Non Helicobacter pylori - Duodenal Ulcer in a Liver Cirrhosis Patient

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ABSTRACT

Peptic ulcer is a clearly marginated ulceration in mucosal membran that can penetrate until muscularis layer and resulted from imbalance between aggressive factor (gastric acid and pepsin) and defensive factor (gastric mucous, bicarbonate and prostaglandin, mucosal blood flow, and cell replacement). Factors that can act as aggressive factor include H. pylori, NSAID, and smoking. Duodenal ulcer is frequently associated with H. pylori, in which Helicobacter pylori is found in 95 - 100% of duodenal ulcer patients..

It was reported, a 39 years old female patient with cirrhosis hepatis who suffered from melena in which endoscopic examination revealed duodenal ulcer as a source of bleeding.There was no H. pylori, based on serologic examination (IgG antiHP) and culture. The ulcer is suspected caused by NSAID based on history of using traditional medicine that may contain NSAID. Treatment with proton pump inhibitor and sucralfate can heal the ulcer after two week treatment.

***Key Words**: H. pylori, duodenal ulcer – liver cirrhosis*

INTRODUCTION

Peptic ulcer is a clearly marginated ulceration of mucosal membrane that extend through the muscularis mucosae, and result from an imbalance between aggressive and defensive factor. Helicobacter pylori associated frequently with duodenal ulcer. Beside H. pylori, there are many factor that can cause the duodenal ulcer.[1,2]

Liver cirrhosis is characterized by a reduction in functional tissue and by disorganization of lobular and vascular architecture leading to parenchymal dysfunction and portal hypertension as the result of chronic wound healing in the liver following chronic damage.[3,4]

It will be reported, a female patient with liver cirrhosis and non-Hp duodenal ulcer that may be a relatively rare case we found.

CASE REPORT

A 39 years old female patient worked in plywood factory in East Kalimantan enterred the hospital with chief complaint dark colour stool. She suffered from dark colour stool three times since one day before admission. Her abdomen enlarged gradually since one year before admission, associated with anorexia and full sensation, and yellowish eyes. There was skin hyperpigmentation and her hair was easy to fall since six month before admission. She frequently took many kind of traditional medicine to treat herself, but she didn't know what the names of those medicine are. She didn't get improvement of her condition.

From physical examination, the patient look moderately ill, fully consciouss, body height 155 cm, body weight 46 kg, body mass index 19.02 kg/m2, blood pressure 100/70 mmHg, pulse rate 108 x/mnt, respiratory rate 24 x/mnt, conjunctiva look anemic, sclera look icteric, hyperpigmentation of skin. No abnormality on chest examina-

tion. There was abdominal enlargement with undulation and shifting dullness. There was ankle edema on both leg.

The results of laboratory examination included: Hb 7.5 gr/dl, leukocyt 8300/uL, ESR 24 mm/hour, thrombocyt 67000 /uL, blood smear: erythrocyt hypochrom mycrocyter, fasting blood glucose 60 mg/dl, postprandial blood glucose 180 mg/dl, ureum 65.5 mg/dl, creatinin 1.19 mg/dl, total bilirubin 5.47 mg/dl, direct bilirubin 0.08 mg/dl, indirect bilirubin 5.39 mg/dl, SGOT 41 mU/ml, SGPT 30 mU/ml, total protein 4.8 gr/dl, albumin 1.8 gr/dl, globulin 3.0 gr/dl, HbsAg (MEIA): (+) abs 1077.7, cut off 6.0. IgG anti H. pylori: negative. There was no colony of H. pylori from culture. Ascites fluid analyse: straw coloured, erythrocyt 50/cmm with 100% crenation, leukocyt 100/cmm with 40% PMN and 60% MN, Rivalta test negative, protein 430 mg/dl, glucose 97 mg/dl. Endoscopic examination showed esophageal varices grade II/III without bleeding sign, multiple ulcer in duodenum with bleeding. Histopathologic examination showed ulceration of duodenal mucosa with shortening and widening of villi and mononuclear inflammatory cells infiltration; this findings usually are found in duodenal ulcer. There was no malignancy in this specimen. Ultrasonography showed the features of liver cirrhosis and ascites.

Treatment included bed rest, soft diet with high calorie and high protein and salt restriction (2 gr NaCl), lansoprazole 1x30 mg/day, sucralfate 4x1 gr/day, spironolacton 1x100 mg/day, lactulose 4x30 ml/day, lavement every 12 hours. In the third day, there was no melena anymore and then lavement was stopped, while other treatment was continued. After two week treatment, reendoscopic examination showed the disappearance of duodenal ulcer. Finely, the treatment was continued with spironolacton 1x100 mg/day, propranolol 2x10 mg/day, lansoprazole 1x30 mg/day, and sucralfate 4x1gr/day.

