Capsule endoscopy in the investigation of patients with portal hypertension and anemia

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INTRODUCTION: Data on small bowel abnormalities in patients with portal hypertension (PHT) are limited. Bleeding from the gastrointestinal tract and anemia are common complications in these patients. Capsule endoscopy (CE) was used to evaluate small bowel (SB) pathology in patients with PHT and anemia, and possible associations with various parameters were examined.

METHODS: Thirty-five patients with PHT referred for CE investigation of the SB for anemia were prospectively enrolled in the study, as well as 70 age- and sex-matched control patients with anemia, normal liver function and no evidence of PHT who underwent CE.

RESULTS: Findings compatible with portal hypertensive enteropathy (PHE) were detected in 65.7% of the patients and in 15.7% of the controls (χ^2=26.641, P=0.000). Abnormalities in PHT patients included varices in 25.7%, diffuse changes of mucosa with inflammatory-like appearance in 42.9%, and angiodysplasias and/or spider angiomas in 22.9% of cases. The presence of PHE was significantly associated only with the presence of severe portal hypertensive gastropathy, while the presence of SB varices alone was significantly associated with the presence of severe portal hypertensive gastropathy, larger esophageal varices and the presence of colonic varices.

CONCLUSIONS: Varices, diffuse changes of mucosa with inflammatory-like appearance, and angiodysplasias and/or spider angiomas are detected more often in patients with PHT than in controls, and probably constitute the endoscopic characteristics of PHE. CE of the SB added a significant number of likely important findings to those detected by conventional endoscopic techniques for the clinical management of patients with PHT and anemia.

Key Words: *Capsule endoscopy; Portal hypertension; Small bowel*

Endoscopie par capsule dans l'examen des patients souffrant d'hypertension portale et d'anémie

INTRODUCTION : Les données sur les anomalies de l'intestin grêle chez les patients souffrant d'hypertension portale (HTP) sont limitées. Les saignements du tractus digestif et l'anémie sont des complications fréquentes chez ces patients. L'endoscopie par capsule (EC) a permis d'évaluer la pathologie du grêle chez des patients atteints d'HTP et les liens possibles avec divers paramètres ont été analysés.

MÉTHODE : Trente-cinq patients atteints d'HTP adressés pour EC du grêle en raison d'anémie ont été inscrits de manière prospective à l'étude, de même que 70 patients témoins, assortis selon l'âge et le sexe, atteints d'anémie, mais ne présentant aucune dysfonction hépatique ni signe d'HTP et ayant subi une EC.

RÉSULTATS : On a noté des signes d'entéropathie hypertensive portale (EHP) chez 65,7 % des patients et chez 15,7 % des témoins (χ^2 = 26,641, p = 0,000). Les anomalies observées chez les patients atteints d'HTP incluaient : varices chez 25,7 %, anomalies diffuses de la muqueuse avec signes pseudo-inflammatoires chez 42,9 % et angiodysplasies et/ou angiomes stellaires chez 22,9 %. La présence d'EHP a été significativement associée uniquement à la présence de gastropathie hypertensive portale sévère, tandis que la présence de varices au niveau du grêle seulement a été significativement associée à la gastropathie hypertensive portale sévère, à la présence de varices œsophagiennes plus volumineuses et à la présence de varices au niveau du côlon.

CONCLUSION : Les varices, les anomalies diffuses de la muqueuse d'aspect pseudo-inflammatoire et les angiodysplasies et/ou angiomes stellaires s'observent plus souvent chez les patients atteints d'HTP que chez les témoins et constituent probablement les caractéristiques endoscopiques de l'EHP. L'EC du grêle a révélé un nombre significatif de caractéristiques probablement importantes en plus des signes mis au jour par les techniques endoscopiques habituelles pour la prise en charge clinique des patients qui souffrent d'HTP et d'anémie.

The term portal hypertension (PHT) was first introduced by Gilbert and Carnot in 1902 for patients with ascites, splenomegaly and esophageal hemorrhage. Bleeding from the gastrointestinal tract is a common complication of PHT, and acute or chronic blood loss may contribute to the appearance of anemia, a common hematological complication in these patients. Gastroesophageal varices can be found in up to 70% of patients with cirrhosis and 30% of these varices will bleed within two years of diagnosis (1). Portal hypertensive gastropathy (PHG), first described in 1985 (2), accounts for 10% to 20% of acute bleeding, but it has been mainly identified as a cause of chronic blood loss. Nonvariceal bleeding from peptic ulcers, gastric erosions and the Mallory-Weiss syndrome is not uncommon. Less common and less significant causes of blood

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loss are hemorrhoids and portal hypertensive colopathy (PHC), a term comprising anorectal varices, spider angiomas and inflammatory changes with or without spontaneous mucosal bleeding (3-7).

