Case Report

Management of a Portal Hypertensive Polyp: Case Report of a Rare Entity

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Gastric outlet · Obstruction · Portal hypertension · Polyp

Abstract

Portal hypertensive polyposis is a rare finding represented in about 2.5% of all patients with portal hypertension. The diagnostic criteria are not yet clearly defined. It has been mentioned in a few case reports; its distribution was mainly duodenal and less frequently gastric. Here, a patient with type 2 diabetes and liver cirrhosis was hospitalized for vomiting, abdominal pain, and melena. The patient was admitted to the intensive care unit for stabilization and urgent esophagogastroduodenoscopy (EGD). EGD revealed a single antral polyp occluding the pyloric ring which was the cause of gastric outlet obstruction. Complete debulking by argon plasma was done which improved gastric outlet obstruction and melena. We conclude that argon plasma coagulation is a safe, rapid, and effective method for treating portal hypertensive polyposis.

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Introduction

Portal hypertension is an important vascular decompensation of advanced liver disease. It is defined by an elevated portal pressure gradient due to increased portal blood flow or increased resistance to blood flow due to irreversible architectural changes in the hepatic parenchyma. Portal hypertension will eventually open the portosystemic collaterals to relieve the splanchnic pressure leading to esophageal and gastric varices, portal hypertensive gastropathy, enterocolopathy, and splenomegaly with thrombocytopenia due to hypersplenism [1]. The severity and extent of the reopening of the portosystemic collaterals depend

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mostly on the differential gradient augmenting the flow between the portal and the systemic circulations and the duration of elevated portal pressure [2].

Venous drainage of the stomach either directly or indirectly into the portal vein works as follows: short gastric veins drain from the fundus to the splenic vein, the left gastroepiploic vein along the greater curvature to the splenic vein, the right gastroepiploic vein from the right side of the greater curvature to the superior mesenteric vein, the left gastric vein from the lesser curvature of the stomach to the portal vein, and the right gastric vein moves from the lesser curvature of the stomach to the portal vein [3].

Portal hypertensive gastropathy (PHG) occurs in up to 80% of all patients with liver cirrhosis and is characterized by macroscopic change in the gastric mucosa associated with mucosal and submucosal vascular dilatation in the absence of any inflammatory insult [4]. It has the potential to cause acute and/or chronic bleeding in the context of liver cirrhosis and portal hypertension, it is caused by increased gastric blood flow in case of portal hypertension augmented by a disturbed equilibrium of the vascular tone chemical regulators in liver cirrhosis such as nitric oxide (NO), tumor necrosis factor alpha (TNF- α), endothelins, glucagon, and prostaglandins [5].

The endoscopic appearance of PHG may include fine pink speckling, scarlatina-like petechiae, multiple hemorrhagic spots, papules, snakeskin-like appearance, cherry-red spots which are classified according to McCormack [6], who divided PHG into mild and severe, or NIEC (New Italian Endoscopic Club for the Study and Therapy of Esophageal Varices) [7] and Tanoue classifications [8], which divided PHG into mild, moderate, and severe. Some authors refine the classification of McCormack, and others depend on the three-stage scale [9].

A recent approach to PHG classification and severity was performed in a two-step classification, suggested by the Baveno consensus workshop, as follows:

- (A) mucosal mosaic pattern (mild: 1; severe: 2);
- (B) red markings (isolated: 1; confluent: 2);
- (C) gastric antral vascular ectasia (absent: 1; present: 2).

Mild PHG: ≤ 3 ; severe PHG: ≥ 4 [10].

Histologically, PHG shows a marked dilatation of the capillaries and collecting venules in the gastric mucosa with an enlarged mean mucosal capillary cross-sectional area; in case of GAVE, fibrin thrombi, ectasia, and myofibroblastic hyperplasia in the superficial mucosa and mucosal blood vessels are dominant [11].

