx, 8 (32%) patients had at middle esophagus, 15 (60%) patients had at lower esophagus, 4 (16%) had stricture at pylorus.

After 2 months reassessment out of 25 patients 2 patients did not follow up in our hospital and 2 patients expired.

Out of 21 patients remained,12patient's OGD endoscopy were normal, 5 Oesophageal and 4 Pyloric stricture patients required repeated pneumatic dilatation. 2 patients further required Gastrojejunostomy after 6 months due to pyloric stenosis.

All 25 patients had esophageal stricture at presentation out of which 5 (20%) patients required repeated pneumatic dilation after 2 months. Out of 4 patients who presented with pyloric stricture initially all 4 (100%) had to undergo pneumatic dilation after 2 months.

Discussion

There are wide variety of chemicals commonly available in household that can be ingested either accidentally or intentionally. Prompt recognition of the seriousness of the condition and proper initiation of therapy will substantially reduce morbidity and mortality.

As compared to common accidental ingestion in children, ingestion in adults is more often intentional, and therefore, tend to be more serious[3].The mortality rate is between 10% to 20% and rises to 78% in cases of attempted suicide(4).

The grade and extensiveness of the injury depends on many factors like quality of the agent consumed, quantity and concentration, exposure duration, food in stomach. The most commonly ingested strong alkali is (sodium or potassium hydroxide) contained in drain cleaners, household cleaning chemicals, 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 also ingested.

There is great degree of variation in oesophageal versus gastric injury in cases of acid and alkali ingestion [5].

It is noted that acid ingestion "lick the oesophagus and bite the pyloric antrum", and alkali tends to produce a uniform severe oesophageal mucosal injury [3,6].

Usually, acid injury is limited to the stomach but around 6%-20% of patients have associated oesophageal and small intestinal injuries[7]

Acid will cause "coagulation necrosis" of the tissue in contact; hence the coagulum formed will hinder any further tissue penetration [3,8]. Alkali will cause "liquefaction necrosis", which leads to the saponification of fats, dissolution of protein and collagen, dehydration of tissues and thrombosis of blood vessels, ultimately causing deeper penetrating tissue injury[5].

In a study done by Zargar et al it was observed that 85.4% of the patients who ingested acid had acute gastric injury, mainly affecting the distal part of the stomach out of which 44.4% had late complications such as pyloric stenosis[9]. In acid ingestion the resultant pyloric spasm along with alkaline pH of duodenum will have relative duodenum sparing which characteristic.[9]

The most appropriate and definitive intervention is early endoscopy which helps in clinical decision and further plan of management[10,11,12] Early endoscopy is essential in the management of patients with corrosive injury as it helps to directly verify mucosal healing and predicting the outcome requiring further intervention[13,14]

Study done by Hawkins et al showed that within 36hr of corrosive ingestion diagnostic endoscopy to be done which has better clinical outcomes. However, endoscopy is deferred up to 1 week in patients with extensive severe oropharyngeal injury, in order to allow the oedema to subside, which in turn reduces airway complication [11)

In the absence of perforation endoscopy is considered as the gold standard diagnostic procedure of choice. Patients diagnosed with perforation require immediate surgery[3].

Gastric acid suppression with PPIs and H2antagonists are used as oesophagitis and gastritis are common and patients have been kept fasting. (15)

Stricture formation, is seen most commonly hence it is the main long-term complication. Around 90% of patients with third-degree burns and 15%-30% of second-degree burns develop stricture (3). Patients with esophageal strictures are managed with repeated dilatation.

Hawkins et al had high success rate with esophageal dilatation.[11] Dilatation is done either anterograde, retrograde or a combination of both. Repeat dilation sessions are done in most cases with a target of attaining a luminal diameter of 12 mm or more in order to relieve solid dysphagia[17] It is noted that

combination of intense PPI therapy and repeated dilatation will reduce the number of impassable strictures which otherwise will require oesophageal resection and reconstructive surgery. (18)

As for acid burns of the gastrointestinal tract which commonly leads to gastric outlet obstruction, it usually responds to balloon dilatation if not surgical bypass might be required.

Bilroth I reconstruction with hemigastrectomy and resection of the first part of the duodenum is an alternative surgical reconstruction procedure. To plan management of patients presenting with corrosive injury the pathophysiology of mucosal injury is very important as improper management can lead to significant patient morbidity. Stricture and obstructive signs begin to appear by 14 days to 6 months as the retraction of the scar begins to occur during this period full blown fibrosis is seen in next 6 to 12 months' time. (19)

Conclusion

In Planning the management of patients presenting with corrosive injury, understanding the pathophysiology of mucosal injury plays a very important role as improper management can lead to significant patient morbidity.

UGI Endoscopy plays the major role in diagnosis and treating patients with patients of corrosive injuries of both oesophagus and pylorus.

The most common sequele of corrosive ingestion is formation of strictures which should me managed well to improve the mortality and morbidity of patient.

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POISONING	% OF PATIENTS
Harpic	50%
Phenol	20%
Sulphuric acid	10%
Other	20%

Table 1: % of patients with type of poisoning

 Table 2 : Endoscopic Zargar's classification

ENDOSCOPIC ZARGER' S CLASSIFICATION

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GRADE	FEATURES
0	Normal
1	Superficial mucosal edema and erythema
2	Mucosal and submucosal ulceration
2A	Superficial ulceration, erosion, exudate
2B	Deep discrete or circumferential ulcer
3	Transmural ulceration with necrosis
3A	Focal necrosis
3B	Extensive necrosis
4	Perforation





 $\frac{1}{2}$



Fig 2 : EGD scopy findings after 2 months





