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Case Report

Portal hypertensive enteropathy: multimodality assessment through computed tomography and magnetic resonance enterography ☆

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ABSTRACT

Portal hypertension consists in an increased portal vein pressure due to prehepatic, hepatic, or posthepatic conditions, with cirrhosis representing the most common cause. The gastrointestinal tract can be secondarily affected not only with varices formation, whose rupture is one of the most dangerous complications, but also with small and large bowel involvement which can predispose to chronic intestinal bleeding. These conditions respectively take the name of portal hypertensive enteropathy and portal colonopathy and their assessment are of almost exclusive pertinence of endoscopic techniques. Up to now, only few reports have described this condition from the radiological point of view. Nevertheless, imaging modalities are not burdened by the invasiveness of endoscopic procedures and are also capable in providing useful information about the intestinal tract as well as the surrounding tissues. This is the first case reporting a diffuse involvement of the small bowel and the right colon in a patient suffering of portal hypertension due to cirrhosis evaluated through the performance of computed tomography and magnetic resonance enterography.

Introduction

Portal hypertension implies an increased portal vein pressure with a hepatic venous pressure gradient, measured between portal and hepatic veins, higher than 5 mm Hg.

Portal hypertension can be classified as prehepatic, hepatic, or posthepatic on the base of the clogging point.

Among the different underlying causes, cirrhosis is definitely the most common.

Higher resistance to portal venous flow is determined by parenchymal changes, mainly consisting in fibrotic involution
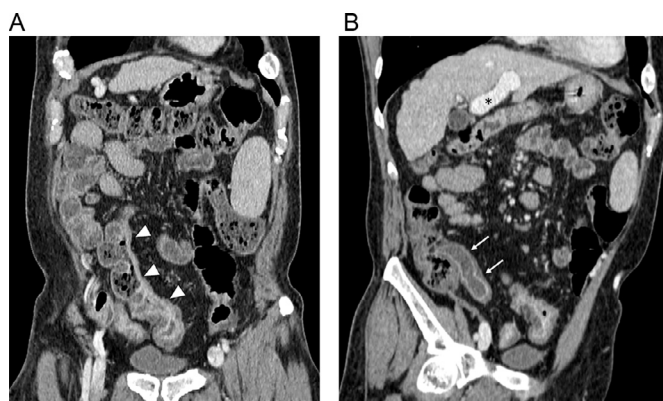
Fig. 1 – Contrast-enhanced CT-scan on coronal (a) and coronal-oblique (b) planes. Diffuse mural thickening of the last ileal loops and the right hemicolon is detectable and more evident along the mesenteric borders (arrowheads). Contrast enhancement had a layered appearance (arrows). Irregular edges of the liver and increased caliber of the left intra-hepatic brunch of portal vein (asterisk) are also visible.

with regenerative nodules formation, and increased vascular tone.

Beyond the value of approximately 10-12 mm Hg, the portal circulation is shunted into the systemic one with a steady dilation of collateral vessels and development of mesenteric varices [1–3].

Although fibrosis and hepatic venous pressure gradient can be respectively quantified through biopsy and balloon catheter positioning within a main intrahepatic vein, imaging modalities have been shown capability to contribute in assessing cirrhosis degree and portal hypertension estimation through a less invasive approach [4–9].

Consequences of portal hypertension on the gastrointestinal tract are often underrated and seldom reported.

In fact, beyond the major risk of esophageal or gastric varices hemorrhage, portal hypertensive enteropathy (PHE), and portal colonopathy may arise [10,11].

These conditions consist in a bowel wall thickening due to submucosal edema from hypoproteinemia, possible polypoid appearance of the mucosa and engorgement of intestinal vessels.

Considering the increased weakness of the mucosal layer and vascular congestion, the risk of acute or chronic bleeding is high and can explain anemic states in cirrhotic patients [10].

Up to now, poor consideration has been given to intestinal consequences of portal hypertension from the radiologic point of view, which mainly relies on the endoscopic approach [11,12].

Nevertheless, computed tomography (CT) and magnetic resonance enterography (MRE) represent wide available and noninvasive alternatives for intestinal assessment.

