# Gastric antral vascular ectasia: A case report and literature review

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## **ABSTRACT**

Gastric antral vascular ectasia (GAVE) is a rare but an important cause of upper gastrointestinal bleeding (UGIB) and commonly presents as occult bleeding that manifests as iron deficiency anemia (IDA). GAVE is commonly associated with chronic illnesses, most frequently liver cirrhosis and connective tissue diseases. The pathogenesis of GAVE is still obscure, and many hypotheses such as mechanical stress, hormonal factors, and autoimmune factors, have been proposed. Upper gastrointestinal endoscopy has a major role in the diagnosis and treatment of GAVE.

Key words: endoscopy, gastric antral vascular ectasia, gastrointestinal bleeding

### **INTRODUCTION**

Gastric antral vascular ectasia (GAVE) is a rare cause of upper gastrointestinal bleeding (UGIB), accounting for about 4% of non-variceal UGIB and commonly presents as occult bleeding that manifests in iron deficiency anemia (IDA). GAVE is commonly associated with chronic illnesses, most frequently liver cirrhosis and connective tissue diseases.<sup>[1]</sup> In this report, we present one case of GAVE, discussing the etiology, endoscopic features, histology, pathogenesis, and optimal management for this rare entity.

# **CASE REPORT**

A 76-years-old male patient, known to have multiple medical problems, was admitted electively in late July 2013 for further work up for symptomatic anemia. Patient was complaining of fatigability, dizziness, and occasional black stool for about 6 months before admission. No history of hematemesis or rectal bleeding was noted. Patient's past medical history includes hypertension, diabetes mellitus, dyslipidemia, ischemic heart disease, atrial fibrillation, congestive heart failure, pulmonary hypertension, severe tricuspid valve regurgitation, chronic kidney disease, benign prostatic hypertrophy, and vitiligo. His past surgical history was significant

for bilateral total knee replacement for osteoarthritis and cataract surgery. His medications are tamsulosin, acetaminophen, darbepoetin alpha, folic acid, esomeprazole, metoprolol, warfarin, and torsemide. Family history was unremarkable. He is a nonsmoker and nonalcohol drinker.

Upon examination, he looked pale and lethargic but with stable vital signs and without postural hypotension. Unremarkable abdominal examination was noted, and digital rectal examination revealed no melena at this time. The cardiovascular examination revealed slow atrial fibrillation, a pansystolic murmur, a prominent jugular venous pressure, and bilateral lower limb edema. The chest examination was remarkable, with signs of right pleural effusion. Skin examination showed vitiligo.

At the time of present admission, laboratory data revealed low hemoglobin (7.6 g/dL) and hematocrit (0.242) with normal mean corpuscular volume. Platelets and white blood cells count were normal. Serum iron study showed low serum iron and ferritin levels. Vitamin B12 and red blood cell (RBC) folate levels were normal. Serology for celiac disease was negative. Urea and creatinine levels were elevated, 21 mmol/L and 192 µmol/L, respectively. Liver profile was normal, and his international normalized

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ratio (INR) was therapeutic at 2.5. Before admission, he has undergone upper and lower endoscopy. Upper endoscopy was reported as mild to moderate gastritis involving the antrum and the distal body (Figure 1) and the gastric biopsy from the antrum was reported as mild *Helicobacter pylori*negative gastritis. Colonoscopy revealed only diminutive polyp in the sigmoid colon. RBC nuclear scan was negative.

During admission, the patient received blood transfusion, and his heart failure treatment optimized then capsule endoscopy (CE) was done. CE showed multiple red punctuate lesions around the pylorus (Figure 2) suggestive of angioectasias or GAVE. Small bowel was looking normal on CE. A repeat gastroscopy was performed and confirmed endoscopic diagnosis of GAVE. Active bleeding was noted from the lesions during endoscopy. Argon plasma coagulation (APC) was applied and good homeostasis was achieved. Warfarin was held temporarily during active bleeding episodes and during endoscopic therapy but was resumed thereafter. His hemoglobin was stable over a period of 2 months and follow up at 10 g/ dL; then he developed melena and drop in hemoglobin level to 8.4 g/dL and required three sessions of APC before stabilization of the hemoglobin level at 11.7 g/dL. No further gastrointestinal (GI) bleeding event was noted until writing this article.

## **DISCUSSION**

GAVE was reported for the first time by Rider *et al.* in 1953,<sup>[2]</sup> and since then, multiple cases were reported with more understanding of its clinical and endoscopic features. Although GAVE is a rare cause of UGIB, it can cause significant and serious GI bleeding specially in the elderly patients with multiple medical problems. GAVE affects most commonly females (71%), with an average age of 73 years at presentation. GAVE usually presents with IDA (89% of patients) due to chronic blood loss but occasionally causes severe acute GI bleeding.<sup>[3]</sup>

GAVE is usually associated with chronic illnesses, most commonly liver and connective tissue diseases. Liver cirrhosis has been found in 30% of the cases. It has also been reported to be common in scleroderma and calcinosis, Raynaud phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia syndrome. Case reports of GAVE in patients with essential hypertension, chronic renal failure, acute myeloid leukemia and GAVE in patients who have undergone bone marrow transplant have been published.

