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Navigating Diagnostic Challenges: Chronic Anemia Leading to the Diagnosis of Gastric Antral Vascular Ectasia (GAVE) in the Absence of Typical Symptoms

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ABSTRACT

Gastric antral vascular ectasia (GAVE) is a rare acquired vascular dysplasia usually associated with portal hypertension or CREST syndrome. Here we present a case of a 62-year-old female with no significant related comorbidities who presented to our clinic due to severe acute on chronic anemia. On initial review of systems, the only complaint the patient had was fatigue, shortness of breath and being lightheaded. Patient denied hematochezia or melena, no hematemesis or other gastrointestinal complaints, no history of NSAIDs or aspirin use. Initial labs were significant for iron-deficiency anemia with hemoglobin of 8.5 g/dL. Recent colonoscopy was non diagnostic for the cause of anemia as well. Due to unrevealing previous workup patient was referred to gastroenterologist and undergone esophagogastroduodenoscopy (EGD) with findings of ecstatic vessels in stomach antrum suggestive of GAVE, successfully treated with argon plasma coagulation. Total in a span of 4 years due to recurrence of anemia patient required 3 EGDs, while GAVE was successfully treated each time with argon plasma coagulation. Due to absence of gastrointestinal (GI) complaints patient was recommended to have regular laboratory workup to monitor for silent blood loss. This atypical presentation of upper GI bleeding warrants physicians to refer patients to gastroenterologists to perform EGD for further evaluation and treatment in cases of unknown etiology of iron deficiency anemia even in the absence of gastrointestinal (GI) complaints and non-revealing previous GI workup with colonoscopy.

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Introduction

Gastric Antral Vascular Ectasia (GAVE) is a rare acquired vascular disease responsible for around 4% of non-variceal upper gastrointestinal bleeding that may cause chronic blood loss with iron-deficiency anemia [1]. The cause is uncertain, and several mechanisms have been suspected to develop GAVE. The lesion may represent a response to mucosal trauma from contraction waves in the antrum due to altered antrum motility and dysfunction, subsequently inducing chronic mucosa trauma and subsequent submucosa fibromuscular hyperplasia and dilation of mucosal capillary leading to development of GAVE syndrome overtime [2-4].

GAVE affects mainly women of middle age and is usually associated with portal hypertension, achlorhydria, atrophic gastritis, and CREST syndrome [5]. Here, we represent a case of an elderly patient with no significant related comorbidities who presented to our clinic due to severe acute on chronic anemia with unknown etiology.

Case

A 62-year-old female with a past medical history of osteopenia, osteoarthritis, asthma, deep vein thrombosis, breast cancer, and anxiety came for evaluation of her anemia. The patient complained of feeling fatigued and lightheaded, short of breath, and unable to complete her workouts. The patient denied hematochezia or melena, no hematemesis, no abdominal pain, only occasional alcohol, and no history of NSAIDs or aspirin use regularly. Initial labs positive for iron deficiency anemia with hemoglobin down to 8.5 g/dL, hematocrit 28.4%, MCV 81 f/L, MCH 24.1 pg., iron 22 ug/dL, iron-binding 471ug/dL, iron % saturation-5, ferritin 14ng/mL, liver enzymes mildly elevated with AST to 62 U/L, ALT normal at 57 U/L, lipid panel significant for mildly elevated cholesterol of 209 mg/dL. US liver was positive for steatosis. CT abdomen was positive for diffuse hepatic steatosis, stable small hypodense lesions within the inferior pole of the spleen likely consistent with small cysts and/or hemangiomas, and extensive colonic diverticulosis. The last colonoscopy was performed a year ago with findings of 2 sessile polyps 4- 5 mm in size and diverticulosis, small internal hemorrhoids. On the 1st EGD, esophageal wall mucosa was normal, with no signs of esophagitis; however, in the stomach antrum, there were findings of ectatic vessels suggestive of GAVE (Figure 1). Treatment with argon plasma coagulation was performed with minimal bleeding. Duodenal mucosa was normal. After the initial evaluation, the patient came back in a year complaining of fatigue and darker than usual stools; however, was lost to follow-up, and EGD was not performed at that time.

