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Gastric Erosion due to Hydrochloric Acid Ingestion

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Abstract: ***Female, 39 years old, Javanese came with nausea and vomitting 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 afterthat 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 historynorprevious 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 10^3, Hemoglobin: 11.8 gr/dl, Platelet: 325x10^3. 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 ondansentron 4 mg intravenous every 8 hours and also planned for esofagogastroduodenoscopy to assess the extent of the upper gastrointestinal damage.

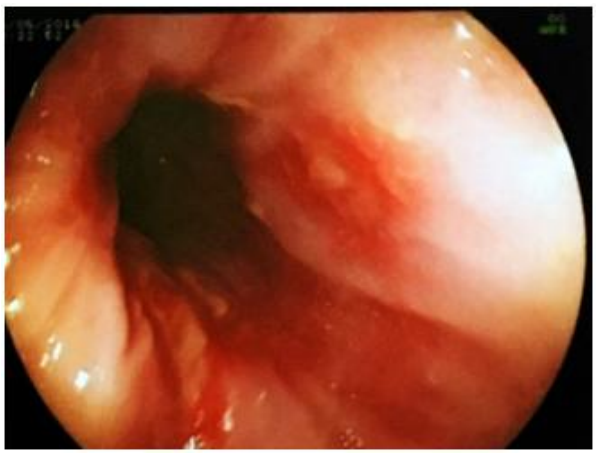

Figure 1: Erosion on the esophagus

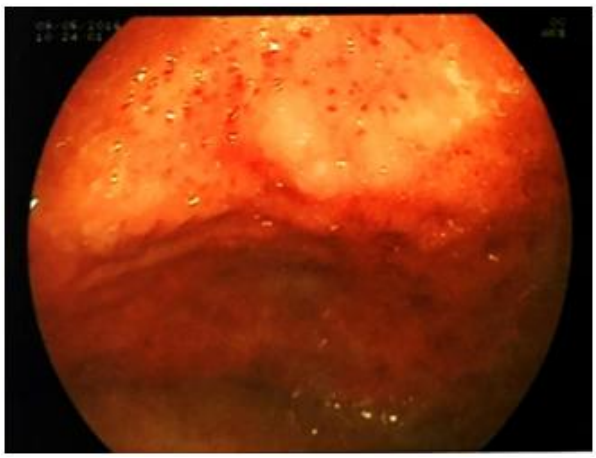

Figure 2: Erosion on the gaster

Esofagogastroduodenoscopy result was showed erosions on the esophagus and also gaster. So the diagnosis was erosions on the esophagus and gaster due to floor cleaning agents ingestion 5 years ago.

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.[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 dermal 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.[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, salivation, chills, fever, shock and renal failure as well as burns, ulceration and perforation of the gastrointestinal tract. Peritonitis or oesophageal, gastric or pyloric strictures may also occur.[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 induced by several chemicals, diseases and mechanical or physical factors.[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.*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.[10, 11, 12]

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.[13, 14, 15]

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. *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.[11, 16]

Table 1. Endoscopical classification of gastric erosions and gastric conditions with erosions as a predominant change.

Main class	Subclass	Characteristic features	Publication
I Complete		Innumerable, pinpoint-sized hemorrhages on the mucosal surface	Roesch & Ottenjann (1970)
	Ia Mature type	The surrounding mucosal elevation is irreversible due to fibrosis	Kawai *et al.* (1970)
	Ib Immature type	The bulging border is due to oedema	Kawai *et al.* (1970)
II Incomplete		A simple defect of the mucosal layer without reaction to surroundings	Roesch & Ottenjann (1970)
	IIa	Erosion located on flat mucosa	Karvonen *et al.* (1983)
	IIb	Erosion located on the prominent folds of the prepyloric region	Karvonen *et al.* (1983)
III Haemorrhagic-erosive gastritis		Innumerable, pinpoint-sized hemorrhages on the mucosal surface with erythrodiapedesis and engorged blood vessels within mucosa and submucosa	Roesch & Ottenjann (1970)
Erosive prepyloric changes (EPC)	EPC grade 1	Standing mucosal prepyloric fold, independent of peristalsis and running transverse or parallel into relation of antral lumen	Nesland & Berstad (1985)
	EPC grade 2	EPC grade 1 plus red spots and/or streaks situated on the top of the folds	Nesland & Berstad (1985)
	EPC grade 3 (equals Incomplete type IIb erosions)	EPC grade 2 plus the presence of macroscopic erosions	Nesland & Berstad (1985)

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 acid of gaster, we use PPI and to prevent vomitting we use antiemetic. Work mechanism PPI is blocking the action of enzyme K^+H^+ATPase (proton pump) which will break up K^+H^+ATP produces energy which is used to remove HCl acid of the cell canal parietal into the lumen of the stomach. PPI prevents acid expenditure the stomach of the canal cell, causing pain reduction ulcer patients, reducing the activity of aggressive factors pepsin with pH> 4.The antiemetic agents work within a neuronal region to counteract the complex act of vomiting through interactions with cranial nerves and neural networks. Each of the individual agents has varying mechanisms of action. Antihistamines work by directly inhibiting histamine at the histamine1-receptor and indirectly inhibiting histamine in the vestibular system. These two actions combine to produce decreased stimulation in the vomiting center.[17, 18, 19]

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 treatment, and the result showed good outcome.

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