

CORROSIVE ESOPHAGOGASTRODUODENITIS FOLLOWING ACETIC ACID INGESTIONS IN CHILDREN: A CASE REPORT

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Abstract

Introduction Corrosive esophagogastrroduodenitis is caustic damage due to ingestion of chemical agents and is a serious medical and social issue, with eighty percent of corrosive injuries occurring in children. Corrosive substances, also described as caustic substances, consist of alkali and acidic agents. Acetic acid (C₂H₄O₂) is a caustic that has acidic properties and widely used in Indonesia as a stimulation of sap production as a form of wood vinegar that used as a stimulant to increase pine sap flow.

Case A 15-year-old girl came to the emergency department with profuse nausea and vomiting after ingestion of acetic acid 3 days ago. She vomited more than 3 times a day, the contents were yellowish liquid without blood. Afterward, the patient felt burning pain in her mouth and throat. She progressively had trouble eating and drinking because of pain and difficulty swallowing. Gastroscopy examination showed erythema ulcer and erosion on the lingual, hypopharynx, esophagus, gaster and duodenum. Patient was diagnosed with dysphagia and odynophagia due to corrosive agent + Esophagoduodenitis and then was treated with Nasogastric tube (NGT), Kaen 3b, Paracetamol injection as analgetic, Dexamethasone injection, Omeprazole injection, Rebamipide, Sucralfate, Nystatin, Diet 50-100 cc/ 3 hour orally via NGT.

Conclusion The ingestion of acetic acid remains widespread in many developing countries. Individuals who consume acetic acid may be asymptomatic or display a variety of initial signs and symptoms. Endoscopy is acknowledged as the gold standard for evaluating both the location and severity of injuries caused by corrosive substances.

Keywords: Esophagogastrroduodenitis ; Corrosive; Caustic; Acetic Acid

1.INTRODUCTION

Corrosive esophagogastrroduodenitis is caustic damage due to ingestion of chemical agents and is a serious medical and social issue, with eighty percent of the corrosive injuries occurring in children. Corrosive substance ingestion may happen accidentally in children who have started to walk or crawl or due to suicidal intention and Destruction of tissue will result in airway, esophagus, or stomach injury, which often lead to death or other adverse effects on the gastrointestinal and respiratory tracts [1]. Corrosive substances, also described as caustic substances, consist of alkali and acidic agents, although some literature implies caustic substances only as alkaline substances. Both alkali and acidic agents can cause organ damage upon direct contact with the internal and external organs of the body [2]. While alkali agents cause damage due to liquefaction necrosis leading to deeper injuries, acidic agents cause damage due to coagulation necrosis which

limits the depth of the injury. However, concentrated alkali and acidic agents will lead to similar pathological changes [3].

Acetic acid ($C_2H_4O_2$) is an organic compound that has acidic properties and is known as the main component in vinegar. It is also widely used in Indonesia as a stimulation of sap production as a form of wood vinegar that used as a stimulant to increase pine sap flow [4,5]. In this case report, we describe an 15-year-old girl who had esophagogastrroduodenitis following accidental acetic acid ingestions, to increase awareness of this serious long term consequence but preventable of acetic acid ingestions.

2.CASE REPORT

A 15-year-old girl came to the emergency department with profuse nausea and vomiting after ingestion of acetic acid 3 days ago. She vomited more than 3 times a day, the contents were yellowish liquid without blood. According to her parents, acetic acid is used to spray pine trees. However, the patient accidentally poured acid on her hand causing a stinging and burning sensation thus making the patient spontaneously suck on the spilled acid on her hand. Afterward, the patient felt burning pain in her mouth and throat. She progressively had trouble eating and drinking because of pain and difficulty swallowing. Bowel movement and urination were within normal limit. The patient has no remarkable previous medical history nor any medication. No fever was noted.

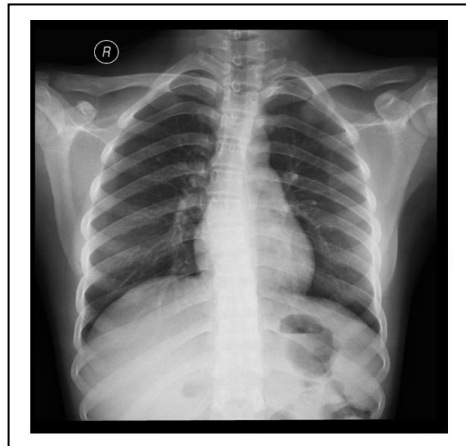
At the time of admission, clinical examination revealed patient's sensorium as alert (Glasgow Coma Scale 15 (Eye 4, Verbal 5, Motor 6)), blood pressure 99/58 mmHg, heart rate 100 times/minute, regular pattern, respiratory rate 23 times/minute with regular pattern, Oxygen Saturation (SpO_2) 98% on room air, axillary temperature $37.2^\circ C$, Visual Analogue Scale (VAS) was 5. Body Weight was 45.2 kg. Body Height was 145 cm. There were no abnormalities observed in head, eyes, ears, nose, and neck examination. Oral Examination shows reddening and swelling of the mucosa of the lips and tongue, and a hint of whitish membrane on the tongue suggestive of candida infestation (**Picture 1**). No murmur, rhonchi, and wheezing were found. Bleeding in the mouth may sometimes be found.



