Gastric Outlet Obstruction due to Peptic Ulcer Disease

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ABSTRACT

Gastric outlet obstruction is a rare complication of peptic ulcer disease, resulting from acute or chronic inflammatory changes. Patient may present asymptomatic or may have mild gastrointestinal symptoms. Some complications may include indirect systemic disorders such as water, acid-base, and electrolyte imbalance, which could be fatal. Acute management should include gastric decompression, correction of water and electrolytes abnormalities, as well as reduction of spasm and edema by using acid-supressants. After the patient has been stabilized, more definite measures should be taken such as endoscopic dilatation or surgery and treatment of peptic ulcer itself. Nowadays, endoscopic dilatation has been performed by using through-the-scope balloon dilating catheters. The diameter of balloon is usually increased gradually over several sessions. Long-term recurrence after endoscopic baloon dilatation has been reportedly low.

Keywords: *peptic ulcer, gastric outlet obstruction, endoscopic baloon dilatation*

ABSTRAK

Obstruksi jalan keluar lambung merupakan komplikasi tukak peptik yang jarang. Obstruksi tersebut disebabkan oleh perubahan-perubahan akut maupun kronik akibat inflamasi. Pasien dapat datang dengan keluhan gastrointestinal yang jelas maupun samar. Terkadang obstruksi diperberat dengan kelainan sistemik tidak langsung yang dapat berakibat fatal seperti gangguan cairan, asam-basa, dan elektrolit. Tatalaksana akut meliputi dekompresi lambung, koreksi cairan dan elektrolit, dan penggunaan obat-obat supresan asam lambung untuk mengurangi spasme dan edema. Setelah pasien stabil, tindakan-tindakan yang definitif seperti dilatasi endoskopi atau pembedahan perlu dilakukan, disertai dengan terapi tukak peptik. Saat ini dilatasi endoskopi dilakukan menggunakan kateter dengan balon yang dapat dikembangkan. Diameter balon dapat dikembangkan lebih besar secara bertahap melalui beberapa sesi. Dilatasi balon endoskopi ini memiliki tingkat kekambuhan jangka panjang yang rendah.

Kata kunci: *tukak peptik, obstruksi jalan keluar lambung, dilatasi balon endoskopi*

INTRODUCTION

Peptic ulcer disease is a problem of the gastrointestinal tract characterized by mucosal damage, secondary to pepsin and gastric acid secretion. After better acid-supressing drugs have been developed and the association of *Helicobacter pylori* (*H. pylori*) has been recognized, there is decreasing frequency of hospitalizations, rates of operations, and deaths associated with peptic ulceration. However, especially for those who has been untreated, complications resulting from peptic ulcer disease may develop. These complication include gastrointestinal bleeding, perforation, and gastric outlet obstruction.[1]

Gastric outlet obstruction is the least frequent complication of peptic ulcer disease, accounting only 1-5%; however, it carries the risk of significant morbidity, and mortality.[2] Long-standing obstruction results not only mechanical and nutritional, but also hydration, electrolytes, and acid-base disturbances.

Management should be aimed at correcting water and electrolyte abnormalities, maintaining adequate nutrition, and relieving obstruction by medications, endoscopic interventions, or surgery, especially for cases with advanced stages.[1]

CASE ILLUSTRATION

A 54-year-old male visited the gastroenterology clinic and had a complaint of recurrent vomiting during the last three weeks. Three years ago, before admission, the patient had suffered from burning pain in epigastrium, which was localized and did not radiate to other regions. The pain was felt especially before a delayed meal and at midnight. Sometimes, it was accompanied with bloating. One year later, the bloating was worse, exacerbated by vomiting, which was most pronounced after a meal. The vomit contained undigested food. He has lost 5 kg of weight over the past 1 month and he felt very week. Subsequently, he visited the gastroenterology clinic at Cipto Mangunkusumo Hospital. He reported no fever, abdominal pain other than the epigastric burning pain. No unusual lump, night sweating, diarrhea, constipation, or passing coffee-ground stool has been reported. His medical history is positively significant for ischemic stroke, which occurs four years ago, leaving him with sequel of slight left hemiparetic sign. He had no history of hypertension, diabetes, asthma, or tuberculosis. He took over-the-counter antacids to relieve his epigastric pain occasionally and over the counter analgetic (containing paracetamol) once daily for about 1 years to manage his headache. He denied any routine consumption of other pain relievers, anti-coagulants, traditional herbal medicines, alcohol, or corrosive substances.

