STUDY OF CORROSIVE POISONING AND ITS EFFECTS ON UPPER GASTROINTESTINAL TRACT AND SURGICAL MANAGEMENT- A SINGLE INSTITUTION EXPERIENCE

Ramesh Lingala[1], Ramesh Kota[2]

[1]Assistant Professor, Department of General Surgery, Gandhi Medical College and Hospital, Secunderabad, Telangana.
[2]Assistant Professor, Department of General Surgery, Gandhi Medical College and Hospital, Secunderabad, Telangana.

ABSTRACT

BACKGROUND

Corrosive injury to the upper gastrointestinal tract is an agonising experience for both the patient and surgeon. Caustic ingestion may cause wide spread injury to the lips, oral cavity, pharynx and the upper airway. The effect that these agents have on the oesophagus accounts for most of the serious injuries and on stomach, which may result in perforation and death in the acute phase.[1,2] If the patient survives the acute effects of caustic ingestion, the reparative response leads to the development of oesophageal and gastric strictures. There is also an increased incidence of oesophageal and gastric cancer in the longterm.[3,4,5] These patients present with the most distressing symptoms of dysphagia and are not able to swallow even liquids sometimes. For a surgeon, it is necessary to restore the GI continuity, so that the patients may be relieved of symptoms and can take food naturally for the rest of their lives. An insight is made into the various modalities of treatments available for corrosive effects of oesophagus and stomach.[6,7] Even though majority of oesophageal strictures can be managed by modern endoscopic interventional methods, surgery is mandatory in few cases. Surgery is the only modality of treatment for gastric complications. Corrosive injuries continue to result in high morbidity and mortality until more conclusive diagnostic and treatment recommendations can be made.

Present study was done to know the effects of corrosive poisoning on upper gastrointestinal tract to evaluate the common surgical procedures in the management of corrosive poisoning and to know the mortality and morbidity after corrosive poisoning.

MATERIALS AND METHODS

It is a prospective observational study conducted at a tertiary care hospital in between December 2014 to December 2016. All the patients were evaluated by history, clinical examination and radiological examination. Treatment was given according to the severity of the injury. The modes of presentation, injury to the upper GI tract, surgical methods of treatment, morbidity and mortality were observed.

RESULTS

There were 50 corrosive ingestions, 43 patients with acid injury, 7 patients with alkali injury. Of 50 cases, 32 patients underwent FJ of which 4 patients were lost to follow up. Of 43 cases of acid injury- 8 were oesophageal strictures, 3 cases had (37.5%) gastric transposition and 5 cases (62.5%) had colonic interposition. Of 7 cases of alkali injury- 1 case (14.28%) had gastric transposition and 1 case (14.28%) had colonic interposition. All of them were done through substernal route. Of 43 patients of acid injury- 19 were gastric strictures, 7 cases (36.84%) managed by gastrojejunostomy and 5 cases by Billroth-I gastrectomy (26.31%) and 7 cases (36.84%) by Billroth-II gastrectomy. Of 7 cases of alkali injury, 1 case (14.28%) was managed by gastrojejunostomy. Of 8 cases of oesophageal stricture of acid injury, 3 cases (37.5%) postoperative period was uneventful, 2 cases (25%) developed wound infection, 2 cases (25%) developed hoarseness of voice and anastomotic leak and 1 case (12.5%) developed chest pain. Of all 2 cases of oesophageal stricture of alkali injury had normal outcome. Of 19 cases of gastric strictures, 17 cases (89.47%) had normal outcome, 1 case (5.26%) developed dumping syndrome, 1 case (5.26%) developed wound infection. Of 1 case of gastric stricture of alkali injury had normal outcome. Of 50 cases studied, 46 (92%) cases were survived, 4 cases (8%) were expired due to extensive injury of oesophagus and stomach, thus success rate of 92% and mortality of 8%.

CONCLUSION

1). Acid corrosive injury is more common in India unlike in the west. 2). Acid affects both oesophagus and stomach, thus oesophagus is not immune from injury due to acid ingestion. 3). Long segment of oesophagus is most commonly involved in corrosive injury, and in stomach, antrum is most commonly involved. 4). All the aforementioned procedures for oesophageal and gastric strictures were fruitful. 5). Health education regarding the effects of corrosive ingestion should be carried out in a large scale. 6). A long-term surveillance of the patients is required to detect early malignancy changes.

