Portal hypertensive duodenal polyp: A case report

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Abstract

Abnormalities of gastric mucosa in patients with portal hypertension are well documented. Manifestations of portal hypertension in small bowel and colon are less common. Colonic polypoid lesions microscopically consisting of a normal mucosa, with dilatation of submucosal vessels, have been described. We here report the first case of portal hypertensive duodenal polyp, responsible for gastro-intestinal bleeding. Endoscopic treatment turned out to be successful.

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Key words: Gastrointestinal bleeding; Cirrhosis; Portal hypertension; Small bowel; Endoscopy

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INTRODUCTION

Esophagogastric varices are common manifestations of portal hypertension, but other lesions such as gastric mucosal changes, called portal hypertensive gastropathy^[1], or colonic mucosal abnormalities^[2,3] may occur. Duodenopathy has also been reported, with various forms^[4-6] but never under the appearance of a polyp. We describe a patient who was found to have a portal hypertensive duodenal polyp, responsible for gastro-intestinal bleeding. Endoscopic treatment was performed with success.

CASE REPORT

A 70-year old man, with alcoholic cirrhosis, was

hospitalized for a melena. Physical examination found superficial venous engorgement of the abdominal wall, with ascitis. Laboratory data revealed anemia and liver dysfunction. International normalized ratio (INR) value was 1.33.

An upper endoscopy showed large esophageal varices without red spots, and portal hypertensive gastropathy. In the descending portion of the duodenum, a 3-cm long, ulcerated and haemorrhagic polypoid lesion was seen (Figure 1).

We performed polypectomy using hemostatic clips on the pedicle, then a polypectomy snare. Anatomopathologic examination of the specimen showed a fibrous and inflammatory polyp, with numerous thick-walled capillaries in its subepithelial portion, and a few vascular ectasias were associated (Figure 2). This histological appearance was considered as portal hypertensive duodenopathy. There was no epithelial proliferation in favor of an inverted polyp or hamartomatous lesion.

No complication occurred, and control endoscopy was performed three days later. The duodenal mucosa appeared normal. Colonoscopy revealed an 8-mm polyp located in the left portion of the colon, which was removed with a polypectomy snare. Anemia resolved and the patient was still free of haemorrhagic recurrence six months later.

DISCUSSION

McCormack *et al*^[1] first described portal hypertensive gastropathy, its appearance consists of mosaic pattern, cherry red spots and scarlatina rash. Other entities have then been documented, such as portal hypertensive enteropathy^[4-10] and colonopathy^[2,3].

Duodenum can thus be involved and with the exception of varices, lesions reported are erythema, scattered petechiae, friable mucosa, erosion, ulcer and edema.

In our patient, portal hypertensive duodenopathy took the unusual presentation of a polyp. Misra *et al*^[11] and Ryushi Shudo *et al*^[4] reported that the predominant pathological features of portal hypertension duodenopathy (PHD) are subepithelial edema, and increase of diameter and wall thickness of the capillaries. Now, numerous capillaries with a thickening wall were found in our specimen, suggesting that the polyp is due to portal hypertension duodenopathy. On the other hand, colonic polypoid lesions due to portal hypertensive colonopathy have already been described^[12].

Moreover, many authors insist on the uncertain clinical significance of portal hypertensive enteropathy^[13], in particular whether it can be responsible for gastro-

Figure 1 Endoscopic view of the duodenal polyp.

Figure 2 Polypoid lesion of the duodenum demonstrating the presence of multiples capillaries in the lamina propria, immediately beneath the surface epithelium.

intestinal bleeding.

In the reported case, the only haemorrhagic lesion was the duodenal polyp. Additionally, anemia resolved after polypectomy. This illustrates that portal hypertensive duodenopathy is a potential aetiology of gastro-intestinal bleeding.

We removed the polyp using hemostatic clips before applying polypectomy snare, to avoid the potential risk of post polypectomy hemorrhage. Polypectomy appeared to be a safe procedure and allowed histological diagnosis.

In conclusion, we report the case of a cirrhotic patient who had upper gastro-intestinal haemorrhage due to a portal hypertensive duodenal polyp. To our knowledge, this is the first case of this form of portal hypertensive duodenopathy. Endoscopic treatment was performed successfully. Although the new data suggest that portal hypertensive enteropathy may present as a source of bleeding, its exact clinical significance has to be defined subsequently.

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