Unusual Presentation and Complication of Caustic Ingestion. Case Report

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Abstract

Caustic substances cause tissue destruction through liquefaction or coagulation reactions and the intensity of destruction depends on the type, concentration, time of contact and amount of the substance ingested. We report an unusual presentation and complication of caustic ingestion in a patient, who accidentally ingested sodium hydroxide. Our patient presented respiratory failure soon after admission and developed necrotizing esophagitis with progression to esophageal stenosis, which required surgical treatment. The complications were related to the amount of caustic soda ingested.

Key words

Caustic soda - necrotizing esophagitis - respiratory failure - caustic stenosis

Introduction

The ingestion of caustic substances is a common condition, which may result in serious injuries of the upper gastrointestinal system and upper airways. Although the incidence of these injuries has declined due to stricter packaging standards, there are still about 5,000 cases affecting children every year in the U.S. In adults, the annual incidence is reported to be 5,000 to 15,000 cases. In the adult population, the injuries are frequently more serious because they are intentional, with larger volumes of ingestion, or with ingestion of industrial toxic compounds. These can result in serious lifelong debilitating conditions such as corrosive esophagitis (CE), esophageal stricture, laryngeal stenosis and later, development of esophageal cancer. Mortality rates after caustic ingestion are reported to be as high as 20% (1,2).

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Caustic soda (NaOH) is a strong base with a very high corrosive potential. In households, caustic soda is used for making soap by an empirical method in many poor regions (3). Since it can easily be confused with water or alcoholic beverages, people may drink it accidentally. Upper respiratory complications of caustic ingestion are uncommon (4). Early signs and symptoms after caustic ingestion are not consistent with the extent of damage, and endoscopy is used to assess injury (5). The first 6-12 hours is the best time to perform an endoscopy (6). Endoscopic ultrasound offers a more accurate evaluation of the depth of the lesions compared to standard endoscopy or computed tomography (7). Medical and surgical treatments are controversial and are centered to prevent esophageal strictures (1,8). We report a case of accidental caustic soda ingestion, developing soon after ingestion respiratory failure (RF) secondary to glottal edema, complicated with acute necrotizing esophagitis.

Case presentation

A 65-year old female presented in E.R. with complete dysphagia, oral pain and odynophagia, dysphonia, chest and epigastric pain, nausea and vomiting, 6 hours after accidental ingestion of 100 ml NaOH, used in households to manufacture soap. She mistook the solution after previously drinking alcohol. At home, she was given 100 ml milk, after which she repeatedly vomited.

The patient had a medical history of depression and chronic alcoholism. Clinical examination showed oropharyngeal burns, lips edema, breath alcohol odor, facial telangiectasias, epigastric pain, anxiety and agitation, blood pressure150/80 mmHg, heart rate 110/min, absence of fever. The patient had an ESR 50 mm and leukocytosis (16,000/mm^3). Glycemia, renal and liver function tests, electrolytes were normal, and alcohol concentration was 150 mg/dl on admission. Other toxicological examinations were negative. Direct X-ray examination of the abdomen and chest were negative. The initial therapy consisted of intravenous fluid administration, proton pump inhibitors (omeprazole 40 mg i.v.), steroids (HHC 150 mg i.v.), antibiotics (ampicillin) and analgesics.

One hour after admission, the patient developed stridor, hoarseness, aphonia, tachypnea (32/min), and cyanosis. Vitals: blood pressure 100/60 mmHg, heart rate 136/min, SaO2 84%, pO2 72 mmHg, pCO2 58 mmHg. RF did not improve after increasing the doses of i.v. steroids, and oxygen therapy. Emergency laryngoscopy revealed upper airway obstruction secondary to pharyngeal, epiglottal and vocal cords edema, and inflammation in glottal area. There was a resolution of RF after tracheostomy. Despite intensive care, in the first days the patient was hemodynamically unstable and required administration of vasoactive agents, thus endoscopic evaluation was delayed. On the 5th day after ingestion, she eliminated a tissue fragment of 18/5 cm, after vomiting (Fig.1). Some tissue fragments were also eliminated in stools. The histological examination of the largest tissue fragment revealed esophageal mucosa with complete necrosis, thickened, edematous submucous area, infiltrated with inflammatory cells. Histological examination of tissue fragments eliminated in stools revealed short fragments of necrotic tissue with isolated granular calcium deposits. On the 7th day after ingestion, the tracheal tube was removed. Dysphagia improved for a period, and reoccurred the 3rd week after admission. Barium esophagogram showed caustic stenosis developed in the distal 2/3 of esophagus (Fig.2), which required surgical therapy (esophagectomy and reconstruction with colonic interposition graft).