DISCUSSION

Peptic ulcer disease is thought to result from an imbalance between aggressive factors, especially gastric acid and pepsin, and protective factors, including gastric mucus, bicarbonate, prostaglandin, mucosal blood flow, and epithel cell replacement. H. pylori, NSAIDs and smoking can act as aggressive factors.[1,2] H. pylori is found in 95-100% patients with duodenal ulcer.[1] Endoscopy is a sensitive, spesific, and safe method for the diagnosis of peptic ulcer disease that is followed with biopsy for histopathologic examination.[2] Liver cirrhosis represents chronic wound healing in the liver (fibrinogenesis) following chronic damage caused by many factors include viral agents (e.g., HBV and HCV), toxins, drugs, metabolic diseases (hemochromatosis, alpha-1 antitrypsin deficiency), autoimmune disease, and biliary disease. The diagnosis of cirrhosis is made by evaluation of clinical signs, laboratory studies of liver function, ultrasonography and laparoscopy-liver biopsy. Laparoscopy is the gold standard for the investigation of liver cirrhosis that allows both macroscopic and microscopic (histopathologic) examination by directed liver biopsy.[4]

In this case, the diagnosis of liver cirrhosis was based on the result of abdominal ultrasonography that shows the features of liver cirrhosis and ascites. This finding was supported by clinical and laboratory evaluation that showed the features of chronic liver disease with the suspected causative factor is hepatitis B virus infection. The diagnosis of duodenal ulcer was based on the result of endoscopy that show multiple ulcer with bleeding in duodenum, with the result of histopathologic examination reveal the features of duodenal ulcer. There was no Helicobacter pylori from serologic examination (IgG anti Hp) and culture. Therefore, the patient is diagnosed as liver cirrhosis with non-Hp duodenal ulcer. The suspected causative agent of this ulcer is NSAID, based on history of using traditional medicine that may contain NSAID.

NSAIDs are directly toxic to the gastric mucosa, and they deplete protective endogenous mucosal prostaglandins by inhibiting prostaglandin synthesis. They reduce gastric mucus secretion, gastric and duodenal bicarbonate secretion, increase gastric acid secretion, and also impair epithelial cell replacement. Some drugs that can treat peptic ulcer include antacids, antisecretory drug (H_2-reseptor antagonis, proton pump inhibitor, anticholinergic agents, and prostaglandin), and cytoprotective drug (sucralfate). H_2-receptor antagonis and proton pump inhibitor (PPI) can effectively prevent and heal NSAID-induced ulcer. PPI may heal ulcer within 2-4 week treatment. PPI may accelerate the healing of such ulcers even if NSAID use is continued.[1,2,5]

In this case, the patient was treated with lansoprazole 1x30 mg/day as PPI that inhibit acid secretion, sucralfate 4x1gr/day as cytoprotective drug that protect gastric mucosa. PPI is an inhibitor of acid secretion that more potent than H_2-receptor antagonis and effective in healing NSAID-induced ulcers, while sucralfate can protect ulcer and mucosa from gastric acid, can bind bile acid, increase endogenous tissue prostaglandin, and may bind epidermal growth factors and present them to the ulcer, thereby increasing mucosal defense.[1,2,5]

Treatment of liver cirrhosis includes: treatment of the underlying disease, administration of antifibrotic drugs, prevention and treatment of complications (portal hypertension, ascites, hepatic encephalopathy). In cases of HBV and HCV, giving antiviral agent in compensated liver cirrhosis may be effective in eliminating HBV and HCV, but it should not be used routinely in patients with liver cirrhosis because decompensation and severe side effects may occur.[4] Therefore, in this patient who have decompensated liver cirrhosis, such treatment was not given. The use of antifibrotic drugs which can stop fibrosis to treat cirrhosis is still experimental and not yet of clinical relevance. Beta-blockers and nitrates are useful in reducing portal hypertension and preventing variceal bleeding.[4,6] In this case, propranolol was given to the patient in order to reduce portal hypertension. The treatment of ascites should be started by instituting a low sodium (max 3 gr NaCl/day), low fluid intake (1-1,5 liter/day), albumin substitution, and diuretic therapy (spironolactone, xipamide). In pronounced ascites, the treatment can be done by paracentesis with concomitant albumin substitution.[4,6] The treatment of ascites for this patient included low salt diet (2 gr NaCl), potassium sparing diuretic (spironolacton 100mg/day) to enhance diuresis, whereas albumin substitution cannot be given because of high cost.

Gastrointestinal bleeding can become a precipitating factor of hepatic encephalopathy. Blood has to be evacuated from the bowel, and the bleeding has to be stopped. Lactulose can act as laxative agent, supress production and absorption of ammonia in the bowel [4,6]. In this case, the patient suffered from melena that caused by bleeding from duodenal ulcer. The drug that treat the ulcer can stop the bleeding. Gastric Lavage every 12 hours was performed to evacuated blood from the bowel, and lactulose could prevent hepatic encephalopathy. The treatment of duodenal ulcer has succesfully treated and made healing of the ulcer after two week treatment in which re-endoscopic examination showed the disappearance of the ulcer.

CONCLUSION

A case was reported, a 39 years old female patient with liver cirrhosis had multiple ulcer in duodenum, based on endoscopic examination. Most of duodenal ulcer is associated with Helicobacter pylori as a cusative agent, but in this case, there was no H. pylori on serologic examination and culture. The suspected causative agent is NSAID, based on history of using traditional medicine that may contain NSAID.

Treatment of duodenal ulcer included proton pump inhibitor and cytoprotective drug that can make ulcer healing after two week treatment, whereas the treatment of cirrhosis was directed to reduce ascites by using potasium sparring diuretic, and reduce portal hypertension by using beta-blocker.

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