Gastric Erosion due to Hydrochloric Acid Ingestion

Ni Made Savitri Devi
General Practitioner, Wanggaya General Hospital, Denpasar, Bali, Indonesia

Abstract: Female, 39 years old, Javanese came with nausea and vomiting every ingestion any food or water. It is found that there is no previous history taking of NSAIDs. However, she has history drink floor cleaning agents 5 years ago. She was in abdominal discomfort since 5 years ago and worsen since 3 months prior admission to hospital. She also complained feel fullness on her stomach every ingestion any food or water. Her esofagogastroduodenoscopy revealed erosions in esophagus and gaster. She was in restoration therapy using PPI and showing good outcome.

Keywords: Esophagus Erosion, Gaster Erosion

1. Background

Chemical injury of upper gastrointestinal tract may cause by acute poisonings with hydrochloric acid, the most common location being the esophagus and the stomach. If the patient survives the acute phase of the poisoning, regenerative response may result in esophageal and/or gastric erosion or stenosis and increased risk for esophageal cancer. Accidental ingestion, particularly in children, due to careless storing of chemicals and ingestion with suicidal intent and due to free availability of the caustic agents contribute to their occurrence. Establishing the diagnosis of acute corrosive poisonings, the severity of the post-corrosive endoscopic changes of the esophagus, stomach and duodenum is of major importance.1,2

Gastric erosions have been defined as endoscopically detectable mucosal breaks that do not penetrate the muscularis mucosae. The duration of erosion can be short-term, chronic or recurrent. An erosion is formed either as a consequence of epithelial cell death or of epithelial detachment, resulting in loss of epithelial cells that exceeds the epithelial regeneration capacity. Secondly, the loss of epithelial cells evokes luminal factors e.g. gastric acid release which can start to destroy subepithelial structures. This kind of loss of mucosal integrity can be induced by several chemicals, diseases and mechanical or physical factors.3,4,5

2. Case Presentation

Female, 39 years old, Javanese, came with chief complain had nausea and vomiting every ingestion any food or water. She complained felt fullness on her stomach and also felt pain on her epigastric site. The pain presented in the case of an empty stomach or after meal and sometimes at night. The patient has felt nausea and abdominal discomfort since 5 years ago and worsen since 3 months ago. Patient confessed there was history of drinking floor cleaning agents 5 years ago and after that patient said felt uncomfortable in her stomach every eat, so that we also find decreased of body weight because of difficulty to eat. There was no any alcohol and smoking history; nor previous illness such as diabetes mellitus and cardiovascular diseases not reported.

On physical examination, it was found that his blood pressure was normal: 110/70 mmHg, pulse rate 84 times/minute, respiratory rate 20x/minute. Tenderness on palpation of epigastrium. Laboratory result showed a normal on cell blood count: WBC: 9.12x 10³, Hemoglobin: 11.8 gr/dl, Platelet: 325x10³. Liver function test and kidney function test were also normal. But, there is a slight decrease on electrolyte count: Na: 131 mmol/L, K: 3 mmol/L, Chlor: 88 mmol/L. The patient was diagnosed with dyspepsia fungsional due to suspect gastric erosive. The patient was then admitted to ward and giving infusion normal saline, proton pump inhibitor esomeprazole bolus 40 mg intravenous and continuous with 40 mg/24 hours, antiemetic ondansetron 4 mg intravenous every 8 hours and also planned for esofagogastroduodenoscopy to assess the extent of the upper gastrointestinal damage.

Figure 1: Erosion on the esophagus

Figure 2: Erosion on the gaster
3. Discussion

Chemical injury of upper gastrointestinal tract may cause by acute poisonings with hydrochloric acid, the most common location being the esophagus and the stomach. If the patient survives the acute phase of the poisoning, regenerative response may result in esophageal and/or gastric erosion or stenosis and increased risk for esophageal cancer. Accidental ingestion, particularly in children, due to careless storing of chemicals and ingestion with suicidal intent and due to free availability of the caustic agents contribute to their occurrence. Establishing the diagnosis of acute corrosive poisonings, the severity of the post-corrosive endoscopic changes of the esophagus, stomach and duodenum is of major importance.\textsuperscript{1,2}

