Gastric Antral Vascular Ectasia

Anita Basavaraj*, Rahul Kulkarni**, DB Kadam#

Abstract
Iron deficiency anaemia secondary to gastrointestinal bleeding is very common in the elderly. Gastric antral vascular ectasia (GAVE) syndrome, also known as watermelon stomach is an uncommon but significant cause of acute or chronic gastrointestinal blood loss in the elderly. It is characterised endoscopically by “watermelon stripes.” It is more common in females than males, and manifests mostly as iron deficiency anaemia due to the gradual blood loss. Pathogenesis is unknown though several humoral factors have been proposed. Diagnosis is based on the clinical history and endoscopic appearance and histological changes. We describe elderly patient who presented with haematemesis and iron deficiency anaemia and was diagnosed to have GAVE and was treated successfully with endoscopic band ligation.

Introduction
Gastric antral vascular ectasia or GAVE syndrome is an uncommon cause of gastrointestinal bleeding. The mean age of presentation is 70 years. The diagnosis of GAVE syndrome in patients with renal or hepatic disease is often problematic because there are more frequent causes of gastrointestinal bleeding in these diseases (vascular malformations, peptic ulcer disease, oesophageal or gastric varices, and colonic and rectal ulcers) that overshadow GAVE syndrome. Furthermore, diagnosis may be challenging because gastrointestinal bleeding may be occult or overt, and the endoscopic appearance of GAVE syndrome resembles that in portal hypertensive gastropathy (PHG) or antral gastritis. However, differentiation of GAVE syndrome from these other causes is critical because of the vastly disparate therapies required for each. The lesions are treated by endoscopic methods, the most commonly used modalities are band ligation, heater probe, electrocoagulation, argon photocoagulation and laser. The purpose of this article is to review an uncommon cause of gastrointestinal bleeding in elderly.

Case Report
56 yrs old male, farmer by occupation, resident of Pune was admitted with complaints of loose motions 4-5 episodes since 4 days, nausea and vomiting since 4 days and two episodes of haematemesis on the day of admission. There was no history of abdominal pain, tenesmus, bleeding per rectum. There was history of passing black coloured stools intermittently. There was no history of fever, yellowish discolouration of skin/eyes/urine or history of altered sensorium in past. There was no history of hypertension, diabetes, ischaemic heart disease, tuberculosis, asthma, herpes zoster. There was no history of haematemesis in past. On enquiry patient gave history of blood transfusion 1 year back for anaemia. Upper gastrointestinal endoscopy done outside 1 year back was suggestive of antral gastritis. Patient was a chronic alcoholic since 20 years. Family and drug history was not contributory. On examination patient was conscious, oriented, afebrile. Pulse was 100/min, Blood pressure 110/70 mmHg. Pallor was present. There was no icterus, cyanosis, clubbing, lymphadenopathy, oedema. Tongue was dry suggestive of dehydration. JVP was not raised. Koilonychia was present.

Cardiovascular system, respiratory and central nervous system examination was within normal limits. Per abdomen was soft, non tender with hepatomegaly of 2.0 centimeters, tender, soft to firm. Spleen was palpable 2 cm below the left costal margin.

Lab investigations revealed Haemoglobin of 7.8 gm%, MCV-74.3 fl, MCH-23.0, MCHC-26.4, HCT-35%, RDW-11.0%, PLT-180000, WBC-7200/cmm, RBC-5.28*106. Serum
iron-60, TIBC-320, Retic Count-1.5%. Peripheral smear examination revealed microcytic hypochromic RBCs with a TLC of 8200/cmm (P76, L22, M02, E-0, B-0). Ultrasonography of abdomen revealed hepatomegaly with splenomegaly with dilated splenic vein. Rest was within normal limits. Other laboratory tests revealed Sr. creatine -2.67mg/dl, Blood urea level-200 mg/dl, Sodium-140 Meq/l, Potassium - 3.8 Meq/l, Bilirubin-2.2 mg/dl, SGOT-42 U/dl, SGPT-38 U/dl. Sr. proteins-4.1 gms (Alb-2.1 gms, Glb-2.0 gms).

Prothrombin time was normal (16 sec). HIV and HBsAg was negative. Urine examination was within normal limits. However stool examination was positive for occult blood.

Upper gastro-intestinal endoscopy was done. Oesophagus and duodenum were within normal limits.

Stomach showed prominent erythematous streaks traversing the antrum and converging on the pylorus. (Watermelon strips appearance). Fundus showed mild portal hypertensive gastropathy.

Thus the final diagnosis was haematemesis with Iron deficiency anaemia due to gastric antral vascular ectasia (GAVE syndrome).

Patient was advised Argon plasma coagulation. As Argon plasma coagulation costs 10000-12000 rupees, patient was not affording for the same and hence patient underwent gastric band ligation over the prominent ectatic vessels (Procedure similar to oesophageal variceal band ligation in which vessels are sucked through endoscope and bands applied) and
was advised regular followup. Gastric band ligation costs 1500 rupees in our hospital.