In particular, CT-scan is largely performed on cirrhotic patients for evaluation of the primary disease and the related complications.

On the other hand, MRE due to the high soft tissue resolution and the luminal dilation can further help in detecting and defining intestinal lesions without any additional radiation exposure [13,14].

To the best of our knowledge, this is the first case describing at once the performance of CT-scan and MRE for assessment of PHE in a cirrhotic patient.

Case report

A 62-year-old male patient affected by Hepatitis B virus related cirrhosis (Child Pugh Class A score 6, MELD 10) was admitted to our hospital due to severe anaemia and abdominal pain.

At clinical examination, engorgement of cutaneous veins of the anterior abdominal wall (the so-called "caput medusae" sign) were noted.

Laboratory tests were as follows: hemoglobin 7.1 g/dL (normal range: 13.8–17.2 g/dL), platelets 67,000/mcL of blood (normal range: 135,000 and 317,000/mcL of blood), serum bilirubin 0.64 mg/dL (normal range: 0.3-1.2 mg/dL), alanine aminotransferase 15 U/L (normal range: 0-57 U/L), aspartate transaminase 19 U/L (normal range: 0-48 U/L), serum total protein 5.2 g/dL (normal range: 6.0-8.3 g/dL), and international normalized ratio (INR) of 1.22 (normal range: 0.8-1.2). Stool tested positive for occult blood test.

In order to exclude active abdominal bleeding, a contrast-enhanced CT-scan (Siemens Somatom definition 64-slice) was performed before and after injection of intravenous contrast medium (Iomeron 400 mg iodine/mL; Bracco Imaging SpA, Milan, Italy; iodine delivery rate: 1.2 mL/Kg; flow rate 3.5 mL/s).

CT findings consisted in shrunken liver with irregular edges and portal hypertension stigmata, including portal vein dilation (17 mm), recanalization of paraumbilical veins, engorgement of oesophageal, peri-splenic, left gastric, and anal canal portosystemic vascular collaterals as well as splenomegaly (maximum diameter: 17 cm).

A diffuse thickening of the ileal loops and the right hemicolon walls was also detectable, with a stratified appearance of mural enhancement (Fig. 1).
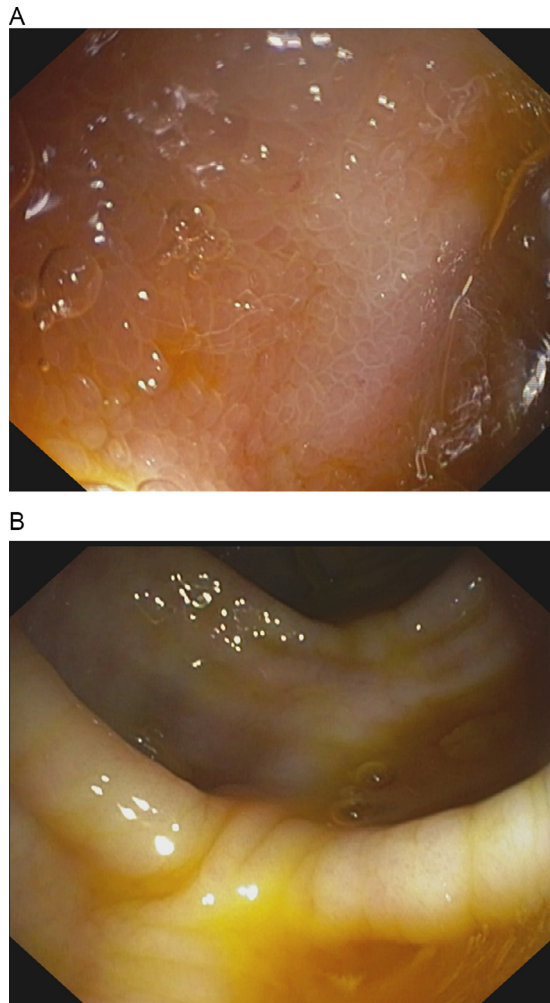
Fig. 2 – Endoscopic appearance of the ileum (a) and of the colon (b) at the time of patient's admission showing a normal mucosal surface.

The latter findings raised the clinical suspicion of an inflammatory bowel disease.

