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CASE REPORT

Diagnosis of autoimmune gastritis by high resolution magnification endoscopy

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Abstract

Endoscopic visualisation of gastric atrophy is usually not feasible with conventional endoscopy. Magnifying endoscopy is helpful to analyze the subepithelial microvascular architecture as well as the mucosal surface microstructure without tissue biopsy. Using this technique we were able to describe the normal gastric microvasculature pattern and we also identified characteristic patterns in two cases of autoimmune atrophic gastritis.

Key words: Magnification endoscopy; Autoimmune gastritis; Collecting venules; Subepithelial capillary network

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INTRODUCTION

Endoscopic visualisation of gastric atrophy is usually not feasible with conventional endoscopy. Except for the absence of rugae and visible vessels in the gastric corpus, macroscopic features, as observed during gastroscopy, are of very limited value in the evaluation of the presence of gastric atrophy[1]. Although histology may be considered as a gold standard for detection of gastric atrophy, neither the original nor the revised version of the Sydney system reliably identifies more than half of the cases in patients with confirmed atrophy[2].

Magnifying endoscopy is a helpful procedure to analyze the subepithelial microvascular architecture as well as the mucosal surface microstructure without tissue biopsy[3]. Using this technique we were able to observe the normal gastric microvasculature pattern and we also identified characteristic patterns in two cases of autoimmune atrophic gastritis.

CASE REPORT

Two male patients (aged 53 and 62 years), underwent upper gastrointestinal endoscopy due to anaemia and low serum B12 levels. Endoscopy was performed using the GIF-Q240Z (Olympus, Keymed, UK) high-resolution magnifying endoscope (× 115 on a 20" screen, 7.9 μm resolution). Prior to endoscopy, a soft black hood was mounted on the tip of the scope, to enable the endoscopist to fix the focal distance at 2 mm between the tip of the scope and the gastric mucosa. Ordinary endoscopic findings were normal, however, magnified observation of the gastric mucosa showed normal gastric antral microvasculature (coil-shaped subepithelial capillary network (SECN) pattern) (Figure 1A), with disappearance of the normal honeycomb-like SECN and irregular collecting venules and areas of tubular structures in the gastric body mucosa (Figures 1B). Targeted biopsies from the gastric body showed glandular atrophy, intestinal metaplasia and inflammatory infiltrate in the gastric corpus, sparing the antrum, as in autoimmune gastritis (Figure 1C). On the other hand, the histological findings in the biopsied specimens from the antral mucosa showed no pathological changes. Histology showed no Helicobacter pylori micro-organisms. Serum anti-parietal cell antibodies were positive.

DISCUSSION

Autoimmune gastritis is characterized by autoimmune destruction of fundic and body glands. The marker for the most severe, end-stage form of diffuse corporal atrophic gastritis is pernicious anemia[4]. However, no endoscopic findings specific for this entity have been reported.

The microvascular pattern of human gastric mucosa has recently been investigated[5-7]. The normal gastric body and fundus mucosal microvessels show two major components: (1) subepithelial capillaries; and (2) collecting venules. Polygonal loops of subepithelial capillaries surround the neck of gastric pits. These loops form a honeycomb-like SECN and converge onto mucosal collecting

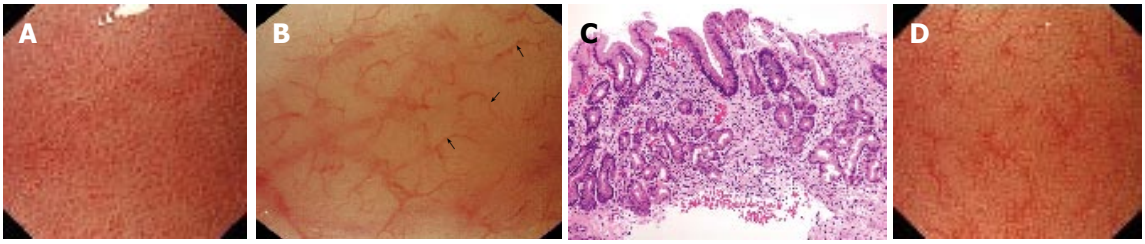

Figure 1 **A**: Magnified view of gastric antral mucosa in our patient. All parts of the antral mucosa demonstrated a coil-shaped subepithelial capillary network (SECN) in regular arrangement; **B**: Magnified endoscopic view of the gastric body mucosa in our patient. Loss of the normal SECN and collecting venules (arrows) in irregular shape and arrangement were evident. These findings are compatible with those of gastric atrophy; **C**: Histology reveales decreased density of glands, loss of specialised glands in the gastric body; **D**: Magnified view of normal gastric body mucosa in another healthy patient Honeycomb-like SECN with collecting venules in regular arrangement can be seen clearly.

venules that drain down into the submucosa (Figure 1D). In the antral mucosa, the normal subepithelial capillaries form a coil-shaped network, while collecting venules are rarely seen.

Yao *et al* have demonstrated that the resolution of the GIF-Q240Z gastroscope is 7.9 μm. Since the minimal diameter of subepithelial capillaries in the gastric mucosa, as described in anatomic studies, is 8 μm, this endoscope is ideally suited to visualize microvessels and capillaries[6].

Nakagawa *et al* have recently studied the usefulness of magnifying endoscopy for the diagnosis of *H pylori*-induced histopathologic gastritis[8]. The observed morphology of collecting venules was divided into the 3 patterns: (1) regular (R), which had the qualities of regularity in venules size, visible second or third order branches, and a uniform distance between venules; (2) irregular (I), which had the qualities of irregularity in size, inability to observe second or third order branches, and a lack of a uniform distance between the collecting venules, with venules sometimes fused to adjacent venules and sometimes lying horizontally; and (3) obscured (O), in which no collecting venules were visible. Observation of an R pattern indicates an absence of *H pylori* infection and histopathologic gastritis. Observation of an O or I pattern indicates the presence of histopathologic gastritis from *H pylori* infection, and an I pattern suggests the presence of gastric mucosal atrophy. We have also shown that in cases of gastric atrophy associated with *H pylori* infection, the normal honeycomb-like subepithelial capillary network (SECN) in gastric body disappears and the collecting venules become irregular[9].

The magnified endoscopic findings of diffuse atrophy in gastric body mucosa and normal antrum associated with autoimmune gastritis have not yet been described. In our cases, the magnified views in gastric antrum revealed a normal coil-shaped subepithelial capillary network. On the other hand, in gastric body mucosa we demonstrated loss of the normal honeycomb-like SECN pattern with irregular collecting venules, suggestive of corporal gastric atrophy, as well as areas with tubulovillous structures as seen in intestinal metaplasia. These magnified endoscopic findings were constantly detected in all parts of the gastric corpus that were examined. These patterns are suggested to be characteristic of autoimmune gastritis and were confirmed by histology.

In summary, it seems that irregular collecting venules can be recognised both in autoimmune- and *H pylori*-associated gastric atrophy. The dissemination of this finding can be used to help differentiate between these two types of gastritis. Gastric body predominant atrophy visualised by magnifying endoscopy, can be very useful in making a precise diagnosis of autoimmune gastritis.

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