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Hematemesis Melena due to Helicobacter Pylori Infection In Duodenal Ulcer: A Case Report and Literature Review

Ayu Budhi Trisna Dewi Rahayu Sutanto[1], I Made Suma Wirawan[2]

[1]General Practitioner Wangaya Hospital Denpasar Bali Indonesia

[2] Endoscopy Unit of Internal Medicine Wangaya Hospital Denpasar Bali Indoensia

Abstract: ***A Balinese woman, 60 years old complaint of hematemesis and melena. Esophagogastroduodenoscopy performed one day after admission and revealed a soliter ulcer at duodenum bulb. Histopathology examination revealed a spherical like organism suspected Helicobacter pylori (H. pylori) infection. Eradication of H. pylori by triple drug consisting of omeprazole, amoxicillin and chlarythromycin as the standard protocol of eradication within 14 days. Reevaluation by esophagogastroduodenoscopy examination will perform in the next 3 months to evaluate the treatment succesfull.***

Keywords: peptic ulcer, duodenum, H. pylori

1. Background

Approximately 500,000 persons develop peptic ulcer disease in the United States each year. in 70 percent of patients it occurs between the ages of 25 and 64 years. The annual direct and indirect health care costs of the disease are estimated at about $10 billion. However, the incidence of peptic ulcers is declining, possibly as a result of the increasing use of proton pump inhibitors and decreasing rates of *Helicobacter pylori (H. pylori)*infection. *H. pylori*, a gram-negative, helical, rod-shaped bacterium, colonizes the gastric mucosa of approximately one-half of the world populationand an estimated 30% to 40% of the US population.*H. pylori*is present in 95% of patients with duodenal ulcers and in 70% of those with gastric ulcers.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 and is a risk factor for mucosa-associated lymphoid tissue (MALT) lymphoma and gastric adenocarcinoma.

2. Case Presentation

Female, 60 years old, Balinese, came with chief complaint of hematemesis since one day prior to admission.Patient also complaint of melena since 1 week ago. No history of taking analgeticnor NSAIDs. The patient has felt nausea, and abdominal discomfort since 1 month ago. Pain in the middle of abdomen and getting worse at night, do not getting better by antacid. 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 herblood pressure was normal: 110/80 mmHg, pulse rate 88 times/ minute, respiratori rate 18x/minute. Tenderness on palpation of epigastrium. Laboratory result showed a normal on cell blood count: WBC: 8x 10^3,Hemoglobin: 10 gr/dl, platelet: 156x10^3. Liver function test and kidney fuction 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: Soliter ulcer at duodenal bulbus

Esophagogastroduodenoscopy result: Oesophagus was normal, there was asoliter ulcer on duodenum bulb. The diagnosis was soliterulcer on duodenul bulb. Biopsy done in the duodenum area.The speciment evaluated by Patology Anatomy Specialist. Biopsy result revealed discontinuity of duodenum superficial epithel mucosa, a lot of polimorfonuclear and limfoplasmacytic cells. Also found spherical and rod like organism. Inferred: duodenum ulcer and found Helicobacter pylori like organism.

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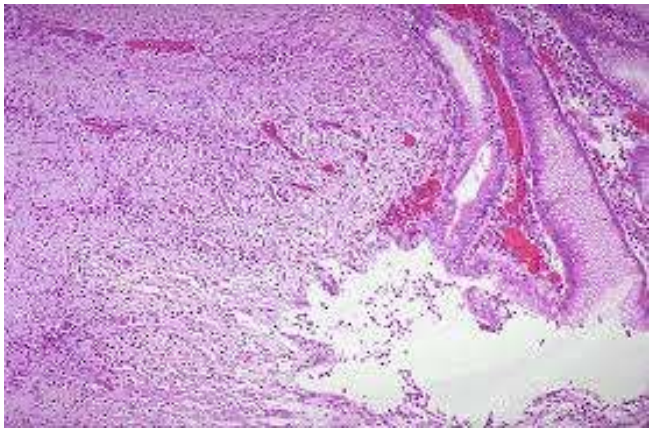


Figure 2: Discontinuity of epithelial duodenum layer, and also found spherical or rod like organism.

