stopped PPI therapy and gastric mucosa was healed completely (Fig. 1e).

Only a few cases of hepatic penetration by peptic ulcer disease endoscopically diagnosed have been reported.[5] Like in this case, imaging methods were not conclusive and most presented with upper gastrointestinal bleeding without abdominal pain.[5] Hp infection and the use of NSAIDs were the identified risk factors for PU in this case. The reported most common endoscopic findings in this situation are giant ulcers that can mimic malignancy (as seen in our patient) and pseudotumoral mass protruding from the ulcer bed or a mass without ulcer[4,5]; the diagnosis also was established after endoscopic biopsies revealing hepatic tissue.[4,5] The histological changes found in our patient are similar to those reported in literature[4,5], which result from advanced peptic ulcer digestion, a process called peptic hepatitis.[5] This usually does not result in changes in liver function tests.[5] Therefore, abnormal results in this group of patients is of limited diagnostic value.[4,5] The majority of patients required surgery for the management of hepatic penetration and in less than one third of the reported cases, medical conservative treatment was possible.[4,5] In conclusion, we report a very rare complication of PU ulcer that requires a high index of suspicion for the diagnosis and in which the medical treatment was effective.

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Joana Rita Carvalho[a,*], Patrícia Sousa[a], João Lopes[a], Francisco Tortosa[b], Santiago Ortiz[b], José Velosa[a]

[a] *Department of Gastroenterology, CHLN – Hospital de Santa Maria, Portugal*
[b] *Department of Pathology, CHLN – Hospital de Santa Maria, Portugal*

* Corresponding author.
E-mail address: joana.rita.carvalho@gmail.com (J.R. Carvalho).
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Pneumatosis intestinalis in Crohn's disease☆

Neumatosis intestinal en la enfermedad de Crohn

Pneumatosis intestinalis (PI), or the presence of gas in the interstitium of the bowel wall, is a radiological sign present in diseases of varying severity which involves several diagnostic and therapeutic approximations. We discuss the cases of 2 patients with Crohn's disease (CD) who presented with asymptomatic cystic PI as a CT scan finding, who made good progress after conservative treatment.

The first case concerns a 60-year-old female patient with long-standing fistulising CD (phenotype A2L3B3+P). She had taken various treatments which she abandoned because they were ineffective or due to side effects and required 2 ileocolic resections, one of them complicated by anastomotic dehiscence. Consequently, she herself decided to discontinue treatment, and her course was characterised by well-tolerated clinical signs and symptoms and very mild elevation of acute phase reactants. At the same time, she presented with signs and symptoms of renal and ureteral calculi which required a nephrostomy and placement of a double-J catheter. The findings on the follow-up CT scan included cystic PI in the ascending and transverse colon (Fig. 1). At the time, the patient only experienced her usual mild abdominal discomfort, with no peritoneal irritation. Alerted by the department of radiodiagnostics, we informed the patient and obtained laboratory test results which showed c-reactive protein (CRP) of 8 mg/l (normal: <5), with no leukocytosis.

The second case involves a 58-year-old female patient with long-standing CD with strictures (phenotype A1L3B2) which required 2 ileal resections and treatment with Salazopirina® [sulfasalazine] and prednisone. These were followed by azathioprine, which was discontinued as she developed signs of portal hypertension. Subsequently, she took no treatment and experienced good clinical control. Over the course of her follow-up, the patient experienced repeated episodes of urolithiasis requiring the placement of a double-J catheter and a left nephrectomy, without subsequent recurrences. Three years later an abdominal CT scan was requested because of mechanical low back pain in