However, due to the lack of luminal dilation, an accurate intestinal assessment was not achievable.

Endoscopy of upper gastrointestinal tract revealed esophageal varices (1st grade), with no evidence of active or recent bleeding, in addition to congestive gastropathy.

Colonoscopy showed normal mucosa along the small and large bowel as well as hemorrhoids (1st grade) (Fig. 2).

Targeted biopsies were obtained in terminal ileum (explored for 10 cm), ascending colon, descending colon, sigma, and rectum. Histopathological examinations revealed a mild, nonspecific inflammation with preserved architecture of the mucosal layer.

In order to better assess the wall thickening of the small bowel and to spare the patient an additional amount of ionizing radiation, a MRE was performed.

After prior ingestion of 1500 mL of polyethylene glycol-water solution, the patient was placed in supine position inside the scanner. The following sequences were ac-

quired: coronal thick-section T2-weighted rapid acquisition with relaxation enhancement (RARE) acquisition, axial, and coronal T2-weighted true fast imaging with steady-state precession (True-FISP, repetition time/echo time: 4.20/2.10 ms, flip angle: 60°), and half-Fourier acquisition single-shot turbo spin echo (HASTE, repetition time/echo time: ∞ /80 ms) with and with- out fat suppression were performed, diffusion-weighted imaging (DWI) scans, obtained on the axial plane using a diffusion factor b fixed at 0, 400, and 800 s/mm^2.

MRE allowed recognizing a concentrical mural thickening of the small bowel extending proximally for 55 cm from the ileocecal valve, with a segmental distribution due to the presence of narrowed and dilated tracts. Wall thickness was mild to moderate, reaching a maximum of 9 mm in width.

Pathologic loops were widely hyperintense on T2-weighted images with fat saturation, due to mural oedema.

Diffusion-weighted and apparent diffusion coefficient calculation showed water restriction, particularly evident in the thickest loops mucosal border.

Enlarged mesenteric blood vessels, hypertrophy of mesenteric fat and lymphadenopathy were also detected on True-FISP acquisitions (Fig. 3).

Hepatic and extrahepatic findings of cirrhosis, such as irregular hepatic edges, splenomegaly, mild ascites, and the portosystemic collateral pathways, were also appreciable.

The patient was subsequently discharged with home therapy and a clinic follow-up scheduled at 3 months. At that time, Hb was 12.7 g/dL and cytolysis and cholestasis enzymes were negative. Faecal calprotectin testing was 2380 mg/kg (negative values: <50 mg/kg), demonstrating persistent intestinal inflammation.

Discussion

The term PHE refers to mucosal layer alterations within the small bowel caused by an increased portal pressure [12].

It is well known that cirrhotic patients can present intestinal dysmotility, impaired absorption and alteration of the local microbiota [15,16].

However, the exact pathogenesis of PHE has not been completely understood so far.

One of the possible underlying causes is that vascular congestion due to portal hypertension could lead to ischemia and arteriovenous shunt formation.

Other theories point out an increased permeability of the intestinal barrier with an overstated inflammatory process or a goblet cells hyperplasia.

PHE usually arise in case of compromised or decompensated cirrhotic disease with coexistent esophageal varices.

When symptomatic, PHE manifests with chronic intestinal bleeding, with consequent iron-deficiency anemia, or acute hemorrhage with melena, hematochezia or, less frequently, hematemesis [12,17].

