Double Pylorus in Upper Gastrointestinal Bleeding

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Case Report

Double Pylorus in Upper Gastrointestinal Bleeding

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Abstract

Double pylorus, also known as acquired double pylorus, is a rare condition defined as a gastrointestinal fistula connecting stomach antrum and duodenal bulb. The prevalence of double pylorus ranges from 0.001% to 0.4% from esophagogastroduodenoscopy (EGD). Although the etiology is unknown, the formation of double pylorus is related to *Helicobacter pylori* infection and the use of non-steroidal anti-inflammatory drugs (NSAID). The development of the occurrence of double pylorus is still unknown, but many systemic diseases play a role. We presented case of a 59-years-old man was admitted to Dr. Soetomo General Hospital with hematemesis and melena. The patient had a history of diabetes mellitus since three years ago and consuming medicinal herbs for myalgia (montalin), which was suspected of NSAIDs for past 5 months. Anemia with hemoglobin (Hb) was 8.3 g/dL, enterogenous azotemia with blood urea nitrogen (BUN) 28 mg/dL and serum creatinine 1.14 mg/dl were obtained to this patient. At EGD, double pylorus was found and accompanied by gastric ulcer, a giant white base ulcer, part of it covered by clotting without any sign of active bleeding. The results of biopsy, chronic inactive gastritis, and no *H. pylori* were found. Treatment mainly depends on gastrointestinal acid suppression through a proton pump inhibitor (PPI).The patient was given a high dose PPI and mucosal protective agent. He was treated for one week and had improved complaints. Chronic inactive gastritis and no *H. pylori* were found in biopsy.

Introduction

Double pylorus, also known as the acquired double pylorus, is a rare condition that is defined as a gastrointestinal fistula connecting stomach antrum and duodenal bulb, which was first reported by Smith and Tuttle in 1969 [1–3]. Double pylorus can occur as a congenital disorder that can be accompanied by other congenital abnormalities, which were reported by Christine in 1971 [4,5]. Double pylorus is a very rare disorder [3,6]. The frequency of double pylorus varies from 0.001% to 0.4% from upper gastrointestinal tract esophagogastroduodenoscopy (EGD) examination. [2,4,7,8]. Cases are reported mainly in Asian countries and are more common in man than woman [9]. For the prevalence in Indonesia, no supporting data have been found.

Though etiology is uncertain, the development of double pylorus is associated with *Helicobacter pylori* infection and the use of non-steroidal anti-inflammatory drugs (NSAID). [9]. In most cases, the double pylorus is a complication of chronic peptic ulcer. The double pylorus diagnosis is essential because it can be caused by recurrent ulceration and failure of epithelial formation in the fistula duct. Double pylorus development is still unknown, but many systemic diseases play a role [10,11]. In congenital double pylorus, defects occur in formation of pyloric ducts in early embryonic phase [5].

Double pylorus has no pathognomonic signs and symptoms. Usually there are symptoms of dyspepsia such as epigastric pain, nausea, vomiting, bloating and gastrointestinal bleeding. Double pylorus is nevertheless identified only incidentally during EGD examination [12,13]. The treatment is focused on factors that influence healing process of mucosa in gastric ulcer. Surgery on patients with refractive symptoms such as recurrent ulcers, proton pump inhibitor (PPI), and gastric obstruction is performed [14].

Case Report/Case Presentation

A male patient, Mr. Rb, 59 years, was admitted to General Hospital dr. Soetomo with hematemesis and melena. The patient complained of hematemesis for 6 hours before admitted. The patient vomited three times with an amount of 200-300 ml and had lower abdominal pain. Defecate liquid two times since 6 hours before admitted, black, and very bad small. The patient complained of dizziness accompanied by nausea and had no appetite since five days ago. There had been shortness of breath when on a heavy activity, reduced when resting, no fever.

The patient had a history of diabetes mellitus since three years ago. He using insulin and oral medication but irregular. History of hypertension, heart, lung, and kidney diseases was denied. There was a history of consuming medicinal herbs for myalgia (montalin). The patient consumed it every day. He felt weak when he did not drink. He used this drug for the past five months. Psycho-social history, he works as a construction worker and smoking three packs a day since young.

