Caustic esophageal injury with tracheoesophageal fistula

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Abstract A 23-year old Saudi female patient is reported, who ingested an alkaline cleaning solution. The patient was managed in a conservative fashion at a local hospital for one month. At the end of that time she developed a tracheoesophageal fistula that caused respiratory failure and prompted transfer to a tertiary institution. Her degree of injury and late transfer made definitive care impossible. This case is presented to discuss the early and late management of caustic esophageal injury and to call for prompt transfer to a referral institution for definitive diagnosis and immediate management.

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Ingestion of caustic substances carries a high rate of mortality and significant and devastating morbidity stemming from injury to the proximal upper gastrointestinal tract. The majority of accidental ingestion occurs in the pediatric population, while suicide gestures account for the majority of adult cases. The management of caustic ingestion depends on an accurate history of the type of substance ingested: acid vs alkali, granule vs liquid and time interval from ingestion. The management of the spectrum of corrosive injuries to the upper gastrointestinal tract is divided into early phase to prevent further injury and sepsis and late phase dealing with stricture and tracheoesophageal (TE) fistula. Current management involves immediate and accurate staging via prompt assessment of extent and location of injury. Aggressive early surgical evaluation and intervention in severe injury is indicated.

In this case, management was initially non-operative. This led to severe complications and no possibility of definitive surgical management. This case report is offered as a review of early and late management of caustic esophageal injury, and a call for immediate transfer to a specialized institution capable of the complex surgical, intensive care, and gastroenterological management of these cases.

Case report A twenty-three year old Saudi woman was referred to King Faisal Specialist Hospital and Research Center four weeks after ingestion of a commercial alkali based cleaning solution (Flash). The patient had been managed at a local institution conservatively with surgical intervention limited to tracheostomy and feeding jejunostomy. Her condition had remained grave and the development of pulmonary insufficiency and sepsis prompted transfer. On arrival the patient was unresponsive, septic and anuric. Initial examination revealed that the patient was sedated with intact tracheostomy with no evidence of cuff leak. There was poor air entry to mid-lung fields bilaterally and evidence of 50 ml air leak with each cycled ventilation. It was not possible to adequately ventilate the patient. The patient had a grossly distended abdomen with tympany and scant bowel sounds. Extremities were pale and cool.

Investigations Abdominal plain film showed massive pneumoperitoneum (Fig.1).

Bronchoscopy performed revealed extensive necrosis and granulation tissue with collapse of right mainstem bronchus. No fistula was visualized.

Bronchography performed revealed suggestion of

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tracheoesophageal fistula on the left, approximately 3 cm proximal to the carina (Fig. 2).

Esophagoscopy revealed severe ulceration and edema beginning at the soft palate with severe stricture at 17 cm precluding further passage of the endoscope. No fistula was visualized.

The patient was taken to the operating room with a diagnosis of severe caustic tracheoesophageal injury with stricture and fistula formation and evidence of perforated viscus with significant ventilatory compromise. At laparotomy gross peritoneal soilage was encountered with adhesion and chronic inflammatory response in the lesser sac and small bowel in the area of the feeding jejunostomy. The jejunostomy was intact. Peritoneal lavage and instillation of warm saline revealed gross air leakage in the area of the antrum and duodenum. The lesser sac was entered and the stomach and duodenum were mobilized. A posterior, well-circumscribed pyloric channel perforation was encountered measuring 2 cm in diameter. No other perforations were seen. The antrum and pylorus were resected utilizing GIA and TA-90 stapling devices. The stapled margins were oversewn. With this maneuver there was cessation of the air leak and ventilation improved. Gastrostomy for decompression and jejunostomy were performed. Esophagectomy and cervical esophagostomy were not performed due to hemodynamic instability. The patient's ventilatory parameters improved over the following 36 hours, but she remained anuric and septic, and hemodynamically unstable. Due to her instability, further operative management could not be considered. In spite of institution of dialysis (CAVHD) and continued isotropic support, the patient remained overtly septic and hemodynamically unstable. With the development of a disseminated intravascular coagulopathy on postoperative day 4, support was withdrawn at the family's request and the patient died on postoperative day 5.

