**INTRODUCTION**

Portal Hypertensive Gastropathy (PHG) is an uncommon single cause of significant Upper Gastrointestinal (GI) bleeding in patients with portal hypertension. When PHG is the sole cause of bleeding, there is diffuse mucosal oozing and there are no other lesions such as varices, to account for the GI bleeding and anemia. Mucosa is friable and bleeding presumably occurs when the gastric vessels rupture. This case report describes the uncommon condition in a lady with Chronic Liver Disease (CLD) due to hepatitis C.

**CASE REPORT**

A 46-year-old female from Karachi, a known case of HCV CLD for 3 years, presented in the emergency room in June 2007 with 6 months history of melena and coffee ground vomitings. For these complaints, three OGDs were done in different peripheral hospitals, which were variably reported as gastric ulcer/gastritis/gastric erosions/duodenal ulcer with no oesophageal or fundal varices. She had been transfused 28 pints of packed red blood cells in last 6 months. There was no history of jaundice, but there was a history of caesarian section and blood transfusion from 15 years back.

On examination, she was fully oriented, tachycardiac, hypotensive, anemic, dehydrated and had pedal edema.

On abdominal examination, mild tenderness was present in the epigastrium. The investigation showed anemia and mildly deranged liver functions and clotting profile (Hb: 9.6 g/dL, total serum bilirubin: 1.87 g/dL and direct bilirubin: 0.69 g/dL, serum SGPT: 33 u/L, serum albumin: 3.59 g/dL, serum globulin: 4.93 g/dL, A/G ratio: 0.73, prothrombin time: 16/12 seconds, activated partial thromboplastin time: 29 seconds. An ultrasound of her liver was suggestive of CLD. The patient underwent esophagogastroduodenoscopy, which revealed markedly congested gastric mucosa with diffuse mucosal oozing and a mosaic pattern, consistent with severe PHG (Figure 1).

No oesophageal or fundal varices were seen. Antral biopsy was taken, which showed lympho-plasmacytic infiltrate, dilated blood vessels and marked congestion, consistent with severe PHG. As the patient had continuous melena TIPSS was advised (Figure 2) and the patient was referred to AKUH for this procedure. Post-procedure patient's hemoglobin was maintained.

After 3 months, a repeat OGD was done, which showed marked improvement in PHG. She is currently symptom free and enjoying good health.

**DISCUSSION**

PHG is characterized by prolonged elevation of portal vein pressure; complications usually develop when portal vein pressure exceeds 12 mmHg. PHG is...
associated with histological and biochemical severity of liver disease. Gastric mucosal blood flow is increased in patients with cirrhosis and PHG. PHG is characterized endoscopically by a mosaic pattern with or without red signs and a proximal distribution. PHG characteristically appears as fine white reticular pattern separating areas of pinkish mucosa on endoscopy, giving the mucosa a “snake skin” appearance. Mucosal changes are mostly evident in fundus and body. Treatment is directed at decreasing portal pressure. This can be accomplished by a variety of measures including portal caval shunt surgery, TIPSS, propanolol and liver transplantation. Only limited data on the efficacy of TIPSS for PHG is available. In this case, the patient had Child-A disease. TIPSS was considered to be the best option. TIPSS is associated with improvement in endoscopic findings and a decrease in transfusion requirements. Endoscopic improvement with mild gastropathy occurs within 6 weeks and for severe PHG, it occurs within 3 to 6 months. Other options, if bleeding continues, are porta caval shunt depending on liver function and liver transplantation for decompensated CLD.

REFERENCES


