GASTROENTEROLOGY IN INDIA

Corrosive Injury to the Upper Gastrointestinal Tract

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Corrosive injury to the upper gastrointestinal (UGI) tract is an agonizing experience for both the patient and the treating physician. Though formulations of household cleansing agents have become safer in recent times, corrosive injury still remains a common clinical problem in many countries, particularly India. While lye is the most frequently ingested caustic in the West, acid ingestion is a common cause of UGI tract injury in our country. Childhood injuries are usually accidental while suicide is a frequent motive in young adults.

The scenario of corrosive injury has changed over the last 30 years. Prior to 1967, most reported cases were due to ingestion of solid lye in the form of particles, flakes or granules. Ingestion of solid lye usually produces damage to the lips, oral cavity and larynx. Esophageal injury and strictures were less common because intense burning of lips or buccal mucosa limited the quantity of solid alkali ingested. Introduction of liquid lye as a drain cleaner led to an increase in the incidence of esophageal and gastric burns. Since 1970, many such products for household use have been reformulated or withdrawn from the market in countries like the USA. However, these agents, especially acids, are easily available in the Indian market. Corrosive agents commonly ingested include strong alkalis like sodium hydroxide and potassium hydroxide and strong acids like sulfuric acid and hydrochloric acid. Ammonium and sodium hypochlorite are examples of weak alkali and weak acid respectively. Acid injuries seem to be more common in India because acids, being cheaper than alkalis, are more commonly used as toilet-bowl and drain cleaners.

Pathophysiology

The extent of injury depends on the nature of corrosive agent consumed (acid or alkali), its physical state, amount and concentration of the agent consumed, and duration of contact with the mucosa.

Alkaline substances produce more severe tissue injury by causing liquefactive necrosis which allows deeper penetration of the corrosive agent and further injury. This process of tissue destruction stops only when the destroyed tissue dilutes or neutralizes the alkali sufficiently to render it harmless. Acids, on the other hand, produce coagulative necrosis resulting in formation of an eschar which protects against further damage. It has been generally believed that acids predominantly injure the stomach and spare the esophagus because of rapid transit through the esophagus, its alkaline environment and relative resistance of esophageal mucosa to acids. However, several workers have reported esophageal injury after acid ingestion. Zargar et al reported that 88% of patients with acid ingestion had esophageal injury and 38% developed esophageal strictures; these figures are comparable to those observed with alkalis. Similarly, gastric injury is believed to be less common with alkalis as these may be partly neutralized by acidic contents of the stomach. However, in one study, 93% of patients with alkali ingestion had evidence of gastric injury. Strong acids produce severe injury while weak acids produce significant injury only in case of prolonged contact. For alkali, the critical pH is 12.5, though the titrable base content is also important. Lye, which has a pH of 12.5, is therefore a strong corrosive agent.

Granules or flakes of lye tend to stick to the mucosa and commonly produce local burns in the buccal cavity. Due to intense burning in the mouth, solid lye is less likely to be ingested and esophagogastric injury is thus uncommon. On the other hand, liquid lye, which has a high specific gravity, rapidly passes into the esophagus and stomach producing more extensive damage. Encapsulated lye may produce discrete ulcers in the stomach.

Though at times difficult to estimate, the amount of corrosive substance ingested may be important. Zargar et al reported that grade II injury predominated when the amount of alkali ingested was less than 50 mL, while ingestion of larger quantities resulted in grade III injuries.