In the literature, two characteristic endoscopic appearances of GAVE have been reported. First is the diffuse punctuate lesions in the antrum, similar to that observed in our patient (Figure 2). This type of GAVE is typically seen in male, patients with cirrhosis and commonly accompanied by acute bleeding. Our patient is a male, does not have liver disease, and is presented with chronic blood loss rather than acute bleeding. Second one is the red lesions organized in stripes radially departing from the pylorus, known as watermelon stomach (Figure 3). This type is mostly common in females with connective tissue diseases and usually present with occult bleeding.<sup>[10-11]</sup>

GAVE is typically located in the gastric antrum; however, it may be also found rarely in other areas of the GI tract, including cardia, <sup>[12-13]</sup> duodenum, jejunum, <sup>[14]</sup> and rectum <sup>[15-16]</sup> The involvement of the proximal part of the stomach is very rare and commonly located within a diaphragmatic hernia. <sup>[17,18]</sup>



Figure 1: Upper endoscopic image labeled as gastritis.

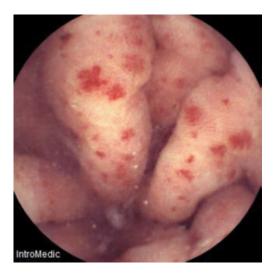


Figure 2: Picture obtained with capsule endoscopy (CE): multiple red punctuate lesions around the pylorus.

The main differential diagnosis of GAVE on endoscopy is portal hypertensive gastropathy (PHG) and antral gastritis. PHG is typically more predominant in the fundus and the body of the stomach, whereas GAVE mainly involves the antrum. Furthermore, severe PHG can extend up to the antrum and can resemble GAVE. GAVE can be easily confused with gastritis on upper GI endoscopy; however, the histological pattern of GAVE, although not pathognomonic, can differentiate between the two entities. On histology, GAVE is characterized by vascular ectasia of the mucosal capillaries, focal thrombosis, spindle cell proliferation (smooth muscle cell and myofibroblast hyperplasia), and fibrohyalinosis, which consist of homogeneous substance around the ectatic capillaries of the lamina propria (Figure 4). [19-21]

The peculiar thing in our patient is that GAVE was misinterpreted as gastritis on upper GI endoscopy and the diagnosis was detected through CE. Sidhu *et al.* reported similar scenario in six patients.<sup>[18]</sup> It is not clear why the diagnosis in our patient was missed on an initial upper GI endoscopy that was performed by an expert endoscopy consultant. One possible explanation of this is the variation in endoscopic appearances of GAVE over time, possibly related to blood flow, hemoglobin concentration, or synchronous other gastric pathology such as gastritis. CE can be regarded as "physiologic" endoscopy, without the need for air insufflation and subsequent compression of the gastric vasculature, and this may make lesions such as GAVE more prominent on CE.

The pathogenesis of GAVE is still obscure, and many hypotheses such as mechanical stress, hemodynamic alterations, and hormonal and autoimmune factors have been proposed. Mechanical stress represented by strong gastric peristalsis is thought to induce prolapse and trauma of antral mucosa and intermittent obstruction of blood vessels, which can lead to fibromuscular hyperplasia and vascular ectasia. [22] Increased levels of hormones with vasodilating properties, such as gastrin and prostaglandin E2, have been observed in patients with GAVE, and it has been suggested that failure of liver processing functions may lead to a build-up of these hormones, contributing to the pathogenesis of GAVE. [23] Up to 60% of patients with GAVE have associated autoimmune diseases, specially connective tissue diseases, and test positive for autoantibodies such as antinuclear antibodies and anti-centromere antibodies; therefore, an autoimmune pathogenesis is suggested.[9]

The mainstay of therapy for GAVE remains endoscopic. Neodymium:yttrium-aluminum-garnet (Nd:YAG) laser has been widely used in the treatment of GAVE. All series have confirmed the efficacy of this endoscopic thermal therapy by reducing or abolishing the need of blood transfusions in 50–80% of cases. [24-27] However, APC has been found to be equally effective in the treatment of GAVE and is superior to ND:YAG laser in cost, convenience, and complication rates. [28] Multiple sessions of APC may be required to reduce bleeding episodes and/or decrease transfusion dependence. Several other endoscopic therapies, such as cryotherapy, [29] band ligation, [30-32] and radiofrequency ablation, [33] have been proposed in the past years. Although initial reports of these endoscopic modalities are encouraging, well-performed, larger, prospective studies are needed before providing any definitive conclusion.

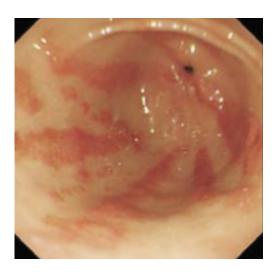
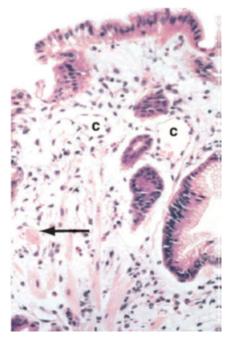


Figure 3: Striped watermelon appearance of gastric antral vascular ectasia.



**Figure 4:** Gastric antral vascular ectasia (GAVE). Histology shows dilated capillaries (c) in the superficial gastric mucosa with occasional thrombi (arrow). Fibromuscular spindle cells are prominent.

Multiple drugs, such as hormonal (estrogen-progesterone) therapy, [34-35] octreotide, [36-37] steroids, [38-39] and transexamic acid, [40] have been tried to control GAVE-related bleeding. After all, no one has clearly shown satisfactory results in order to consider medical therapy as a valid alternative to an invasive approach.

Before the advent of endoscopic therapy, GAVE was commonly treated by antrectomy. However, surgical resection is often associated with significant morbidity and mortality and should be reserved only for patients who are refractory to medical and endoscopic therapy.

## **CONCLUSION**

GAVE is a rare but an important cause of UGIB. Diagnosis of GAVE can be overlooked during upper GI endoscopy and must always be considered specially in obscure GI bleeding. Endoscopic therapy using APC is the main treatment option.

## Conflict of interest

None for all authors.

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