3. Discussion

The stomach and the duodenal lining have several mechanisms that prevent ulcers developing. A coating of mucus protects the stomach lining from the effects of acidic digestive juices. Food and other substances in the stomach neutralize acid. Certain chemicals produced by the stomach protect the cells lining the stomach. Peptic Ulcers can be broadly classified into Gastric or stomach ulcer and Duodenal Ulcer. PUD is one of the most common human ailments, affecting approximately 50% of the world population. Gastric Ulcers occur mainly in the elderly, on the lesser curve. Ulcers elsewhere are often malignant. Duodenal Ulcers are four fold commoner than gastric ulcer. [1] Peptic ulcer disease (PUD) may or may not have symptoms. When the symptoms occur, they include a burning pain in the middle or upper stomach between meals or at night, bloating, heart burn, nausea or vomiting. In severe cases, the symptoms include dark or black stool, vomiting of blood, weight loss and sever pain in the mid to upper abdomen.[1,2]In this case we found hematemesis and also melena, pain usually at night, approximately 4 hours after meal.

Etiology of PUD include H.pyloriinfection, NSAIDS, pepsin, smoking, alcohol,bile-acids, steroids, stress, and changes in gastric mucin consistency(may be genetically determined). Other causes include Zollinger-Ellison syndrome,Crohn disease and liver cirrhosis, and similar symptoms stomach cancer, coronary heart disease, and inflammation of the stomach lining or gallbladder. Symptoms of PUD are nonspecific and diagnosis is unreliable on history, frequent symptoms include, epigastric pain, nausea, flatulence and bloating, heartburn, a posterior ulcer may cause pain radiating to the back,symptoms. In this case, we suspected the cause of duodenum ulcer is H. pylori infection, because on the histhopathology examination we found spherical or rod like organism. [3,4,5]

H. pylori cause PUD at first by infection. Then it causes gastritis in the antral region. Then there is a defective inhibition of gastrin release and acid secretion. The gastric acid is then hyper secreted. The duodenal acid load is increased. This is followed by metaplasia in the duodenal bulb. Thus duodenal H. pylori infection is caused. Peptic ulcers can heal spontaneously and may occur intermittently. But they can also have a serious fate. The complications might be life threatening without any warning signs. This is most common in elderly patients on NSAID. The most complication that occur include bleeding and perforation. Bleeding can be both gradual and abrupt. If abrupt bleeding occurs, it causes black, tarry stools and a drop in blood pressure. Only about 2 to 5 percent of people with a peptic require surgery. Perforation usually causes abdominal pain suddenly and usually requires surgery.[2]

If the initial clinical presentation suggests the diagnosis of peptic ulcer disease, the patient should be evaluated for alarm symptoms. Anemia, hematemesis, melena, or hemepositive stool suggests bleeding, vomiting suggests obstruction, anorexia or weight loss suggests cancer persisting upper abdominal pain radiating to the back suggests penetration; and severe, spreading upper abdominal pain suggests perforation. Patients older than 55 years and those with alarm symptoms should be referred for prompt upper endos- copy. Esophagogastroduodenoscopy (EGD) is more sensitive and specific for peptic ulcer disease than upper gastrointestinal barium studies and allows biopsy of gastric lesions. Presence of *H. pylori* can be confirmed with a serum enzyme-linked immunosorbent assay (ELISA), urea breathtest, stool antigen test, or endoscopic biopsy. Eradicating *H. pylori* is often sufficient in patients with small duodenal ulcers. We do esopfagogastroduodenoscopy in this case one day after admission.the biopsy performed in the duodenum area, to know the cause of ulcer. Before endoscopy performed, we give treatment proton pump inhibitor infusion, bolus 80 mg and then continues with 8 mg/ hours until 72 hours. Antacid and also sucralfat given to this patient.

The 2015 European Society of Gastrointestinal Endoscopy guideline made a strong recommendation with high-quality evidence for intravenous bolus with continuous PPI infusion for 72 hours in patients with endoscopic hemostasis and for patients not receiving endoscopic hemostasis with adherent clot.The organization suggests that intermittent intravenous bolus dosing (at least twice daily) for 72 hours postendoscopy may be considered, with a weak recommendation and moderate-quality evidence. [5]Patients with overt upper gastrointestinal bleeding, Hemodynamic status is first assessed, and resuscitation initiated as needed. Patients are risk-stratified based on features such as hemodynamic status, comorbidities, age, and laboratory tests. Pre-endoscopic proton pump inhibitor (PPI) may be considered to decrease the need for endoscopic therapy but does not improve clinical outcomes. Upper endoscopy is generally performed within 24h. The endoscopic features of ulcers direct further management. Patients with active bleeding or non-bleeding visible vessels receive endoscopic therapy (e.g., bipolar electrocoagulation, heater probe, sclerosant, clips) and those with an adherent clot may receive endoscopic therapy. [6]

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Table 1: Differential diagnosis of peptic ulcer disease[2]