Concentration of the corrosive is also important. In animal studies, Krey demonstrated that the depth of injury increased as the concentration of NaOH applied to an isolated esophagus was increased from 3.85% to 22.5%. The weakest solution caused damage only to the mucosa and submucosa while strong solutions caused transmural injuries and peri-esophageal reaction. Duration of mucosal exposure to the corrosive agent is another
important determinant. Even weak caustics may produce significant injury if allowed to remain in contact with the mucosa for a long period of time.7 

Clinistest tablets and disc batteries produce injury by slightly different mechanisms. Clinistest tablets, used to test urine for sugar, may produce esophageal ulceration and stricture secondary to liberation of intense heat on hydration.12 Disc batteries produce damage secondary to local pressure and release of electrolyte solution which contains 45% ROH.13

In animals pathological changes due to caustic injury can be divided into three phases.14 In the first or acute phase immediately after contact, the superficial epithelium is destroyed and necrosis extends to a variable depth from superficial to full thickness of the esophageal wall. Polymorphonuclear leukocytes and bacterial invasion soon follow. Vascular thrombosis may further accentuate the injury. Progression of inflammatory response and vascular thrombosis lead to sloughing off of superficial layers between the second and fifth days. This is followed by the second or reparative phase with appearance of granulation tissue at the periphery of the injury. Collagen deposition starts early and peaks during the second week but continues for weeks to months. Re-epithelialization also occurs during this phase but may not be complete for a long time. In the third phase which may begin as early as the end of the second week, collagen contracts both circumferentially and longitudinally resulting in stricture formation. Perforations, with involvement of adjacent organs including pleura, pericardium, pancreas and spleen, may result.15 Functional abnormalities of the vagal nerves16 and esophagus and gastric motility have also been reported after acid ingestion.17

Clinical Presentation

Clinical features are variable and depend largely on the physical state, concentration and amount of the caustic ingested. It may be emphasized here that presence or absence of oropharyngeal involvement does not accurately predict the extent of esophageal or gastric injury.18

Typically, the clinical course is triphasic, ie acute, latent and retractor phases.4 In the acute phase, the patient complains of pain in the oral cavity, retrosternal area or abdomen. Odynophagia and dysphagia are common and gradually decrease over the next 3-5 days with decrease in mucosal edema and spasm. Aspiration of caustic material may lead to hoarseness, stridor and dyspnea necessitating urgent tracheostomy. Hematemesis may occur but is usually mild and self-limiting. Severe hematemesis occurring a few days after caustic ingestion suggests development of aortoesophageal fistula.19 Full thickness necrosis can produce perforation leading to mediastinitis, pleural effusion, empyema, or peritonitis.19

Septicemic shock is a common cause of death. In the latent phase, there is transient decrease in symptoms like dysphagia, giving a false sense of improvement. The retractor phase is characterized by progressive fibrosis which gives rise to symptoms of esophageal or gastric outlet obstruction. Obstructive symptoms usually appear within 2-3 weeks of corrosive ingestion but may sometimes be delayed for many months. About 10%-33% of patients with documented esophageal injury develop clinically apparent esophageal strictures which appear early. It within 3-8 weeks of caustic injury, progress rapidly while those appearing more than 8 weeks after the injury have a slower progression.4

Diagnosis

The diagnosis is easy in most cases since history of corrosive ingestion is available. At times, however, for fear of medicolegal action, accurate history may not be forthcoming. Besides examination of mouth and pharynx which may provide important clues, endoscopic and radiologic examination of the UGI tract not only helps in making a diagnosis but also in assessing the extent and severity of injury and presence of complications, which is not possible on the basis of symptoms alone.18

Upper Gastrointestinal Endoscopy

Only 20%-40% of patients with caustic ingestion have identifiable esophageal injury20 and this may present without any oral involvement.21 Thus, endoscopy performed soon after corrosive ingestion is an excellent tool for assessment and staging of injury and tailoring the treatment accordingly. Patients with minimal or no injury to the esophagus and stomach can be discharged safely while those with severe injury need hospitalization. Endoscopic examination with a flexible pediatric endoscope should be done after hemodynamic stabilization is achieved. At times, edema and erythema may develop 12-24 hours after suble injury and may be missed at endoscopy done at an earlier time.13 General anesthesia and endotracheal intubation are required only if the patient has respiratory distress. It is wise to withhold endoscopy in the subacute phase (5-15 days after caustic ingestion) because of higher chances of perforation. Some advocate limiting endoscopic examination to documenting the most proximal evidence of injury21 while others favor a complete though cautious screening of the entire UGI tract.21,22

