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A Rare Cause of Upper Gastrointestinal Bleeding, Gastric Antral Vascular Ectasia: A Case Report

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Abstract Gastric antral vascular ectasia (GAVE) is a vascular malformation that causes upper gastrointestinal bleeding (UGIB). It is often discovered incidentally, and it is usually secondary to iron deficiency anemia due to chronic blood loss. Diagnosis of this pathology is primarily done by endoscopy, with direct visualization of the lesions. Multiple treatment options including medical, endoscopic, or surgical are available. We describe a case of a 69-year-old lady who presented with symptomatic anemia and who was found to have Gastric antral vascular ectasia on endoscopy.

Keywords: *GAVE, iron deficiency anemia, endoscopy, liver cirrhosis*

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1. Introduction

Gastric antral vascular ectasia is a rare but serious vascular malformation that causes upper gastrointestinal bleeding. This condition was first reported in 1953 and is often referred to as "watermelon stomach" due to the appearance of longitudinal antral folds containing visible columns of tortuous red ecstatic vessels resembling stripes on a watermelon [1,2].

GAVE is most commonly discovered incidentally secondary to iron deficiency anemia (IDA) due to chronic blood loss that is severe enough to require blood transfusions [3]. GAVE is strongly associated with liver cirrhosis, autoimmune diseases, and other chronic or autoimmune conditions [4]. Diagnosis of GAVE is primarily done using endoscopy, with visualization of the unique lesions within the stomach being enough to come to a conclusive diagnosis. There are multiple treatment options for GAVE including medical, endoscopic, and surgical therapy.

Here, we are presenting the case of a 69-year-old morbidly obese lady who presented to our Emergency Department with symptomatic anemia and the Esophagogastroduodenoscopy showed changes consistent with GAVE. Her hospital course was complicated with acute renal failure, multiple infections, and liver failure and despite all the efforts, the patient expired.

2. Case Presentation

This is a 69-year-old-lady with a past medical history of morbid obesity, diskitis, osteomyelitis of L4-L5, septic left hip, paroxysmal atrial fibrillation, hypertension, cirrhosis possible secondary to NASH, and chronic anemia, who was sent to the Emergency Department (ED) by her primary care physician after she was found to be anemic with a Hb of 5.2 in routine blood work. The patient complained of generalized weakness, and she did endorse two episodes of bright red blood per rectum in the last few days prior to the presentation. The patient has never had an Esophagogastroduodenoscopy (EGD) or colonoscopy in the past. Pertinent negatives included dyspnea, palpitations, chest or abdominal pain, nausea, vomiting, melena, hematemesis, hematuria, or hemoptysis.

In the ED, the patient was found to be afebrile, hypertensive with BP: 160/68 mmHg, non-tachycardic, non-tachypneic, and saturating 98% on room air. On physical examination, the patient was a bedbound morbidly obese female, with cardiovascular and lung examination without positive findings, positive guaiac stool test, and the bilateral lower extremities with edematous changes. Initial lab work was remarkable for hemoglobin 5.5 [12 - 16 GM/DL], hematocrit 16.6 [37 - 47%], MCV 91.1 [80 - 94 FL], creatinine 1.44 [0.4 - 1 MG/DL]. FOBT was positive. EKG showed normal sinus rhythm. Chest x-ray revealed linear opacity in the left lung base which was consistent with possible subsegmental atelectasis. The patient was initially admitted for blood transfusion and further GI assessment.

She received 3 units of PRBC with evidence of improvement in hemoglobin levels from 5.5 to 8.1 [12 - 16 GM/DL]. Further labs revealed a reticulocyte count of 3.3 [0.5 - 1.5%], iron saturation of 40.7%, vitamin B12 level 959 [180 – 914 pg/ml], and folic acid 4.9 [5.9 – 24.8 ng/ml]. The viral hepatitis panel was negative. EGD was performed and it showed changes consistent with Gastric Antral Vascular Ectasia (GAVE) treated with Argon Plasma Coagulation (APC). In the following days, the patient remained stable, however, her hemoglobin level dropped to 6.6 [12 - 16 GM/DL], and the decision was made to transfuse 1 more unit of PRBC.

