CLINICAL CASE

EDEMOASCITIS DECOMPENSATION. IS IT ALWAYS LIVER CIRRHOSIS?

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Abstract

Portal hypertension (PH) is a clinical syndrome characterized by an increase in the portal hepatic venous pressure gradient (HVPG), defined as the gradient between the portal vein and the inferior vena cava. When there is clinically significant PH, it is usually associated with clinical events such as the development of esophagogastric varices (EGV), oedemoascitic decompensation, encephalopathy... these being more frequent in the case of PH due to liver cirrhosis.

In all etiologies of PH there is an increase in resistance to portal blood flow, followed by an increase in said flow. Depending on the location of the increased resistance, it will be classified as prehepatic, intrahepatic or posthepatic PH[1]. Posthepatic portal hypertension occurs when there is a deterioration in hepatic venous outflow, increasing resistance at this level.

We present the case of a patient where a striking portal dilation led to the diagnosis of posthepatic PH.

Keywords: posthepatic portal hypertension, ascites, membranous occlusion of the inferior vena cava.

Clinical case

A 72-year-old man with no personal history of interest or toxic habits was admitted for diarrhoea of two months' duration, associated with constitutional syndrome, ascites and new-onset oedema.

Laboratory tests showed slight leukocytosis with neutrophilia, slight deterioration of renal function with creatinine 1.34mg/dl and hypoalbuminaemia, with normal platelets, hepatobiliary profile and coagulation.

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During admission, abdominal ultrasound, computed tomography (CT) and angioCT (imaging) were performed, showing findings suggestive of chronic liver disease and very striking dilatation of the main portal and both intrahepatic branches, with reduced calibre at the level of the inferior vena cava, in the segment entering the liver that appears practically filiform, as well as reduced calibre of the proximal segment of the suprahepatic veins, these being permeable, with no associated splenomegaly. Upper gastrointestinal endoscopy ruled out the presence of varicose veins and echocardiography was performed, with no abnormalities.

During admission, the patient evolved favourably, maintaining a good diuresis rate, decreasing oedema and abdominal perimeter with depletive treatment, reaching the diagnosis of post-hepatic PH secondary to membranous obliteration of the inferior vena cava.

Discussion

Post-hepatic portal hypertension occurs when there is impaired hepatic venous outflow and increased resistance at this level[1]. The most common cause of posthepatic PH is Budd-Chiari syndrome (BCS), right heart failure or obstruction of the inferior vena cava by tumours, thrombosis or caudate enlargement[2].

BCS is a clinical entity that encompasses a group of syndromes that can cause obstruction of hepatic venous drainage at any level, from the small hepatic veins to the junction of the IVC with the right atrium[2].

It is a rare entity. The main causes are neoplastic, haematological, inflammatory, infectious and pharmacological. However, after ruling out these aetiologies, other less common ones should be considered, such as membranous occlusion of the IVC (MOIVC), also known as obliterative hepatocavopathy, or hepatic vena cava syndrome[3].

Regarding the pathophysiology, a congenital origin due to a failure in the embryogenesis of the inferior vena cava (IVC) has been proposed, although different studies have established the probable acquired origin or the possible membranous transformation that causes stenosis at the level of the inferior vena cava[4].

Clinically, it manifests with signs of PH. Analytically, there is no major alteration of liver function. It has also been associated with the development of hepatocarcinoma, a priori less aggressive than that due to other aetiologies. Imaging tests will provide the diagnosis, with the "IVC reverse flow" sign

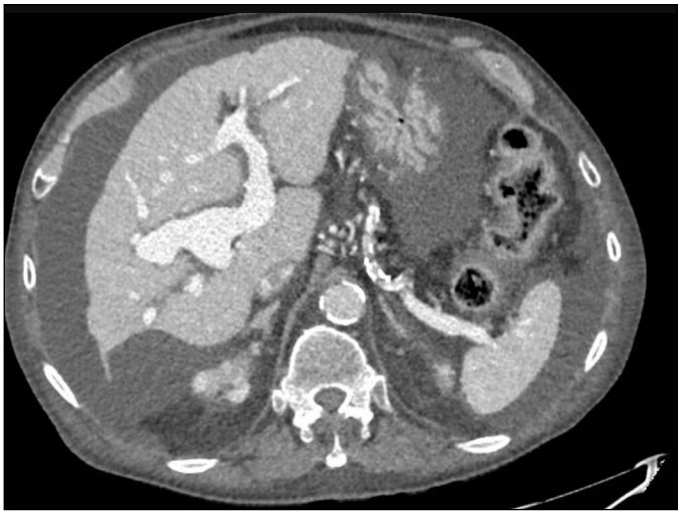

Figure 1. ***Abdominal CT image in venous phase showing large dilatation of both intrahepatic portal branches.***

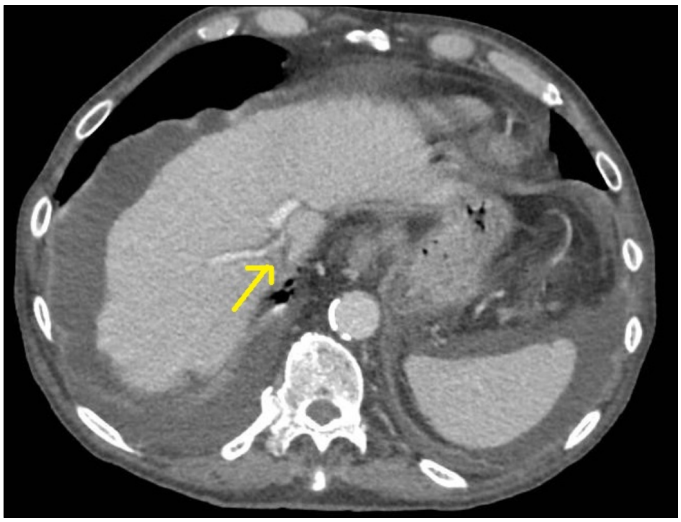

Figure 2. ***AngioCT image in the venous phase showing a decrease in the calibre of the inferior vena cava in the segment entering the liver, appearing practically filiform, with reduced calibre of the proximal segment of the suprahepatic veins.***

and the "jet bleed" sign on CT and MRI being specific to BCS subtype membranous obliteration[5].

Diagnosis is based on clinical features mentioned above and visualisation on imaging tests, and can distinguish complete from partial occlusion. Computed tomography or cavography by contrast administration can highlight the level of obstruction at which a retrograde leak or IVC reverse flow sign occurs[3].

The treatment of choice for MOIVC is endovascular recanalisation of the obstructed area by percutaneous balloon angioplasty, which is effective in up to 91% of cases. Other less commonly used surgical techniques include membranotomy, membranectomy or shunts[1,3]. Complications associated with PH are treated similarly to other aetiologies.

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Currently, MOIVC is included among the possible causes of Budd-Chiari syndrome, although some authors suggest that it could be considered a distinct syndrome[3]. The importance lies in the possibility of curative treatment, which would avoid the high morbidity and mortality secondary to PH decompensation, as well as the risk of developing hepatocarcinoma.

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