Esophageal burns due to corrosive ingestion have been divided into three grades.26 In first degree burns, there is mucosal erythema and edema; subsequently, the mucosa sloughs without any scar or stricture formation. Second degree burns include ulceration with necrotic tissue and white plaques that are less than circumferential, while the third degree burns include circumferential involvement of the esophageal wall. A majority of stree
tures develop following severe and circumferential involvement. Recently, Zangar et al. have modified the classification of esophageal burns to help in better prognostic and therapeutic guidance. They divided Grade 2 injuries into Grade 2a (fibrillation, hemorrhages, erosions, blisters, exudates and superficial ulcers) and Grade 2b (Grade 2a plus deep discrete or circumferential ulcers). Grade 3 injuries were classified into Grade 3a (Grade 2 plus small scattered areas of necrosis) and 3b (Grade 2 plus extensive area of necrosis). All patients with Grades 1 and 2a injuries recovered without sequelae while a majority of patients with Grade 2b (71%) and all survivors with Grade 3 injuries developed strictures. Most deaths also occurred in association with Grade 3 injuries.

Radiological Evaluation
In the acute phase of corrosive injury, plain skigrams of the chest and abdomen may show evidence of esophageal or gastric perforation. Contrast studies with water soluble agents performed within two days may not give accurate information about esophageal and gastric involvement. In fact, information obtained may be misleading because of atonia or spasm. Later changes include blurring of mucosa, intramural pseudocysticosis, deep linear ulcers with intramural dissection, retention and pocketing of contrast and intramural gas collections. The stomach may show evidence of ulceration, bullae and pseudopolyps. Contrast studies are very useful for early detection of strictures. After about two weeks, signs of acute injury subside and barium examination may show development of esophageal or gastric strictures.

Complications

Early complications
These include perforation, UGI bleeding, infection, septicemia, shock and death. Airway obstruction may occur within hours due to injury to the epiglottis or larynx. Aspiration may be followed by severe pneumonitis with opportunistic infections and adult respiratory distress syndrome. Perforation usually occurs a few days after ingestion because of progressive necrosis, and commonly presents as slowly progressing mediastinitis. Early perforation (within hours) occurs after ingestion of large amounts of a strong corrosive agent. Tracheoesophageal or bronchoesophageal fistulae may form. Shock is due to hypovolemia or sepsis. Most series report early mortality in the range of 1% to 4%. Mortality is generally lower in children.

Late Complications
Esophageal stricture. Stricture formation depends upon the depth of injury. Almost all survivors of Grade 3 injury will develop stricture while those with Grade 1 injury recover without sequelae.

Development of a dense fibrous stricture takes about 4-6 weeks though dysphagia may appear earlier. Strictures may be long, tortuous, multiple and very tight. Barium contrast studies are the best method to demonstrate the presence, number, length and location of the stricture. These may also reveal associated gastric narrowing. A barium study should be obtained about 3 weeks after injury irrespective of the presence or absence of symptoms, and repeated as required. Eighty percent of strictures become apparent between 2 to 8 weeks after injury though some patients become symptomatic several months after initial injury.

Gastric cicatrization. Corrosive injury must often cause pyloric or antral stenosis, though other deformities like shortening and irregularity of lesser curvature and hour glass deformity of the stomach may also occur. Gastric outlet obstruction due to antral or pyloric stenosis is less common as compared to esophageal stricture. In one large series, only 4 of 216 patients with history of caustic ingestion developed gastric outlet obstruction. Symptoms of gastric obstruction usually appear within 5-6 weeks of injury but in some cases may appear after a few years. They may at times become apparent after esophageal stricture is dilated, enabling the patient to eat.