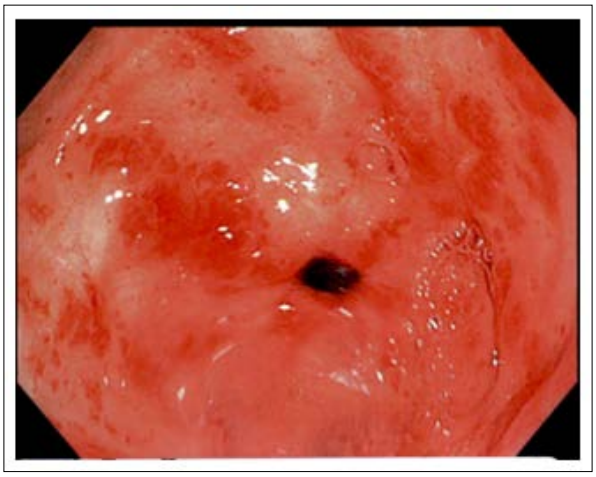

Figure 1: Gastric Antrum with dilated small blood vessels. GAVE pre-treatment

In 3 years, she was started on Xarelto, and shortly after that, her labs were significant for a drop in hemoglobin to 5.9 g/dL and hematocrit to 20%; however, no apparent gastrointestinal bleeding was observed, such as hematemesis, melena, or hematochezia. Due to signs of GAVE seen on second EGD -argon plasma coagulation was performed with treatment of approximately 80% of gastric antral vascular ectasia; post procedure, there were no complications or bleeding observed. The patient was instructed to closely monitor hemoglobin since that was the only sign of slow, continuous, subtle GI bleeding in her case. Due to the risks outweighing the benefits, the patient was stopped on Xarelto; she also received iron transfusions with restoration of a normal hematocrit shortly after that.

In less than a year, the patient came back to the gastroenterologist's office once again due to anemia with no signs of melena or bright red blood per rectum. Third EGD was performed with argon plasma coagulation of most of marked gastric antral vascular ectasia (Figure 2) distributed in a mosaic pattern from antrum to the stomach fundus, typically suggestive of portal hypertensive gastropathy. However, no history of liver cirrhosis and no esophageal varices were seen on EGD.

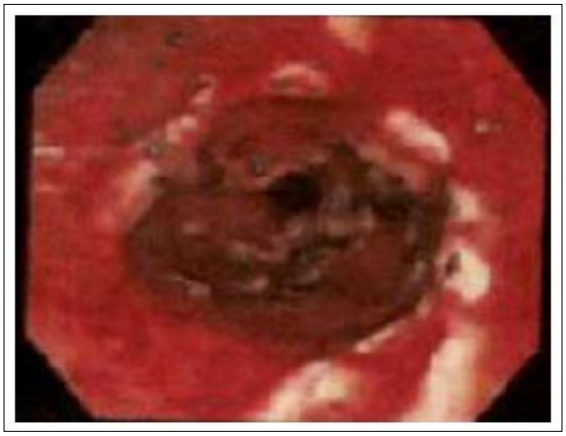

Figure 2: Pre-Pyloric Stomach with coagulated blood vessels. GAVE post treatment with argon plasma

In general, in 4 years, due to severe fatigue and iron deficiency anemia with the lowest hemoglobin of 5.8 g/dL, she had several iron transfusions and underwent 3 EGDs with argon plasma treatment each time due to marked progressive GAVE syndrome.

Discussion

GAVE syndrome, or watermelon stomach, is a rare condition with the most common presentation as GI bleeding. While GAVE syndrome is more typical for people with liver cirrhosis and hypertensive gastropathy or in noncirrhotic patients with autoimmune disorders and more specific connective tissue diseases, our patient did not have any of those, which tells about the unique presentation of our case [6]. Due to profound anemia and recurrence of bleeding requiring treatment with argon plasma, the patient eventually had to stop Xarelto.

While there is no consensus for the optimal therapeutic approach, several treatment options in patients with GAVE are available, such as argon plasma coagulation, radiofrequency ablation, band ligation, YAG laser coagulation, cryotherapy, laser treatment options, and even surgical antrectomy. Argon plasma coagulation is considered one of the most successful treatment approaches with the best outcomes. However, that remains debatable since it is unclear if superficial abrasions are sufficient for an abnormal vascular network of GAVE since patients need a mean of 5.6 treatment sessions [6, 7]. In patients with vascular ectasia due to portal hypertension the treatment would be concentrated at lowering portal pressure by using non selective beta blockers or in certain cases by performing TIPS [8].

However, the clinical presentation of GAVE syndrome was unique in our case since our patient's only symptom was fatigue caused by severe anemia. A review of systems was persistently negative for melena, hematochezia, or hematemesis, with no nausea, vomiting, or abdominal pain. Even though traditionally, lower gastrointestinal bleeding and malignancy are thought first in patients with chronic anemia, upper GI sources could also be the case. This warrants the necessity to perform EGD evaluation in patients with chronic anemia to rule out silent upper gastrointestinal blood losses presented without other clinical signs such as melena or hematemesis.

By this day, our patient had three treatment sessions with argon plasma coagulation over four years. Due to the high possibility of recurrence of symptoms due to progressive recurrent vessel erosion and potential bleeding, the patient was advised to systematically monitor her hemoglobin.

Conclusion

GAVE syndrome is a rare source of non-variceal upper GI bleeding associated with various systematic autoimmune diseases, liver cirrhosis, or chronic renal failure. Our case presentation is engaging such that our patient did not have any other comorbidities, and severe anemia was the only presentation of subtle, silent, but chronic blood loss. This atypical presentation of upper GI bleeding warrants physicians to refer patients to gastroenterologists to perform EGD for further evaluation and treatment in cases of unknown etiology of iron deficiency, even if previous GI workup with colonoscopies was nonrevealing.

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