Nowhere in the literature is the occurrence of portal hypertensive polyposis (PHP) mentioned; portal hypertensive polyps have been mentioned in scarce case reports; however, mainly duodenal and less frequently gastric polyps. Grossly and histologically, portal hypertensive polyps have the appearance of normal hyperplastic polyps with epithelial hyperplasia and proliferating ectatic capillaries in the lamina propria [12].

Case Report

A patient with type 2 diabetes – on insulin therapy – and advanced liver cirrhosis was hospitalized for vomiting, abdominal pain, and melena. The patient was admitted to the intensive care unit. The laboratory examination revealed low hemoglobin (7.1 g/dL), mean corpuscular volume (75.8 fL), mean corpuscular hemoglobin (24.6 pg), thrombocytopenia 25,000, a prothrombin concentration of 54%, and an international normalized ratio of 1.7. Abdominal ultrasound showed liver cirrhosis, splenomegaly with a splenic diameter of 18.5 cm, and a portal vein diameter of 15.4 mm. The patient received proton pump inhibitor infusion and intravenous fluids and had undergone urgent esophagogastroduodenoscopy which revealed a single antral polyp occluding the pyloric ring which was the cause of gastric outlet obstruction (Fig. 1). The gastric polyp was biopsied showing foveolar hyperplasia with ectatic capillaries. The gastric mucosa showed PHG-typical features (Fig. 2). Debulking of the polyp was successfully done using argon plasma coagulation (APC; ERBE

Fig. 1. Portal hypertensive polyp occluding the pyloric ring.

Fig. 2. Endoscopic appearance of the portal hypertension.

Fig. 3. **a** Complete debulking using argon plasma coagulation. **b** Adequate healing of the debulked area.

VIO D 200, with safe pulse mode, effect 2, 40 W) (Fig. 3). There was a dramatic improvement in gastric outlet obstruction, and the patient was discharged on oral proton pump inhibitor, carvedilol 6.25 mg once daily. A follow-up esophagogastroduodenoscopy was done 4 weeks later and showed complete healing of the raw area (Fig. 3) with control of melena and an improved hemoglobin level of 10.6 gm/dL ($p = 0.001$).

Discussion

The pathogenesis of PHP can be explained by an increased congestion due to portal pressure with the induction of angiogenesis and cellular proliferation [13]. Independent risk factors of PHP are thrombocytopenia, worsening of Child-Pugh score, also previous treatment with band ligation due to increased formation of portosystemic shunts, and increased portal blood flow to the gastric mucosa. It is a rare finding represented in about 2.5% of all patients with portal hypertension. The diagnostic criteria for portal hypertensive polyp are not yet clearly defined [14].

Gastric polyps are commonly present in the fundus and antrum. Gastric polyps are mostly associated with familial adenomatous polyposis or found in patients who are on long-term proton pump inhibitors, also chronic *Helicobacter pylori* infection can cause a chronic inflammation which may lead to polyp formation. All of these were excluded in the present case [15]. It is rare for PHG to cause gastrointestinal bleeding; anemia due to chronic blood loss occurs in nearly 9–10%, acute upper gastrointestinal bleeding occurs in <2.5% [16].

APC is a therapeutic option. Research showed that a combination of APC and non-selective beta-blockers was highly efficacious and safe in controlling bleeding from PHG; and APC alone is rapid and effective when beta-blockers are contraindicated [17]. Our patient had a bleeding tendency due to reduced platelet count and prolonged international normalized ratio, and this is the end result of advanced cirrhosis; however, argon plasma therapy was administered safely without bleeding complications.

PHP is a rare entity which presented in our case with vomiting, gastric outlet obstruction, and melena due to a single polyp occluding the pyloric ring. PHP was relieved by complete debulking of the polyp using APC with improved anemia and melena.

Statement of Ethics

The research was conducted in accordance with the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee. Written informed consent was obtained from the patient for the medical intervention and to publish his images.

Disclosure Statement

The authors report no conflicts of interest.

Author Contributions

Waseem M. Seleem performed the endoscopy. Amr S. Hanafy wrote the paper. Both authors reviewed the final version of the paper.

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