Cancer of the esophagus. Several anecdotal reports and small series suggest an increased risk of squamous cell carcinoma of the esophagus following corrosive injury. The risk is estimated to be about 1000-3000 fold higher as compared to the general population. History of caustic ingestion is present in 1.4% to 2.6% of patients with esophageal carcinoma. Younger age of these patients and the development of carcinoma at the stricture site suggest an etiological relationship with the caustic injury. Esophageal cancer has been reported in a 15 year old patient with past history of caustic ingestion. These patients develop carcinoma a decade earlier than other patients with esophageal cancer. The reason for this increased incidence is not known but chronic inflammation may be responsible.

The average time interval between corrosive intake and development of carcinoma is about 40 years. Most cancers present with the usual symptoms, ie dysphagia, weight loss, pain, etc. Most of these tumors occur in functional esophagi only. Since patients seek medical help earlier because of dysphagia, their tumors are diagnosed early enough to be resectable in 30% to 40% of cases. The prognosis after resection is good with reports of 75% five-year survival. The factors which may account for such good prognosis include younger age of the patient, early diagnosis and limitation to the spread of carcinoma by the surrounding avascular scar tissue.

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Large prospective studies are required to find out the exact incidence of cancer after caustic ingestion. There is no information whether development of cancer is influenced by the nature of corrosive ingested, age at injury or extent of initial esophageal damage. The effect of successful dilatation of esophageal stricture on incidence of cancer is also not known. Whether the bypassed esophagus is also prone to high risk of cancer is not clear. However, chances of development of carcinoma in a scarred esophagus left in situ are minimal because of lack of mechanical, chemical and thermal irritation to the damaged esophagus. There is so far only a single case reported of cancer arising in an excluded esophagus.

Gastric Cancer. Unlike esophageal carcinoma, there is no good evidence to support an increased risk of gastric cancer after caustic injury. There are few case reports of gastric cancer and squamous metaplasia in persons with history of caustic ingestion, but the etiological relationship is not well established.

Treatment

Emergency Management

In the absence of perforation, treatment is largely symptomatic and aimed at prevention of early and late complications. Assessment of airway status is a very important part of the emergency management, since laryngeal injury can progress very rapidly and produce total airway obstruction. If there is evidence of laryngeal injury with respiratory difficulty, proper airway should be immediately established by endotracheal intubation or tracheostomy.

Unlike many other poisonings, emesis should not be induced as it will again expose the esophagus, pharynx, buccal cavity and larynx to the corrosive material. Similarly, gastric lavage is also not of value because of the extreme rapidity with which tissue necrosis occurs after contact with the caustic substances. Further, gastric lavage may add to the injury by producing heat and gases as a result of neutralization of the caustic material. Small sips of water can be given to dilute any fluids of caustic still suspected to be in the esophagus. Use of nasoendogastric tube is not widely advocated but can be helpful in aspiration of gastric contents and determination of pH. Intravenous fluids are given to correct hypovolemia, maintain nutrition and correct any acid-base imbalance. Broad-spectrum antibiotics may be needed to cover superimposed infection. Early surgery is needed for perforation of esophagus or stomach following ingestion of corrosives. Some workers advocate immediate surgical intervention if endoscopic findings suggest full thickness necrosis or gastric pH is alkaline. Surgical approach usually consists of aggressive resection of necrotic tissue and tissue with doubtful viability, appropriate diversion and bypass, and adequate drainage.

Prevention of Stricture

Although animal studies have provided promising results with some agents, as yet no treatment is effective in preventing stricture formation after corrosive injury in man. Poor understanding of the pathophysiology of stricture formation and lack of controlled trials have made the interpretation of existing data difficult. Various modalities used for prevention of stricture formation include steroids, antibiotics, esophageal stents, prophylactic bougienage and antifibrotic drugs.

