Original Research Paper

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General Surgery

A CASE SERIES OF CORROSIVE INJURIES OF GASTROINTESTINAL TRACT

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ABSTRACT **Background:** Corrosive ingestion is responsible for spectrum of upper gastrointestinal tract injury from self limited to perforation. Endoscopy determines severity of corrosive ingestion which is of great importance in choosing treatment. This article reviews all aspects of corrosive injury of oesophagus, stomach and duodenum focusing on endoscopy role. **Aim:** This study aimed at evaluating the endoscopy outcomes of corrosive injuries upper of gastrointestinal tract **Material And Methods:** Our 2 years study between may 2022 - may 2024 include 25 patients with clinical evidence of corrosive ingestion, admitted in our institution (Jaipur National Institute of Medical Sciences and Research center). Patients evaluated for injury to oesophagus, stomach and duodenum by endoscopy and findings have shown maximum injury in oesophagus. **Results:** 25 pateints with age between 16-50 years were evaluated. In 15 patients, the injury in oesophagus which includes upper, middle and lower oesophagus i.e 3 (upper), 10 (middle) and 2 (lower) oesophagus. Injury to oesophagus and stomach were present in 6 patients, injury to only stomach present in 2 patients, injury to oesophagus, stomach, duodenum, pylorus present in 2 patients. Accidental ingestion of corrosive agent was present in 18 patients, suicidal attempt with corrosive agent were present in 7 patients. corrosive agent which was ingested were HARPIC(hydrochloric acid), PHENYL which contains phenolic acid, carbolic acid, ALKALI which contains sodium hydrochloride and some other unknown corrosive agents. The main symptom of corrosive ingestion were dysphagia, burning sensation and chest tightness . endoscopy done within 48 hours of ingestion of corrosive agent, after that not gone for endoscopy because of chances of perforation and mortalilty.

KEYWORDS : Corrosive Injury, Stricture, Acids And Alkali.

INTRODUCTION

Corrosive injury of the upper gastrointestinal tract is a worldwide clinical problem. Majority of the corrosive injuries occur in children that is mainly accidental ingestion whereas Adults usually ingest corrosive agent with a suicidal intent and associated with morbidity and mortality.

Corrosive ingestion mainly affects from oral cavity to stomach. In acute stage injury varies from mild inflammation to lethal form and in chronic stage stricture formation occurs leading to dysphagia and malnutrition.

A corrosive agent causes destruction of the tissue with which it comes in contact. They are broadly classified into acid and alkali. Acids have a pungent odour and noxious taste, less viscous and cause coagulative necrosis with formation of an eschar.[1] This acts as a barrier for further penetration of the acid, limiting the depth of injury. Secondly acid causes pyloro-spasm, resulting in stasis and increased contact time of the ingested agent in the pre-pyloric region and thus, development of an antropyloric stricture. The common acids which are commercially available are in the form of toilet cleaners (hydrochloric acid), storage battery acids (sulphuric acid), jewellery cleaners (hydrochloric and nitric acid in a 3:1 proportion).

Alkalis are tasteless and odourless, which leads to longer contact time with the tissue and cause liquefactive necrosis, thus resulting in deeper penetration and increased risk of adjacent organ injury[1]. Alkalis have neutralising action on the acid in the stomach and avoid pyloric spasm, making the stomach less prone to injury.

The common alkalis available are drain cleaners (30% liquid sodium hydroxide) and household cleaner. In the presence of relevant history, the diagnosis is obvious. Initially patients present with chest pain, vomiting, excessive salivation, Haematemesis, Upper airway involvement leads to respiratory distress, stridor and hoarseness. Severe chest pain radiating to the back, with episodes of fever and cough may suggest oesophageal perforation.

In the chronic phase, scarring and fibrosis lead to oesophageal stricture which present as dysphagia, regurgitation. Gastric strictures present with vomiting, early satiety and weight loss.

AIM:

This study aimed at evaluating the endoscopy outcomes of corrosive injuries of gastrointestinal tract

MATERIALS AND METHODS :

The study of cases with corrosive injury of gastrointestinal tract was done at department of general surgery at JNUIMSRC, JAIPUR from a period of 2 years from May 2022 to May 2024 with follow up of 3 months.

Patient Preparation:

Pateints were admitted to hospital before endoscopy. Proper history and clinical examination was done and full routine investigations , viral markers were carried out. Informed consent was taken before endoscopy.

Investigations

Routine blood investigation- showing Leukocytosis and raised C-reactive protein indicates a severe acute inflammatory response to corrosive injury.

Arterial blood gas analysis alteration occurs with airway involvement. Severe electrolyte imbalance seen in some cases is due to large amount of fluid loss in the third space. Coagulation profile is important in bleeding patients. Altered renal and liver functions occur in response to hypotension and systemic infection.

Imaging Modalities

Chest and abdominal radiographs are usually the initial investigations carried out in the emergency setting. The findings on the chest radiograph may include pleural effusion, pneumomediastinum, pneumothorax as well as nodules and consolidation secondary to aspiration pneumonitis .