Discussion Exposure to caustic agents is a common problem affecting several thousands of people annually. The literature shows an evolution from anecdotal considerations to a current body of publications that includes several large series and even prospective studies of acid and alkali ingestion. Even so, prediction of corrosive injury is poorly correlated with the following standard parameters: pH, solid or liquid agent, viscosity of agent, concentration of active ingredients, volume ingested, contact time, time of intervention and premorbid esophageal condition. A study published in 1989 attempted to define the total acidity or alkalinity of solutions commonly ingested by defining the titratable alkaline/acid reserve, or TAR. TAR was defined as the volume of standard 0.1 M solution of HCL or NaOH required to titrate 100 ml of a one per cent solution of the product in question to a pH of 8.00. A relationship between the
TAR of 4 categories of products and esophageal injury in an animal model was verified, and is applied as a measure of corrosive potential.

**Mechanism of injury** The patient presented here ingested an alkaline substance, and the literature indicates that alkali products are ingested more frequently. In a large retrospective study from France, 484 cases were analyzed, with alkali ingestion found in 357 of the 484 cases. Similarly, a large series from India found alkali ingestion more common in adults attempting suicide. The mechanism of injury in alkaline ingestion is a liquefaction necrosis with lipid saponification, dehydration of tissue and propagation of the pathologic process as unattached alkali molecules are carried into the tissues surrounding the esophagus. Venous thrombosis, tissue edema, arterial thrombosis and necrosis ensue. Since commercial preparations are odorless and tasteless, large volumes of 200 to 300 ml are commonly ingested in suicide attempts. The esophagus is severely injured at its areas of narrowing: the cricopharyngeal area, the aortic arch, and the lower esophageal sphincter. Three phases of the pathophysiology of alkaline injury have been defined.

1. Acute inflammatory phase with necrosis and sloughing (one to four days);
2. Subacute phase with sloughing and granulation tissue formation, fibroplasia and collagen deposition (four to fifteen days);
3. Scarring phase with active fibroblast proliferation and transmural fibrous tissue formation (fifteen days to four weeks).

The mechanism of acid injury is more straightforward due to direct tissue damage and lack of propagation of destruction as in alkali injury. Acids produce binding free electrons in tissue proteins with formation of an eschar, stopping acid penetration. As the acid pools in the stomach along the greater curvature, pylorospasm occurs and severe antral damage results. There is direct burn of the tissue, and no liquefaction necrosis. Although the etiology of tissue destruction is different with acids and alkali, the ultimate management is directed at the same principles of caustic substance dilution, rapid assessment via endoscopy, maintenance of NPO status, IV fluids, consideration of antibiotics and steroids, and immediate surgical consultation.

**Staging** The degree of injury as assessed by endoscopy is standardized to an analogue of skin injury where:

- **First degree:** involvement of mucosa only.
- **Second degree:** transmucosal involvement with no extension into periesophageal or perigastric tissue.
- **Third degree:** full-thickness with extension into periesophageal or perigastric tissues with mediastinal or intraperitoneal involvement.

The degree of initial involvement correlates well with the eventual development of stricture and the uncommon, but devastating complication of tracheoesophageal fistula. Early endoscopy to define extent of injury and esophageal stenting for second and selected third-degree involvement is advocated in some series. Initial supportive care and stabilization as well as use of antibiotics, steroids and TPN is appropriate, but aggressive early surgical resection of severe esophagogastric injury is more commonly advocated in the literature. Although use of sucralfate, n-acetylcysteine, lathyrhetic compounds and steroids have shown benefit in vivo and in some studies, they are reserved as adjunct therapies, their main benefit is seen in the reduction of stricture rate in the management of second-degree and early third-degree burns. Early series in the literature dictate diversion of saliva and oral flora from the affected esophageal segment via esophagostomy to allow healing to occur. Later series suggest early esophageal resection is preferable.