KEYWORDS

Corrosive Poisoning, Upper Gastrointestinal tract, Surgical Management.

HOW TO CITE THIS ARTICLE: Lingala R, Kota R. Study of corrosive poisoning and its effects on upper gastrointestinal tract and surgical management- A single institution experience. J. Evid. Based Med. Healthc. 2017; 4(44), 2691-2695. DOI: 10.18410/jebmh/2017/535

Financial or Other, Competing Interest: None.
Submission 04-05-2017, Peer Review 10-05-2017,
Acceptance 22-05-2017, Published 01-06-2017.
Corresponding Author:
Dr. Ramesh Lingala,
House No. 4-6-58, Venkatasai Nagar, Lane 5C,
Chengicherla Boduppal Muncipality, Telangana-500092.
E-mail: rameshlingala23@gmail.com
DOI: 10.18410/jebmh/2017/535

BACKGROUND

Corrosive injury to the upper gastrointestinal tract is an agonising experience for both the patient and surgeon. Caustic ingestion may cause wide spread injury to the lips, oral cavity, pharynx and the upper airway. The effect that these agents have on the oesophagus accounts for most of the serious injuries and on stomach, which may result in perforation and death in the acute phase.[1,2] If the patient survives the acute effects of caustic ingestion, the reparative response leads to the development of oesophageal and gastric strictures. There is also an increased incidence of oesophageal and gastric cancer in the longterm.[3,4,5] These patients present with the most distressing symptoms of dysphagia and are not able to swallow even liquids sometimes. For a surgeon, it is necessary to restore the GI continuity, so that the patients may be relieved of symptoms and can take food naturally for the rest of their lives. An insight is made into the various modalities of treatments available for corrosive effects of oesophagus and stomach.[6,7] Even though majority of oesophageal strictures can be managed by modern endoscopic interventional methods, surgery is mandatory in few cases. Surgery is the only modality of treatment for gastric complications. Corrosive injuries continue to result in high morbidity and mortality until more conclusive diagnostic and treatment recommendations can be made.

MATERIALS AND METHODS

It is a prospective observational study conducted at a tertiary care hospital in between December 2014 to December 2016. All cases who had accidentally or intentionally taken corrosive poisoning are included in the study. Cases of below 12 years of age group are excluded. During the initial clinical evaluation, the history was directed towards the details of corrosive agent like the type of corrosive (acid or alkali), quantity taken, concentration and whether the corrosive ingestion was suicidal or accidental. Then, the symptoms were evaluated and also the time of presentation after the corrosive injury was taken into the consideration. Patient was then subjected to clinical examination including vital data, oropharyngeal evaluation, chest and abdominal examinations. After routine serum chemistry and blood grouping, each patient was subjected to plain radiographs of chest and abdomen for evidence of perforation. When the condition of the patient has been stabilised, each patient with no clinical evidence of perforation was subjected within the next 48 hours under local Xylocaine anaesthesia. Oesophagogastroduodenoscopy using a flexible endoscope was attempted in all patients to assess the location, extent and severity of the injury to the upper GI tract. The injury was graded according to the modified criteria given by Zargar and colleagues.

Treatment

Condition of the patient assessed clinically and in severe injuries if needed, emergency surgical intervention was done. In mild-to-moderate injuries, patients were treated conservatively for few days. If they could tolerate, oral feeds were allowed for liquid or soft diet. If they could not tolerate oral feeds, feeding jejunostomy was done for improving nutrition. Patients were followed up every week for the first month, then every month for six months, thereafter alternate month for one year. During each visit, the patients were evaluated by upper GI endoscopy to know the status of strictures and clinical improvement. Barium contrast studies were also done to evaluate the level of strictures. CECT abdomen was done to know the status of remaining bowel. Patients with oesophageal strictures were subjected to dilatation till a sufficient lumen (to a minimum of 15 mm) was established for considerable amount of time. Patients with failed endoscopic oesophageal dilatations were taken up for oesophageal replacement surgery.