Data on small bowel (SB) abnormalities in patients with portal hypertensive enteropathy (PHE) are limited, due to the difficulty of exploring the whole length of the SB (2,8-12). In view of the scarcity of information, we conducted the present study and used capsule endoscopy (CE) to evaluate SB pathology in patients with PHT. This novel method is well tolerated and allows complete visual investigation of the SB (13,14). Moreover, the diagnostic yield of CE in anemia is well documented and significantly higher than that of any other method, including push enteroscopy, small bowel follow-through, computed tomography, angiography, colonoscopy and gastroscopy (15). To our knowledge, there is only one study published in this field (16).

PATIENTS AND METHODS

The present prospective study was conducted from April 2004 until December 2007, and included consecutive patients with PHT and iron deficiency anemia referred to the General Hospital of Athens 'Helena Venizelou' (Athens, Greece) for SB investigation with CE. An open-access system was followed (ie, any physician who wished to refer a patient to the hospital could do so. All patients had been recently investigated with esophagogastroduodenoscopy (EGD) and colonoscopy. Exclusion criteria were congestive heart disease, liver transplantation, hepatocellular carcinoma, history of abdominal surgery and the use of acetylsalicylic acid or nonsteroidal anti-inflammatory drugs.

Patients' clinical characteristics, including sex, age, etiology of PHT, Child-Pugh score, prior history of upper gastrointestinal and variceal bleeding, and endoscopic intervention (sclerotherapy or ligation), were recorded. Biochemical test results (ferrum, ferritin, liver function), prothrombin time and hemoglobin levels were obtained. In EGD, esophageal varices were graded according to the system proposed by the North Italian Endoscopic Club for the Study and Treatment of Esophageal Varices (17), and congestive gastropathy was classified according to McCormack et al (2).

Age- and sex-matched patients with anemia, negative EGD and colonoscopy, normal liver function and no evidence of PHT who were investigated with CE during the same period, were used as controls for the interpretation of findings.

The M2A capsule (Given Imaging, Israel) was used in the present study. Contraindications for the CE procedure were the generally accepted contraindications described previously (18). Written informed consent was obtained in all cases. Patients' preparation and CE procedure followed the generally recommended guidelines (19). All patients were advised to abstain from solid food on the day before the procedure and to ingest a 1 L solution of polyethylene glycol. Prokinetic medications were not administered. Nine hours after ingestion, the sensory array and recording device were removed.

CE videos were studied and all SB abnormal findings were recorded. A careful search was performed for signs of PHE, which, according to Misra et al (12), include diffuse hyperemia and edema, spider angiomata, patchy hyperemia and severe acute enteritis with spontaneous bleeding from the mucosa. SB varices were also considered as part of the spectrum of PHE.

For the interpretation of CE results, a single gastroenterologist initially screened all videos and selected images of potential abnormalities. Then, two gastroenterologists experienced in interpreting CE independently reviewed the selected images. All videos were extensively discussed; findings identified by both reviewers were considered as definitive and were included in the report. The procedure was defined as complete or incomplete depending on the passage of the capsule into the cecum throughout the duration of the examination.

Then, a search was conducted for any association between the presence of PHE and the following parameters: sex, age, etiology of cirrhosis, Child-Pugh class, size of esophageal varices, presence and severity of PHG, history of upper gastrointestinal bleeding, history of endoscopic intervention on esophageal varices and presence of colonic varices or PHC.

Statistical analysis

The SPSS program, version 13.0 (SPSS Inc, USA) was used for statistical analysis. Continuous data with normal distributions are presented as mean ± SEM. Differences between groups were evaluated by the χ^2 test or Fisher's exact test for qualitative variables. Student's *t* test was used to compare quantitive variables. $P<0.05$ was considered to be statistically significant.

RESULTS

A total of 35 patients fulfilled the inclusion criteria during the study period. There were 33 cirrhotic patients and two patients with portal vein thrombosis. The control group consisted of 70 patients. Demographic and clinical characteristics of patients, as well as findings of upper and lower gastrointestinal tract endoscopies are listed in Table 1.