Hydrochloric acid has many uses. It is used in the production of chlorides, fertilizers, and dyes, in electroplating, and in the photographic, textile, and rubber industries. Hydrochloric acid is corrosive to the eyes, skin, and mucous membranes. Acute (short-term) inhalation exposure may cause eye, nose, and respiratory tract irritation and inflammation and pulmonary edema in humans. Acute oral exposure may cause corrosion of the mucous membranes, esophagus, and stomach and dernal contact may produce severe burns, ulceration, and scarring in humans. Chronic (long-term) occupational exposure to hydrochloric acid has been reported to cause gastritis, chronic bronchitis, dermatitis, and photosensitization in workers. Prolonged exposure to low concentrations may also cause dental discoloration and erosion.\textsuperscript{6,7}

Ingestion of concentrated hydrochloric acid at concentrations sufficient to produce mucosal irritation, can cause severe corrosive injury to lips, mouth, throat, oesophagus and stomach, pain, irritation, dysphagia, nausea, vomiting, thirst, salivaition, chills, fever, shock and renal failure as well as burns, ulceration and perforation of the gastrointestinal tract. Peritonitis or esophageal, gastric or pyloric strictures may also occur.\textsuperscript{8,9}

Gastric erosion was defined as a superficial defect in the mucosa that did not penetrate through the muscularis mucosae layer. The duration of erosion can be short-term, chronic or recurrent. The usual finding is a white base of erosion, although occasionally a blackened base may be seen as a mark of recent haemorrhage; the lesions are flat or minimally depressed and usually are surrounded by a narrow rim of erythema. Erosion has also been described as a defect in the mucosa with a necrotic base that is less than 3–5 mm in diameter. This kind of loss of mucosal integrity can be caused by several chemicals, diseases and mechanical or physical factors.\textsuperscript{3,4,5}

The pathogenesis of gastric erosion is multifactorial, so too the etiology also multifactorial. Erosions are a result of an imbalance between protective and destructive factors. The factors that participate in the pathogenesis of gastric ulcer can be divided into luminal, epithelial cell, apoptotic and regenerative factors. \textit{Luminal factors.} Gastric acid is most important luminal factor. It is well known that there is hyperacidity in stress ulcers. Secondly, there is elevated pepsin formation. Thirdly, increased retrograde peristaltic waves with low pyloric tone and reduced local motility can lead to more intensified biliary and pancreatic duodenogastric reflux. In erosions patients, the mucoprotective index (the ratio of neutral to total mucoproteins) has been reported to be decreased, and weakening of mucosal defences has been speculated to be a major factor in the pathogenesis of gastric erosions.

\textit{Epithelial cells and mucus.} Protection against gastric acid is provided by epithelial cells which secrete mucus and bicarbonate. Mucosal protection becomes weakened when there is a decline in mucus secretion, mucosal blood flow, the synthesis of DNA and prostaglandins. This changes the mucus composition and low molecular glycoprotein production increases, leading to conditions favouring proteolysis and rediffusion of H+ -ion, which then enhance ulcer formation.\textsuperscript{10,11,12}

Increased apoptosis leading to loss of mucosal integrity is one factor in the development of ulcers. When reperfusion takes place, then neutrophils can produce toxic oxygen free radicals. \textit{Regeneration.} Ulcer associated cell lineage (UACL) has been found at sites of chronic gastric ulcers. Gastric stem cells induce UACL and this assists in the healing of the ulcer. This involves coordinated localisation of mucins and trefoil peptides, which are potent mitogens of GI epithelium in conjunction with epidermal growth factor. In addition, healing can be assisted also by an increased mucosal blood flow. The classification is summarized in Table 1. Gastric erosions are divided into three categories: complete erosion, incomplete erosion and hemorrhagic-erosive gastritis.\textsuperscript{11,16}
In this case, the cause of gastric erosion is due to hydrochloric acid ingestion. No history of NSAIDs usage and from esofagogastroduodenoscopy result showed pin point sized hemorrhages on the mucosal surface of the esophagus and also the gaster.

Management of this case was to reduce the stomach acid and prevent vomiting so that patients can get the nutrients from the food that she ate. To reduce the acidic of gaster, we use PPI and to prevent vomiting we use antiemetic.

This case report presented a 39 years old female patient was treated with PPI and antiemetic for 7 days treatment, and the result showed good outcome.

4. Conclusion

This case report presented a 39 years old female with nausea and vomiting every ingestion any food or water and tenderness on palpation of epigastrium. Patient was has history of drink floor cleaning agents that contains hydrochloric acid. Esofagogastroduodenoscopy result was showed erosions on the esophagus and also gaster. The patient was treated with double drug combination, PPIand antiemetic for 7 days treatments, and the result showed good outcome.

References