Endoscopy

Endoscopy plays a crucial role in the diagnosis and management of patients with corrosive injury. Endoscopy is usually performed within 24–48H after ingestion, and initial endoscopy after 96H[3] of corrosive

ingestion is not advised because the injured oesophagus is in the phase of ulceration and granulation tissue formation, when it is fragile and easily perforated.[2]

The Zargar classification is used for grading the early endoscopic findings and treatment varies based on the severity of grading.

Table No.1 – Zargar Classification

Zargar classification	Description
Grade 0	Normal mucosa
Grade I	Edema and erythema of the mucosa
Grade II A	Hemorrhage, erosions, blisters, superficial ulcers
Grade II B	Circumferential lesions
Grade III A	Focal deep gray or brownish-black ulcers
Grade III B	Extensive deep gray or brownish-black ulcers
Grade IV	Perforation

Patients with no evidence of mucosal injury may be discharged timely leading to reduced cost of hospital stay[4].

RESULTS

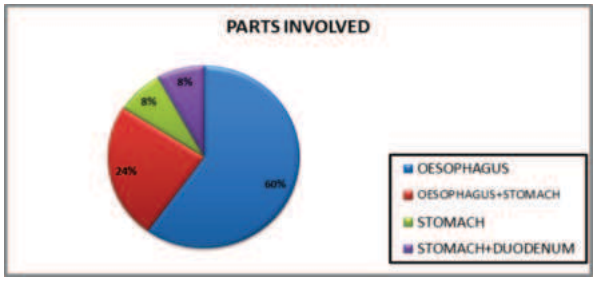


Chart 1 - Upper Git Parts Involvged

The procedure which we have done are :
1. Diagnostic : Upper Gi Endoscopy
2. Therapeutic Are :
A. Baloon Dilatation
B. Bougie Dilatation

3 . Supportive Treatment:
After 2 or 3 days of establishment of corrosive injury , which shows that in future there may be stricture formation from cricopharynx to duodenum , we went for feeding jejunostomy (FJ) in 16 patients out of 25 patients and after 3-4 days , we gave proper feeding from feeding jejunostomy and prepared the patient for further surgical or endoscopy treatment.

We evaluated patient endoscopy after 2 months of ingestion of corrosive agent and then went for the dilatation or surgical treatment. We did balloon dilatation of oesophagus in 8 patients out of 15 patients, bougie dilatation in 6 out of 15 patients and in 1 patient a strong stricture was there and we referred the patient for colonic transposition to the gastroenterologist .

Out of 6 patients which are having oesophagus and gastric injury, oesophageal dilatation done in 4 patients and they recovered , and in 2 patients we did the oesophageal dilatation with (GJ) gastrojejunostomy. 2 patients in which stomach is only involved causing gastric outlet obstruction were treated by gastrojejunostomy.

In pyloric injury, patient got pyloric stenosis , so in 1 patient we dilated pyloric stenosis by balloon dilatation and 1 patient developed gastric otlet obstruction in which we did gastrojejunostomy.

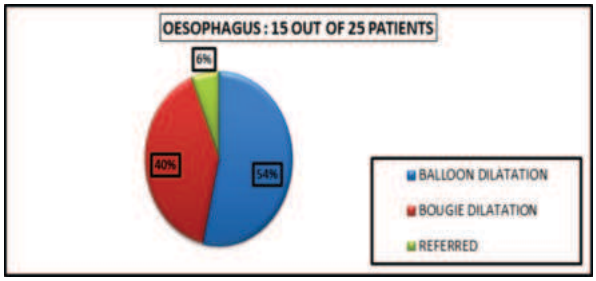
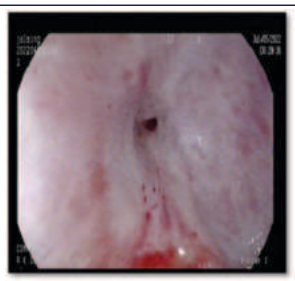
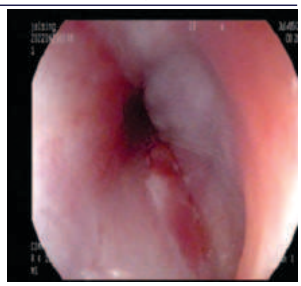


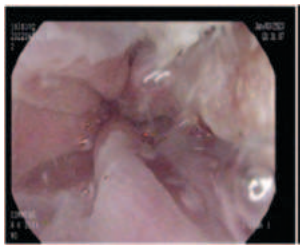
Chart No.2 – Oesophagus Involved Patients



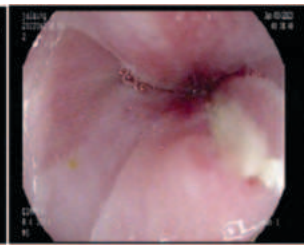
Pre-balloon Dilatation Of Upper Oesophageal Stricture - Fig No.1