All patients completed the procedure uneventfully. No cases of capsule retention were observed and the exit of the capsule was confirmed in all cases. Complete visualization of the SB was achieved in 29 of 35 patients with PHT and in 55 of 70 controls ($\chi^2=0.268$, degrees of freedom =1, $P=0.605$). Causes of failure of the capsule to reach the colon within the recording time among patients with PHT were slow gastric passage (n=1), presence of food that impaired capsule progression (n=1) and no clear reason (n=4). Gastric emptying time ranged from 4 min to 198 min (median 13 min) and SB transit time ranged from 139 min to 438 min (median 278 min).

SB findings detected by CE in both patients and controls are listed in Table 2. Twenty-three of the 35 patients with PHT (65.7%) were found to have signs of PHE. SB varices (Figures 1 and 2) were evident in nine of 35 patients (25.7%), diffuse changes of mucosa with inflammatory-like appearance in 15 of 35 (42.9%) (Figures 3 and 4), and angiodysplasias and/or spider angiomas in eight of 35 patients (22.9%). In eight patients with PHT, CE revealed more than one abnormality. The jejunum and ileum were equally involved. In the control group, no patient had evidence of SB varices, while diffuse changes of the mucosa with inflammatory-like appearance were revealed in three patients (4.3%) and angiodysplastic lesions were revealed in eight (11.4%). Findings compatible with PHE were detected in 65.7% of the patients and in 15.7% of the controls ($\chi^2=26.641$, $P=0.000$).

The presence of PHE was significantly associated only with the presence of severe PHG. No association was found with sex, age, etiology of PHT, Child-Pugh score, history of upper gastrointestinal bleeding, size of esophageal varices, history of endoscopic intervention, PHC and colonic varices (Table 3).

TABLE 1
Demographic, clinical and endoscopic characteristics of patients and controls

Characteristic	Patients with PHT	Controls
Patients, n	35	70
Male:female	28:7	56:14
Age, years, mean ± SEM	53.0±1.7	52.9±1.1
Hemoglobin, g/L, mean ± SEM	81.0±0.2	84.0±0.1
Cirrhosis/portal vein thrombosis, n	33/2	0
Etiology of cirrhosis, n (%)		NA
Alcohol	15 (45.4)	
Hepatitis C	7 (21.2)	
Hepatitis B	6 (18.2)	
Cryptogenic	3 (9.1)	
Primary biliary cirrhosis	2 (6.1)	
Child-Pugh class, n (%)		NA
A	8 (24.2)	
B	17 (51.6)	
C	8 (24.2)	
History of upper GI bleeding, n (%)	19 (54.3)	10 (14.3)
Variceal bleeding	13 (37.1)	0
Nonvariceal bleeding	6 (17.1)	10 (14.3)
History of endoscopic intervention, n (%)	14 (40.0)	NA
Endoscopic findings of upper and lower GI tract related to PHT		NA
Esophageal varices, n (%)	30 (85.7)	
Small	10	
Medium	18	
Large	2	
Gastric varices, n (%)	5 (14.3)	
Duodenal varices, n (%)	1 (2.9)	
Portal gastropathy, n (%)	28 (80.0)	
Mild	16	
Severe	12	
Colonic varices, n (%)	9 (25.7)	
Portal colopathy, n (%)	16 (45.7)	

Some patients showed more than one finding. GI Gastrointestinal; NA Not applicable; PHT Portal hypertension

TABLE 2
Small bowel findings detected by capsule endoscopy

Small bowel findings compatible with portal hypertension	Patients with portal hypertension (n=35)	Controls (n=70)	Statistics
Varices, n (%)	9 (25.7)	0 (0)	χ^2=19.688,
Duodenum	1		df=1,
Jejunum	3		P=0.000
Ileum	4		
Jejunum and ileum	1		
Diffuse changes of mucosa with inflammatory-like appearance, n (%)	15 (42.9)	3 (4.3)	χ^2=24.440, df=1, P=0.000
Duodenum	1	0	
Jejunum	5	1	
Ileum	3	2	
Jejunum and ileum	6	0	
Spider angiomas and/or angiodysplasias, n (%)	8 (22.9)	8 (11.4)	χ^2=2.360, df=1, P=0.125
Jejunum	3	4	
Ileum	2	2	
Jejunum and ileum	3	2	
Total, n (%)	23 (65.7)	11 (15.7)	χ^2=26.641, df=1, P=0.000
Other small bowel findings, n (%)			NA
Scalloping, fissures	0 (0)	1 (1.4)	
Single ulcer	0 (0)	2 (2.9)	
Ulceration – cobblestoning	0 (0)	1 (1.4)	
Multiple diverticulae	0 (0)	1 (1.4)	
Submucosal mass	0 (0)	2 (2.9)	
Polyp/tumour	0 (0)	2 (2.9)	

Eight patients with portal hypertension showed more than one finding. df Degrees of freedom; NA Not applicable

Considering SB varices separately, their presence was significantly associated with the presence of severe PHG, larger esophageal varices and colonic varices (Table 4).

