An uncommon lesion: Gastric Xanthelasma

Yayğın olmayan bir lezyon: Gastrik Ksantalezma

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Although the clinical significance of gastric xanthelasmas is unclear, they are important lesions because they may be confused with malignant lesions. The etiopathogenesis is also unclear, but chronic gastritis, Helicobacter pylori (H. pylori) infection, diabetes mellitus and hyperlipidemia have been implicated. Xanthelasma is more frequent in women and its incidence increases with age. The lesions are frequently located in the stomach, and less frequently in the esophagus, duodenum and the colon. The lesions have a yellowish-white appearance, are between 0.5 and 10 mm in size and can be single or multiple. Xanthelasmas were found to be associated with chronic gastritis, gastrointestinal anastomoses, intestinal metaplasia, and H. pylori infection. These lesions are predisposing conditions for gastric cancer. Therefore, endoscopic biopsy is mandatory and careful follow-up is required. In this paper, four patients who attended hospital with abdominal pain and dyspepsia and by chance were found to have xanthelasmas on endoscopic examination are presented, and gastric xanthelasmas are discussed.

Key words: Gastric xanthelasma, intestinal metaplasia, malignancy

INTRODUCTION

Xanthelasmas of the gastrointestinal tract are uncommon benign lesions. Chronic gastritis, Helicobacter pylori (H. pylori) infection, diabetes mellitus and hyperlipidemia have been implicated in the etiopathogenesis. Because xanthelasmas may be confused with malignant lesions, endoscopic biopsy is mandatory. In this paper, four patients who attended hospital with abdominal pain and dyspepsia and by chance were found to have xanthelasmas on endoscopic examination are presented.

Case 1

A 59-year-old man applied with abdominal pain and weight loss. He had experienced one episode of vomiting ingested food 10 days previously. He had bilateral truncal vagotomy and pyloroplasty for gastric ulcer 25 years ago. Physical examination was unremarkable except for a vertical upper midline surgical scar and an incisional hernia. The results of the complete blood count, standard biochemical analyses and thyroid function tests were within normal limits. Upper gastrointestinal endoscopy revealed decreased gastric peristalsis, erythema and edema on the gastric mucosa. There was a 10x10 mm, yellowish-white plaque on the anterior wall of the distal corpus and a 6x7 mm ulcer on the incisura angularis. Histopathological examination of the plaque revealed dense histioyte accumulation that led to the diagnosis of xanthelasma. The microscopic findings of the biopsies from the ulcer were compatible with a benign ulcer. H. pylori was not detected.

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Case 2
A 35-year-old man applied with epigastric pain, burning and occasional diarrhea with yellowish stools not containing blood or mucus. Findings of physical examination, complete blood count and biochemical tests were normal. Endoscopy revealed stage A peptic esophagitis (Los Angeles classification), and three slightly elevated, yellowish-white plaques (largest size 7x8 mm) at the cardia near the Z –line (Figure 1). There was a 5x5 mm ulcer on the anterior wall of the bulbis. Biopsies were taken from the lesions at the cardia. Histopathological examination revealed multiple foamy macrophages in the lamina propria. *H. pylori* was not observed.

Case 3
A 70-year-old woman attended hospital with abdominal pain radiating to the back. She had hypertension for 30 years and had Billroth II operation 20 years ago. Her blood pressure was 140/80 mm Hg. A vertical upper midline incision and epigastric tenderness were noted. The erythrocyte sedimentation rate and the results of the complete blood count and standard biochemistry tests were within normal limits. Endoscopic examination demonstrated a 15x15 mm slightly elevated, yellowish-white plaque just near the gastrointestinal anastomosis. Biopsy specimens showed histiocytes with foamy cytoplasms filling the lamina propria. *H. pylori* was not observed.