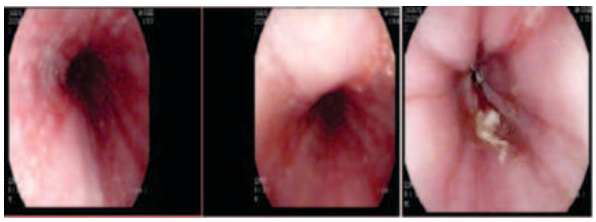
Post Balloon Dilatation Of Upper Oesophageal Stricture Fig No.2



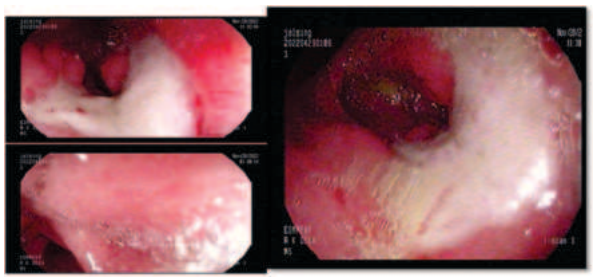
Within 48 Hours Of Upper Oesophageal Injury- Fig No.3



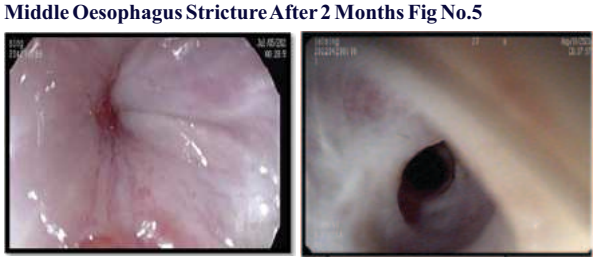
Lower Oesophageal Stricture After 2 Months



Upper Oesophageal Stricture After 2 Months Fig No.4

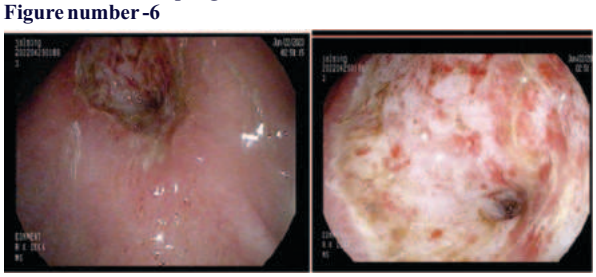


Middle Oesophagus Stricture After 2 Months Fig No.5

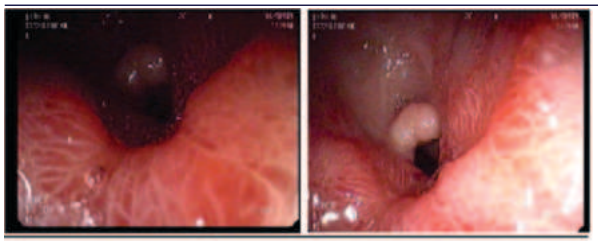


Pre Procedure **Post Procedure**

Corrosive Injury With Harpic Causing Mid Oesophageal Stricture And Oesophageal Baloon Dilatation Done
Figure number -6



After 2 Months Stricture **Pylorus Causing Gastric Outlet Obstruction Fig No.7**



After 2 Months Distortion Of Pylorus- Fig No.8

DISCUSSION

The factors that determine the extent of injury are nature (acid / alkali), physical form (liquid / solid), and quantity of ingested corrosive. Acid causes coagulation necrosis and alkali produces liquifactive necrosis and causes paraoesophageal injury with damage to adjacent organs like damage to respiratory tract. Stricture formation is most common, causing dysphagia and malnutrition[2]. Endoscopy has a pivotal role in the diagnosis and management, with a few shortcomings,

Acid licks the oesophagus but bites the stomach[4]. Clinical features depend upon extent, in mildest form patient may be asymptomatic. Management includes initial resuscitation, evaluation of the grade of injury, treatment of early complication and maintanence of nutrition and prevention of stricture.

Treatment According To Zargar Grading – Table 2

GRADE 1	Stricture formation unlikely and patient can be given oral feeds
GRADE II	Stricture in 30-70% patients and balloon dilatation preferred
GRADE III	Stricture in 90% with risk of perforation
GRADE IV/V	Emergency surgery recommended

Types Of Caustic Substances

- Sulfuric acid ingestion, found in lead-acid batteries and some metal cleaners, pool cleaners, drain cleaners and anti-rust product. Signs and symptoms include : Brown to black streak from angle of mouth, Brown to black vomitus.[10]

Nitric acid, hydrochloric acid and carbolic acid poisoning causes pulmonary oedema and ulceration of tongue.

The pathological changes are usually similar with almost all agents. Based on the time elapsed after ingestion of the corrosive agent, pathological changes in the upper GIT vary and have been divided into 3 phases.

Phase I (within 24 h): 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.

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.

Pulmonary complications include pneumonia secondary to aspiration and opportunistic infections in the lung

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