

Keynote: Corrosive Poisoning

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Introduction : The term corrosive poison encompasses alkaline and acidic agents that cause tissue destruction when they come in contact with mucous membranes. At our Institute suicidal acid ingestion in the form of toilet cleaning agents has been the commonest form of corrosive poisoning for the last 25 years. Contrary to the belief that alkalis bite the esophagus and lick the stomach due to dilution by its acidic contents¹ and acids spare the esophagus and damage the stomach. However we have shown that concentrated alkalis and acids, both cause similar damage to esophagus as well as the stomach^{2,3}. **Pathophysiology** : Alkaline substances produce more severe tissue injury by causing liquefactive necrosis which allows deeper penetration of the corrosive agent and further injury. Acids produce coagulative necrosis resulting in formation of an eschar which protects against further damage¹. In animals pathological changes due to caustic injury are divided into three phases⁴. In the acute phase, the superficial epithelium is destroyed and necrosis extends to a variable depth. Vascular thrombosis may further accentuate the injury and lead to sloughing off of superficial layers. This is followed by the second or reparative phase with appearance of granulation tissue. Collagen deposition starts early and peaks during the second week but continues for weeks to months. In the third phase collagen contracts both circumferentially and longitudinally resulting in stricture formation. **Role of endoscopy** : Symptoms and signs are poor indicators of degree and extent of damage to the GI tract. Radiological studies are also not sensitive. The best method to stratify patients with acute corrosive ingestion is upper GI endoscopy. We had modified the endoscopic grading of corrosive injury and shown that perforation and mortality occur in grade 3b injury with strictures develop in grade 2b and 3a injury⁵. Patients with injury upto grade 2a escape from developing any sequelae. Recently we have shown that occurrence of hematemesis and / or melaena in these patients is a reliable predictor of severe injury. We have also observed that a re-look endoscopy at 5 day may be more useful than endoscopy on day 1-2 in assessing severity of injury. **Management**:

Management in the acute phase revolves around early recognition of complications like perforation, maintenance of airways and endoscopic assessment of the injury. Use of steroids and antibiotics has not been shown to prevent late complications. Once perforation has been ruled out, patients can be stratified as per endoscopic grading of injuries. Those with grade 1 or 2a injury can be allowed oral feeds while those with grade 3b injury should be observed for complications and be managed in consultation with the surgical team. Those with grade 2b and 3a injury can be managed conservatively. They require nutritional support for 6-8 weeks. Nutrition can be supported by total parenteral nutrition or jejunostomy feeding⁶. We have been instituting nasoenteral tube feeding in these patients for the last 10 years. Placement of a nasojejunal tube ensures feeding and acts as a stent. Most strictures develop within the first two months of corrosive ingestion. A contrast study should be done at this stage to plan further treatment. Those with tortuous or long esophageal strictures can be subjected to surgery while others can be dilated endoscopically. Corrosive esophageal strictures are difficult to dilate but over three fourths of them respond to endoscopic dilatation⁷. We have shown that combining intralesional steroid injections with dilatation is very

rewarding in difficult strictures⁸. Surgery has often been the choice of treatment for corrosive induced gastric outlet obstruction. We have, however, shown that short segment gastric cicatrization can be effectively treated with endoscopic balloon dilatation⁹. Those with extensive gastric cicatrization or with > 2.5 cm segmental narrowing should be subjected to surgery. **Late sequelae:** Carcinoma of esophagus has been reported in patients with corrosive injury after 2 to 3 decades. We have shown that patients with severe injury at lower end of esophagus may develop features of vagal dysfunction due to entrapment of vagal fibers in the cicatrization process¹⁰. This can lead to slowing of small bowel transit time, bacterial overgrowth, gallbladder stasis and hypochlorhydria. Corrosive induced gastric damage can result in delayed gastric emptying and achlorhydria even in the absence of any structural cicatrization.

Implications of these sequelae need to be studied. **References:** 1. Gumaste VV, Dave PB. Ingestion of Corrosive Substances by Adults. *Am J Gastroenterol* 1992;87:1-5. 2. Zargar SA, Kochhar R, Nagi B, Mehta SK. Corrosive acid ingestion: spectrum of injury to upper gastrointestinal tract and natural history. *GASTROENTEROLOGY* 1989; 97: 702- 707. 3. Zargar SA, Kochhar R, Nagi B, Mehta S, Mehata SK. Ingestion of strong corrosive alkalis : Spectrum .of injury to upper gastrointestinal tract and natural history. *AMERICAN JOURNAL OF GASTROENTEROLOGY* 1992; 87: 337- 41. 4. Johnson EC. A study of corrosive esophagitis. *Laryngoscope* 1990;73:1651- 96. 5. Zargar SA, Kochhar R, Mehta SK. The role of fiberoptic endoscopy in the management of corrosive ingestion: need to modify endoscopic classification of burns. *GASTROINTESTINAL ENDOSCOPY* 1991; 37: 242-246. 6. Dilawari JB, Sing S, Rao PN et al. Corrosive acid ingestion in man : a clinical and endoscopic study. *Gut* 1984;25:183-7. 7. Broor SL, Kumar A, Chari ST et al. Corrosive esophageal strictures following acid ingestion; clinical profile and results of endoscopic dilatation. *J Gastroenterol Hepatol* 1989;4:55-61. 8. Kochhar R, Ray JD, Sriram PVJ, Kumar S, Singh K. Intralesional steroids augment the effects of endoscopic dilation in corrosive esophagealstrictures. *GASTROINTESTINAL-EDOSCOPY* 1999; 49: 509-13. 9. Kochhar R, Sethy PK, Nagi B, Wig JD. Endoscopic balloon dilatation of benign gastric outlet obstruction. *J.Gastroenterol Hepatol* 2004;19:418-22. 10. Khan BA, Kochhar R, Nagi B, Raja K, Singh K. Gallbladder emptying in patients with corrosive induced esophageal strictures. *Dig Dis Sci* 2005;50:111-5.