Portal Hypertension

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Contents

The major consequence of portal hypertension is bleeding, particularly from esophageal varices (Fig. 23.1) [1]. Digestive bleeding has also been found to occur from gastric sources (Fig. 23.2) [2] and colorectal sources [3, 4]. The recognized existence of portal hypertensive gastropathy [5] and portal hypertensive colopathy [6] suggests that the small bowel may also present endoscopic lesions related to portal hypertension. In fact, Thiruvengadam and Gostout [7] reported in 1989 on three patients presenting with blood loss, who had diffuse ervthema and scattered petechiae in the stomach and in the duodenum and jejunum. Since then, the small bowel, previously considered to be the most difficult segment of the gut to study, has come to be easily explored using new endoscopic methods such as capsule endoscopy and double-balloon enteroscopy. This development in the field of small bowel endoscopy has allowed significant progress



Fig. 23.1 Esophageal varices

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Fig. 23.2 Portal hypertensive gastropathy. (a) Mild portal hypertensive gastropathy.(b) Severe portal hypertensive gastropathy



in the study of small bowel diseases, including the implications of portal hypertension.

The concept of portal hypertensive vasculopathy, including portal hypertensive gastropathy, portal hypertensive colopathy, and portal hypertensive enteropathy (PHE), proposed by Viggiano and Gostout [8] to describe the effects of portal hypertension in the gut, is not unanimously accepted, however. Although the association between portal hypertensive gastropathy and portal hypertensive colopathy is well documented in a large series of cirrhotic patients [6], the studies that address this issue and include PHE show conflicting results. A paper by De Palma et al. [9] found that PHE is significantly more common in the presence of hypertensive gastropathy and portal hypertensive colopathy, but two other studies [10, 11] found no association between the presence of PHE (documented by capsule endoscopy) and the presence of portal hypertensive gastropathy and portal hypertensive colopathy.

De Palma et al. [9] reported the changes found in the mucosa of the small bowel in patients with portal hypertension. They used capsule endoscopy to study 37 cirrhotic patients and 34 controls and considered the presence of abnormalities resembling mucosal inflammation and/or vascular lesions to be manifestations of PHE. These findings were detected in 67.5 % of the cirrhotic patients and in none of the controls. In accordance with this, the endoscopic lesions in the small bowel of patients with portal hypertension may be classified as mucosal abnormalities, which include a reticulate pattern (Fig. 23.3), sometimes with erythema (Fig. 23.4), or as vascular lesions, which include angiectasia-like lesions (Fig. 23.5) and varices (Fig. 23.6). These findings are detected in 65-69 % of patients with portal hypertension [9, 11–14], regardless of the presence of cirrhosis [11]. The same papers report the presence of varices in up to 27 % of the patients [11], but in others, only 8 % of the patients present this finding [9]. There is also huge variability in the prevalence of angiectasia-like lesions, ranging from 63 % [15] to 22 % [14], and in abnormalities resembling mucosal inflammation, found in 13 % of patients by De Palma et al. [9] but in 63 % by Canlas et al. [16].

An important issue is whether angiectasia-like lesions detected by capsule endoscopy should be considered a small bowel manifestation of portal hypertension. Although angiectasia seems to be the main cause of acute bleeding from the small bowel in patients with portal hypertension, documented in all patients in the series presented by De Palma et al. [9] and in one of two patients in another series [11], the frequency of detection of these lesions was similar in patients with portal hypertension and in the control group [11]. In fact, in a recently published paper [17], angiectasia and red spots, which were reported as portal hypertensive lesions in previous studies, showed no correlation with the hepatic venous pressure gradient. Therefore, angiectasia-like lesions probably should not be considered a small bowel manifestation of portal hypertension. Further studies aimed at recognizing the clinical consequences of the small bowel endoscopic findings in these patients (as regards bleeding episodes or transfusion requirements, for example) should probably consider separately those who present angiectasia-like lesions.

The main question is the clinical impact of these findings, as the significance of the endoscopic lesions suggestive of PHE is uncertain. Some studies report active bleeding from the small bowel of cirrhotic patients [9, 11], but a recently published study involving 40 patients with liver cirrhosis (50 % presenting abnormal vascular findings in the small bowel), who were observed for 5–27 months (median 16.3), suggested that capsule endoscopy findings had no impact on the clinical course [18].

Two options exist when dealing with small bowel manifestations of portal hypertension. The first is to perform Fig. 23.3 Reticulate pattern.(a, b) Reticulate pattern.(c) Reticulate pattern with a polyp

Fig. 23.4 Reticulate pattern with erythema. (a, b) Reticulate pattern with erythema. (c) Ileocecal valve with erythema



Fig. 23.5 Angiectasia-like lesions. (**a**–**c**) Small angiectasia-like lesions



Fig. 23.6 Varices in the small bowel. (**a**–**d**) Blue lesions, compatible with varices

enteroscopy only in patients who present obscure digestive bleeding. This was the choice of De Palma et al. [9], who found active bleeding in four patients (10.8 %), the source being angiectasia-like lesions. The other diagnostic option is to include enteroscopy in the workup of all cirrhotic patients, regardless of the presence of hemorrhage. Not surprisingly, when including patients without a previous history of obscure digestive bleeding, fewer patients with portal hypertension present active bleeding [11]. In fact, enteroscopy probably should be used only in patients who present obscure digestive bleeding [19]. In this context, capsule endoscopy is the preferred method for two reasons: (1) It allows a complete examination of the small bowel and is obviously more efficient in detecting small bowel manifestations of portal hypertension than other diagnostic modalities, such as retrograde ileoscopy [20, 21]. (2) An accurate diagnosis may enable a therapeutic intervention. In fact, endoscopic treatments for angiectasia-like lesions, such as clipping and argon plasma coagulation (APC), can be performed in patients with portal hypertension using doubleballoon enteroscopy [22].

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