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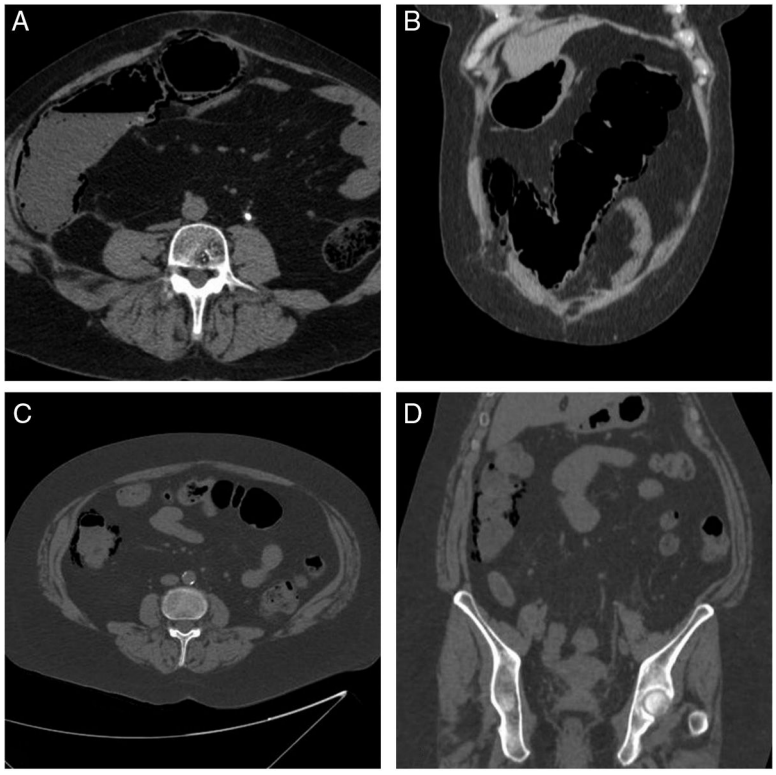

Figure 1 Case 1: axial (A) and coronal (B) slices from the abdominal and pelvic CT scan without contrast medium in which extensive pneumatosis intestinalis is observed in the ascending and transverse colon. Case 2: axial (C) and coronal (D) slices from the abdominal and pelvic CT scan without contrast medium in which pneumatosis intestinalis limited to the ascending colon is observed.

which cystic PI was observed in the ascending colon (Fig. 1), resulting in her being referred to gastroenterology as an emergency case. At the time she was asymptomatic, and the laboratory tests showed CRP at 1.2 mg/l (normal: <5), with no leukocytosis.

In both cases, the PI was considered a finding and was treated conservatively with clinical and radiological monitoring. Partial resolution of the episode was observed on the follow-up CT scans after three weeks and four months, respectively.

Historically, PI has been considered as a sign of severity associated with intestinal ischaemia. However, it can be secondary to highly diverse causes, including among others infection, inflammation and iatrogenic reasons.[1] The generalisation of cross-sectional imaging techniques and their increased sensitivity have resulted in increased diagnoses of this radiological finding. This has led to a search for indicators which enable the image to be correlated with the severity of the underlying disease and to determine whether or not emergency surgery is required.

The theories most accepted to explain the onset of PI are bacterial, mechanical and pulmonary.[2] PI is not a common finding in patients with inflammatory bowel disease (IBD), but it has been described previously, sometimes as an endoscopic complication.[3] A case-control study showed a higher incidence in patients with IBD than in the general population and was associated significantly with corticosteroid therapy.[4] No association has been described between renal and ureteral calculi or manipulation of the urinary system and pneumatosis intestinalis, although this has been the case with abdominal surgery,[5] which both patients required.

Abdominal pain and peritoneal irritation are among the clinical findings which correlate PI with intestinal ischaemia.[5] In laboratory tests, lactic acid and CRP levels as well as leukocytosis are notable, although the degree of significance associated with each of these varied in the different studies.[1,5]

Concerning the radiological pattern, several authors have described the association between the linear pattern of PI and ischaemia respective of the benign nature of the cystic pattern.[2] It has not been possible to correlate gas localisation or distribution with severity of the finding or underlying disease, although, generally, isolated colonic involvement is considered benign with respect to small intestine involvement. Other warning signs of ischaemia include reduced mural contrast enhancement, dilatation of the intestinal loops, ascites and mesenteric fat stranding.[2]

For management of idiopathic or benign PI there is consensus that clinical monitoring and personalised conservative treatment are sufficient.[4] Hyperbaric oxygen therapy[2] and antibiotic therapy with metronidazole[4] have been suggested.

