Multiple regenerative liver nodules uncovering a constrictive pericarditis
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Section: Abdominal imaging
Area of Interest: Abdomen Thorax
Procedure: Diagnostic procedure
Imaging Technique: MR
Imaging Technique: Ultrasound
Special Focus: Hyperplasia / Hypertrophy Calcifications / Calculi Dilatation Case Type: Clinical Cases
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Patient: 24 years, male

Clinical History:
A 24-year-old male patient, with history of asthma, underwent an abdominal ultrasound (US) in the context of elevated serum gamma-glutamyltransferase and bilirubin. Several hepatic isoechogenic nodules were discovered (Fig. 1) and an abdominal magnetic resonance (MR) was performed to investigate these lesions.

Imaging Findings:
MR revealed hepatomegaly with dilatation of hepatic veins and inferior vena cava (IVC), periportal lymphoedema, mosaic attenuation pattern of the liver and discrete peri-hepatic ascites (Figs. 2, 3). Several hepatic nodules were found, the largest measuring 4 cm, in segment III. Their features included: iso to hyperintensity on T1, iso to slight hyperintensity on T2, and after GdEOB-DTPA, absence to homogeneous enhancement in the arterial phase and iso- to slight hyperintensity in the portal phase. In the hepatobiliary phase, the lesions were iso- to hyperintense (Figs. 2-9). Although heterogeneous, these findings are compatible with regenerative nodules in a congestive liver.

Constrictive pericarditis was subsequently diagnosed through pericardal calcification detected in computed tomography (CT) and by echocardiography (calcified pericardial thickening, right chambers dilatation, absence of IVC inspiratory collapse and dyssynergic motion of the interventricular septum, with preserved left ventricular function). We retrospectively identified pericardial thickening on the abdominal MR (Fig. 10).

Discussion:
Background
Constrictive pericarditis results from scarring of the pericardium with impairment of diastolic filling [1]. Tuberculosis, cardiac surgery, radiation therapy and connective tissue diseases are frequent causes, but the majority is idiopathic or viral [2]. In constrictive pericarditis, congestive hepatopathy results from hepatic venous outflow obstruction at the level of the heart. With chronic compromise of the hepatic venous system, the portal vein tends to become a draining vein (instead of the main role in hepatic blood supply), leading to an increase of the hepatic arterial blood flow, which may lead to the development of hepatocellular tumours [3].

Clinical Perspective
Diagnosing constrictive pericarditis and secondary congestive hepatopathy is challenging [4] and it has occurred
after liver transplantation for cryptogenic cirrhosis in a reported case [5]. Symptoms of constrictive pericarditis will result from systemic venous congestion and low cardiac output [1] and the pattern of hepatic injury will depend on the relative contribution of these two features [6]: our patient showed signs of right-sided heart failure with preserved left ventricular function. Patients with congestive hepatopathy are asymptomatic in most cases, but some may present with jaundice, abdominal discomfort and ascites [6]. Elevations in alkaline phosphatase, gamma-glutamyltransferase and bilirubin [6] are expected.

Imaging Perspective

Imaging diagnosis of constrictive pericarditis may rely on CT and MRI, echocardiography findings (as the ones describing in our case) or cardiac catheterization (elevated and equalized diastolic pressures are the rule [1]). In addition to the recognized signs of passive congestion on cross-sectional imaging [7, 8], a fine reticular and coarse linear pattern of progressive enhancement seen at MR on venous and delayed phases is consistent with irreversible chronic liver disease/ fibrosis [9], which was not detected in our case.

Large regenerative hyperplastic nodules are described in Budd-Chiari syndrome [10], but regenerative nodules have also been reported in an explanted liver of a patient with constrictive pericarditis [5]; in addition, they are known to occur frequently in congestive hepatopathy, with an imaging appearance similar to that of focal nodular hyperplasia [11]. These nodules, described in conditions such as Budd-Chiari syndrome and dilated cardiomyopathy, are usually multiple, measuring 0.5-4 cm, bright on T1 (perhaps due to copper deposits), iso- to hypointense on T2, hyperintense on the arterial phase and present prolonged enhancement on hepatobiliary phase (due to ductular proliferation) [10].

Outcome

The treatment of constrictive pericarditis is surgical pericardiectomy [1]. The timely diagnosis is important to prevent the development of cirrhosis and hepatocellular carcinoma. [4]

Differential Diagnosis List: Constrictive pericarditis causing congestive liver with large regenerative hyperplastic nodules, Focal nodular hyperplasia, Hepatocellular carcinoma

Final Diagnosis: Constrictive pericarditis causing congestive liver with large regenerative hyperplastic nodules

References:

Description: A solid nodule at segment III is shown, almost isoechoic to the remaining hepatic parenchyma. Several similar nodules were also found. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal.
Description: T2 SPAIR, showing dilatation of the inferior vena cava and hepatic veins. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Figure 3

Description: T1 out-of-phase, showing a 4 cm-nodule at segment III, slightly hyperintense. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: T1 out-of-phase, showing a smaller nodule at segment IVa, with similar features to the one in Fig. 4a. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: The nodule in segment III is isointense on T2 without fat suppression. **Origin:** Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal

Description: The nodule in segment III is isointense on T2 SPAIR. **Origin:** Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: The nodule in segment IVa is isointense on T2 without fat suppression. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal.

Description: The nodule in segment IVa is isointense on T2 SPAIR. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal.
Description: Other nodule on segment VII/VI is slightly hyperintense on T2 without fat suppression.
Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal

Description: Other nodule on segment VII/VI is slightly hyperintense on T2 SPAIR.
Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
**Description:** The nodule at segment III is isointense on T1 GRE fat sat (THRIVE) without contrast.

**Origin:** Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: The nodule in segment IVa is isointense on T1 GRE fat sat (THRIVE) without contrast.

Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Figure 6

Description: The nodule in segment III is slightly hyperintense on T1 GRE fat sat (THRIVE), arterial phase. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: When subtraction images are employed, the nodule segment III is nearly isointense to the rest of the hepatic parenchyma on T1 GRE fat sat (THRIVE), arterial phase. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: The nodule in segment IVa is slightly hyperintense on T1 GRE fat sat (THRIVE), arterial phase. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: When subtraction images are employed, the enhancement of the nodule on segment IVa on T1 GRE fat sat (THRIVE), arterial phase is confirmed. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: The nodule in segment III is isointense on T1 GRE fat sat (THRIVE), portal venous phase.
Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
**Description:** The nodule in segment IVa is slightly hyperintense on T1 GRE fat sat (THRIVE), portal venous phase. **Origin:** Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: The nodule in segment III is isointense on T1 GRE fat sat (THRIVE), hepatobiliary phase.
Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: The nodule in segment III is isointense with a central element on T1 GRE fat sat (THRIVE), hepatobiliary phase. Other nodules were hyperintense in this phase, such as the one at segment VII/VIII. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
Description: Pericardial thickening seen at the upper slices of T1 out-of-phase. Origin: Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal
**Figure 10**

*Description:* T1 GRE fat sat (THRIVE) after Gd EOB-DTPA, arterial phase, depicting mottled pattern of enhancement of the liver parenchyma and periportal hypointensity in keeping with perivascular lymphoedema. **Origin:** Radiology Department, Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, EPE, Lisboa, Portugal