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Helicobacter Pylori Infection as the Cause of Giant Ulcer in Anthrum Gaster

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Abstract: ***Male, 70 years old, Balinese came with hematemesis. It is found that there is no previous history taking of NSAIDs. However, he was in abdominal discomfort since 2 month prior admission to hospital. His esofagogastroduodenoscopy revealed giant ulcer in anthrumgaster. Histopathologic examination revealed a spherical and rod like organism and limphoplasmacytic, polymophonuclear cell appearance, specific for Helicobacter pylori gastritis. He was in eradication therapy using triple drug regiment for 10 days showing a good outcome.***

Keyword: Helicobacter Pylori, Giant ulcer

1. Background

Helicobacter pylori (Hp) is a gram-negative spiral bacterium whose ecological niche is the human stomach. Hp gastritis is etiologically associated with peptic ulcer, primary gastric B-cell lymphoma and gastric carcinoma. Despite a general decline in the incidence of gastric cancer, it remains the fourth most common cancer and second leading cause of cancer-related deaths worldwide. Hp colonizes the gastric mucosa of approximately one-half of the world population and estimated 30% to 40% of the U.S. population. Hp is present in 95% of patients with duodenal ulcers and in 70% of those with gastric ulcers.[1,2] The most common causes of peptic ulcer disease (PUD) are Hp infection and use of nonsteroidal anti-inflammatory drugs (NSAIDs). We report one case gastric ulcer due to Hp infection.

2. Case Presentation

Male, 70 years old, Balinese, came with chief complaint of hematemesis since one day prior to admission. These were also accompanied by pain in the centre of stomach. 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 2 month ago. There was no decreased of body weight and using analgeticor NSAIDs. We also did not find 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: 120/80 mmHg, pulse rate 98 times/ minute, respiratori rate 20x/minute. Tenderness on palpation of epigastrium. Laboratory result showed a normal on cell blood count: WBC: 9x 10^3, Hemoglobin: 12 gr/dl, platelet: 256x10^3. Liver function test and kidney function test were also normal. The patient diagnosed with hematemesis suspect peptic ulcer. The patient was then admitted to ward and giving infusion ringer lactat, proton pump inhibitor esomeprazole bolus 40 mg intravenous and continuous with 8 mg/ hours and planned for esofagogastroduodenoscopy to evaluate the source of hematemesis.

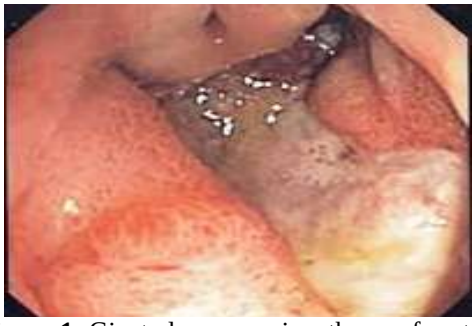

Figure 1: Giant ulcer on regioanthrum of gaster

Esofagosatroduodenoscopy result: esophagus was normal, there was a giant ulcer on gaster at the anthrum region. Duodenum was normal. So the diagnosis was giant ulcer on regioanthrum. Biopsy done in the anthrum area, and also corpus area. The specimen evaluated by Pathology Anatomy department. Biopsy result shown: discontinuity of superficial epithel mucosa, a lot of polimorfonuclear and limfoplasmacytic cells. Also found spherical and rod like organism. Inferred: Gastric ulcer and found Helicobacter pylori like organism.

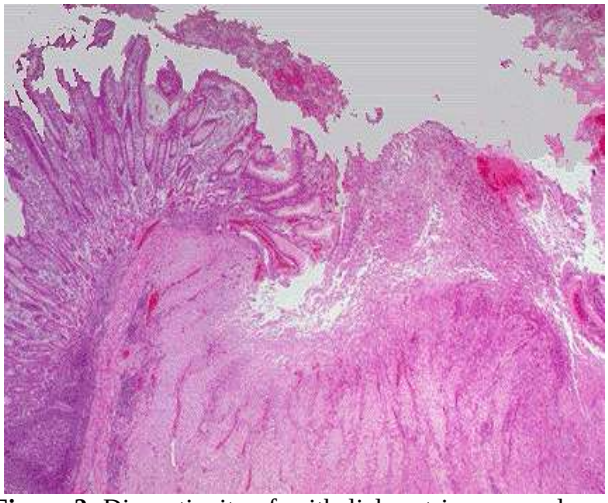

Figure 2: Discontinuity of epithelial gastric mucoca layer. Limfoplasmacytic and polimorphonuclear cells and also found spherical or rod like organism.