Esophagogastroduodenoscopy (EGD) was subsequently performed and revealed pyloric stenosis, multiple giant ulcers in the gastric cardia, and pangastritis. Duodenum could not be examined because of the stenosis. Endoscopic dilatation with 10 mm baloon was performed along with endoscopic placement of 16F-diameter nasoduodenal tubes.

The patient was advised to have further baloon dilatation the week after, but he refused due to financial problems. He was then discharged from hospital and the tube was pulled off at the out-patient clinic. He was able to have soft diet and gradually began to eat solid food. After felling well, he has never had further visit and did not take his medications.

He remained uneventful until two years later when his previous symptoms recurred. Laboratory examination showed hypokalemia (2.72 mEq/L) which was corrected with oral potassium. EGD was performed and revealed pyloric stenosis, pangastritis, esophagitis, and ulcers in the esophagus and antrum (Figure 1). Biopsy was performed and showed chronic atrophic gastritis, *H. pylori* was not found.

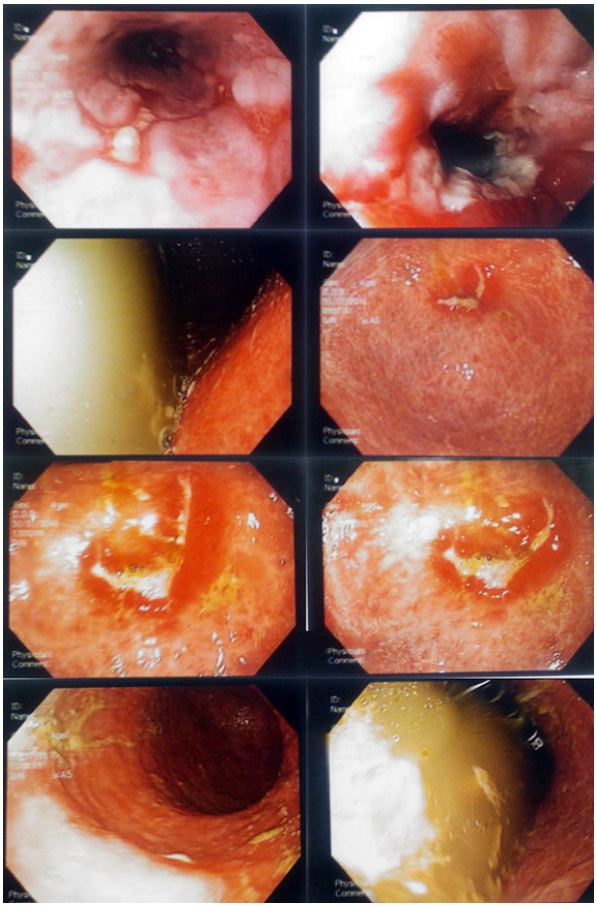

Figure 1. Esophagogastroduodenoscopy showed pyloric stenosis, esophagitis, pangastritis, and ulcers in the esophagus and cardia

The patient underwent a repeat dilatation of 15-mm baloon followed by pyloric triamcinolone injection. A 18F nasoduodenal tube was inserted and subsequently pulled out at outpatient clinic. Until four months later, the patient reported no recurrent symptoms and has gradually gained weight.

DISCUSSION

About 25% of patients with peptic ulcer disease have serious complications such as hemorrhage, perforation, penetration or gastric outlet obstruction. The gastric outlet obstruction is the last, but the least frequent complication.[1,3] Peptic ulcer disease was the most common cause of gastric outlet obstruction in the past, but nowadays, the frequency of obstruction due to peptic ulceration has declined and malignancy is now the leading cause of gastric outlet obstruction.[3]

Less than 5% patients with duodenal ulcer disease and less than 1-2% with gastric ulcer disease have developed this complication.[2]

Several elements may contribute to the development of gastric outlet obstruction:[4] (1) Rapidly reversible elements including spasm, edema, inflammation, and pyloric dysmotility related to the ulcer or inflammatory changes; (2) Fibrosis, scarring, and deformity underlying slowly reversible or irreversible obstruction; (3) Gastric atony, which develops after prolonged obstruction and contributes to gastric retention.

