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Review Article

An Endoscopic Impression of Gastric Antral Vascular Ectasia Amidst Multifocal Gastrointestinal Inflammatory Findings: A Case Report

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ABSTRACT

Gastric antral vascular ectasia (GAVE), commonly referred to as watermelon stomach, is an uncommon vascular disorder of the stomach that may present with chronic occult gastrointestinal blood loss, iron-deficiency anaemia or overt upper gastrointestinal bleeding. It is an uncommon vascular abnormality of the gastric antrum, primarily recognised by its characteristic endoscopic appearance. Chronic active gastritis associated with *Helicobacter pylori* represents a distinct inflammatory gastric condition, while colitis involves inflammation of the colon. A 40-year-old woman presented with abdominal pain, multiple episodes of loose stools, and vomiting for two days. Initial clinical assessment suggested colitis and acute peptic disease. Computed tomography (CT) of the abdomen and pelvis revealed circumferential mural thickening of the pyloric region and first part of the duodenum, along with long-segment thickening of the ascending colon associated with pericolonic fat stranding and prominent adjacent vessels, suggestive of an inflammatory or infective process. Subsequent oesophagogastroduodenoscopy revealed a focal area of abnormal mucosa in the gastric antrum, with an endoscopic impression of GAVE. However, histopathological examination of the antral biopsy demonstrated chronic active gastritis with *H. pylori*-like organisms on the mucosal surface and within the gastric pits. The rapid urease test was also positive, with no evidence of intestinal metaplasia, dysplasia, or malignancy.

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This case highlights the coexistence of an endoscopic impression of GAVE, histologically confirmed *H. pylori*-associated chronic active gastritis, and radiological findings suggestive of ascending colitis. It emphasises the importance of correlating clinical, radiological, endoscopic, and histopathological findings, particularly when the endoscopic impression and biopsy diagnosis differ.

INTRODUCTION

Gastric antral vascular ectasia (GAVE), commonly referred to as watermelon stomach, is an uncommon gastrointestinal vascular abnormality characterised by ectatic mucosal and submucosal vessels predominantly involving the gastric antrum. It was first described by Rider and colleagues in 1953. The term watermelon stomach was subsequently introduced by Jabbari and colleagues in 1984, following recognition of the characteristic longitudinal erythematous stripes radiating from the pylorus across the antral mucosa [1,3] . GAVE is an established cause of gastrointestinal blood loss and may present with chronic iron-deficiency anaemia or overt gastrointestinal haemorrhage. Its diagnosis is primarily based on characteristic endoscopic appearances, with histopathological examination providing supportive evidence in selected cases [3–6]. The characteristic endoscopic patterns include longitudinal erythematous stripes radiating from the pylorus, classically described as the watermelon appearance, and a diffuse punctate or honeycomb pattern. Histologically, GAVE may demonstrate mucosal vascular ectasia, fibrin thrombi, spindle-cell proliferation, and fibrohyalinosis [3,6,7]. Differentiation from portal hypertensive gastropathy is clinically important because the two entities differ in their underlying pathophysiology and management [3,4]. Chronic active gastritis is a histopathological diagnosis characterised by chronic inflammatory cell infiltration, predominantly lymphocytes and plasma cells, accompanied by neutrophilic activity. Helicobacter pylori is a major aetiological factor associated with chronic active gastritis and is implicated in several gastroduodenal disorders. Histological identification of the organism and a positive rapid urease test provide complementary evidence supporting active infection [10–14]. Colitis represents inflammation of the colonic wall and may result from infectious, inflammatory, ischaemic, or other pathological processes. Cross-sectional imaging may demonstrate bowel-wall thickening, mural oedema, pericolonic fat stranding, and regional lymphadenopathy. However, these findings are not specific to a single aetiology and require correlation with clinical, laboratory, microbiological, and endoscopic findings [15–18]. Management of GAVE depends on the severity of bleeding, haemoglobin levels, associated comorbidities and the patient's clinical condition. Initial management generally involves correction of anaemia through iron supplementation and blood transfusion when clinically indicated. Endoscopic treatment is considered the preferred approach for patients with persistent or recurrent bleeding. Argon plasma coagulation (APC) is one of the commonly used endoscopic techniques, in which thermal energy is applied to the abnormal vascular areas to achieve coagulation and reduce bleeding. Other therapeutic options include endoscopic band ligation and radiofrequency ablation, which may be considered in selected patients, particularly when conventional treatment provides an inadequate response [4–6] . Despite treatment, GAVE may have a recurrent or chronic course, requiring repeated endoscopic assessment and monitoring of haemoglobin and iron parameters. Surgical management is rarely required and is generally reserved for patients with severe, persistent bleeding who do not respond adequately to medical and endoscopic interventions. Antrectomy, which involves surgical removal of the gastric antrum, has been described as a definitive treatment option in selected refractory cases. Follow-up is particularly important in patients with persistent anaemia or recurrent gastrointestinal bleeding[6].