Case 4
An 80-year-old woman attended hospital with abdominal pain and fatigue. She had been treated for one year for type 2 diabetes mellitus regulated by diet. In physical examination, conjunctivae were pale and she had epigastric tenderness. Her hemoglobin level was 11.6 g/dl and the erythrocytes were macrocytic. The results of biochemical examinations were within normal limits. Upper gastrointestinal endoscopic examination revealed two slightly elevated, irregularly-edged, yellowish-white lesions (7-8 mm) on the posterior wall of the distal corpus and incisura angularis; the remaining mucosa was atrophic (Figure 2). Histopatho-
logical examination showed adenoid structures including goblet cells among multiple foamy macrophages, thus diagnosis was gastric xanthelasma and intestinal metaplasia (Figure 3).

DISCUSSION

Although the clinical significance of gastric xanthelasmas is unclear, they are important lesions because they may be confused with malignant lesions. The incidence of upper-gastrointestinal xanthelasmas was reported as 0.23% (1). Approximately 76% of the lesions are located in the stomach, particularly the antrum and the pyloric region (70%); they occur less frequently in the esophagus (12%), duodenum (12%) and the colon (1-4). Most of the reported lesions were located in the antrum and the pylorus, whereas in the present report one of the lesions was in the cardia and the others in the corpus. Xanthelasma is more frequent in women and its incidence increases with age. The lesions have a yellowish-white appearance, are between 0.5 and 10 mm in size and are multiple in 13 to 24% of the cases; 17% of the patients have more than five lesions (5). In our study, the lesions in the cardia were multiple and one of the lesions in the corpus was larger than those reported before.

The etiopathogenesis is unclear. The detection of H. pylori antigens in the cytoplasms of xanthelasma cells in some studies led to the hypothesis that these lesions may be initiated by H. pylori infection (6, 7). In contrast to these reports, H. pylori could not be demonstrated in our patients although one had a duodenal ulcer. The prevalence of H. pylori infection is high in our country, so we think that the existence of H. pylori infection and gastric xanthelasma may be coincidental. Because histochemical characteristics resemble those of skin lesions, a possible relationship with lipid metabolism has been investigated, but no obvious association with lipid metabolism disorders or hypercholesterolemia was found (2, 8, 9). The lipid profiles of our four patients were within normal limits. Fasting blood glucose levels of the patients were within normal limits; only one patient had mild diabetes mellitus controlled by dietary restriction. In our study no etiological agent could be identified in three patients; one patient had mild type II diabetes.

In various studies, the gastric glands around the lesions were found to exhibit moderate-severe atrophy (89%) and intestinal metaplasia (13%) (2, 7, 10). Xanthelasmas were also found to be associated with chronic gastritis and gastrointestinal anastomoses (10, 11). Similarly, we determined that one of the patients had atrophic gastritis; the other had chemical gastritis due to Billroth II operation. The lesion was near the anastomosis in one patient. No malignant change was detected in any patient; however, glandular structures suggesting intestinal metaplasia were noted among foamy macrophages in one patient. This finding suggests that xanthelasma may be confused with or accompany intestinal metaplasia, thus these lesions should be differentiated from each other by biopsies since intestinal metaplasia is a premalignant lesion.

The endoscopic appearances of gastric xanthelasmas may be confused with gastric carcinoid tumors. Histologically, xanthelasmas are composed of uniform polygonal cells with foamy cytoplasm. The ‘packaged’ appearance of these cells and the vascular stroma may resemble neuroendocrine tumors; thus these lesions should be differentiated from clear cell carcinoids of the stomach. This has to be confirmed by immunoreactivity for chromogranin A and the demonstration of dense core granules ultrastructurally (12). Muraoka et al. (13) observed an association between type IIa early gastric cancer and xanthoma and speculated that cancer cells may have caused xanthoma cell proliferation via an autocrine mechanism. In other studies, it was recommended that xanthelasma should be differentiated from signet ring cell carcinoma by histochemistry and immunohistochemistry (14, 15). Atrophic gastritis, intestinal metaplasia, H. pylori infection and chronic gastritis, which are predisposing conditions for gastric cancer, may accompany xanthelasmas.

In conclusion, although the clinical significance of gastric xanthelasmas is unclear, similarities with malignancies and association with premalignant lesions require endoscopic biopsy and histopathological examination.

REFERENCES


