

CAUSTIC INJURY

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Caustic injury to the oesophagus can be mild, but also is - potentially lethal. Most caustic ingestions occur in children, in whom it is usually accidental, or in adults with suicidal intent. The severity of the injury depends on the type, pH, quantity strong acid, causing coagulative necrosis with eschar formation, which may limit penetration to deeper layers, or strong alkali, leading to liquefactive necrosis. The latter potentially penetrates deeper into the oesophageal wall, producing a more severe injury pattern. Diagnosis is usually not difficult. When the injurious agent is identified, it should be tested for its pH, and suicidal attempt considered. Patients may have pain in the neck, throat, chest or even the abdomen. Drooling of saliva, dysphagia and odynophagia can be present. Hoarseness of voice is an important sign to look for as it may signify laryngeal injury and potential airway obstruction. If the airway is judged to be compromised, careful assessment and emergency intubation using fiberoptic guidance, or even a surgical airway, are indicated. There is no role for gastric lavage or attempts to neutralise the acid or alkali. Initial treatment after securing an adequate airway is supportive, with intravenous fluid, oxygen supplementation and cardiorespiratory monitoring. Once the patient is stabilised, an endoscopy and CT scan with intravenous contrast should be considered. Careful endoscopy allows assessment of the extent of the injury (Figure 66.34). The Zargar grading can be used (Table 66.2). In general, the longer and more circumferential the injury, the more likely that stricture will form. The stomach is assessed as well for injury, and a nasogastric/duodenal tube can be placed with endoscopic guidance. This can be used for alimentary nutritional support; if a stricture forms, there is still a potential route of access through to the stomach. A CT scan can assess oesophageal oedema and also surrounding soft-tissue infiltration. Most caustic injuries can be managed conservatively with supportive measures. Deterioration requires surgical treatment, with emergency oesophagectomy. The oesophagus can be mobilised transhiatally or via a thoracoscopic approach. Immediate reconstruction is not recommended. A cervical oesophagostomy and a gastrostomy can be done and future reconstruction planned. A feeding jejunostomy is an alternative for nutritional support if the stomach also requires resection. Delayed complications include stricture and malignancy. A stricture can form early and may be resistant to dilatation (Figure 66.35). There is not enough evidence to support routine use of systemic steroids, intralesional injection of steroid or topical mitomycin C to reduce stricture formation. Endoscopic dilatation should be gradual, as the perforation rate is higher than in other forms of strictures. Long strictures are often resistant to dilatation. Oesophagectomy or bypass surgery may be required. Oesophagectomy has the advantage of removing the oesophagus with its long-term risk of malignancy. However, surgery is difficult because of scarring and adhesions to the mediastinum, thus a bypass operation may be preferable. The gastric conduit is placed in the retrosternal route to reach the neck. When it is also damaged and cannot be used, a colonic interposition is the alternative. The native oesophagus can be left in situ as the risk of dilatation and resultant mucocele is low. Showkat Ali Zargar, contemporary, gastroenterologist, Sher-i-Kashmir Institute of Medical Sciences, Srinagar, India. Burrill Bernard Crohn, 1884-1983, gastroenterologist, Mount Sinai Hospital, New York, NY, USA,

described regional ileitis in 1932. - - -). - - - -

injury to the oesophagus. Zargar classification Description grade 0 Normal appearance 1
Oedema and hyperaemia 2a Superficial ulceration and friability 2b Deep ulceration or
circumferential ulceration 3a Multiple deep ulceration and scattered necrosis 3b Extensive necrosis 4
Perforation Figure 66.34 Endoscopy picture showing caustic burn to the oesophagus.

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