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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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