Picture 1. Patient's Mouth shows hyperemia and swelling on the lips and whitish membrane on top of tongue suggesting suspected candida infestation

Laboratory Investigation revealed normal hemoglobin, white blood cells and Platelets (12.1 g/dl , $8.69 \times 10^3/\text{cu mm}$, and $362 \times 10^3/\text{cu mm}$). Slight Increase in neutrophil: 72.2% (reference range: 50-70%), decrease on lymphocyte 10.6% (reference range: 20-40%), high monocyte at 10.1% (reference range: 0.0-7.0%). Urea was increased at 51.0 mg/dl (reference range: 18.0-45.0) and creatinine was normal at 0.84 mg/dl (reference

range: 0,50-1.20). There is no marked finding on liver enzyme. No abnormalities were found in electrolytes result. Chest X-ray showed no abnormalities (**Picture 2**).

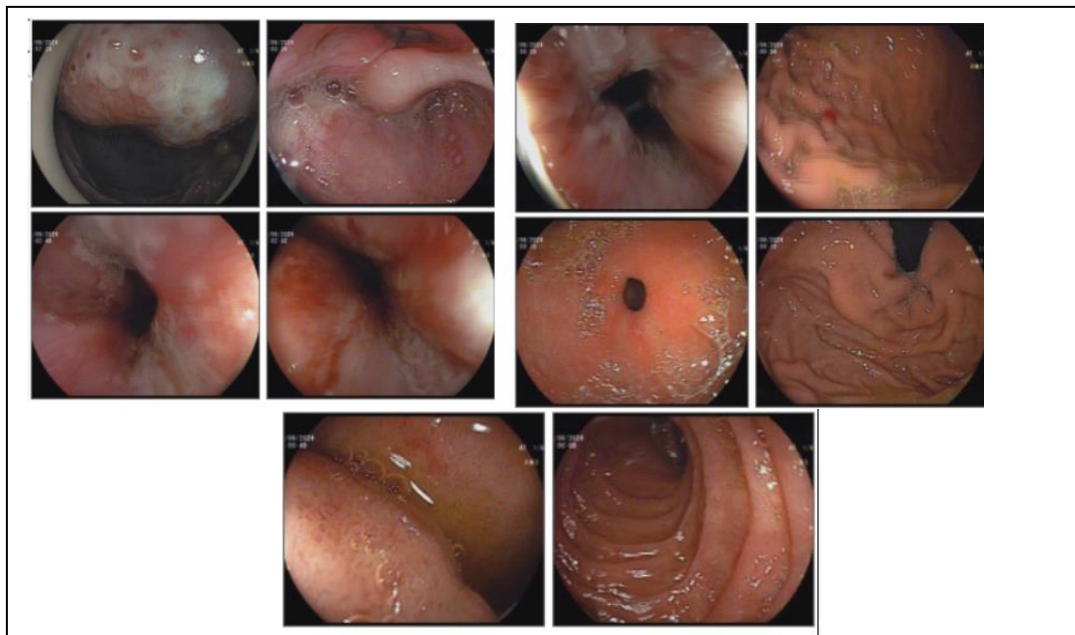


Picture 2. Thorax Xray

Esophagogastroduodenoscopy examination was done to the patient (**picture 3**):

1. The lingual and hypopharynx was found to have multiple ulcers and hyperemic arytenoid edema
2. Esophagus: there are multiple ulcers, erosions and hyperemia can be found on proximal, middle and distal esophageal mucosa. Mucosal breaks >5mm at the esophagogastric junction (EGJ) around 36cm
3. Stomach: the mucosa of the corpus and the antrum show hyperemia and multiple erosions. Cardia and fundus are normal. The pyloric ring was normal. No pyloric gaping. Intact lower esophageal sphincter.
4. Duodenum: bulb mucosa hyperemic (+), multiple erosions.

Current gastroscopy examination found no stenosis or esophageal stricture but there is potential for future stricture. There is adhesion near the upper esophageal sphincter.



Picture 3. Esophagogastroduodenoscopy shows hyperemic, ulcer and multiple erosion on multiple sites

The patient was then diagnosed with dysphagia and odynophagia due to corrosive agent + Esophagoduodenitis. Patient was treated with Nasogastric tube (NGT) insertion for 2-4 weeks, Kaen 3b fluid 2000cc daily, Paracetamol injection 500 mg/6 hour as analgetic, Dexamethasone injection 5 mg/6 hour, Omeprazole injection 40 mg/12 hour, Rebamipide 3 times a day, Sucralfate 3x5 ml a day, Nystatin 4x1 ml a day, Diet 50-100 cc/ 3 hour orally via NGT.