CASE REPORT

A 40-year-old woman presented with abdominal pain, multiple episodes of loose stools, and vomiting of two days' duration. The initial clinical assessment documented colitis and acute peptic disease. Abdominal examination revealed epigastric tenderness. Considering the presenting gastrointestinal symptoms, further investigations, including computed tomography (CT) of the abdomen and pelvis and upper gastrointestinal endoscopy, were performed.

Plain CT of the abdomen and pelvis, reveals smooth circumferential mural thickening involving the gastric

pylorus (approximately 5 mm) and the first part of the duodenum (D1) (approximately 5.6 mm), suggestive of an inflammatory process. Long-segment apparent mural thickening of the ascending colon, measuring approximately 5.5 mm, was also observed, accompanied by pericolonic fat stranding and prominent adjacent vessels, suggestive of colitis of possible inflammatory or infective aetiology. A few subcentimetric mesenteric lymph nodes were identified in the central abdomen and right iliac fossa, with the largest measuring approximately 5.7 mm in short-axis diameter, possibly representing reactive lymph nodes. The liver, gallbladder, pancreas, spleen, kidneys, urinary bladder, and uterus were unremarkable. No ascites or free intraperitoneal air was identified.

Subsequent oesophagogastroduodenoscopy demonstrated normal findings in the oesophagus, gastro-oesophageal junction, gastric fundus, body, pylorus, and duodenum. However, a focal area of abnormal mucosa was identified in the gastric antrum, from which a biopsy specimen was obtained. The endoscopic impression was gastric antral vascular ectasia (GAVE) (Figure 1). A rapid urease test performed on the antral biopsy specimen was positive, suggesting Helicobacter pylori infection.

Histopathological examination of the gastric antral biopsy revealed gastric antral-type mucosa with focal denudation of the foveolar epithelium and oedematous lamina propria showing moderate-to-dense inflammatory cell infiltration, predominantly composed of lymphocytes and plasma cells, along with a few neutrophils and occasional eosinophils. Features of active inflammation were evident, and H. pylori-like organisms were identified on the mucosal surface and within the gastric pits. No intestinal metaplasia, epithelial dysplasia, or malignancy was identified in the sections examined. The final histopathological diagnosis was chronic active gastritis of the gastric antrum associated with H. pylori-like organisms. The overall clinical and diagnostic findings demonstrated coexisting gastrointestinal abnormalities, including an endoscopic impression of GAVE, histopathologically confirmed chronic active antral gastritis associated with H. pylori-like organisms, and radiological findings suggestive of ascending colitis. Although GAVE was suspected based on the endoscopic appearance, the available biopsy report documented chronic active gastritis without describing characteristic histological features of GAVE. Therefore, GAVE is considered an endoscopic impression rather than a histopathologically confirmed diagnosis.

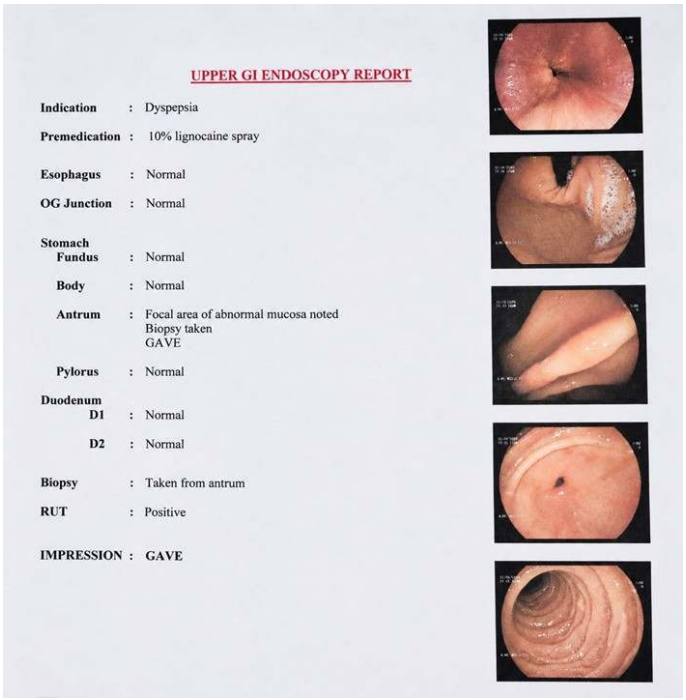


Figure 1: ***Upper Gastrointestinal endoscopic appearance of GAVE in the gastric antrum***

DISCUSSION

The present case illustrates the identification of multiple gastrointestinal abnormalities during the evaluation of a 40-year-old woman presenting with acute abdominal symptoms. The diagnostic findings involved the gastric antrum, gastroduodenal region, and ascending colon, highlighting the importance of correlating radiological, endoscopic, and histopathological findings.