In the following days, the patient's mentation deteriorated, and she became more lethargic and confused however she was still able to protect her airways. ABG was performed, and it showed pH 7.18 [7.35 -7.45], pCO2 57 [35 - 45 mmHg], pO2 68 [>80 mmHg], and HCO3 19.7 [22 - 26 mmol/L], ammonia level was 79 [16 – 60 umol/L]. At that time, the patient was receiving lactulose 30 ml and she was placed on BiPAP. In the setting of continued dropping in hemoglobin, the patient underwent a follow-up EGD on 03/08 with prior intubation for securing airways. This study showed non-bleeding gastric ulcers with no stigmata of bleeding, and gastric antral vascular ectasia without bleeding. Colonoscopy has been planned however poor bowel preparation preclude the study at that time. After the EGD, since the patient had a low threshold for intubation due to altering mentation, the decision was made to transfer the patient to the ICU for closing monitoring.

During her ICU stay, the patient was not able to protect her airways developing a hypercapnic respiratory failure requiring intubation on 3/10. Ammonia level, creatinine, and BUN trended up to 105 [16 - 60 umol/L], 4.59 [0.4 - 1 MG/DL], and 31 [8 – 20 MG/DL] respectively; consistent with hepatic and uremic encephalopathy. In the setting of acute renal failure, a Udall catheter was placed in the right femoral area for hemodialysis. ANA and anti-mitochondrial antibodies were negative.

Subsequently, chest imaging including chest x-ray and CT scan of the chest revealed bilateral infiltrates and the patient became increasingly hypotensive. She required multi-vasopressor support, and she was started on broad-spectrum antibiotics with renal dose IV Meropenem 1000 mg and IV Vancomycin 1000 mg intermittent with hemodialysis sessions. Respiratory cultures grew Pseudomonas aeruginosa, however, blood cultures were negative. After completion of the antibiotic regimen, Meropenem and Vancomycin were discontinued on 3/24.

The decision was made to take the patient to the Operative room for PermCath placement however, her dependence on multi-vasopressor support preclude her from the surgical procedure.

Unfortunately, the patient was restarted on empiric meropenem 1 g IV daily, vancomycin 2 g IV once, and micafungin 100 mg IV daily on 4/3 due to worsening fevers. At that time, the right femoral Udall was removed, and a right internal jugular vein Udall was placed by surgery. Subsequently, AST increased to 1715 [15 - 41 U/L] and ALT increased to 263 [14 – 54 U/L] with total bilirubin 10 [0.4 – 2 MG/DL], consistent with shock liver. On 4/13, her ventilator FiO2 was maximized to 100%, however, her oxygen saturation remained at 73% despite this. On 4/16 she became pulseless, at that time full ACLS protocol was performed, however, despite all the efforts, the patient expired.

3. Discussion

Gastric antral vascular ectasia is a rare but serious vascular malformation of capillary-type vessels that causes UGIB. It was first reported in 1953 by Rider et al. as “an erosive type of gastritis with marked veno-capillary ectasia” with a “fiery red” antrum “marked with hypertrophic mucosal changes” [1]. The term “watermelon stomach” that is used readily today would later be coined by Jabbari et al. in 1984. Jabbari defined GAVE as “longitudinal antral folds” that were “converging on the pylorus, containing visible columns of tortuous red ecstatic vessels” resembling stripes on a watermelon, hence the name [2].

Since its discovery, GAVE has been an uncommon and enigmatic source of UGIB, especially in older females with multiple comorbidities. The average age of presentation is 73 years old with females being 71% of the patient population. GAVE is most commonly discovered incidentally secondary to IDA due to chronic blood loss that is severe enough to require blood transfusions [3].

In addition to IDA, GAVE has numerous other medical associations. Most GAVE patients suffer from a constellation of other medical conditions, most of them chronic or autoimmune. The most common association is liver cirrhosis (30% of GAVE patients). 1 in 40 patients undergoing liver transplantation for end-stage liver disease was found to have GAVE. Autoimmune diseases are the second most common comorbid condition GAVE patients suffer, mostly in non-cirrhotic patients. 62% of non-cirrhotic GAVE patients were found to have a form of autoimmune connective tissue disorder, 31% suffered Raynaud’s phenomenon, and 20% had sclerodactyly. Other common conditions include scleroderma, chronic renal failure, acute myeloid leukemia, and ischemic heart disease [4].

