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Case Report

A case of severe upper alimentary tract necrosis by corrosive acid poisoning

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ABSTRACT

Corrosive poisoning is common in South East Asian countries than in the West. It can be accidental or suicidal and can cause gastrointestinal tract injuries. The grade of injuries depends on several factors related to the patient and the substance causing injury. Dilemmas arise at different management levels, whether to resort to a radical surgical approach or consider more conservative approaches. We present a case of suicidal corrosive acid injury in a 23-year-old male with extensive upper gastrointestinal tract injury managed surgically. Ampullojejunostomy may be a feasible option in patients with stomach and duodenal necrosis following corrosive acid poisoning if ampulla is normal. However, its role in the emergency setting may be questionable.

Keywords: Acid, Hydrochoric acid, Ampulla of vater, Surgery, Necrosis

INTRODUCTION

Hydrochloric acid is commonly available in local Indian markets as cheap toilet cleaners and tile washers. Various dilutions of acid are available, and products with 20-30% dilution, which removes hardened soaps, scum, and iron rust, are more common. The marketing of many of these products is unregulated, unregistered, and insufficiently labeled. Due to these reasons, hydrochloric acid consumption, accidentally or for suicide, is more common in India than in Western countries. Extensive injuries occur in patients who have consumed lethal amounts. Management of extensive injuries is challenging and creates a considerable dilemma over the appropriate approach. Here we present a rare case of suicidal corrosive acid injury in a young male with extensive upper gastrointestinal tract necrosis, managed surgically.

CASE REPORT

A 23-year-old male had presented to surgery casualty with an alleged history of toilet cleaner consumption (30 ml of 20% hydrochloric acid). The patient presented with severe epigastric and retrosternal burning sensation and one episode of vomiting. On presentation, the patient was conscious, well oriented, but restless and irritable due to severe pain. His blood pressure was 90/60 mmHg, pulse rate was 98/min, and oxygen saturation of 98%. His oral mucosa and teeth showed erosions. The abdomen had diffuse tenderness.

The patient was started on intravenous hydration, antibiotics, antacids, steroids, and analgesics. Urine was found to be dark-colored containing few flakes. X-ray chest and abdomen revealed no evidence of pneumoperitoneum or pneumomediastinum (Figure 1).

Routine blood investigations were done, which showed severe hyperkalemia and acute kidney injury (AKI). Indirect laryngoscopy showed erosions at the base of the tongue, edematous larynx, and true vocal cords could not be seen. Endotracheal intubation was tried but was difficult due to severe and extensive edematous changes in the throat; hence the patient underwent emergency tracheostomy. A repeat chest X-ray was taken, and found to have pneumoperitoneum (Figure 2).

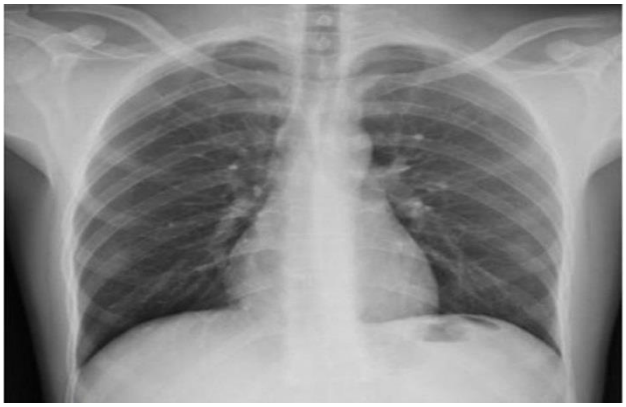

Figure 1: Chest X-ray did immediately after admission without any evidence of pneumomediastinum or pneumoperitoneum.

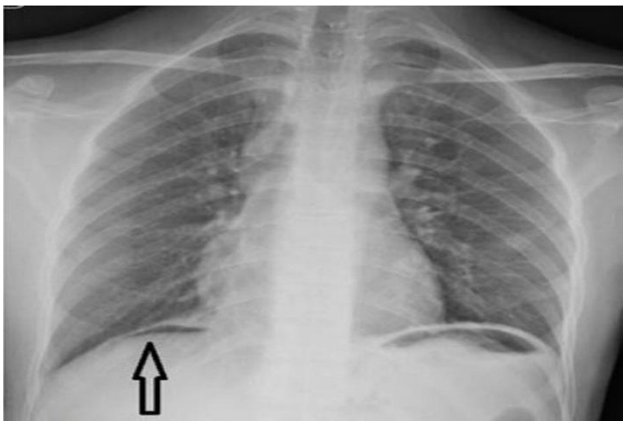

Figure 2: Chest X-ray following tracheostomy showing pneumoperitoneum (arrow).

