



An Interesting Finding in Upper Gastrointestinal Endoscopy

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Here we report a case of a 62-year-old man who presented to the gastrointestinal clinic during the past 6 months with symptoms of chronic abdominal pain, accompanied by dyspepsia and weakness. He suffered from type 2 diabetes and hypertension and had received drug treatment for both diseases. Over the past year, he had repeatedly taken non-steroidal anti-inflammatory drugs (NSAIDs) due to arthralgia without the use of gastric acid inhibitors. He refused the presence of melena or bleeding when describing his condition. Physical examination showed that he only had a sensitivity of the upper abdomen, and in blood tests, microcytic anemia with serum iron level 7.1 µg/dL and iron deficiency with serum ferritin level 10 ng/mL, and hemoglobin 10.1 g/L were detected. He was examined for gastrointestinal microcytic anemia and the symptoms. Esophagogastroduodenoscopy showed normal esophagus, erosive gastritis, normal duodenum, and Double pylorus. Colonoscopy was normal. The endoscope passed both channels separately and the bulb of the duodenum was seen. (figure 1). The patient's double pylorus was confirmed by contrast radiography (figure 2). Also, urea testing of rapid *Helicobacter pylori* was conclusive, while histological studies of the gastric corpus and antrum revealed that chronic active gastritis



Fig.1: Endoscopy of the patient's pyloric area



Fig.2: Contrast radiography of the patient

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was present in both and many *Helicobacter pylori* bacteria were found in H&E staining. The patient was treated to eradicate *Helicobacter pylori* with clarithromycin 500 mg, amoxicillin 1 g, and omeprazole 20 mg, twice daily for 14 days. Eradication was confirmed by a carbon-13 breath test, two months after treatment. Proton pump inhibitors were continued to be used once daily and NSAIDs use was restricted. Symptoms disappeared after a few weeks of treatment



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and the patient's anemia normalized after 6 months of treatment with the iron pill.

What is your diagnosis?

Answer: **Acquired double pylorus**

CONCLUSION

Double pylorus (DP) is a rare disease caused by a gastrointestinal fistula that spreads from the gastric antrum to the duodenal bulb. This rare disease may be acquired or congenital.^{1,2} The primary cause of congenital DP (CDP)¹ was described by Christine in 1971 and only a few cases have been reported since then. In the CDP, defects in pyloric canal formation appear in the initial stages of embryonic life.³

However, presented cases occur in gastric cancer or as a complication of gastric ulcer and as a result of the formation of a fistula between the antrum and the duodenal bulb or prepyloric region.⁴ This rare anomaly is observed in 0.001% to 0.4% of endoscopic cases.⁵

Acquired DP² alone has no specific clinical manifestation and can present with vomiting, abdominal pain, gastrointestinal bleeding, and dyspepsia⁶ as a result of concomitant gastric ulcer or other diseases.

The diagnosis of this lesion could be endoscopically considered. In the endoscopic examination, the antrum should adequately open by air to make the fistula visible through the thickened gastric folds. Most fistulas lie on the small curvature of the gastric antrum.

At endoscopy, the gastric antrum may appear normal¹ and in some cases, inflammation or scarring is also seen.⁷ Enough air needs to be blown into the stomach to open the gastric folds and see the fistula if present. The size of the fistula may vary from millimeters to a few centimeters, and in many patients, these fistulas are based in the small curvature of the upper wall of the duodenal bulb and the gastric antrum.²

Although the DP has a definite appearance at endoscopy, it may be easily misdiagnosed with gastric diverticulum or the double pylorus may be reported as a polyp, tumor, or a large mucosal fold in upper gastrointestinal endoscopy.

As reported in a study by Hu and colleagues² who followed patients with DP, the secondary pyloric canal remained in most cases (about 60% of patients) throughout the patient's lifetime; however, in some of the patients (25%), lateral pylorus was occluded or sometimes (only 5% of patients) a communication was established with the real pylorus and a canal was formed.²⁻⁸

Various mechanisms and reasons have been proposed for creating acquired pylorus. Some researchers believe that gastric mucosal microcirculation following systemic diseases such as diabetes, chronic pulmonary disease, rheumatoid arthritis, cirrhosis, systemic lupus erythematosus, and chronic kidney failure may be associated with acquired DP.^{9,10}

It has also been suggested that a long history of treatment with drugs, including corticosteroids and NSAIDs, can influence the course of gastric ulcer. *Helicobacter pylori* play an important role in the pathogenesis of duodenal ulcer and most gastric peptic diseases. *Helicobacter pylori* are potentially responsible for refractory and non-healing peptic ulcer cases.¹¹ The mentioned mechanisms or their combination can cause non-healing gastric ulcers and its complications over time and in rare cases cause fistula and pyloric duodenum. DP can rarely cause gastric outlet obstruction.¹²

Treatment of patients with DP should focus on removing the factors that affect mucosal healing of gastric ulcers. In some cases, endoscopic treatment may be performed using biliary sphincterotomy to divide the living bridge tissue. Surgical procedures should be used for patients who may not receive endoscopic treatment or patients with refractory symptoms such as recurrent ulcers or repeated bleeding with adequate drug treatment with proton pump inhibitors.¹³ Table 1 summarizes the patients' characteristics and features of the DP.

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There is nothing to be declared. (Informed consent was taken from the patient)

CONFLICT OF INTEREST

The authors declared no conflict of interest related to this research.

1. Congenital double pylorus
2. Acquired double pylorus

Table 1: Summary of the patients' characteristics with double pylorus

Patients' characteristics	Chronic abdominal pain, vomiting, dyspepsia, and gastrointestinal bleeding due to concomitant gastric ulcers or other diseases.
Risk factors	Use of drugs causing ulcers, adrenal adenoma, <i>Helicobacter pylori</i> , diabetes, chronic obstructive pulmonary disease, chronic kidney failure, Behcet's disease
Reasons (for causing the disease)	duodenal ulcer, gastric cancer, gastric ulcer
Incidence rate	0.001% to 0.4% of upper gastrointestinal endoscopy
Complications	Gastric outlet obstruction, refractory gastric ulcer
Laboratory findings	Blood biochemistry and complete blood count are usually normal, but in patients with gastrointestinal bleeding, complete blood counts may indicate anemia and blood biochemistry may indicate intravenous azotemia (e.g. increased normal creatinine and blood urea nitrogen). <i>Helicobacter pylori</i> test results are often positive.
Differential diagnosis	Gastric diverticulum
Treatment	Drugs, endoscopic or surgical treatment

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