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Irritable Bowel Syndrome with predominant diarrhea and giardiasis: Is it one or the other?☆

Síndrome del intestino irritable con predominio de diarrea y giardiasis: ¿es uno u otra?

Diarrhea-predominant Irritable Bowel Syndrome (IBS-D) is where more than a quarter (25%) of bowel movements are type 6 or 7 on the Bristol Stool Form Scale, and less than a quarter are type 1 or 2. The symptoms must meet the corresponding Rome IV criteria: recurrent abdominal pain, at least one day per week (on average) in the last three months, associated with at least two of the following criteria: (1) Related to defaecation; (2) Associated with a change in stool frequency; and (3) Associated with a change in stool form (appearance).[1]

Giardiasis is a common cause of infectious gastroenteritis worldwide, being associated with poverty, with a prevalence that varies from 2% (high-income countries) to 30% (low-income countries).[2]

The typical symptoms of giardiasis which include diarrhea, often explosive, especially in the morning, without blood or mucus, flatulence, abdominal pain and swelling,[2] often suggest IBS-D, with which a differential diagnosis must be made. However, with certain frequency, this can become difficult with the usual procedures and end up going unnoticed.

We present the case of a 29-year-old woman who reported having travelled to Mexico over a year beforehand, where she had acute gastroenteritis that took a long time to resolve. She reported weight loss, abdominal distension and pain, and chronic explosive diarrhea, with numerous soft-liquid stools, urgency and relief of pain when expelling wind and/or faeces, since her return from the trip. All tests, including coeliac serology, thyroid function, serial stool cultures/ova and parasite exams and a colonoscopy, were completely normal, and her symptoms were interpreted as a post-infectious-type IBS-D, probably in relation to some viral or parasitic infection acquired in Mexico.

The conventional treatment provided no improvement. With the abdominal pain and diarrhea persisting, treatment was recommended with paroxetine 20 mg/day.

As there was only slight improvement after several months of treatment, the patient was advised to increase the dose to 50 mg/day (30-0-20). After another two months, still with no improvement, further stool cultures/ova and parasite exams were performed, which were again negative, and it was decided to try treatment with rifaximin (400 mg/twice daily, one week a month, repeating for another month), with no results.

Finally, an endoscopy was performed with duodenal biopsies to rule out another malabsorption disorder (biochemically, there were no data to suggest that), and possible duodenal giardiasis, although the stool cultures/ova and parasite exams had been repeatedly negative, with no eggs or trophozoites detected. Duodenal biopsies were also normal, with no evidence of atrophy or giardiasis. We requested that a PCR for *Giardia duodenalis* also be performed on the biopsies and this came back positive, clarifying a diagnosis which had been eluding us for months. The patient was treated with tinidazole 50 mg/kg body weight, in a single oral dose, which led to rapid improvement in symptoms within a few weeks.

There are studies[3] that find a strong association between giardiasis and post-infectious IBS (PI-IBS) in young people. D'Anchino et al.[4] studied 100 patients with symptomatic giardiasis and found that in 82 of them IBS had previously been identified, suggesting that the symptoms attributed to giardiasis may, in fact, be the result of pre-existing IBS, exacerbated by *Giardia* infection.

Moreover, individuals with giardiasis are approximately four times more likely to be diagnosed with IBS 90 days after the diagnosis of giardiasis than those without giardiasis.[3]

In terms of the relationship between the two disorders, giardiasis may be a trigger for the exacerbation of IBS, but the parasitic infection is no longer necessary for the symptoms to persist once they have become established.[4]

From our experience, we recommend performing a PCR assay on a stool sample in patients with symptoms suggestive of IBS-D who have travelled abroad, particularly to low-income areas (risk factor for giardiasis), who have persistent diarrhea, even if stool investigations, including stool cultures and ova and parasite exam, are persistently negative.

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Portal hypertension as a complication of hepatic hydatidosis☆

Hipertensión portal como complicación de hidatidosis hepática

Approximately 75% of the hydatid cysts that develop in humans are caused by the larvae of *Echinococcus granulosus* that settle in the liver. They tend to be paucisymptomatic for years, with the most common complications being infection and rupture.[1]

We describe the case of a patient with multiple hepatic hydatidosis and portal hypertension apparently caused by compression which began as severe gastrointestinal bleeding secondary to the development of oesophageal varices.

This was a 40-year-old patient from Morocco with a previous history of hepatic hydatid disease with partial hepatectomy performed 30 years previously due to rupture of a hepatic cyst. He was admitted for a first episode of upper gastrointestinal bleeding due to oesophageal varices, with normal liver function. Somatostatin and endoscopic ligation were used to stabilise the patient's condition.

Magnetic resonance imaging and CT-angiogram of the liver identified two giant hepatic cysts, one measuring 9 cm and the other measuring 10 cm, causing portal hypertension as a result of extrinsic compression at the bifurcation and the main portal branches. The liver parenchyma showed no clear signs of cirrhosis, there was an increase in the size of the splenic vein, splenomegaly and portosystemic oesophageal and fundal collaterals. A liver biopsy was also taken which ruled out liver cirrhosis.

The patient made poor progress with early recurrence of the bleeding in the form of hematemesis, requiring admission to the ICU and further medical and endoscopic treatment. In addition, urgent transjugular intrahepatic porto-systemic shunt (TIPS) was performed, without great technical difficulties, this time achieving definitive haemostasis.

The patient was assessed by the general surgery department of the referral hospital to assess the possibility of liver transplantation versus elective cystectomy. In the end, they opted for surgical resection of the hepatic cysts, at least the larger one compressing the portal vein.

When such cysts are located close to the hilum of the liver they can compress it or cause it to rupture into the main bile ducts and cause jaundice.

In the literature, rare cases of portal hypertension secondary to cystic compression have been reported, this time associated with the alveolar pattern caused by E. multilocularis, and in polycystic disease caused by E. vogeli and oligarthrus.[2,3]

The hydatid cysts caused by *E. granulosus* are usually benign and can be asymptomatic for years. However, in the long term they can cause different complications and symptoms depending on which part of the liver they are located in.

The predominant symptom is right hypochondriac abdominal pain, which can radiate towards the shoulder and be accompanied by abdominal distension, cholestasis, portal hypertension and/or ascites.

Hepatic hydatid disease is a rare cause of portal hypertension. However, it should be considered above all in patients who live in endemic areas and have a hepatic mass.[3–5]

Several mechanisms have been described to explain portal hypertension in patients with hepatic hydatid disease: compression of the portal vein or its branches, as happened in our case; the cavernous transformation of the portal vein itself and obstruction of the splenic vein (with development of segmental portal hypertension); and compression and obstruction of the suprahepatic veins (Budd-Chiari syndrome). The main clinical manifestation of this complication is usually gastrointestinal bleeding; although in the last of the above cases it can also present as ascites.

The treatment of choice for these patients continues to be surgery. However, the fact that patients also tend to have a coagulation disorder means attempts are made with more conservative management, combining medical treatment with interventional radiology techniques. In addition to medical treatment with albendazole, these patients should be given beta-blockers as prophylaxis for variceal bleeding. If the patient's liver function deteriorates or they develop untreatable portal hypertension, they may be candidates for liver transplant.

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