Even among cirrhotic patients, patients with non-alcoholic steatohepatitis (NASH) were found to develop GAVE most frequently. A retrospective study of 855 cirrhotic patients from 2009 to 2011 stratified different etiologies of cirrhosis: NASH (18%), autoimmune (15.1%), HBV (6.3%), HCV (19.4%), alcohol (25.7%), alcohol plus HCV (7.8%), cryptogenic (2.8%) and other (4.8%). Out of all the etiologies, GAVE was most frequently found in NASH cirrhotic patients compared to cirrhosis of any other etiology (29.2% vs. 9.4%; $p < 0.001$). Further analysis using a univariate analysis indicated NASH cirrhosis as a major risk factor for GAVE determined by an odds ratio of 3.73 (95% CI: 2.36 – 5.90, $p < 0.001$); this significance remained even in a multivariate analysis [5].

Diagnosis of GAVE is primarily done using endoscopy, with visualization of the unique lesions within the stomach being enough to come to a conclusive diagnosis without the need for histologic confirmation. GAVE is divided into two categories depending on endoscopic appearances: raised or flat longitudinal stripes of vascular tissue extending from the pylorus (known as “watermelon

stomach") (70%) or diffusely scattered punctuate lesions (30%). The punctuate type of GAVE is significantly linked to cirrhosis and the male population, while the more common form of GAVE is most often associated with other comorbidities, such as connective tissue disease with a female prevalence [6,7].

Two main differential diagnoses of GAVE are portal hypertensive gastropathy (PHG) and antral gastritis. GAVE can commonly be mistaken for antral gastritis and histology is the main distinguishing factor. Histological features largely pathognomonic for GAVE include vascular ectasia of the mucosa, fibrin thrombi, spindle cell proliferation, and fibrohyalinosis. PHG and GAVE can be differentiated mainly by location. PHG is predominant in the fundus and body of the stomach, whereas GAVE rarely progresses distally to the antrum of the stomach. GAVE patients also have a more severe degree of concurrent liver disease (higher Child-Pugh score), greater blood loss, and lower serum gastrin levels. Spindle cell proliferation and fibrohyalinosis are also significant histological features that yield an 85% diagnostic accuracy for GAVE if the endoscopic examination is inconclusive for either differential diagnosis [8].

There are multiple treatment options for GAVE chosen depending on desired therapeutic outcome: medical, endoscopic, and surgical. Medical therapy is largely aimed at controlling chronic blood loss from GAVE lesions. Such therapies include corticosteroids (mainly in auto-immune-associated GAVE), hormone replacement therapy (in postmenopausal women), octreotide, tranexamic acid, and thalidomide therapy. Each medical regimen is tailored to an intended symptomatic outcome and side effects are weighed against therapeutic success depending on patient circumstances. Endoscopic management includes cryotherapy and different forms of coagulation and ablations [neodymium-yttrium-aluminum garnet (Nd: YAG) laser, argon plasma coagulation (APC), endoscopic band ligation, and radiofrequency ablation]. Of all endoscopic treatments and all treatments available, argon plasma coagulation is the most commonly used, having replaced Nd: YAG laser treatment. APC is overall easier, safer, and more cost-effective making it the treatment of choice and most widely used. APC uses high-frequency electrical currents to cause tissue coagulation. Nonetheless, the efficacy of this treatment in achieving recurrence-free survival at one year is less than 50% for the patients. Furthermore, there have been reports indicating therapy failure rates of up to 14%. Equally noteworthy is the high incidence of complications, ranging from 20% to 30% [9]. Surgery is a last resort reserved only for patients' refractory to both medical and endoscopic therapy. When GAVE cannot be controlled, Antrectomies, such as Billroth I or II, and Roux-en-Y procedures, partial or total gastrectomy, or esophagogastrostomy are performed [10]. In some cases, treatment of the underlying disease, such as cirrhosis or systemic sclerosis can also cause the resolution of GAVE.

When encountering a patient with symptoms suggestive of upper gastrointestinal bleeding, it is essential to consider gastric antral vascular ectasia as a potential underlying case for achieving a timely and effective intervention.

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