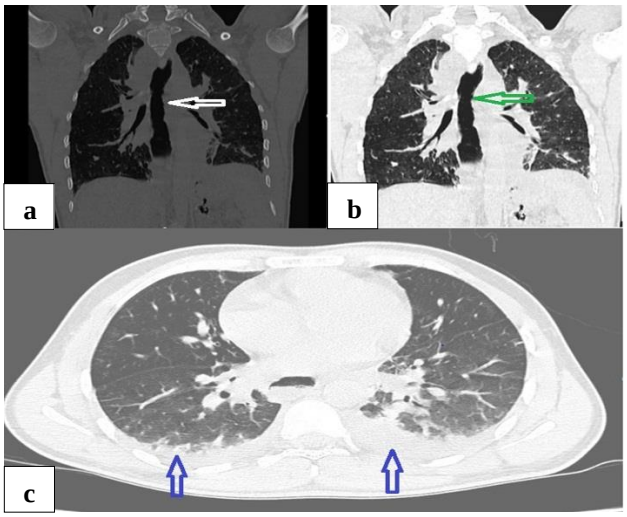

Figure 3: Computed tomography showing aspiration pneumonitis changes with the normal esophagus (green arrow) with bilateral minimal pleural effusion (blue arrows) without evidence of pneumomediastinum and no evidence of any air foci around the esophagus (white arrow), (a) and (b) coronal sections, and (c) axial section.

High-resolution computed tomography thorax (HRCT) was taken, which revealed pneumoperitoneum and air around the tracheostomy tube without any evidence of pneumomediastinum. There were minimal bilateral pleural effusions and aspiration changes in both lung fields (Figure 3).

The patient was taken up for emergency exploratory laparotomy after dialysis. On abdominal exploration, there were two liters of dirty brown fluid with mild biliary staining. The entire stomach and duodenum up to the second part were found to be tarry black and necrosed (Figure 4).

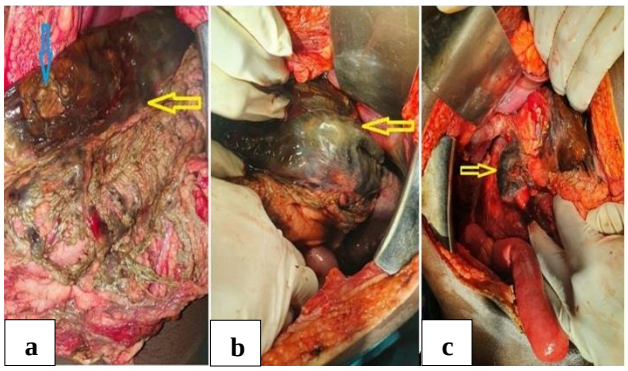

Figure 4: (a-c) Intra-operative images showing necrosed fundus, greater curvature of the stomach, ad duodenum (yellow arrow) with perforation (blue arrow).

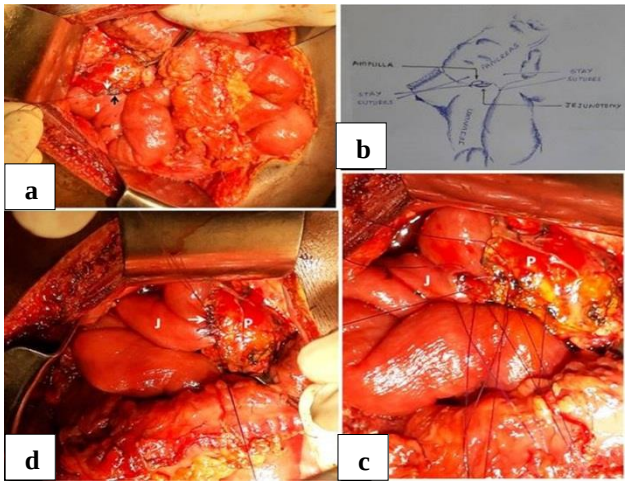

Figure 5: Intra-operative images of ampullo-jejunostomy (a) proximally advanced jejunum with jejunostomy (black arrow), and pancreas with ampulla of Vater (white arrow), approximated by two stay sutures; (b) diagrammatic depiction of Figure A; (c) pre-placed sutures before tying; and (d) completed ampullo-jejunostomy (arrow) with stay sutures in-situ.