Steroids. Although corticosteroids have been used by many workers during the last four decades to prevent the formation of corrosive esophageal strictures (CES) after caustic injury, their exact role remains uncertain. Given for 6-8 weeks soon after injury, they have been shown to prevent stricture formation in several animal studies. Follow up in these studies was however very short and development of a delayed stricture could not be ruled out. Also, the incidence of infections was higher in the steroid treated group. Steroid therapy has been reported useful for preventing CES formation in man by some workers but the evidence is not convincing. In a recent prospective controlled trial that included 60 children with acid or lye injuries, steroids (2 to 2.5 mg/kg/day of prednisolone for 3 weeks and then tapered over the next 2.3 weeks) proved to be of no value in preventing stricture formation. In animal studies, longer duration of steroid treatment was required to demonstrate maximum benefit. It is possible that results of steroid treatment may be different in adults, or with longer durations of use. Further, as the total number of children treated with steroids was relatively small, analysis of their efficacy in different grades of injury was not possible. However, it is clear that steroids are not needed in Grade 1 injury and are not useful in Grade 3 injuries. Since their efficacy in Grade 2 injuries is doubtful and side effects significant, we are not in favor of use of steroids for prophylaxis against CES.

Antibiotics. Antibiotics have not been shown to have any attenuating effect on scar formation either in animal or human studies. These are given only if evidence of infection is present or as an adjunct to steroid therapy.

Nutrition. Since oral feeding may cause further damage to the esophagus or stomach, intravenous hyperalimentation is recommended in the acute phase. Further, these patients not only suffer from dysphagia but also have high nutritional demands due to catabolic stress. Di Costanzo et al suggested that total parenteral nutrition (TPN) (40-50 Kcal/Kg body weight) may help in preventing
striction formation. None of their patients with Grade 2 and only 36% of those with Grade 3 injuries showed stricture formation while receiving TPN. No controlled trials have, however, been reported. TPN is preferable to feeding through nasogastric tube in patients with laryngeal involvement, severe antroploric injury, or gastroesophageal reflux.

Placement of stents. A wide bore silicone tube stent placed across the injured area may theoretically prevent tissue contraction and formation of CES. Stents placed prophylactically for a period of 3 weeks in cats resulted in fewer and less severe stricture formation. Prophylactic placement of stents has been found useful for prevention of CES in children also. However, experience with stent placement is rather limited. The procedure may result in significant complications like perforation. Presently, therefore, esophageal stents are not recommended outside research protocols.

Prophylactic bougienage. The rationale for this is similar to that for stent placement. Bougienage is started at the beginning of the third week with a moderate sized dilator and is carried out regularly for several weeks. A large study showed no benefit from prophylactic bougienage. On the contrary, it may cause further trauma to the injured mucosa, accelerating collagen deposition.

Antifibrotic drugs. Various drugs used in animal experiments include collagenase, proline analogues, aminopropionitrile, N-acetylcysteine, penicillamine and colchicine. These lathyrogens act at different levels of collagen synthesis preventing dense scar and stricture formation. Though promising in animal models, there is no experience with these drugs in humans.

In the absence of any definitive therapy to prevent stricture formation, the approach at present is essentially one of vigilant anticipation to detect stricture formation early.

Treatment of Esophageal Stricture

Dilation. In view of its efficacy and safety, endoscopic dilation has become the first-line treatment of uncomplicated CES in both adults and children. It is started about 4 weeks after a severe corrosive injury, since the rate of perforation is high with early dilatation. Prograde dilation over an endoscopically placed guide wire is the method of choice. Fluoroscopic guidance is used in initial dilatations and with difficult strictures. Various types of dilators used are: i) Savary-Gilliard, ii) Eder-Puestow, iii) advanced Keymed, and iv) balloon dilators.

Retrograde dilation is not commonly used except in rare instances where prograde dilation fails. In most cases, dilation is carried out on an outpatient basis. At our center, dilation is performed at weekly intervals till an adequate lumen size (45F) is obtained. Initially 1-6 bougies are passed during each session according to tightness of the stricture. Some tight strictures may benefit by twice weekly dilations early in their course. Our experience shows that many patients with tight and long strictures who were earlier considered unsuitable for dilatation and were advised surgery can now be successfully dilated with fluoroscopic guidance. Forceful dilatation or too rapid a jump in dilator size in a single sitting may produce further injury and poor response to subsequent dilatations. Complications of dilatation include pain, perforation, mediastinitis and peritonitis.