Gastric antral vascular ectasia (GAVE) is a recognised vascular lesion of the stomach, predominantly involving the antral mucosa. It is an established cause of chronic gastrointestinal blood loss and may present with iron-deficiency anaemia, occult gastrointestinal bleeding, or overt haemorrhage. Its characteristic endoscopic appearance includes longitudinal erythematous vascular stripes radiating from the pylorus or a diffuse punctate pattern involving the antral mucosa. Histopathological features may include dilated mucosal capillaries, fibrin thrombi, spindle-cell proliferation, and fibrohyalinosis. In the present case, upper gastrointestinal endoscopy identified a focal area of abnormal antral mucosa, with an endoscopic impression of GAVE. However, the subsequent antral biopsy demonstrated chronic active gastritis with H. pylori-like organisms rather than explicitly documented vascular ectasia or fibrin thrombi.

The antral biopsy also demonstrated oedematous lamina propria with moderate-to-dense lymphoplasmacytic infiltration and accompanying neutrophilic activity, consistent with chronic active gastritis. The identification of H. pylori-like organisms on the mucosal surface and within the gastric pits, together with a positive rapid urease test, provides concordant evidence supporting H. pylori infection. The updated Sydney System provides a standardised framework for the histopathological assessment of gastritis, incorporating the distribution and severity of chronic inflammation, neutrophilic activity, glandular atrophy, intestinal metaplasia, and H. pylori density. The findings in this patient are consistent with the reported diagnosis of chronic active gastritis. The absence of intestinal metaplasia, epithelial dysplasia, and malignancy is also noteworthy, indicating that none of these changes was identified in the tissue sections examined. Current international consensus recommendations and the American College of Gastroenterology guideline emphasise the importance of identifying and treating confirmed H. pylori infection, followed by confirmation of eradication using an appropriate test. In addition to the gastric findings, CT examination demonstrated long-segment mural thickening of the ascending colon, accompanied by pericolonic fat stranding and prominent adjacent vessels. A few subcentimetric mesenteric lymph nodes were also observed and considered possibly reactive. Bowel-wall thickening on CT may arise from inflammatory, infectious, ischaemic, neoplastic, or other causes. Its interpretation depends on the extent, distribution, degree of mural thickening, enhancement pattern, and associated mesenteric or pericolonic changes. Pericolonic fat stranding and mural thickening may be encountered in infectious and inflammatory colitis, although neither finding is specific to a particular organism or disease. In the present case, the radiological findings were interpreted as suggestive of ascending colitis, possibly of inflammatory or infective aetiology. Clinical correlation and, where indicated, stool microbiological investigations, inflammatory markers, and lower gastrointestinal endoscopic evaluation with biopsy may help determine the underlying aetiology. Therefore, the available diagnostic evidence supports the description of radiologically suspected ascending colitis rather than a definitive diagnosis of infectious colitis or inflammatory bowel disease.

The coexistence of an endoscopic impression of GAVE, histologically demonstrated chronic active gastritis with H. pylori-like organisms, and radiological evidence suggestive of ascending colitis provides a clinically relevant diagnostic combination. This case demonstrates the importance of distinguishing between an endoscopic impression and a tissue-based diagnosis. Accurate integration of clinical, radiological, endoscopic, and histopathological evidence is essential for appropriate diagnostic interpretation and scientific reporting.

CONCLUSION

This case describes a 40-year-old woman presenting with acute gastrointestinal symptoms in whom upper gastrointestinal endoscopy suggested gastric antral

vascular ectasia (GAVE). Histopathological examination of the antral biopsy established chronic active gastritis associated with H. pylori-like organisms, without evidence of intestinal metaplasia, epithelial dysplasia, or malignancy in the sections examined. A positive rapid urease test provided additional evidence supporting H. pylori infection. Concurrent abdominal CT demonstrated long-segment ascending colonic mural thickening with pericolonic inflammatory changes, suggestive of colitis of possible inflammatory or infective aetiology.

This case highlights the importance of integrating radiological, endoscopic, and histopathological findings while maintaining diagnostic precision. In particular, the endoscopic impression of GAVE should be distinguished from the histopathological diagnosis of chronic active gastritis, as the available biopsy did not document characteristic vascular changes. This distinction emphasises the importance of correlating different diagnostic modalities to ensure accurate clinical interpretation and reporting.

The manuscript has been structured in accordance with the principal components of the CARE case-reporting framework. Additional clinical information, including relevant medical and medication history, laboratory investigations, stool investigations, treatment details, clinical timeline, follow-up findings, and final clinical outcome, should be incorporated wherever available before journal submission. Endoscopic photographs demonstrating the suspected GAVE pattern and any subsequent investigations or interventions would further strengthen the report.

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