On physical examination, it was found that compos mentis GCS 456, blood pressure was 125/75 mmHg, heart rates was 96 x/minute regular, strong pulse, adequate content, respiratory rates was 20 x/minute, the axillary temperature was 36.5°C, and SpO2 was 99 % of free air. The examination of head and neck conjunctiva anemic was present, no jaundice, no cyanosis, no dyspnea, no enlarged lymph nodes, jugular venous pressure does not increase. The thorax was normal, the abdomen appeared flat and soepel, liver and spleen enlargement were not palpable, normal bowel sounds, abdominal pain in umbilical region. Extremities were warm, dry acral, capillary refill time <2 seconds. No edema was found on both legs.

Laboratory tests showed that Hb was 8.3 g/dL, hematocrit was 23.6%, MCV 78.1; MCH 27.5; MCHC 35.2; platelets was 356,000 mm^3, leukocytes was 14,369 mm^3, neutrophils was 79.1%, lymphocytes was 14.5%, sodium was 132 mmol/L, potassium was 4.4 mmol/L, chloride was 106 mmol/L, HbA1C was 11.2%, random blood sugar was 202 mg/dL, SGOT was 16 U/L, SGPT was 27 U/L, BUN was 28 mg/dL, serum creatinine was 1.14 mg/dL, and albumin was 3 gr/ dL. The examination of chest radiograph showed normal heart and lungs.

The patient was treated with a B1 soft diet 2,100 kcal/day, infusion of NaCl 0.9% 1,500 ml in 24 hours iv, injection of omeprazole 40 mg every 8 hours iv, sucralfat syrup 15 ml every 8 hours per oral, novorapid injection 4 units every 8 hours sc ac 15 minutes, transfusion of p*acked red cells* (PRC) 1 colf/day until Hb >10 g/dl, metoclopramide 10 mg every 8 hours iv, amlodipin 10 mg every 24 hours orally, lactulac syrup 15 ml every 8 hours orally, Ceftriaxon 1 gram every 12 hours iv, and paracetamol 500 mg every 8 hours orally.

The patient had been given PRC transfusion until Hb reached 10.3 g/dL, and the upper gastrointestinal EGD showed that the cause of bleeding to this patient was a gastric ulcer which was suspected and caused by consumption of medicinal herbs (montalin). At EGD, double pylorus was accompanied by giant white ulcer with a partially covered in clotting without any sign of active bleeding. It could be concluded that there was a double pylorus with giant ulcer Forrest IIb (Table 1).

In treatment for 1 week at Dr. Soetomo General Hospital, the patient's complaints were improved with no nausea and vomiting, there were no hematemesis and melena, and appetite was improved. The patient discharged from hospital. Planning therapy was B1 soft diet 2,100 kcal/day, omeprazole 20 mg every 12 hours per oral, sucralfat syrup 15 ml every 8 hours per oral, novorapid injection 4 units every 8 hours sc ac 15 minutes, amlodipin 10 mg every 24 hours per oral. The results of biopsy, chronic inactive gastritis, and no *H. pylori* were found. The patient underwent check up in gastroenterology poly after 1 week out of hospital with complaints of decreased appetite, abdominal pain, no nausea and received sucralfate syrup and lansoprazole per oral.

Discussion/Conclusion

Most of double pylorus cases reported previously were in Asian countries and have become case reports in the world including Greece and Peru [3,9]. In world literature, less than 100 cases have been reported and most patients are European or Asian. Only five cases were registered in the US [14]. The mechanism for occurrence of double pylorus is still unknown. A long history of treatment, including NSAIDs and corticosteroids, can influence peptic ulcer healing. As a potential ulcerogenic drug, NSAIDs play an important role in formation of double pylorus [3,4,6,15]. Double pylorus is associated with comorbid diseases that accompany it. Many systemic diseases that play a role in poor healing, such as diabetes mellitus. In diabetes mellitus, damage to gastric mucosal microcirculation can be a cause. Poor healing is also associated with other diseases, such as chronic blockage of the lungs, chronic renal failure, rheumatoid arthritis, and systemic lupus-erythematosis [2,4,6,16]. The cause of poor healing may be due to nonadherence and nonaffordability in treatment or NSAIDs [14]. The patient had a history of consumption of medicinal herbs (montalin) which was suspected of NSAIDs. He took it for 5 months and had a history of diabetes mellitus since 3 years ago.