Discussion

The ingestion of caustic substances has often devastating consequences on the esophagus and the stomach. Corrosive esophagitis as a result of NaOH ingestion has been known for a long time, but scientific studies were only done between 1960 and 1975 (9). Corrosive esophagitis was

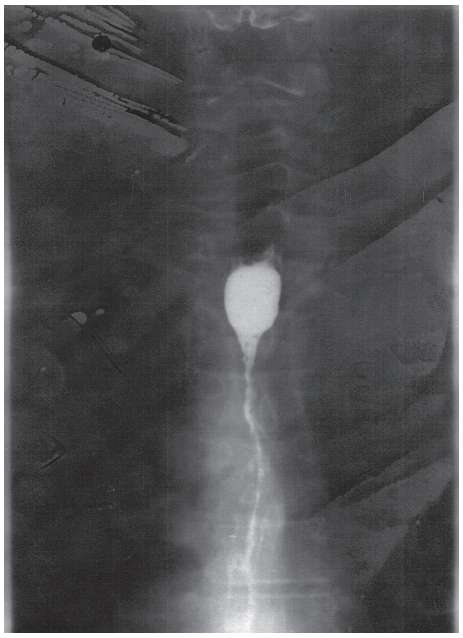

Fig.2 Barium esophagogram reveals caustic stenosis 3 weeks after ingestion.

Fig.1 Fragments of esophageal mucosa eliminate by vomiting.

frequent in Europe, and after the 2nd World War its frequency dramatically increased in France, Germany, Belgium, the Balkan countries and Japan. Medical and social interventions were promptly established, resulting in a considerable decrease in its frequency in Western Europe (3). The severity of the disease and the high cost of treatment made it imperative that NaOH should no longer be sold to the general public. In our region, a 10-year retrospective study on acute poisonings showed a 10.7% prevalence of caustic poisoning in adults (10).

Corrosive esophagitis evolves in three phases: acute esophagitis (Table I), a quiet phase and sequelae (weeks or months after the onset of the disease) (3). Morphologic features in caustic injury are presented in Table II. On average, 50 ml of concentrated liquid are sufficient to cause extremely severe injuries, while 15-30 ml cause severe lesions, and less than 15 ml cause lesions of medium intensity (11). Our patient ingested 100 ml of NaOH and experienced a low flow state in the first days after admission, which required a vasoactive support, which can explain the severity of esophagitis (necrotizing esophagitis, NE).

Necrotizing esophagitis is a rare clinical entity with high mortality (overall rates 50%). Causes include ischemia (especially in elderly with cardiac and vascular disease which experience a low flow state), trauma, caustic ingestion, radiation, and infection (12-15). In a retrospective review comparing surgical versus medical management, mortality was 90% in the medically managed group versus 27% in the surgical group (13).

Necrotizing esophagitis is often discovered on upper endoscopy, and has been described as “black esophagus” (16). Conservative management of NE with antibiotics, fluid and nutritional support and proton pump inhibitors is often effective. Surgical intervention is required when patients fail this therapy. Transmural involvement is also exceedingly rare, with most patients presenting mucosal and submucosal involvement. Frank perforation is an atypical presentation when esophagitis is discovered on endoscopy (12,14).