PHE has been reported in up to 97% of the patients, although the incidence is extremely variable on the basis of the type of endoscopic procedure performed [12,17].
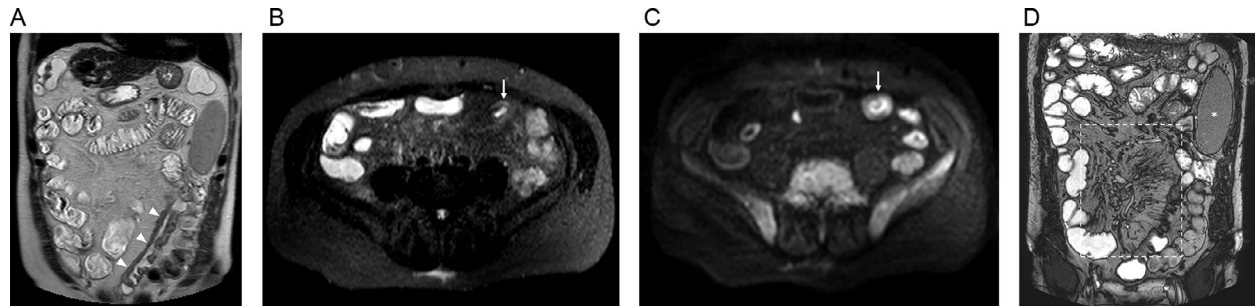
Fig. 3 – Coronal T2-w HASTE (a) confirmed the bowel wall thickening, also characterized by hyperintensity (arrows) on axial T2-w SPAIR (b) and axial DWI at 800 s/mm^2 b-value (c) due to edema and inflammatory cells infiltration. Coronal True-FISP (d) also demonstrated engorgement of mesenteric vessels (within the dotted square) and splenomegaly (asterisk).

Higher rates have been recorded when enteroscopy and capsule endoscopy were performed [17].

The employment of these techniques allowed a better characterization of intestinal lesions, framing them as vascular or inflammatory-related [12,17].

Among the former, telangiectasias, angiodysplastic-like lesions, varices, and active bleeding can be detected, while the latter may include ulcers, edema, strictures, and mucosal erosions [18,19].

The small bowel and the right colon are the preferred localizations of PHE, due to a major effect of portal hypertension on the superior mesenteric vein and a higher number of additional collaterals of the left colon which help in decreasing the hydrostatic pressure.

Otherwise, if the inferior mesenteric vein is more severely affected, intestinal lesions will arise in the remaining large bowel segments [11,20].

Considering that mural thickening is a typical consequence of PHE, this condition should be included in the differential diagnosis of inflammatory bowel diseases [21].

Up to now, radiological evaluation of PHE has been performed through abdominal CT-scan and MRI without distension of intestinal loops.

The findings previously described include wall thickening and mural enhancement which can be homogeneous or with a targeted appearance [11,20].

Pneumatosis and mucosal thumbprinting due to superimposition of Clostridium difficile infection have been reported in case of colonic involvement [11].

In our case, a diffuse and irregular wall thickening of the ileal loops was detected, with a lesser involvement of the right colon.

At CT-scan, a layered enhancement of the intestinal walls was visible and more evident on the mucosal surface.

On MRE, T2-w sequences with fat suppression aided in recognizing mural edema, while True-FISP images demonstrated vasa recta engorgement.

Edema within the intestinal walls can be explained by hypoproteinemia and increased portal pressure with lymphatic insufficiency [11].

DWI scans at the highest b-value overlapped CECT findings showing higher intestinal signal along the mucosal layer, which could be explicated by water molecules restriction due to inflammatory changes.

This case demonstrates that a radiological approach for PHE evaluation is feasible and easily achievable.

Cirrhotic patients often undergo follow-up CECT scan during their lifetime.

However, these exams are generally targeted to the primary disease and its main complications, whereas PHE could pass unnoticed or be underestimated.

A prior dilation of the intestinal loops through oral contrast medium solution would be advisable, but this depends on the clinical suspicion and may vary among different centers.

In such cases, MRE could reasonably complement the information of CT scan without any further radiation exposure of the patient.

Moreover, DWI scan allows avoiding additional administration of intravenous contrast medium.

Conclusion

Anemia in cirrhotic patients can occur due to multiple causes.

Especially in case of chronic anemic states, the diagnostic hypothesis of PHE has to be taken into account.

Currently, endoscopic procedures represent the mainstay of PHE assessment, but their evaluation is invasive and limited to the mucosal surface.

On the other hand, the performance of imaging modalities in this field have been somehow overlooked although they can safely provide useful information about intestinal walls as well as the surrounding structures and the abdominal organs.

This case proves that radiologists and clinicians should be aware of the diagnostic potential of CT-scan and MRE in order to achieve an exhaustive appraisal of PHE through a noninvasive approach.

Patient consent

This work was in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Informed consent was obtained from the patient.

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