There are no specific clinical symptoms of double pylorus. Symptoms include chronic stomach pain, dyspepsia and vibrations and gastrointestinal bleeding due to peptic ulcer or other diseases [1,2,4]. Although the entire blood and blood count is generally normal, full blood tests indicate anemia in patients with gastrointestinal bleeding and biochemistry will increase enterogenous azotemia ureum nitrogen (BUN) with regular serum creatinine [2]. Anemia with Hb was 8.3 g/dL, enterogenous azotemia with BUN 28 mg/dL and serum creatinine 1.14 mg/dl were obtained to this patient.

Diagnosis of double pylorus is made by EGD. Most of double pylorus is located in minor curvatura gastric antrum. From gastric antrum, visualization of biopsy forceps or catheters that have passed through double pylorus and entered pylorus can be described as a technique to diagnose double pylorus [1,15]. In patient with double pylorus, research on *H. pylori* is very important. Some literature has been reported that there is a correlation between occurrence of double pylorus and *H. pylori*. Patient infected with *H. pylori* should be treated [2]. During treatment, EGD showed that the cause of bleeding to patient was a gastric ulcer which was suspected and caused by consumption of medicinal herbs for myalgia (montalin). At EGD (shown in Fig. 1), double pylorus was accompanied by giant white ulcer with a partially covered in clotting without any sign of active bleeding. There was a double pylorus with giant ulcer Forrest IIb. The results of biopsy, chronic inactive gastritis and no *H. pylori* were found.

In general, double pylorus is a by-product of upper gastrointestinal EGD. Most dual pylorus lie on the side of the minor curvatura between the gastric antrum and the superior part of the duodenal bulb. [3,12]. It is believed that fistula formation due to gastric ulcer causes anomalies, but duodenal ulcer can also cause the formation of double pylorus [3]. Our patient with double pylorus that was found incidentally during EGD, double pylorus was located in gastric side of curvatura minor side.

The management should focus on factors of inhibiting mucosal healing. NSAIDs and corticosteroids should be avoided. Double pylorus in majority of patient has good response to medical treatments such as PPIs, H_2 antagonist receptors, antacids and mucosal protective agents, both regardless of whether double pylorus persists or not. Cessation of NSAID use is very beneficial because symptoms will be reduced, recurrence of ulcers can be prevented, and double pylorus will close [1–3,17]. Surgical intervention is not primary therapeutic [1–3]. According to a retrospective study, double pylorus treated with antagonists H_2 receptor or PPI, double pylorus remains open in majority of patient by 64%, merges with normal pylorus is 27% and closed in only 9% of cases[4,8,18]. Complications are gastric outlet obstruction and persistent gastric ulceration[4]. Treatment mainly depends on gastrointestinal acid suppression through PPI. In current case, patient was given a high dose PPI and mucosal protective agent. In the development of ulcers and chronic ulcers, *H. pylori* played an significant role. However, we did not notice *H. pylori* in the current case of biopsy specimen. Patient should be followed up and EGD repeated to assess improvement of ulcers and whether double pylorus remains open, fused with normal or closed pylorus.

Conclusion

It was reported 59-years-old man presented with hematemesis and melena. Laboratory tests showed decreased Hb and increased BUN. The results of EGD confirmed that the cause of bleeding was peptic ulcers caused by excessive consumption of medicinal herbs (montalin). In EGD, double pylorus in gaster with giant ulcer Forrest IIb without any sign of active bleeding were obtained. Chronic inactive gastritis and no *H. pylori* were found in the biopsy. The patient was given high doses of PPI and mucosal protective agents.

Statements

Statement of Ethics

Informed consent was obtained for this case report. Ethics approval was not needed for this paper.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Heasty Oktaricha, Muhammad Miftahussurur performed data acquisition and drafted the manuscript. Muhammad Miftahussurur supervised and provided critical revision of the manuscript. All authors read and approved the final manuscript.

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Figure Legends

Fig. 1. Double pylorus

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