Symptoms of gastric retention include early satiety, bloating, indigestion, anorexia, nausea, vomiting, epigastric pain, and weight loss. The presence of recognizable food more than 8–12 hours after eating is indicative of gastric retention. Some patients with chronic organic disease such as peptic ulcer have decreased visceral sensitivity. This may result high-grade outlet obstruction without perceived gastric distress.[5]

Patients with gastric outlet stenosis may present with symptoms and signs not directly related to gastrointestinal disorder. Dehydration causes hypovolemia and hypotension. It may cause acute kidney injury. In order to compensate, aldosterone secretion is stimulated, which causes sodium and water retention. Aldosterone acts on the renal tubules to reabsorb sodium in exchange for potassium and hydrogen, hence producing hypokalemia and metabolic alkalosis. Vomiting further worsens this condition. Severe metabolic alkalosis is potentially life-threatening. It affects the major organ systems, including cardiac dysrhythmias and vascular collapse, and neurological effects, which are primarily seizures. Hypokalemia may result in cardiac dysrhytmia, muscle weakness, or rhabdomyolysis.[6-9] A fatal ventricular fibrilation and respiratory failure in hypokalemic patients with pyloric stenosis have been reported.[7,9]

Endoscopic inspection and biopsy are indicated in suspected cases of outlet obstruction and usually provide definitive diagnosis of underlying pathology.[5] Malignancy must be excluded in all cases of obstruction since about 50% of outlet obstruction cases are due to malignancy. Patients with malignancy tend to be older and typically do not have a history of peptic ulcer or using non-steroid anti inflammatory drugs (NSAIDs).[5]

The initial step in the management of presumed gastric outlet obstruction is to confirm the diagnosis of gastric retention. If it is present, lavage and decompression should be performed, preferably by large-bore tube. The purposes are to relieve symptoms, deflate the stomach so it can regain tone, and monitor fluid loss. The patient should be rehydrated and given potassium replacement if hypokalemia. Parenteral nutrition should be considered if the patient is severely malnourished.

Intravenous acid-supressants such proton pump inhibitor (PPI) may reduce acid output. PPI treatment also starts ulcer healing, ameliorates inflammatory edema, and assists in resolving obstruction. Approximately one-half of cases respond initially to this regimen, especially when the obsruction is caused by spasm, edema, inflammation, or associated pyloric dysmotility rather than dense scarring. Some initial responders may eventually require surgery or endoscopic dilatation.[3,4,5]

Gastric outlet obstruction is not an emergency; both endoscopic or surgical intervention should be delayed until the patient has been stabilized and having their fluid and electrolyte balance restored. Delays are also appropriate if the patient's nutritional status is compromised (an albumin < 2.8 in general is a strong predictor of a poor surgical outcome) or if the stomach is markedly dilated (post-operative gastric atony appears more likely and may be prevented by preoperative decompression).[4]

A variety of surgeries have been described for obstructing duodenal, pyloric channel, and prepyloric ulcers. They include truncal vagotomy together with either a drainage procedure (either gastrojejunostomy or pyloroplasty) or an antrectomy. Another option is a highly selective vagotomy combined with stricture dilation. Other forms of duodenoplasty have been described for postpyloric obstructions. In the unusual event of an obstructing prepyloric gastric ulcer, an antrectomy followed by a Billroth type I gastroduodenostomy is the procedure of choice.[3] Recently laparoscopy management like truncal vagotomy has been performed and shows promising results.[10,11] Surgery involving vagal denervation always carries risk for prolonged gastric atonia and sometimes requiring further completion gastrectomy. Estimates of postoperative gastric dysfunction range from 10-50%.[12,13]

In the past, patients with gastric outlet obstruction due to peptic ulcer traditionally were sent to surgery if they failed to respond to three days of nasogastric suction. However, in cases where the cause can be reversed (e.g. *H. pylori* or NSAIDs), a more conservative approach deserves consideration.[4]

The initial experience with endoscopic baloon dilatation (EBD) in patients with gastric outlet obstruction was with fluoroscopic guided balloon

catheters. However, with the advent of through-the-scope (TTS) balloon dilating catheters, EBD has become the first line of therapy in a majority of patients with non-malignant obstruction.[14] In the past EBD was associated with high long-term recurrence rate.[15,16] But nowadays, when *H. pylori* elimination making *H. pylory*-related ulcer recurrence unlikely, excellent antisecretory therapy can be offered, and there are a number of endoscopic techniques for dilating stenoses, long-term recurrence after EBD has been reportedly low.[17,18] Symptoms are usually improved with successful dilation to 12 mm. A regimen of gradual dilation over two or three sessions seems sensible. The largest diameter of stenosis at which symptoms occur is unclear. Many authorities recommend dilation to 15 mm, which is often associated with relief of symptoms. The presence of gastric atony also contributes to symptoms. The risk of perforation rises with the size of balloon. Almost all of the perforations in one series occurred after dilation with a 20 mm balloon.[3,5] A risk factor that predicts the need of surgery after EBD is the need of more than two courses of endoscopic balloon dilation to relieve symptoms.[19]