DISCUSSION

Increased resistance to portal blood flow due to liver cirrhosis or thrombosis of the portal vein leads to PHT. PHT results in the development of gastroesophageal and/or ectopic (colonic, enteric) varices, as well as other mucosal lesions in the stomach, small intestine and colon. They are referred to under the term 'portal hypertensive intestinal vasculopathy' (including PHG, PHC and PHE) (10,20). While PHG and PHC are the commonly recognized components of the spectrum, data on PHE are limited. There are only a few reports on PHE concerning the duodenum, the upper jejunum, and the terminal ileum (9-12,21). This lack of information reflects the inability to examine the SB in the era before CE introduction in clinical practice. The above-mentioned studies were based on

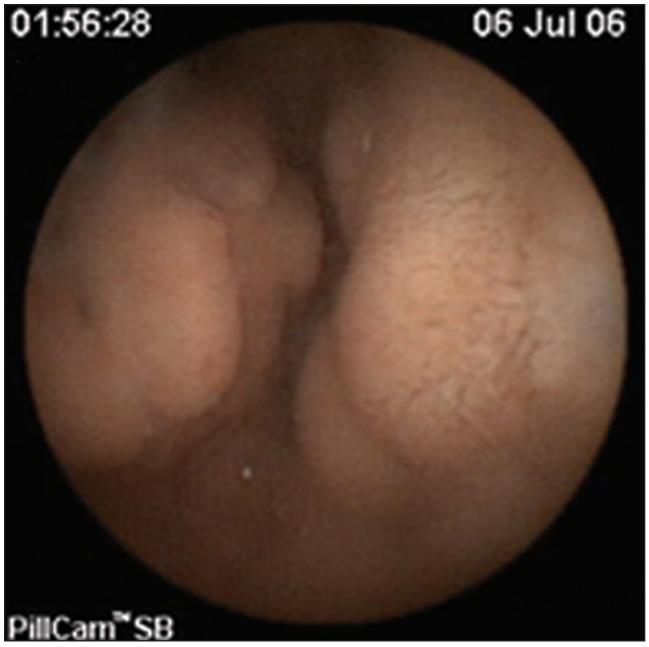

Figure 1) *Jejunal varix in a patient with portal hypertension*

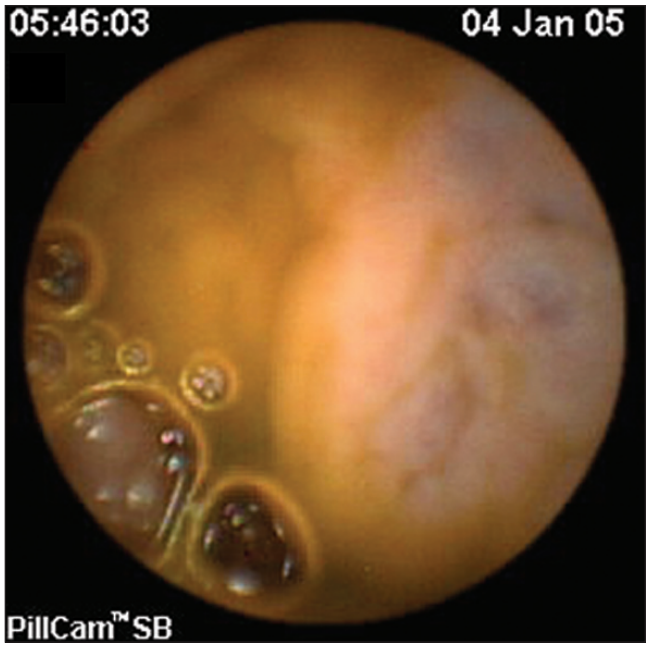

Figure 2) *Ileal varix in a patient with portal hypertension*

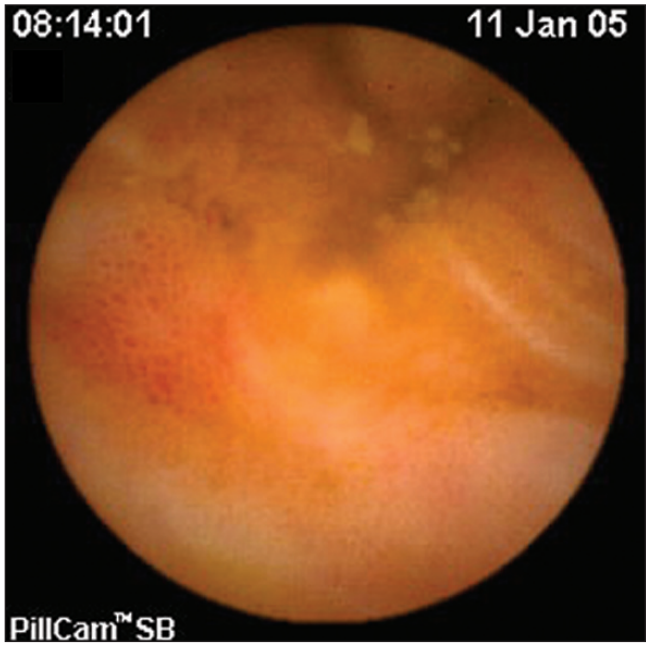

Figure 3) *Portal hypertensive enteropathy in the ileum of a patient with portal hypertension*

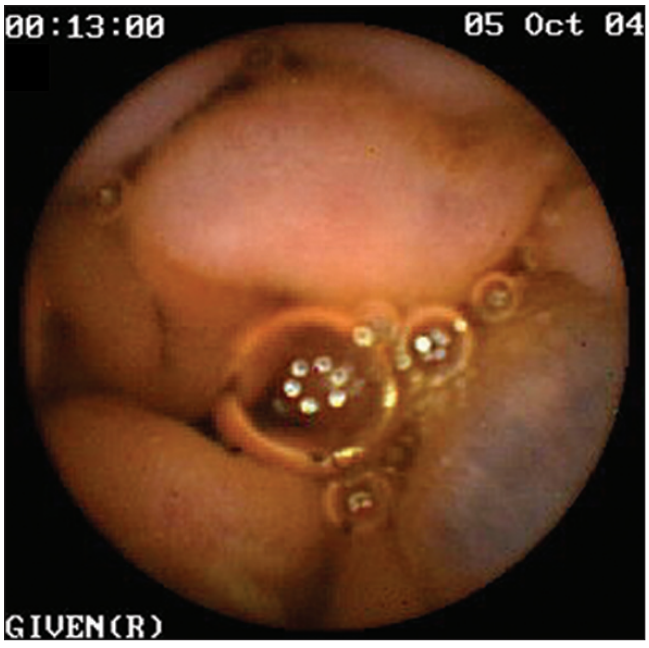

Figure 4) *Portal hypertensive enteropathy and a varix in the jejunum of a patient with portal hypertension*

TABLE 3
Factors associated with portal hypertensive enteropathy

Factor	χ^2	df	P
Sex	0.127	1	1.000
Age	t=0.334	33	0.740
Etiology of cirrhosis	5.507	4	0.239