3. Discussion

Helicobacter pylori (Hp) initially named *Campylobacter pyloridis*, was first identified in humans and cultured by Marshall and Warren. Hp infection is highly associated with gastrointestinal diseases, including gastric inflammation, peptic ulcer, gastric cancer, and gastric mucosa-associated lymphoid-tissue lymphoma. It has been classified as a group 1 carcinogen (infection with Hp is carcinogenic in humans) by the International Agency for Research on Cancer consensus group since 1994.[1,2,3]

The exact routes of Hp transmission remain unclear. However, epidemiologic studies have shown that exposure of food to contaminated water or soil may increase the risk of Hp infection, suggesting that person-to-person transmission by oral-oral, fecal-oral, or gastro-oral exposure is the most likely path for Hp infection. it is typically transmitted via the fecal-oral route during early childhood and persists for decades. The bacterium is a known cause of gastric and duodenal ulcers. Hp colonization itself is not a disease, but an infection can lead to various clinical disorders in the upper gastrointestinal tract. In most cases Hp colonization induces histological gastritis, but pronounced clinical signs seldom develop. It is estimated that Hp positive patients have a 10% to 20% lifetime risk of developing ulcer disease and a 1% to 2% risk of developing distal gastric cancer. Hp bacteria mainly adhere to gastric epithelial cells and release cytotoxins causing duodenal ulcer. Several infection-associated factors of Hp, such as urease, catalase, lipase, adhesion molecules, cytotoxin-associated gene protein (CagA), a homologue of the Bordetella pertussis toxin secretion protein and vacuolatingcytotoxin (VacA), contribute to gastric ulceration.[4,5,6]

A common causative factor for gastric ulceration is an invasion of Hp a micro-aerophilic, gram-negative, flagellated, spiral-shaped bacterium. Half of all gastric ulcer cases are associated with infection by Hp. The bacterium's spiral shape and high motility allow it to penetrate the deep portions of the mucus gel layer, restrict gastric emptying and survive in the grooves between epithelial cells under the protective gastric mucosal layer of the stomach. There, it causes local damage by inducing inflammatory mediator influx. Prostaglandins are involved in promoting the defense mechanisms of the stomach, and Hp may promote gastric mucosal prostaglandin secretion by up to 50% to maintain its preferred environmental conditions. Because prostaglandin levels in the gastric mucosa are decreased in elderly patients, ageing is associated with a diminished epithelial cell turnover rate and a reduced capacity to repair the gastric mucosa. Advanced age is therefore a major risk factor for complicated peptic ulcer disease. [7,8]

In this case, the cause of peptic ulcer is Hp infection. No history of NSAIDs usage and the main reason is on histopathologyc examination revealed a spherical or rod like organism. Limphoplasmacytic and polymorphonuclear cell that a specific finding for chronic active gastritis due to Hp infection. PUD (peptic ulcer disease) was defined on the basis of the endoscopy findings as a deep mucosal defect with suspected submucosal invasion in the stomach or duodenum in the active or healing stage. [6]Peptic ulcer disease (PUD) is classified as: (1) *Helicobacter pylori*-associated PUD, (2) nonsteroidal anti-inflammatory drug (NSAID) or aspirin-associated PUD, and (3) idiopathic PUD. [6]PUD presents with various symptoms, such as epigastric pain, dyspepsia, nausea, or anorexia. In some PUD patients, however, serious complications such as bleeding or perforation may be the first sign of a problem without any other warning symptoms, and these occur in about 25% of PUD patients.PUD presents with various symptoms, such as epigastric pain, dyspepsia, nausea, or anorexia. In some PUD patients, however, serious complications such as bleeding or perforation may be the first sign of a problem without any other warning symptoms, and these occur in about 25% of PUD patients.[8]Upper gastrointestinal bleeding (UGIB) is a critical condition that requires prompt and effective medical and endoscopic management. Peptic ulcer disease is the most common cause of UGIB, accounting for more than 50% of cases of nonvariceal UGIB (NVUGIB). The incidence of AUGIB in the USA ranges from 48 to 160 cases per 100,000 adults per year. [8,9]

The role of Hp in PUD has been well documented in the literature since the initial landmark *Lancet* article by Marshall and Warren in 1983. The current international consensus guidelines support testing patients with bleeding peptic ulcers for Hp, and administering eradication therapy if present, with confirmation of eradication. This was confirmed in a meta-analysis, which showed that treatment of Hp infection is more effective than antisecretorynoneradication therapy (with or without long-term maintenance antisecretory treatment) in the prevention of recurrent bleeding from peptic ulcer. However, the timing of Hp testing is unclear due to the potential false-negatives in the setting of acute UGIB, which is thought to be partly due to the alkalotic milieu imparted by the presence of blood in the gastric lumen and the resultant proximal migration of the bacterium, as well as concurrent PPI use.[6, 10,11]