Table 1. Differential Diagnosis of Peptic Ulcer Disease		
Commonly mistaken for peptic ulcer disease	**Less commonly mistaken**	**Rarely mistaken**
Esophagitis	Celiac disease	Abdominal aortic aneurysm
Functional dyspepsia	Cholangitis	Acute coronary syndrome
Gastritis	Cholecystitis	Barrett esophagus
Gastroenteritis	Cholelithiasis	Gastric cancer
Gastroesophageal reflux disease	Esophageal perforation	Viral hepatitis
	Inflammatory bowel disease	Zollinger-Ellison syndrome
	Irritable bowel syndrome	

Recommendations emphasize early risk stratification, by using validated prognostic scales, and early endoscopy (within 24 hours). Endoscopic hemostasis remains indicated for high-risk lesions, whereas data support attempts to dislodge clots with hemostatic, pharmacologic, or combination treatment of the underlying stigmata. Clips or thermocoagulation, alone or with epinephrine injection, are effective methods, epinephrine injection alone is not recommended. Ulcer bleeding is first treated by endoscopic hemostasis. If it fails, surgery or interventional radiology is chosen. Second, medical therapy is provided. In cases of NSAID-related ulcers, use of NSAIDs is stopped, and anti-ulcer therapy is provided. If NSAID use must continue, the ulcer is treated with a proton pump inhibitor (PPI) or prostaglandin analog. In cases with no NSAID use, *H. pylori*positive patients receive eradication and anti-ulcer therapy. If first-line eradication therapy fails, second-line therapy is given. In cases of non*H. pylori*, non-NSAID ulcers or *H. pylori*-positive patients with no indication for eradication therapy, non-eradication therapy is provided. The first choice is PPI therapy, and the second choice is histamine 2-receptor antagonist therapy. After initial therapy, maintenance therapy is provided to prevent ulcer relapse. [8,9,10.] In this case we treated the H. pylori infection by giving triple drug consist of omeprazole 2 x 1 capsul, amoxicillin 2 x 1 gram and chlarithromycin 2 x 500 mg for 14 days.

Prognosis of PUD is excellent if the underlying cause such as H.pylori infection or drugs can be addressed.[11] Prognosis of this case is dubius ad bonam. We plan to do esophagogastroduodenoscopy in the further next 3 months to reevaluate the treatment succesfull.

References

[1] Siddique, R. A. (2014). Prevalence of peptic ulcer disease among the patients with abdominal pain attending the department of medicine in Dhaka medical college hospital. *Bangladesh. Bangladesh*, *13*(1), 2279-0861.
[2] Fashner, J., &Gitu, A. C. (2015). Diagnosis and Treatment of Peptic Ulcer Disease and H. pylori Infection. *American family physician*, *91*(4).
[3] Mustafa, M., Menon, J., Muiandy, R. K., Fredie, R., Sein, M. M., &Fariz, A. (2015). Risk Factors, Diagnosis, and Management of Peptic ulcer Disease. *IOSR-JDMS*, *14*(7), 40-6.
[4] Cheung, F. K., & Lau, J. Y. (2009). Management of massive peptic ulcer bleeding. *Gastroenterology Clinics*, *38*(2), 231-243.
[5] Chui, D. (2016). Proton pump inhibitor dosing for acute ulcer bleeding. *Canadian Pharmacists Journal: CPJ*, *149*(3), 119.
[6] Laine, L., & Jensen, D. M. (2012). Management of patients with ulcer bleeding. *The American journal of gastroenterology*, *107*(3), 345.
[7] Barkun, A. N., Bardou, M., Kuipers, E. J., Sung, J., Hunt, R. H., Martel, M., & Sinclair, P. (2010). International consensus recommendations on the management of patients with nonvariceal upper gastrointestinal bleeding. *Annals of internal medicine*, *152*(2), 101-113.
[8] Satoh, K., Yoshino, J., Akamatsu, T., Itoh, T., Kato, M., Kamada, T., ... & Murakami, K. (2016). Evidence-based clinical practice guidelines for peptic ulcer disease 2015. *Journal of gastroenterology*, *51*(3), 177-194.
[9] Ramakrishnan, K., & Salinas, R. C. (2007). Peptic ulcer disease. American family physician, 76(7).
[10] Sung, J. J. Y., Kuipers, E. J., & El-Serag, H. B. (2009). Systematic review: the global incidence and prevalence of peptic ulcer disease. Alimentary pharmacology & therapeutics, 29(9), 938-946.
[11] Barkun, A., &Leontiadis, G. (2010). Systematic review of the symptom burden, quality of life impairment and costs associated with peptic ulcer disease. The American journal of medicine, 123(4), 358-366.

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