Child-Pugh class	0.017	2	0.991
Size of esophageal varices	5.213	3	0.157
Grade of PHG	7.947	2	0.019
History of upper GI bleeding	0.135	1	0.713
History of endoscopic intervention	0.338	1	0.721
Colonic varices	0.783	1	0.450
PHC	1.128	1	0.288

df Degrees of freedom; GI Gastrointestinal; PHC Portal hypertensive colopathy; PHG Portal hypertensive gastropathy

partial endoscopic examination with push enteroscopy and ileocolonoscopy and, in some cases, on histology.

Although there are occasional reports on SB lesions in patients with PHT, their true prevalence, as well as their implication in overt and occult bleeding are unknown (9-12,22-27). In the present study, we used CE to investigate the whole length of the SB in patients with PHT and anemia. Our main objective was to search for SB findings that could be of potential clinical significance among patients with PHT. Signs of PHE – varices, diffuse changes of mucosa with inflammatory-like appearance, and angiodysplasias and/or spider angiomas – were found in 65.7% of our patients. This figure is in accordance with a study reported by De Palma et al in 2005 (16). These endoscopic findings were more common among patients with PHT than among controls. The increased prevalence of SB varices in our study, compared with the study of De Palma et al (25.7% versus 8.1%), could be possibly explained by the presence of esophageal varices in the vast majority of our patients (85.7% versus 32.4%).