RESULTS

The study included 50 patients with a definitive history of corrosive ingestion. The age of the patients was in the range of 13-60 years with a mean age of 36.5 years. The maximum percentage of patients 40% were in the age group of 21-30 years and only 4% in the age group of 51-60 years. Women constituted more numbers than men. Most of them ingested acid while 7 of them ingested caustic soda. The exact volume, the concentration of the acid or alkali was not known. Ingestion was suicidal in 44 (88%) and accidental in 6 (12%). Four patients presented immediately to the hospital within 8-10 hours with a mean of 9 hours. Rest of the patients presented within a week time with a mean of 4.5 days. 66% patients presented with burns of either lips, tongue, buccal mucosa, soft palate, hard palate or pharynx.

Involvement of UGI Tract-

	Acid (n=43)	Alkali (n=7)
Oesophagus	10 (23.25%)	2 (28.57%)
Stomach	5 (11.62%)	1 (14.28%)
Both oesophagus and stomach	28 (65.11%)	4 (57.14%)

Sl. No.	Stricture Sites	Acid	Alkali
1.	Middle 1/3 of oesophagus	2 (25%)	1 (50%)
2.	Lower 1/3 of oesophagus	1 (12.5%)	-
3.	Long segment with gastric injury	4 (50%)	1 (50%)

4.	Antrum	12 (63.5%)	-
5.	Antrum and body	7 (36.84%)	-

Sl. No.	Surgery Done	Acid (n=43)	Alkali (n=7)
1.	**Oesophageal Stricture**		
	a) Gastric transposition	3 (37.5%)	1 (14.28%)
	b) Colonic transposition	5 (62.5%)	1 (14.28%)
2.	**Gastric stricture**		
	Gastrojejunostomy	7 (36.84%)	1 (14.28%)
	Billroth-1 gastrectomy	5 (26.31%)	-
	Billroth-2 gastrectomy	7 (36.84%)	-

Of 50 cases, 4 patients who underwent surgery died postoperatively after2-3 days. Of 50 cases, 32 patients underwent FJ of which 4 patients were lost to follow-up. Of 8 cases of oesophageal stricture of acid injury, 3 cases (37.5%) postoperative period was uneventful, 2 cases (25%) developed wound infection, which were managed by proper antibiotics, 2 cases (25%) developed hoarseness of voice and anastomotic leak, which were managed by conservative management and 1 case (12.5%) developed chest pain, which was managed conservatively. Of all 2 cases of oesophageal stricture of alkali injury had normal outcome. Of 19 cases of gastric strictures, 17 cases (89.47%) had normal outcome, 1 case (5.26%) developed dumping syndrome, which was managed by counselling, 1 case (5.26%) developed wound infection, which was managed by proper antibiotics. Of 1 case of gastric stricture of alkali injury had normal outcome.

The burns visualised by endoscopy have been graded by Zargar[8] and colleagues as follows-

Grade	Endoscopic Findings
Grade 0	Normal examination
Grade - 1	Oedema and hyperaemia of the mucosa
Grade - IIA	Superficial localised ulceration and friability and blisters
Grade - IIB	Grade IIA plus circumferential ulcerations
Grade - IIIA	Deep ulceration, area of focal necrosis
Grade - IIIB	Multiple and deep ulcerations, areas of extensive necrosis
Grade - IV	Perforation

Table 1. Endoscopic Findings

Part Involved	Number of Cases	Percentage
Oesophagus	10	23.25%
Stomach	5	11.62%
Both	28	65.11%

Table 2. Structures Involved in Acid Injury

Part Involved	Number of Cases	Percentage
Oesophagus	2	28.57%
Stomach	1	14.28%
Both	4	57.14%

Table 3. Structures Involved in Alkali Injury

Part Involved	No. of Cases	%
Isolated mid oesophageal stricture	3	30%
Lower segment stricture	2	20%
Long segment stricture	5	50%