In order to facilitate dilatation and reduce recurrence, the use of intralesional steroid injections has been reported. Steroid injections have been shown to inhibit stricture formation by interfering with collagen synthesis, fibrosis and chronic scarring. It has been suggested that triamcinolone presents the cross linking of collagen that results in scar contracture; so if the scar is stretched and steroid injected into it, presumably the contracture will not occur. Steroids also decrease the fibrotic healing that appears after dilation.[2]

REFERENCES

1. Ramakrishnan K, Salinas RC. Peptic ulcer disease. Am Fam Physic 2007;76:1005-12.
2. Kochhar R, Kochhar S. Endoscopic balloon dilation for benign gastric outlet obstruction in adults. World J Gastrointest Endosc 2010;2:29-35.
3. Vakil N. Peptic ulcer disease. In: Feldman M, Friedman LS, Brandt LJ, Sleisenger MH, eds. Sleisenger & Fordtran's Gastrointestinal and Liver Disease. 9th ed. Philladelphia: Saunders Elsevier 2006.p.861-8.
4. Soll AH. Complications of peptic ulcer disease. In: Basow DS, ed. UpToDate. 18.2 ed. Waltham, MA; 2010 [cited Jan 5, 2011]. Available from: URL: http://www.uptodate.com/contents/complications-of-peptic-ulcer-disease.
5. Soll AH, Graham DY. Peptic ulcer disease. In: Yamada T, Alpers DH, Kalloo AN, Kaplowitz N, Owyang C, Powell DW, eds. Textbook of Gastroenterology. 5th ed. Oxford: Blackwell Pub 2009.p.936-66.
6. Moutzouris DA, Manetas S, Mountantonakis SE, Falagas ME. Please treat me with metoclopramide. Emerg Med J 2007;24:735-6.
7. Das AK, Patil V. Adult pyloric stenosis-a forgotten entity. Age Ageing 2006;35:448.
8. Siow SL, Wong CM, Sohail M. Adult pyloric stenosis masquerading as acute renal failure. Med J Malaysia 2009;64:168-9.
9. McCauley M, Gunawardane M, Cowan M. Severe metabolic alkalosis due to pyloric obstruction: case presentation, evaluation, and management. Am J Med Sci 2006;332:346-50.
10. Palanivelu C, Jani K, Rajan PS, Kumar KS, Madhankumar MV, Kavalakat A. Laparoscopic management of acid peptic disease. Surg Laparosc Endosc Percutan Tech 2006;16:312-6.
11. Kim SM, Song J, Oh SJ, Hyung WJ, Choi SH, Noh SH. Comparison of laparoscopic truncal vagotomy with gastrojejunostomy and open surgery in peptic pyloric stenosis. Surg Endosc 2009;23:1326-30.
12. McCallum RW, Polepalle SC, Schirmer B. Completion gastrectomy for refractory gastroparesis following surgery for peptic ulcer disease. Long-term follow-up with subjective and objective parameters. Dig Dis Sci 1991;36:1556-61.
13. Hom S, Sarr MG, Kelly KA, Hench V. Postoperative gastric atony after vagotomy for obstructing peptic ulcer. Am J Surg 1989;157:282-6.
14. Yusuf TE, Brugge WR. Endoscopic therapy of benign pyloric stenosis and gastric outlet obstruction. Curr Opin Gastroenterol 2006;22:570-3.
15. Kuwada SK, Alexander GL. Long-term outcome of endoscopic dilation of nonmalignant pyloric stenosis. Gastrointest Endosc 1995;41:15-7.
16. Lau JY, Chung SC, Sung JJ. Through-the-scope balloon dilation for pyloric stenosis: long-term results. Gastrointest Endosc 1996;43:98-101.
17. Cherian PT, Cherian S, Singh P. Long-term follow-up of patients with gastric outlet obstruction related to peptic ulcer disease treated with endoscopic balloon dilatation and drug therapy. Gastrointest Endosc 2007;66:491-7.
18. Rana S, Bhasin D, Chandail V, Gupta R, Nada R, Kang M. Endoscopic balloon dilatation without fluoroscopy for treating gastric outlet obstruction because of benign etiologies. Surg Endosc 2011;25:1579-84.
19. Peng CL, Lin HJ, Lo WC, Lai CR, Guo WS, Lee SD. Characteristics of patients with benign gastric outlet obstruction requiring surgery after endoscopic balloon dilation. Am J Gastroenterol 1996;91:987-90.

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