Patient was treated with cold saline lavage, IV antibiotics, IV proton pump inhibitors and IV fluids, IV Iron and blood transfusion. Patient was also started on Tab propranolol 10 mg on discharge in view of dilated splenic vein.

After 2 months patient was investigated and was maintaining haemoglobin of 10.2 gm/dl and stool occult blood was negative.

Thus we had a case of gastric antral vascular ectasia presenting with haematemesis and iron deficiency anaemia treated with cost efficient “gastric band ligation”.

Discussion

Gastric antral vascular ectasia (GAVE syndrome) is a relatively rare cause of GI bleed. Although GAVE is considered a rare medical condition, it accounts for up to 4% of all non-variceal upper gastrointestinal bleedings. Gastric antral vascular ectasia (GAVE) was first described by Rider et al. (1953) in a patient with severe chronic iron-deficiency anaemia, as an erosive type of atrophic gastritis with marked venocapillary ectasia. Three decades later, in 1984, Jabbarei and colleagues coined the term “Watermelon Stomach” to describe its classic endoscopic appearance.

GAVE is mainly seen in elderly. The mean age of diagnosis is 73 years for females and 65 yrs for males. However patients in third decade have also been found to have GAVE. It is generally associated with medical conditions like heart, liver, renal diseases, diabetes, connective tissue diseases (esp systemic sclerosis), hypothyroidism and status as bone marrow transplant recipient.

Pathogenesis of GAVE is not fully understood. Several theories have been proposed 1) Mechanical stress due to increased gastric peristalsis. 2) Spindle cell proliferation due to hypergastrinaemia which is frequently observed in GAVE leading to rise in venous hydrostatic pressure. 3) Local proliferation of neuroendocrine cells producing high levels of vasoactive substances (e.g.-vasoactive intestinal peptides and serotonin) that lead to vascular dilatation. 4) Antral mucosal prolapse through the pylorus. 5) Autoimmune vasculopathy

GAVE most commonly present with iron deficiency anaemia from chronic and slow bleeding. The lesions are painless and acute presentations like haematemesis and melaena are not common but have been documented. It may be associated with weakness, severe fatigue, dyspnoea and with other typical findings which reveal manifestations of anaemia. Many patients become unresponsive to iron therapy and become transfusion dependant thus presenting as refractory anaemia.

Endoscopic appearance is pathognomonic of GAVE showing columns of red tortuous ectatic vessels along the longitudinal folds of antrum converging on the pylorus that resemble watermelon strips. Diagnosis is based on clinical history and endoscopic appearance. Endoscopic appearance is sufficient for establishing diagnosis. Histologic findings of vascular ectasia with fibrin clots and fibromuscular hyperplasia of lamina propria are characteristic, however they are not necessary to confirm the diagnosis.

Differential diagnosis of GAVE includes 1) Portal hypertensive gastropathy. 2) Chronic Antral gastritis. 3) Atrophic autoimmune gastritis. 4) Dieulafoy lesion/caliber persistent artery. However characteristic endoscopic appearance and histopathology showing no microthrombi on microscopy in cases of portal hypertensive gastropathy, autoimmune gastritis and dieulafoy lesion helps to differentiate between GAVE and other conditions.

Sometimes watermelon stomach can be wrongly interpreted as refractory haemorrhagic gastritis. In contrast to antral gastritis, lesions of GAVE are typically sharply demarcated with small margintated red spots and vessels blanch with pressure and bleed freely on endoscopic biopsy. There can be a dilemma in trying to differentiate between GAVE syndrome and severe PHG in patients with existing cirrhosis or portal hypertension. Generally those patients with GAVE syndrome had more severe liver disease (by Child-Pugh scoring), greater blood loss, lower serum gastrin levels, and were more likely to have had previous sclerotherapy. It has become clear, that portal hypertension does not play an important role in the development of GAVE. This is supported by findings that there is no significant correlation between the degree of vascular ectasia and degree of portal hypertension.

Mainstay of treatment is endoscopic modality which includes 1. Variceal Band ligation. 2. Sclerotherapy.

However in a study done by Wells et al, it was found that, compared with endoscopic thermal therapy (endoscopic thermal therapy with cautery or Argon plasma coagulation), early band ligation had a significantly higher rate of bleeding cessation (67% vs 23%, P =.04), fewer treatment sessions required for cessation of bleeding (1.9 vs 4.7, P =.05), a greater increase in haemoglobin values (2.8 g/dL vs 0.9 g/dL, P =.05), a greater decrease in transfusion requirements (-12.7 vs -5.2, P =.02), and a greater decrease in hospital admissions (-2.6 vs -0.5, P =.02) during the follow-up period. Also gastric band ligation is an economical modality of treatment and can be offered in resource limited settings with equally good efficacy.

In conclusion, GAVE has been recognised as a cause of gastrointestinal bleeding, though it is uncommon. GAVE Syndrome has to be kept in mind in elderly patients presenting with iron deficiency anaemia. It is important to accurately diagnose this condition and differentiate it from portal hypertensive gastropathy since the treatment and outcomes vary significantly.

References