Prevalence, variability, and outcomes in portal hypertensive colopathy: a study in patients with cirrhosis and paired controls CME

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Background: Management of portal hypertensive colopathy (PHC) has been challenged by controversial results in its prevalence and clinical relevance.

Objective: To describe the PHC prevalence and to evaluate the variability in diagnosis, the relation to severity of liver disease, and the incidence of severe outcomes.

Design: Cross-sectional study.

Setting: Endoscopic unit of a tertiary-care academic center in Rio de Janeiro, Brazil.

Patients: Patients with cirrhosis with portal hypertension and controls paired for age and sex.

Interventions: All patients were submitted to standard and image-enhanced colonoscopies, which were recorded in a coded video file and analyzed twice by a blinded endoscopist.

Main Outcome Measurements: The prevalence of PHC.

Results: A total of 51 patients with cirrhosis (55% male, mean age 59 years) and 51 healthy controls (43% male, mean age 61 years) were included. The top ranking colonoscopic findings were angiodysplasia-like lesions, nonspecific vascular pattern, red spots, and colorectal varices, all significantly more frequent in patients with cirrhosis compared with controls. PHC prevalence was 71% in patients with cirrhosis. For PHC, interobserver and intraobserver agreement (*k* values [standard error]) were 0.68 (0.09) and 0.63 (0.10), respectively. Intraobserver agreement for colonoscopic findings was satisfactory. PHC was not related to more severe liver disease or liver stiffness. Only 5 patients developed severe outcomes during follow-up.

Limitations: The exclusion of patients with cirrhosis without esophageal varices and the absence of an interobserver agreement analysis by double-blinded endoscopists.

Conclusion: PHC was highly prevalent in patients with cirrhosis, and its diagnostic agreement was satisfactory. PHC is not associated with relevant severe outcomes in a 12-month follow-up. (Gastrointest Endosc 2015;82:469-76.)

Liver cirrhosis, the main cause of portal hypertension, causes mucosal and hemodynamic changes in the entire GI tract.[1] Gastroesophageal varices and portal hypertensive gastropathy are highly prevalent manifestations of portal hypertension in patients with cirrhosis, causing severe and life-threatening outcomes such as active bleeding,

Abbreviations: CRC, colorectal cancer; FICE, Fujinon Intelligent Color Enhancement; PHC, portal hypertensive colopathy.

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responsible for high rates of mortality.[2] Vascular changes such as angiodysplasia-like lesions, isolated or diffuse cherry red spots, and colorectal varices have been described as colonoscopic findings of portal hypertensive colopathy (PHC) in cirrhosis.[3,4] The prevalence of PHC in patients with cirrhosis varies from 3% to 84%,[5] and its clinical relevance is not well-clarified.[6] Controversial results have been described concerning the relationship of this condition with the severity of liver disease.[7,8] The diagnostic criteria, prevalence, and management of portal hypertension–related outcomes in the upper GI tract are well-established.[9] However, the management of PHC has been challenged by its variability in diagnostic criteria and controversial results on prevalence and clinical relevance of this condition.

The aims of our study were to (1) describe the prevalence of PHC as well as colonoscopic findings in patients with cirrhosis compared with age and sex paired volunteer controls without liver disease, (2) evaluate the intraobserver and interobserver variability in diagnosis of PHC, (3) evaluate the association of PHC with the severity of liver disease, and (4) evaluate the incidence of severe outcomes in patients with cirrhosis with PHC.

METHODS

Study design

This cross-sectional study was conducted at the University of the State of Rio de Janeiro, Brazil. Patients with cirrhosis aged >18 years with esophageal varices or signs of previous endoscopic band ligation were eligible for inclusion from March 2011 to June 2013. All patients included were submitted to standard colonoscopy complemented with an image-enhanced endoscopy method. Exclusion criteria were (1) diagnosis of sepsis, (2) presence of hepatic encephalopathy grade ≥ 2, (3) any contraindication for colonoscopy performance, or (4) incomplete examination or inadequate bowel preparation. In a second step, patients with cirrhosis were prospectively followed for at least 12 months to evaluate the incidence of severe outcomes related to PHC, such as acute lower GI bleeding or mortality. Controls paired for age and sex from individuals referred to colonoscopy during the recruitment period also were included. The study protocol was conducted in accordance with ethical principles, the guidance of the Helsinki Declaration, and the Good Clinical Practice Guidelines. The study was approved by the local ethics committee. All participants signed the informed consent on enrollment at the study.

Patients with cirrhosis and healthy controls

Diagnosis of cirrhosis was defined by US findings, liver biopsy (METAVIR score = F4),[10] or presence of liver-related outcomes such as ascites, hepatic encephalopathy, or abdominal collateral vessels. All patients with cirrhosis were submitted to laboratory blood analysis and transient elastography, with a maximum delay of 3 months before or after colonoscopy. The results of upper GI endoscopy performed in the previous 6 months were considered. Portal hypertension was defined by the presence of esophageal varices or endoscopic cicatrices of previous endoscopic band ligation. Severity of liver disease was evaluated by the Child-Pugh classification[11] and the Model for End-stage Liver Disease calculation.[12]

Age and sex controls were randomly selected from consecutive individuals aged >18 years who were referred for colonoscopy for colorectal cancer (CRC) screening, lower digestive bleeding, or anemia. Liver disease in the control group was excluded by a normal clinical examination and no fibrosis by transient elastography (liver stiffness measurement <7.1 *k*Pa).[13]

Transient elastography was performed following a validated procedure by using an M probe of FibroScan (Echo Sens, Paris, France) previously described elsewhere.[14] Transient elastography was considered reliable when the following criteria had been met: (1) 10 successful measurements, (2) an interquartile range $<30\%$ of the median value, and (3) a success rate of $>60\%$.[15] Liver stiffness was considered as the median of all valid measurements. Liver stiffness measurement was defined as unreliable if it did not meet the earlier-described criteria.

Colonoscopy

After an overnight fast, patients were administered a 750-mL, 20% mannitol solution as a bowel-cleansing regimen. Colonoscopies were performed with patients under conscious sedation in day-clinic hospitalization by a single experienced endoscopist (R.A.) by using a Fujinon EC450LP model videoscope (Saitama City, Japan). This model had an image-enhanced endoscopy method known as Fujinon Intelligent Color Enhancement (FICE) that is capable of observing the mucosal epithelium of microstructure and capillaries highlighting colon lesions.[16] All standard colonoscopies were complemented by a complete colon examination by using FICE. Colonoscopic examinations (standard colonoscopy plus FICE) from patients and controls were recorded in a high-definition video file and were coded by using a random number generator to be evaluated twice afterward by another experienced endoscopist (F.A.F.F.) blinded for clinical data. Basically, all coded video files were analyzed by the experienced blinded endoscopist in a random order for description of colonoscopic findings and diagnosis of PHC. Both endoscopists (R.A. and F.A.F.F.) underwent a training program for video analysis agreement in a previous pilot study. Interobserver agreement was estimated between nonblinded and blinded endoscopists. Then in a second step, all video files were re-evaluated by the same blinded endoscopist in a different random order to evaluate intraobserver agreement on colonoscopic findings and diagnosis of PHC.