Data on short or long term results of endoscopic dilatation in CES are limited. Excellent results have been reported by two centers. Of our 57 patients with CES, a large majority (90%) could be successfully dilated with complete relief of dysphagia. A mean of 9 sitings was required to achieve adequate (45F) dilatation. Recurrence of dysphagia was highest in the first six months after adequate dilatation and then gradually decreased with time. Many of these patients were dysphagia-free for a long period of time. Perforations were uncommon and could be managed conservatively. Bacteremia was not uncommon and was seen in 21% of cases.

Response to dilatation depends upon various factors including tightness and length of the stricture, grade of dysphagia, and average density of fibres. Strictures which develop soon after acute injury are more dense and difficult to dilate than those which are slow to develop. Among various clinical and radiological features, esophageal wall thickness is determined on contrast enhanced CT scan of the chest was found to be the most sensitive predictor of response to dilatation and short term recurrence.

Surgery. Surgery is indicated when a stricture cannot be dilated or there has been a complication like perforation after dilatation. In the past, patients with multiple strictures or a diffusely scarred esophagus were often referred to the surgeon. With improvement in dilatation techniques, fewer patients require surgery. At our center, we advise surgery only when a stricture cannot be dilated at all, when repeated dilatations are ineffective in maintaining adequate esophageal lumen, and when dilatation results in a complication that cannot be managed conservatively.

Esophageal substitution is the surgical procedure of choice. Various bowel segments available for replacement include colon, stomach, and jejunum. Several areas of disagreement however remain, such as (1) selecting the most appropriate segment, (2) route of transposition, (3) direction of placement, and (4) leaving the injured esophagus in situ.

Use of colon leaves the gastric reservoir intact and
prevents the problem of gastroesophageal reflux. Moreover, the colon has sufficient mobility and dependable blood supply. Inoperability of position is preferred since antiperistaltic position is frequently associated with regurgitation and poor surgical results. Retrosternal route is preferred for esophageal bypass while after transhiatal esophagectomy, transmediastinal route is preferred. Additional esophagectomy increases mortality and therefore is confined to situations where complete stricture of the thoracic portion of the esophagus is seen or malignancy is suspected.

Cervical anastomosis is preferred to intrathoracic anastomosis because the latter is associated with higher risks of recurrence. Stomach tube fashioning, though technically easy, has problems of acid-peptic reflux and reduced gastric reservoir capacity. Jejunum is used after failed colon interposition when stomach is unsuitable for anastomosis. Transhiatal, transcervical esophagectomy which spares the need for thoracotomy is gaining increasing use. Patients with severe hypopharyngeal and laryngeal injuries pose a special problem since anastomosis has to be done above the cricopharyngeus, which compromises swallowing and predisposes to aspiration.

Treatment of Gastric Stricture

Usually these patients require pyloroplasty or a bypass procedure, eg gastrojejunostomy. Recently, hydrostatic balloons and long Savary-Gilliard dilators have been successfully used for dilatation of antral strictures but experience is very limited.

Summary

Corrosive injury to the UGI tract is a common problem and has a wide spectrum of presentations. Unlike in the West, where lye ingestion is more frequent, corrosive injury in India is more commonly due to acids. Depth of the injury is the most important factor which determines the outcome. Endoscopy done soon after corrosive ingestion is safe and is very helpful in assessing the extent and severity of injury and in planning proper management of these patients. At present no therapy has been proven to be effective for prevention of stricture formation. Endoscopic dilatation seems to be the treatment of choice for management of most esophageal strictures with very good short and long-term results. Surgery should be considered only when dilatation fails.

References

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