3.DISCUSSION

Eighty percent of cases involving the ingestion of corrosive substances occur in children, with a significant portion of these incidents taking place in developing nations indonesia. Accidental ingestion is particularly common among children under five, while intentional ingestion is more frequently seen in adolescents. The severity of the injury depends on the type of corrosive substance (alkali or acid), its pH level, the amount ingested, and the site of exposures. Acute ingestion can cause damage to the skin, respiratory tract, or upper gastrointestinal system, ranging from minor to life-threatening complications [6].

Caustic substances inflict both functional and histological damage upon contact with body surfaces. Numerous household and industrial chemicals possess caustic properties. These substances are generally categorized acids (with a pH less than 7) and into alkalis (with a pH greater than 7) [7]. Acids are powerful desiccants that can lead to coagulation necrosis and the formation of eschar in damaged tissues, while alkalis result in liquefaction necrosis. Acids can be divided into two groups: (1) Strong acids, such as mineral or inorganic acids like sulfuric, nitric, and hydrochloric acid, as well as organic acids like carbolic, oxalic, acetic, and salicylic acid; and (2) Strong alkalis, including hydrates and carbonates of sodium, potassium, and ammonia [8]. In this case report, the caustic substance ingested by the patient was acetic acid, which falls under the category of strong acids.

Caustic ingestions can lead to various signs and symptoms, including nausea and vomiting, drooling, oral burns, difficulty swallowing, dyspnea, abdominal pain, hematemesis, and even stridor. Approximately twenty percent of patients may develop esophageal strictures. The absence of lesions in the oropharynx does not rule out the possibility of significant injury to the esophagus or stomach, which could result in perforation or stricture [9]. Upper gastrointestinal strictures are a frequent long-term complication of severe corrosive injuries, typically occurring three weeks after ingestion. Other complications associated with corrosive ingestion that contribute to increased morbidity, alongside stricture formation, include gastroesophageal reflux disease (GERD), dysmotility, and an elevated risk of neoplasms [6].

In acute caustic ingestion, esophagogastroduodenoscopy (EGD) is the gold standard for assessing the severity and location of caustic damage to the esophagus, gaster, and duodenum. Esophageal injury is graded using Zargar classification [8,10]. Patients who intentionally ingest caustic substances should receive early endoscopy, as the cases associated with suicidal intent have the highest likelihood of resulting in significant clinical injuries. In contrast, for unintentional ingestions, especially in children, the choice to conduct endoscopy is not as straightforward [8].

Gupta et al. state that all patients with clinically significant injuries (grades 2 and 3 according to the Zargar classification) should exhibit symptoms during the initial evaluation. No individual symptom or combination

of symptoms can reliably identify all patients with esophageal injuries. Those who are asymptomatic typically show normal results on endoscopic examinations. Therefore, EGD may not be necessary for asymptomatic patients suspected of caustic ingestion, although further large-scale prospective studies are needed to definitively address this important clinical issue [11]. Some literature suggests that endoscopy is most effective when performed within the first 48 to 72 hours following corrosive ingestion, after the patient has been stabilized. Beyond 72 hours, the affected areas may become soft, swollen, and fragile, increasing the risk of perforation during EGD. When performing EGD, it should be done gently, ideally using a thin (5.5 mm) endoscope, with minimal air insufflation and appropriate sedation [12].

The primary focus in managing corrosive injury is hemodynamic stabilization and ensuring the patient's airway is secure, which may require the establishment of a surgical airway. Induced vomiting and gastric lavage are contraindicated because they pose a risk of re-exposing the patient to the corrosive substance, potentially causing additional harm to the esophagus. The effectiveness of milk and water as antidotes or diluents for corrosive agents has not been proven. Acid suppression is a frequently employed treatment for corrosive injuries. It is thought to promote healing of the esophageal mucosa and reduce the risk of stress ulcers. However, evidence supporting this practice is limited; only one small uncontrolled study has demonstrated endoscopic healing after omeprazole infusion in such cases [13,14].

Antibiotics are generally not prescribed for corrosive ingestion injuries classified as grade 1 and 2a. However, for injuries rated higher than grade 2b, antibiotic therapy may be warranted due to the risk of infection from oral microbiota. The role of corticosteroids in this context remains debated; both a meta-analysis of studies conducted from 1991 to 2004 and a broader review covering literature from 1956 to 2006 found no significant advantage of steroid use in preventing strictures. Typically, steroids are reserved for patients exhibiting airway-related symptoms [15, 16].

4.CONCLUSION

The ingestion of acetic acid represents a straightforward and preventable issue; however, it remains prevalent in many developing countries. Individuals who have ingested acetic acid may either remain asymptomatic or exhibit a range of initial signs and symptoms. These can include difficulty swallowing saliva, and reluctance to eat, progressing to severe systemic manifestations that could potentially result in death. Endoscopy is recognized as the gold standard for assessing both the location and severity of injuries resulting from corrosive ingestion. Effective, early management and recognition of the extent of injury is required to prevent deterioration of the patient's condition since endoscopy is best performed within 72 hours.

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