Table 4. Segments of Oesophagus Involved in Corrosive Poisoning

Parts Involved	Number of Cases	Percentage
Antrum	13	65%
Body and antrum	7	35%

Table 5. Parts of Stomach Involved

Result	No. of Cases	%
Normal outcome	5	50%
Chest pain	1	10%
Wound infection	2	20%
Anastomotic leak and hoarseness of voice	2	20%

Table 6. Results of Oesophageal Stricture Surgeries

Result	No. of Cases	%
Normal outcome	18	90%
Infection	1	5%
Dumping syndrome	1	5%

Table7. Results of Gastric Stricture Surgeries

Survival	46	92%
Mortality	4	8%

Table 8. Outcomes of the Study

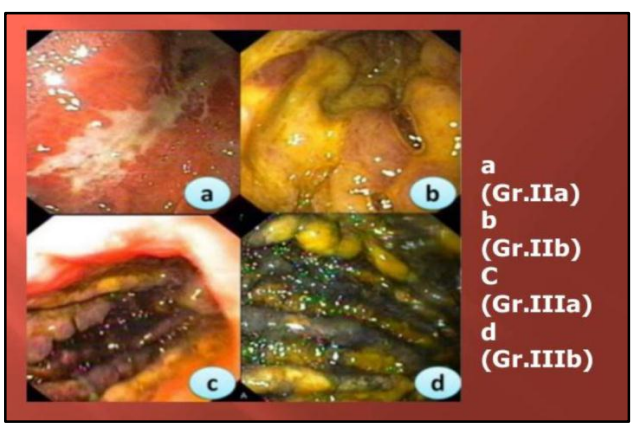

Figure 1. Endoscopic Findings in Corrosive Poisoning

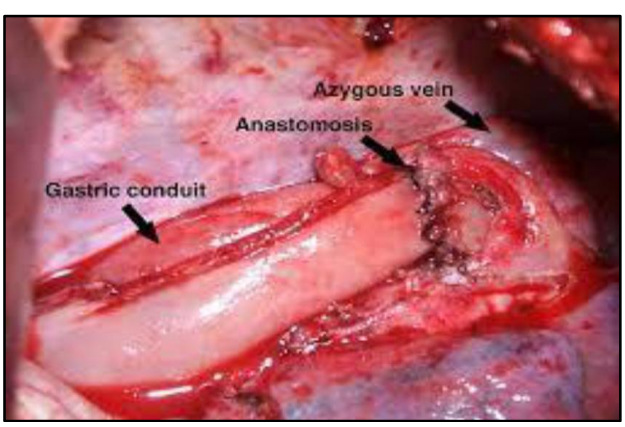

Figure2. Gastric Conduit

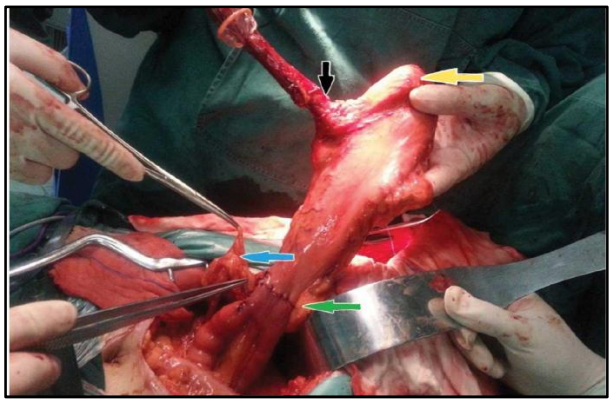

Figure 3. Colonic Conduit

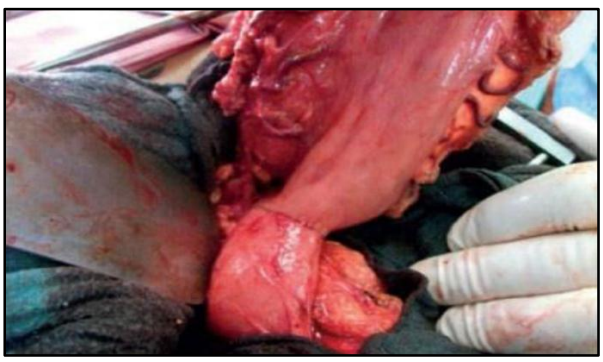

Figure 4. Billroth I Procedure

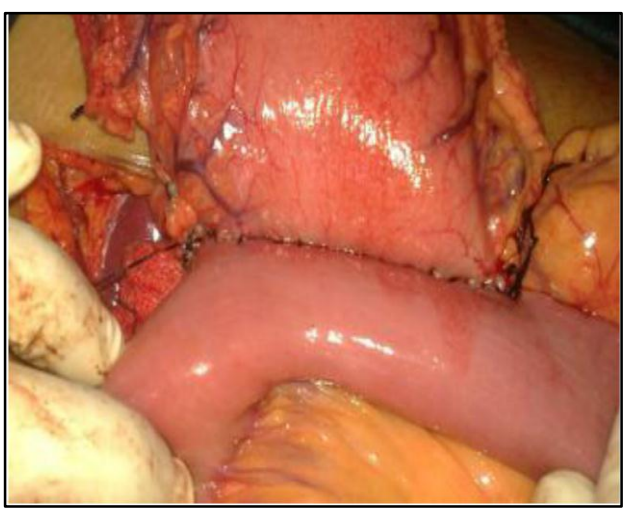

Figure 5. Billroth II Procedure

DISCUSSION

Corrosive injury to the upper gastrointestinal tract is common in India. This occurs due to ingestion of corrosive agents like acids or alkalies. Acid ingestion is more common than alkali ingestion in India. The patients have easy access to acids as these are commonly used as toilet cleansers and are easily available. In India, hydrochloric acid is widely used as cheap toilet cleaner. Individual case studies or studies of small number of patients have led to the suggestion that acids cause maximum damage to the stomach and relatively minor damage to the oesophagus because of rapid transit and great resistance of squamous epithelium to acid. In general, caustic alkali causes more profound injury than acid does, because acid produces coagulative necrosis, which acts a barrier to limit deeper levels of injury, whereas alkali tends to cause liquefactive necrosis, thereby allowing deeper penetration. Most of these studies were from the west. The duodenum was relatively spared in all of the cases, Dilwari et al and Zargar et al[8] observed the same pattern, which was attributed to the pyloric spasm induced by acid, the alkaline pH of the duodenum.