Total gastrectomy with pancreas preserving duodenectomy was done. Bilio-pancreato-enteric continuity was restored by performing end to side

ampullo-jejunostomy. A single layer of ampullo-jejunal anastomosis was performed using polypropylene. A total of eight interrupted sutures were applied, three to the posterior wall, three to the anterior wall, and one each at superior and inferior angles (Figure 5). The distal end of the esophagus was examined. It was found that though serosa was looking healthy, mucosa and muscularis was discolored. As the patient was unstable, the distal end of the esophagus was sutured, and cervical esophagostomy was performed. Cervical esophageal mucosa was observed to be healthy. Feeding jejunostomy was performed, abdominal tube drains were placed. After two days patient had increased tachypnoea, tachycardia, progressive circulatory shock, and needed ionotropic support. On the thirteenth post-injury day, the patient had a cardiac arrest and expired.

DISCUSSION

Corrosive injuries of the esophagus and stomach are frequently seen in developing countries.[1] It can be due to acid or alkali ingestion. It can be accidental or suicidal, the former being more common in children. It is fatal in children than in adults, as the amount consumed will be small.[2] Direct mucosal injury caused by the consumed acid depends on the amount, dilution, pH, other chemical components of the product, and mode of consumption. Other factors that influence the severity of injury are the relative resistance provided by stratified epithelial layers of the upper gastrointestinal tract, content, and food or fluid properties present in the stomach. Alkali is consumed in a more considerable amount due to its tasteless nature, whereas acids are consumed in a minimal amount due to its pungent odor and sour taste. Suicidal ingestion of corrosive substances will have predominant oral, pharyngeal, and upper oesophageal lesions, whereas accidental ingestion has predominant gastric injuries, although esophageal injuries may be associated.[3]

Acids are known to cause coagulative necrosis of tissues. The coagulum acts as a barrier to the deeper invasion of the acid. Alkalis are known to cause liquefactive necrosis and are associated with more profound injuries in the tissues.[4] It has been found that a fatal dose of hydrochloric acid is 30-40 ml. In our case, the amount of acid ingested was around 30 ml.

The clinical outcome of corrosive ingestion depends upon the extent and depth of the initial injury. Injuries localized to mucosa will heal without any sequel. Injuries involving the submucosa or muscularis layer results in complications. Rarely perforations can occur when transmural injuries are present.[5] Stomach necrosis resulting from corrosive poisoning is rare, and there are only a few case reports in the literature of such events. In our patient, there was full necrosis of the stomach and duodenum with dusky esophageal mucosa. Few acids are associated with systemic side effects like hypocalcemia, hypokalemia, and acidosis.[6] However, in our patient, there was AKI with hyperkalemia secondary to extensive necrosis.

Patients may present with hypersalivation, odynophagia, vomiting, breathlessness, or hematemesis. Hoarseness of voice and stridor are indicative of laryngeal injury.[2] Our patient presented with complaints of severe retrosternal and epigastric burning pain, and later, he developed breathlessness. The patient underwent an emergency tracheostomy due to the non-visualization of cords.

There is a general concept that ‘acid licks the esophagus and bites the pylorus, disproved in many case reports and injuries mainly seen in the esophagus.[7] Acid exposure to the distal stomach induces pyloric spasm and controls the entry of corrosive into the duodenum. The alkaline pH of the duodenum also helps to negotiate the effects of acid. Hence duodenum is relatively safer when consumed acid is diluted and in lesser amount. There is a case report in literature where isolated full-thickness jejunal perforation was seen following sulphuric acid and cocktail ingestion.[8] Acute gastric injury is seen mainly in the distal stomach, ranging from mucosal injury to perforation. Rarely the stomach gets necrosed. In our case, the whole stomach and duodenum were necrosed with perforation of the stomach.