Studies carried out in Turkey (17) and Denmark (18) have identified a high incidence of esophageal stenosis (72.7%

Table I Endoscopic grading and prognosis of caustic lesions (adapted from 31-33)

Lesion grading	Endoscopic features	Prognosis
Grade I non-ulcerative esophagitis	Mild erythema Edema of the mucosa	Recovery without sequelae
Grade II a	White exudates (patchy or linear) Hemorrhages Erosions, blisters Superficial ulcer Erythema in the area	Recovery without sequelae
Grade II b	Circumferential lesions Ulceration may be apparent, and may extend into the muscle layers	Recovery with sequelae (strictures)
Grade III a	Mucosa appears very dusky, very black Deep ulcerations (transmural tissue is involved) The lumen may be completely obliterated	Recovery with sequelae (strictures)
Grade III b	Multiple deep brownish-black or gray ulcers. Extended necrosis.	Recovery with sequelae
Grade IV	Perforation	Strictures in survivors

and 85%, respectively), among patients who ingested caustic agents. In contrast, the incidence reported in Finland (19) was extremely low, since the authors stated that commerce of NaOH was prohibited in Finland in 1966. The physical status of the caustic agent influences the severity of the lesions in the human esophagus. In our report, the patient had ingested NaOH in the liquid form, after alcohol ingestion.

Soda in the solid form is less aggressive to the esophagus because it sticks to the oral mucosa, where it produces deep lesions (20). The liquid form in concentrations < 10% only causes esophageal stenosis whereas the concentrated liquid form can provoke more lesions in the stomach than the solid form, as well as more severe perforations and stenosis in the esophagus (21). In a study of 202 children, the upper third of the esophagus was involved in 40.6% of cases, followed by the middle third in 23.8%, by the lower third in 23.3%, and the entire esophagus in 12.4% of cases (17). The incidence of lesions of the upper esophageal third was lower in other studies (22). The stenosis occurring in the middle third is due to failure of microcirculation at this level; therefore this is the preferred site for stenosis (23). Our patient developed CS in the middle and lower third of the esophagus, which required surgical treatment.

Table II Morphologic aspects in caustic injury (adapted from 22)

First hours after ingestion	eosinophilic necrosis edema intense hemorrhagic congestion
First days	ulcerations covered with a leukocytic fibrinous layer perforation when ulceration exceeds the muscle plane
4^{th}-5^{th} day after ingestion	fibroblasts reach the site of injury a mold of the lesion is formed, consisting of dead cells, secretions and food remains
10^{th} day - first weeks	repair begins edema and lymphatic ectasia persist in the submucosa sclerosis sets in at the muscle level the autonomic nervous plexus is destroyed fibrosis occurs in layers whose depth depends on the severity of the caustic injury
First month	difficult epithelization of the mucosal ulcerations due to the vascular lesions
The rest of life	new ulcerations followed by re-epithelization due to small traumas provoked by food these traumas increase the scars, reducing even more the lumen of the organ

Females develop more frequently CS because they may be more sensitive to caustic agents or they ingest a larger amount of caustic substance than males. The presence of stenosis of the esophagus is directly related to the amount of caustic agent ingested. With ingestion of two or three tablespoonfuls, the risk of fistulas, perforations or even death is increased (22). The frequency of stenosis has been decreasing over the last few years, because of the changing of home products composition and the introduction of containers that are difficult to handle by children (24-26). Drinking an antidote, passing a naso-gastric tube, and corticoid or antibiotic treatment showed no effect on CS incidence (22).

Vomiting might be a factor contributing to the aggressiveness of injury (27). A 6.9% incidence of complications was observed among patients who vomited during the acute phase, whereas no complications were observed among those who did not vomit (22). Repeated vomiting might have contributed to the severity of complications in our case.

Respiratory complications of caustic ingestions have been described, but usually in the setting of the individual

case report (28-30). Our patient developed soon after admission RF secondary to upper airway obstruction, probably due to aspiration during vomiting. Aspiration of the ingested substance could further injure the tracheobronchial tree, therefore initial evaluation after caustic ingestion should include flexible bronchoscopy, in addition to endoscopy. Any patient presenting respiratory symptoms should be nursed in a high dependency setting with access to advanced airway support at least in the initial stage of their presentation.

Current popular belief is that caustic substances do not kill, which is not true, but when death does not occur, stenosis of the esophagus will inevitably develop, causing the patients to depend on dilatation, surgery, or progression to cancer.

Conclusion

Accidental caustic ingestion may have an unusual presentation and severe complications, but a favorable outcome. The complications are related to the amount of caustic agent and repetitive vomiting after ingestion, and are represented by acute respiratory failure, acute necrotizing esophagitis and stenosis of esophagus. Guidance and education are important preventive tools, but the best approach is to restrict access to caustic agents, by prohibiting their free commercialization.

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