Other studies confirm the presence of PHE, but the reported incidence rates are conflicting. Pennazio et al (28), in a study published recently in abstract form, reported a 10% incidence of SB varices (also detected with CE) in a small number of cirrhotic patients. On the other hand, Misra et al (12) reported a high incidence (18%) of ileal varices in patients with PHT using ileocolonoscopy, and considered that this is a probable underestimation, because they examined only a short segment of the ileum (12).

Diffuse changes of SB mucosa with inflammatory-like appearance (erythema, edema, granularity and/or friability) as part of the spectrum of PHE, were predominant among our patients (42.9%), followed by SB varices (25.7%) and spider angiomas or angiodysplasias (22.9%). These diffuse lesions probably reflect mucosal alterations similar to that of the gastric mucosa in patients with PHG (21).

Based on the findings of this and the previously mentioned studies, the endoscopic characteristics of PHE are well documented. Nevertheless, the question of whether they can be the cause of overt or occult bleeding in patients with PHT remains unanswered. Our study included patients with anemia only and, consequently, failed to identify actively bleeding lesions. The assumption that PHE lesions are implicated in gastrointestinal bleeding is based on reports of bleeding SB varices and other PHE lesions (11,22-27,29,30), as well as on the knowledge that pathogenetically similar lesions, like esophagogastric or colonic varices, PHG and PHC, are well-established causes of bleeding.

PHE affected both the jejunum and the ileum equally, and a significant number of findings were beyond the reach of conventional endoscopic techniques, including push enteroscopy. Although, theoretically, these lesions could be diagnosed by means of double-balloon enteroscopy, this novel method is not yet widely available and is clearly more invasive. The safe and well-tolerated CE technique is currently the preferable method to image areas of the gut such as the distal jejunum and the proximal ileum.

We examined possible associations between PHE and various parameters, and found that the presence of PHE was significantly associated only with the severity of PHG. No association was found with sex, age, etiology of PHT, Child-Pugh score, history of upper gastrointestinal bleeding, size of esophageal varices, history of endoscopic intervention, PHC and colonic varices. The absence of any association between PHE and Child-Pugh class was a surprise to us, although both Misra et al (12) and Pennazio et al (18) found the same. We have also been surprised by the lack of association with history of endoscopic intervention of esophageal varices, because it is well known that variceal ligation and sclerotherapy are predisposing factors for PHG and ectopic varices formation (31,32). SB varices were more common in patients with severe PHG. They were also significantly associated with the presence of larger esophageal varices and colonic varices. Although the pathophysiological basis for, and the clinical importance of these findings needs to be further explored, we can conclude that PHE represents the enteric manifestations of PHT, and that it shares a common pathogenesis with PHG and esophageal varices.

TABLE 4
Factors associated with small bowel varices

Factor	χ^2	df	P
Sex	0.598	1	0.648
Age	t=1.541	33	0.153
Etiology of cirrhosis	1.082	4	0.897
Child-Pugh class	0.850	2	0.654
Size of esophageal varices	9.348	3	0.025
Grade of PHG	6.534	2	0.038
History of upper GI bleeding	0.008	1	1.000
History of endoscopic intervention	3.590	1	0.112
Colonic varices	5.648	1	0.030
PHC	2.143	1	0.245

df Degrees of freedom; GI Gastrointestinal; PHC Portal hypertensive colopathy; PHG Portal hypertensive gastropathy

CONCLUSIONS

The present study reinforces the views that the SB in patients with PHT manifests macroscopically similar changes as the rest of the gut and that these changes (varices and mucosal alterations) most probably share, as a common pathophysiological mechanism, the impaired enteric venous drainage through the portal system. CE of the SB proved to be a useful tool in the investigation of patients with PHT and anemia, and added a significant number of likely important findings to those detected by conventional endoscopic techniques. Our findings did not alter the ongoing management of the patients (ie, treatment with beta-blockers and conventional endoscopic follow-up) because we did not identify patients with active bleeding. However, in case of such a finding, the next step would be to perform push enteroscopy or double-balloon enteroscopy and provide endoscopic treatment for the bleeding lesion. Additionally, patients with already identified PHE lesions could be treated accordingly in the case of a future episode of obscure gastrointestinal bleeding (negative upper and lower endoscopy).

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