A chest X-ray is the first investigation to be done in an emergency. It shows the presence of pneumoperitoneum, pneumomediastinum, or changes in the lung. Contrast esophagogram is also useful, but there is controversy over the contrast or water-soluble contrast. Water-soluble contrast is associated with an increased risk of aspiration, and barium may irritate the pleural and peritoneal cavity in the presence of a perforation. Computed tomography (CT) thorax and abdomen are very useful for grading the corrosive injury. In our patient, the esophageal injury was graded as one, along with definite evidence of pneumoperitoneum. Although we could not comment on the stomach’s vascularity, the CT was not contrast-enhanced due to the patient’s AKI.

Endoscopy is the most crucial investigation in caustic injuries. There are controversies over the timing of the endoscopy. Early endoscopy may under grade the injury, whereas late endoscopy increases the chances of perforation. Injuries can be graded based on endoscopic findings, as described by Zargar et al.[9] We did not do the endoscopy or esophagogram on our patient as the patient was not stable for endoscopy. We did HRCT thorax and abdomen, which was suggestive of pneumoperitoneum, and hence the patient was taken for emergency laparotomy.

The basic plan in managing corrosive acid injury patients includes fluid and electrolyte management, stabilization of the patient, broad-spectrum antibiotics, and timely surgical intervention.[6] The grade of injury will guide the management of the patient. Medical management for caustic injuries includes neutralizing agents, antibiotics, proton pump inhibitors, steroids, and emetics. It is advised

not to use neutralization agents or charcoal as it carries the risk of setting out exothermic reactions, thus further increasing the grade of injury.[10] The role of a nasogastric tube is controversial. It has been said that it prevents further vomiting and may act as a stent in esophageal perforation. At the same time, it also has been said that blind insertion must be avoided as it increases the risk of long segment stricture formation.[11,12] In our case, we did not attempt nasogastric tube insertion or pH neutralizing agents.

There have been reports of caustic injury resulting in upper gastrointestinal necrosis where esophagectomy, gastrectomy, pancreaticoduodenectomy, colectomy, and splenectomy were done in various combinations.[13] Definitive reconstruction has to be deferred in acute settings as it carries a high risk of mortality and morbidity. In our patient, based on the preoperative CT scan, we suspected perforation of the stomach, without any apparent injury to other parts of the gastrointestinal tract. Hence we proceeded with laparotomy. The stomach, the first part of the duodenum, and the second part of the duodenum were necrosed and had perforation. There was doubt about the esophagus' viability as serosa was looking healthy, but the mucosa was discolored. Although transhiatal or trans-thoracic esophagectomy was one option, we did not do esophagectomy because there was intra-operative doubt about esophageal non-viability and absent evidence of esophageal perforation in preoperative CT scan.[14] A study done in Taiwan, reported in 2015, had proposed that survival after corrosive acid poisoning can be predicted based on the intraoperative findings. They proposed and classified the injuries in four classes based on intraoperative findings.[15] Our patient had a class 3 injury, which has a high mortality. We did total gastrectomy, total duodenectomy with the preservation of the pancreas. We did ampullo-jejunostomy as ampulla was healthy. We also did cervical esophagostomy and feeding jejunostomy for the patient.

Although there is no ample evidence that supports ampullo-jejunostomy for patients who undergo duodenectomy, we could find a single case report published by Paul who had done jejunal advancement and anastomosis of jejunum to a patch of duodenum containing ampulla of Vater following subtotal duodenectomy for recurrent colon carcinoma.[16] In the present case, ampullo-jejunostomy was performed to restore the bilio-pancreato-enteric continuity.

In a study of blunt esophageal stripping via cervical esophagostomy for corrosive injury, 11 out of 17 patients showed uneventful postoperative recovery.[17] An esophageal resection is an adverse prognostic predictor for survival for the patient because of the associated morbidities, ending in long-term impacts on the quality of life.[18] However, in our case, esophagectomy was precluded due to absent evidence of perforation, necrosis, and avoidance of prolonged operative time with its morbidity.

CONCLUSION

Acute corrosive injury to the upper gastrointestinal tract is a common condition in developing countries. It poses several challenges and dilemmas over conservative treatment, the timing of endoscopy, and surgical approaches. Esophagectomy may be considered in esophageal perforation or necrosis with mediastinitis for the removal of septic foci involving the esophagus. High-grade corrosive injuries like necrosis of the stomach and duodenum carry higher morbidity and mortality. Management of the high-grade corrosive injury involving the second part of the duodenum is challenging. Ampullo-jejunostomy may be a feasible procedure for selected patients if ampulla is normal; this technique's usefulness in acute emergency conditions needs further evaluation.

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