A case of endoscopic ultrasound diagnosis of gastric amyloidosis

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A 37-year-old female presented to our hospital with the complaint of abdominal distension for one month. A gastroscopy examination showed extensive chyme retention and varying erosion and ulceration of the gastric body. The pathology showed moderate chronic inflammation and erosion of the gastric body mucosa, with accompanying Helicobacter pylori infection. She accepted treatment for one month. The patient then accepted to undergo endoscopic ultrasonography, which showed that normal structure of the gastric body intumescence was discernible. The pathology showed chronic mild-moderate inflammation of the gastric body mucosa associated with interstitial amyloidosis, with accompanying Helicobacter pylori infection. She accepted treatment for the eradication of Helicobacter pylori, and the original ulcer healed.

Key words: Endoscopic ultrasonography, gastric amyloidosis, Helicobacter pylori

INTRODUCTION

Amyloidosis is among a group of diseases caused by a variety of amyloid deposition in the interstitial space that causes damage to cells and organ function, affecting the heart, kidneys, gastrointestinal tract, skin, nervous system, and others. The digestive system is one of the most common encroachment sites of amyloidosis, and it was reported that the gastrointestinal tract was affected in approximately 50-70% of cases (1,2). However, amyloidosis that is limited only to the stomach is rare, and might be prone to misdiagnosis because of its various clinical manifestations and non-specific laboratory tests and imaging examinations. We describe a case of gastric amyloidosis diagnosed by endoscopic ultrasonography (EUS).

CASE REPORT

A 37-year-old female presented to our hospital with the complaint of abdominal distension for one month. In our hospital, she underwent a gastroscopy examination with biopsies, which showed extensive chyme retention and varying erosion and ulceration of the gastric body (Figure 1). The pathology showed chronic mild-moderate inflammation of the gastric body mucosa associated with interstitial amyloidosis, with accompanying Helicobacter pylori infection. She accepted treatment for the eradication of Helicobacter pylori, and the original ulcer healed.
She accepted treatment with proton pump inhibitors and gastric mucosal protective agents for one month. She presented to our hospital again for review, and the endoscopy showed multiple apophyses of the gastric body and gastric angle of unknown nature (Figure 2). The patient then accepted EUS with 12 MHz micro-probe, which showed that normal structure of the gastric body intumescence was discernible, but the boundaries between the hierarchical structures were unclear, the mucous layer was thick with low-echo structure, submucosa and muscularis propria were significantly thicker, and the thickest wall of the gastric body was approximately 1.63 cm (Figure 3). The pathology of the biopsy showed chronic mild-moderate inflammation of the gastric body mucosa associated with interstitial amyloidosis, accompanied by Hp infection (Figures 4, 5). The patient accepted treatment for the eradication of Hp, and the original ulcer healed. She has been under follow-up until the present, and no obvious symptoms have appeared repeatedly.

**DISCUSSION**

Amyloidosis was named in 1860, and is caused by abnormal deposition of specific glycoprotein fibers in the tissues and organs. It was named as such because of the similar reaction as when starch contacts iodine or sulfuric acid. There is no satisfactory classification of the disease at present. It could be divided according to systemic or localized type according to the tissue distribution, or as primary or secondary type according to whether an accompanying exact disease is determined; however, more and more scholars now claim that amyloidosis should be classified according to amyloid peptide constituents (3).
Amyloidosis is often associated with chronic disease such as rheumatoid arthritis, tuberculosis, multiple myeloma, chronic inflammation, and others (4). This patient was diagnosed as HP-positive in the first endoscopy. The pathology showed chronic inflammation of the gastric mucosa. After treatment for the eradication of HP, the original ulcer healed, and clinical symptoms disappeared. This suggested that the patient with amyloidosis could be associated with HP infection, which caused the chronic inflammation of the gastric mucosa. Veniniyoor et al. (5) reported a case with gastric mucosa-associated lymphoid tissue (MALT) lymphoma with simultaneous gastric amyloidosis, and the case was HP-positive; however, Veniniyoor considered that amyloidosis was not caused by the HP. Therefore, the relationship between HP infection and gastric amyloidosis needs to be studied further.

The majority of gastric amyloidosis is systemic amyloidosis performance in the stomach, and localized gastric amyloidosis is extremely rare. The common gastrointestinal manifestations of amyloidosis are gastroparesis, constipation, indigestion, intestinal pseudo-obstruction, and gastrointestinal bleeding, etc. (6). Because these symptoms are nonspecific and are common symptoms of digestive diseases, the clinical diagnosis is difficult and it is easily misdiagnosed. Routine gastroscopy shows fine granular mucosa and polypoid apophysis, mucosal erosion and ulceration, mucosal crisp and easy stripping, and mucosal wall thickening and stiffness due to diffuse amyloid infiltration (7), but as there are no characteristic features, gastric amyloidosis is easily confused with Borrmann type IV gastric cancer and gastric MALT lymphoma. EUS shows gastric mucosa and submucosal thickening, disappearance of normal levels and low echo in gastric amyloidosis. However, EUS shows all or part of the whole layer of diffuse gastric wall thickening, mostly above 1 cm, especially the submucosa and muscularis propria, structural damage, and diffuse hypoechoic or heterogeneous echo in Borrmann type IV gastric cancer and gastric MALT lymphoma. Because EUS has been reported to diagnose amyloidosis with low specificity, there have been few such cases, and we lack experience and relevant statistical data on gastric amyloidosis. Final diagnosis of this disease required pathological examination, which showed amyloid deposits, and Congo red staining was positive. Amyloid deposition of the vessel wall in the submucosal tissue is the most obvious, so the endoscopic biopsy needs to be deep enough, while EUS can improve the positive rate of biopsy.

The prognosis of amyloidosis is poor, and the median survival after diagnosis is approximately 13.8 months (8). There are currently no specific treatment options for amyloidosis, but there are some drugs to treat the disease, including melphalan, cyclophosphamide, dimethyl sulfoxide, and colchicine, though the effects are not ideal. Surgery and lymph node cleaning may be the best treatment strategy for localized amyloidosis and prevention of complications (4). The latest report showed the lesions of gastric amyloidosis had completely disappeared after autologous stem cell transplantation (9); thus, autologous stem cell transplantation may be a new direction for future treatment.

Figure 4. Hematoxylin-eosin staining showed amyloidosis of the gastric body mucosa (10x10).

Figure 5. Congo red staining showed amyloidosis of the gastric body mucosa (10x10).
REFERENCES