In this study, 43 patients who consumed acid and 7 patients who consumed alkali were involved. In contrast, corrosive acid ingestion accounts for a mere 5% of the corrosive consumed in the west. Suicidal ingestion of caustic agents has also increased in severity in the recent past. In this study, suicidal intent was the most common cause for ingestion (88%). In present study, I have excluded children as most of the children are referred to paediatric surgery department. The peak age for caustic ingestion is seen among adults aged 21 years and older. Women preceded more than men in present study.

The most useful investigation in the evaluation of an acute corrosive gastric injury is an upper gastrointestinal endoscopy. Endoscopic evaluation has been advised as soon as possible after corrosive ingestion, since it is believed that the risk of perforation is lowest at this point. Early endoscopy carries the risk of misdiagnosing the extent of transmural damage in the presence of extensive hyperaemia. A repeat study is again required a few days subsequently to assess the true damage. It is our policy, therefore, to do endoscopy 72-96 hours after the corrosive ingestion. In present study of 50 patients, at first, endoscopy examination, the majority of patients had IIA (n=15) and IIB (n=15) with 48% stenosis of oesophagus and stomach noted.

Adherence to the basic tenet of avoiding a gastric lavage in any corrosive poisoning cannot be overemphasised. Attempts at neutralising the acids or alkalies are ill-advised and the resulting exothermic reaction from the neutralisation process may do more harm than good. Similarly, there is not much role for measures to dilute the corrosive with milk, water and so forth, as the definitive extent of the injury is determined within minutes after ingestion.