In conclusion, the onset of PI can occur in patients with inflammatory bowel disease. It is a radiological finding and not a clinical diagnosis, and as such should be interpreted in the context of its onset. There are various elements which can help us evaluate its severity and

the need for emergency surgical intervention. When PI is considered a benign finding, conservative management under which patients show good clinical progress is adequate.

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Carolina González-Olivares[a], Ana Palomera-Rico[b], Nieves Romera-Pintor Blanca[b], Rubén Sánchez-Aldehuelo[a], Alexandre Figueroa-Tubío[a], Irene García de la Filia[a], Antonio López-Sanromán[a,*]

[a] *Servicio de Gastroenterología y Hepatología, Hospital Universitario Ramón y Cajal, Madrid, Spain*
[b] *Servicio de Radiodiagnóstico, Hospital Universitario Ramón y Cajal, Madrid, Spain*

* Corresponding author.
E-mail address: alsanroman@salud.madrid.org
(A. López-Sanromán).

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Acute myocardial infarction in a patient with a recent diagnosis of Crohn's disease☆

Infarto agudo de miocardio en paciente con diagnóstico reciente de enfermedad de Crohn

Introduction

Inflammatory bowel disease (IBD) is an independent risk factor for the onset of thromboembolic events (TE). We know that TE are extraintestinal manifestations that significantly increase morbidity and mortality in these patients.

Case report

Our case study is that of a 19-year old male patient with no substance abuse, recently diagnosed with Crohn's ileocolitis during a moderate flare-up, and receiving outpatient treatment with corticosteroids and azathioprine. He came to the Emergency Department with chest pain associated with autonomic signs and symptoms of onset a few hours earlier. The electrocardiogram showed ST segment elevation with morphology characteristic of a subepicardial injury in V1–V4. Additional findings included increased cardiac enzymes revealed in blood tests and an emergency echocardiography suggestive of extensive anterior wall myocardial infarction. Emergency catheterisation was performed, revealing a thrombotic occlusion of the middle segment of the left anterior descending artery which was treated with a drug-eluting stent. In addition, dual antiplatelet and anticoagulation therapy with acenocoumarol was started. During this episode, he underwent CT angiography, which revealed a renal and splenic infarction and hepatic and colonic perfusion disorder (Fig. 1). The antiphospholipid antibodies, factor deficiencies (protein C, protein S and antithrombin III), mutations associated with thrombophilias (Factor V Leiden, prothrombin) and homocysteine levels were negative or in the normal range. Following his recovery from the acute episode, the patient continued receiving the anticoagulation. The course of the Crohn's disease was indolent; the patient developed a complex perianal fistula and abscesses, requiring surgical drainage and combined treatment with biological therapy and immunosuppressants to control the disease.

Discussion

Venous and arterial thromboembolism is an extraintestinal manifestation which significantly increases morbidity and mortality in patients with IBD.[1] TE are 3 times more common in patients with IBD than in controls, and the relative risk increases 15-fold in disease flare-ups.[2] These patients have an increased risk of developing coronary artery disease, at younger ages and without associated cardiovascular risk factors,[3] as in the case of our patient. Although the principal hypothesis is that the inflammation maintained by the IBD flare-up causes an acceleration of the process of atherosclerosis and development of thrombosis,[4] the underlying thrombotic mechanism is complex, multifactorial and not completely elucidated.

There are some related acquired factors such as treatment with corticosteroids, prolonged bed rest or canalisation from central venous catheters. It has also been speculated that certain genetic mutations in thrombosis factors could be the cause of increased TE in these patients.

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