**Early management** The early treatment of second and third-degree corrosive esophageal injury involves endoscopy and stenting for the less severe injuries and early surgical intervention in cases of extensive esophagogastric transmural involvement, perforation, mediastinitis, or sepsis. One series even advocates exploration of all circumferential Grade II and Grade III esophageal injury, due to an inability to assess periesophageal tissue involvement. This is suggested because of the ability to examine the serosal surface of the esophagus and the surrounding tissues intraoperatively. Several series also emphasize the need for removal of the affected esophagus before the esophageal necrotic process reaches the vulnerable membranous portion of the trachea. The erosion of the corrosive process into the membranous trachea is uniformly associated with mortality in the literature.
In one large series critically ill patients with corrosive esophagitis and gastric perforation were managed with non-thoracotomy esophagogastrectomy (Orringer procedure) in 7 patients and compared with cervical esophagostomy plus total gastrectomy in 5 patients. Two of the 7 patients managed with nonhoracotomy esophagogastrectomy died, while the group undergoing esophagostomy with total gastrectomy had a one hundred per cent mortality. The cause of death in all patients in the control group was sepsis from the retained necrotic esophagus. These results are consistent with other series.

Early non-surgical management advocated in the literature for non-transmural injury involves esophagoscopy observation in Grade I injury with the addition of esophageal stenting for more serious, but still non-surgical injury. Surgical consultation is obtained in all cases. Stent removal only occurs when barium swallow reveals resolution of injury and visualization of the esophageal lumen around the stent. All patients are managed with NPO status.

Early surgical management centers around three principles: (1) Removal of the ongoing corrosive process before it reaches the mediastinum and trachea; (2) Removal of necrotic tissue; (3) Diversion of the salivary stream from the injured esophagus to prevent infection if the esophageal injury is deemed non-transmural and the esophagus felt to be viable.

Late management The late management of caustic injury to the esophagus centers around management of stricture and tracheoesophageal fistula. If strictures caused by the healing process cannot be managed by endoscopic dilatation, elective esophagogastrectomy may be necessary. The literature discussing the management of acquired non-malignant tracheoesophageal fistula generally deals with fistulae arising from cuffed endotracheal intubation, trauma, granulomatous disease, post-irradiation and iatrogenic surgical injury. Cases of acquired TEF fistula from caustic ingestion are rare and difficult to manage. Intrathoracic tracheoesophageal fistulas are rare and approach is via right thoracotomy with interposition of a pedicled intercostal muscle flap. Bronchial resection with end-to-end anastomosis is reserved for circumferential injury. The body of literature contains no reference to satisfactory management of a long segment tracheobronchial injury as seen in this case.

Summary Caustic esophageal injury is generally accidental in the pediatric population and intentional in the adult. Volume ingested is more for the odorless, tasteless alkaline solutions. Damage to the upper digestive tract is esophageal in alkaline injury and antral in acid injury. Alkaline injury is insidious and ongoing compared to acid injury. Laparotomy for accurate staging is indicated if transmural esophageal injury is suspected or if it cannot be ruled out.

The only possibility of patient salvage in this case of such extensive esophageal injury is esophagogastrectomy before the corrosive process reaches the membranous trachea or mediastinal structures. Late transfer of such a patient prevents the possibility of timely surgical intervention. Successful management of these cases requires stabilization, dilution of the caustic substance to minimize ongoing injury, and immediate transfer to a tertiary care institution for accurate staging and operative intervention.

References
تعرض هذه الدراسة قصة مريضة سعودية، عمرها 23 عامًا، ابتعدت كمية من محلول منظف قلوي. عولجت المريضة بأسلوب محافظ في مستشفى محلي لمدة شهر واحد، وأصيبت في نهاية هذه المدة بنشور رغامي مريثي بسبب لها قصورًا في التنفس مما أوجب إحالتها فورًا إلى مستشفى تخصصي. غير أن شدة الإصابة وتأخر الإحالة جعلا العناية الدقيقة أمرًا مستحيلًا. الغرض من عرض هذه الحالة هو مناقشة التدبير المبكر والتأخير لإصابة المريء الناتجة عن ابتلاع مادة كاوية، والتبني إلى ضرورة التحويل العاجل إلى مستشفى تخصصي بهدف التشخيص الدقيق والعلاج الفوري.