Emergency surgical intervention is needed if the patient develops any signs of oesophageal perforation, peritonitis or uncontrolled massive haematemesis. In view of the high probability of slow, but relentless progression of transmural necrosis, there should be a low threshold for consideration of laparotomy at the earliest suspicion. If there is severe oesophageal burn with a high likelihood of stricture formation, a feeding jejunostomy is performed and the stomach is assessed intraoperatively at this time. If the oesophagus is relatively spared with moderate injury to the stomach, the patient is fed through a jejunostomy and kept on regular observation to monitor the progress of the gastric burn. If, on the other hand, the stomach appears soft and necrotic, a gastrotomy is made and the extent of transmural and mucosal necrosis is assessed before planning a resection either a distal gastrectomy or a total gastrectomy. Patients with extensive gastric injury are

often critically ill and do not withstand lengthy reconstructive procedures. Hence, we do total gastrectomy, closure and drainage of the oesophageal and duodenal stump, a cervical oesophagostomy along with a feeding jejunostomy leaving reconstruction (using a jejunal loop) for a more opportune moment should the patient comes out of the acute phase. For less extensive acute gastric injury, a distal gastrectomy may suffice.

Early surgery is essential to improve the prognosis in cases of oesophageal or gastric perforation. The reduced mortality achieved through early detection of impending or actual perforations outweighs the morbidity and mortality rate associated with surgical exploration in patients with endoscopically-diagnosed second-degree burns. However, many surgeons have condemned early surgery because the extent of injury often cannot be delineated, leaks at anastomotic sites can occur and surgery will not be needed in the majority of patients. The use of corticosteroids to prevent stricture formation in patients with severe injury is a subject of debate.

In this study, corticosteroids were not used for any patient. In present study, antibiotics were given to all patients. In present study, 10 patients (20%) have undergone early dilatations and the results were fruitful. Stent placement has shown some success in some series, though timing and type vary. Stents should be left in place for 14 to 21 days to allow for reepithelialisation. Documented effects from artificial feeding in patients intoxicated with corrosive substances are reduction of infections, reduction of predisposition to developing aspiration pneumonia, reduction of the risk for pulmonary embolism.

In present study, patients with grade I and IIA degree of damage, total parenteral nutrition is given in the first 24 hours is followed by a liquid diet until the 10th day. Afterwards, food intake was in a liberal regimen. In patients with grade IIB and III degree of damage, the so called oesophageal rest is recommended. Oesophageal rest may last until the 10th day after corrosive ingestion. Early endoscopic grading of the injury caused by corrosive acid not only helped in planning the management of patients, but also in predicting the prognosis.

SUMMARY AND CONCLUSION

1. Acid corrosive injury is more common in India unlike in the west.
2. Acid effects both oesophagus and stomach, thus oesophagus is not immune from injury due to acid ingestion.
3. Long segment of oesophagus is most commonly involved in corrosive injury and in stomach antrum is most commonly involved.
4. All the aforementioned procedures for oesophageal and gastric strictures were fruitful.
5. Health education regarding the effects of corrosive ingestion should be carried out in a large scale.
6. A long-term surveillance of the patients is required to detect early malignancy changes.

REFERENCES

[1] Gago O, Ritter FN, Martel W, et al. Aggressive surgical treatment for caustic injury of the esophagus and stomach. Ann Thoracic Surgery 1972;13(3):243-250.

[2] Appelberg HR. Corrosive burns of the esophagus and their treatment. Acta-Otolaryngologica 1960;52(Sppl 158)138-143.

[3] Csikos M, Horvath O, Petri A, et al. Late malignant transformation of chronic corrosive esophageal strictures. Langenbecks Arch Chir 1985;365(4):231-238.

[4] Hopkins RA, Postlethwait RW. Caustic burns and carcinoma of esophagus. Ann Surg 1981;194(2):146-148.

[5] Akiyama H. Surgery for carcinoma of the esophagus. Curr Probl Surg 1980;17(2):53-120.

[6] Nicosia JF, Thornton JP, Folk FA, et al. Surgical management of corrosive gastric injuries. Ann Surg 1974;180(2):139-143.

[7] Zhou JH, Jiang YG, Wang RW, et al. Management of corrosive esophageal burns in 149 cases. J Thorac Cardiovasc Surg 2005;130(2):449-455.

[8] Zargar SA, Kochhar R, Nagi B, et al. Ingestion of strong corrosive alkalis: spectrum of injury to upper gastrointestinal tract and natural history. Am J Gastroenterology 1992;87(3):337-341.