Management of this case was eradication of Hp infection as the cause of gastric ulcer by using PPI, clarithromycin, amoxicillin for 10 days. Maintenance with proton pump inhibitor such as omeprazole, antacida and giving sucralfat. Treatment of infection relies on a combination of antimicrobial agents and antisecretory agents, the elevation of the gastric pH by antisecretory agents being required for the bactericidal effect of the antimicrobial agents. While choosing a treatment regimen for Hp patients should be asked about previous antibiotic exposure and this information should be incorporated into the decision-making process. For first-line treatment, clarithromycin triple therapy should be confined to patients with no previous history of macrolide exposure who reside in areas where clarithromycin resistance amongst Hp isolates is known to be low. Most patients will be better served by first-line treatment with bismuth quadruple therapy or concomitant therapy consisting of a PPI, clarithromycin, amoxicillin, and metronidazole. When first-line therapy fails, a salvage regimen should avoid antibiotics that were previously used. If a patient received a first-line treatment

containing clarithromycin, bismuth quadruple therapy or levofloxacin salvage regimens are the preferred treatment options. If a patient received first-line bismuth quadruple therapy, clarithromycin or levofloxacin-containing salvage regimens are the preferred treatment options.[4]

Treatment	Asia-Pacific region[13]	Developing countries[14]	Europe[15]	United States[16]
First-line	Triple therapy (PPI + CLA + AMO/MET) BIS-based quadruple therapy (PPI + BIS + MET + TET)	Triple therapy (PPI + CLA + AMO/FUR) Quadruple therapy (PPI + CLA + AMO + BIS/MET or PPI + BIS + MET + TET) Sequential therapy (PPI + AMO and PPI + CLA + NIT)	Triple therapy (PPI-CLA-containing regimen) BIS-based quadruple therapy (for high clarithromycin resistance) Sequential therapy (for high clarithromycin resistance)	Triple therapy (PPI + CLA + AMO/MET) BIS-based quadruple therapy (BIS + MET + TET + RAN) Sequential therapy (PPI + AMO and PPI + CLA + TIM)
Second-line	BIS-based quadruple therapy (PPI + BIS + MET + TET) LEV-based triple therapy (PPI + LEV + AMO) RIF-based triple therapy (PPI + RIF + AMO)	BIS-based quadruple therapy (PPI + BIS + TET + MET/FUR) LEV-based triple therapy: (PPI + LEV + BIS/FUR/AMO)	BIS-based quadruple therapy LEV-based triple therapy	BIS-based quadruple therapy (PPI + TET + BIS + MET) LEV-based triple therapy (PPI + AMO + LEV)
Third-line	RIF-based triple therapy (PPI + RIF + AMO)	LEV-based or FUR-based triple therapy (PPI + AMO + LEV/RIF or PPI + FUR + LEV)	Guided by antimicrobial susceptibility testing	

AMO: Amoxicillin; BIS: Bismuth; CLA: Clarithromycin; FUR: Furazolidone; LEV: Levofloxacin; MET: Metronidazole; NIT: Nitronidazole; RAN: Ranitidine; RIF: Rifabutin; TET: Tetracycline; TIM: Timidazole; PPI: Proton pump inhibitor.

Table 1: Treatment management for Hp infection in different geographic areas.[4]

Eradication therapy usually consists of a combination of two antibiotics with a gastric acid inhibitor most often a proton-pump inhibitor (PPI) sometimes in combination with bismuth compounds. The recommended eradication regimen in Northern Europe is triple therapy with a PPI in combination with clarithromycin and amoxicillin or metronidazole for 7 days.[9] The clinical and economic benefits of Hp eradication extend to its role in peptic ulcer prevention, a disease that is responsible for a serious burden of morbidity and mortality throughout the world. Hp eradication also reduces peptic ulcer bleeding relapses, the development of NSAID induced ulcers, and unexplained dyspeptic symptoms. [11,12]

4. Conclusion

This case report presented a 70thyear-old male with hematemesis and tenderness on palpation of epigastrium. Histopathologic finding confirm the cause of giant ulcer due to Hp infection. The patient was treated with triple drug for 10 days treatment, which consisted of PPI, amoxicillin and chlarytromycin, the result showed good outcome. The patient scheduled for ulcer reevaluation 3 months later.

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