A case of liver inflammatory pseudotumor: CT and MR findings
Published on 18.10.2010

DOI: 10.1594/EURORAD/CASE.8814
ISSN: 1563-4086
Section: Abdominal imaging
Case Type: Clinical Cases
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Patient: 40 years, female

Clinical History:

A 39-year-old woman presented for further diagnostic work-up after a 3 cm hypoechoic lesion had been discovered in her liver. She had been submitted to ultrasound due to RUQ vague discomfort for the last 2 years, complicated with fever and weight loss during the last 3 months.

Imaging Findings:

The lesion was evaluated with dynamic contrast-enhanced CT as well as dynamic contrast-enhanced MRI. At non-enhanced CT the lesion was hypodense (Fig. 1a). At the dynamic contrast-enhanced CT there was no enhancement during the arterial phase (Fig. 1b). There was a mild degree of peripheral enhancement in the portal venous phase (Fig. 1c). There was no substantial change in the enhancement pattern of the lesion during the delayed phase (Fig. 1d), relative to the portal venous phase. At the dynamic MR examination, the lesion exhibited similar hemodynamics (Fig. 2c-e) with those of the dynamic CT examination. At non-enhanced MR and on the T1-W images (Fig. 2a), the lesion showed an outer isointense ring and a slightly hypointense core, relative to the normal liver parenchyma. On the T2-WI the lesion had an outer hypointense rim, an inner isointense ring and a hyperintense core (Fig. 2b). Biopsy was performed using the coaxial technique with a core biopsy system (QCS-18G-15cm). Tissue core samples as well as aspirates were obtained and sent for examination. Findings included the presence of spindle-cell stromal tissue with regions of dense fibrosis. There was a mixture of acute and chronic inflammatory cells, mainly plasma cells, acellular debris and benign liver cells. No malignant cells were detected. The final pathological diagnosis was inflammatory pseudotumor.

Discussion:

Inflammatory pseudotumour (IPT) is a mass of fibrous stroma, chronic inflammatory infiltrates and no anaplasia. It occurs in many organs, most frequently in the lung. Liver IPT is uncommon and occasionally discovered by US, CT or MRI. IPTs are characterised by a large population of polyclonal plasma cells, a variable amount of fibrosis, foamy histiocytes and other chronic inflammatory cells.

The aetiology of IPT is still unclear. Infection, biliary obstruction and immunological compromise have been suggested causes.

Patients with liver IPTs commonly have symptoms suggestive of inflammation. Fever is the most common symptom and may last for weeks or months. Leukocytosis and elevated ESR are frequent. LFTs may be normal or slightly elevated. Tumour makers are always negative and are important for differential diagnosis. The following CT and MRI imaging characteristics of IPTs have been reported in the literature:

Liver IPTs appear as ill-defined hypoattenuating lesions at unenhanced CT. There are two morphological patterns of CT enhancement: 1. multiseptate with enhancing internal septa and periphery and 2. non-enhancing central areas.
MR features of liver IPTs include iso/hypointensity on T1-W images and iso/hyperintensity on T2-W images. The conspicuity between the lesions and liver parenchyma, however, is less than that seen on dynamic MR imaging. Slightly hyperintense lesions on T2-WI may contain more inflammatory cells. Isointense lesions on T2-WI contain less inflammatory cell and more coagulative necrosis. A dynamic enhanced MRI study of IPTs found no enhancement in the arterial phase. The lesions were well defined in the portal venous and/or delayed phase and their MR enhancement patterns included: 1. peripheral enhancement, 2. small nodular enhancement in central or marginal areas, 3. enhancing septum, either fine/linear or thick/irregular.

Both CT and MRI features reflect the underlying histopathology. The central non-enhancing areas at CT and MRI correspond to chronic inflammatory infiltrates with foamy histiocytes, plasmacytes and lymphocytes. Enhancing internal septa and periphery represent fibroblastic proliferation. This is due to the fact that inflammatory infiltrates do not enhance, while the fibrotic parts may exhibit various degrees of portal venous or delayed enhancement. The latter is attributed to the accumulation of contrast medium in the extravascular space with delayed washout.

Infrequently though IPTs may exhibit arterial enhancement. There have been some reports of liver IPTs in hepatitis B and C patients, mimicking HCC. Tumour markers may help in such cases.

The differential diagnosis of liver IPTs includes liver abscess, metastasis, cholangiocarcinoma, and HCC. The prognosis of liver IPTs is generally excellent, with spontaneous regression having been reported.

In our case the lesion core was hypo/isointense on T1WI and hyperintense on T2WI (water and cellular content). The clue for suspecting an IPT rather than an abscess was the T2WI hypointense peripheral rim (fibrotic part), not typical of an abscess. Rarely chronic abscesses may also have a fibrotic component.

Unfortunately radiological features are nonspecific, so most patients, in the literature, have undergone surgical resection. Familiarity with this uncommon disease may lead radiologists to suspect the diagnosis. Percutaneous biopsy should then be performed, with surgery reserved only for complicated cases.

**Differential Diagnosis List:** Liver inflammatory pseudotumour (inflammatory myofibroblastic tumour).

**Final Diagnosis:** Liver inflammatory pseudotumour (inflammatory myofibroblastic tumour).

**References:**


Figure 1

Description: Non-enhanced CT. Hypodense lesion adjacent to right hepatic vein. The core of the lesion is markedly hypodense and its periphery less hypodense relative to normal liver parenchyma.

Origin:

Description: Contrast enhanced CT, arterial phase: The lesion does not exhibit arterial phase enhancement.

Origin:
Description: Contrast enhanced CT, portal venous phase: Subtle peripheral enhancement. The inner part of the lesion remains markedly hypodense. Note the presence of an incidental small nearby cyst.

Origin:

Description: Contrast enhanced CT, delayed (3 min) phase: No substantial change of the lesion enhancement pattern relative to the portal venous phase. Origin:
**Figure 2**

**a**

**Description:** Non-enhanced T1-W. The lesion has a hypointense core and an iso/hypointense periphery relative to liver parenchyma. **Origin:**

**b**

**Description:** T2W: Markedly hyperintense core, while the periphery of the lesion exhibits and inner isointense and an outer hypointense ring. **Origin:**
**Description:** T1-WI post Gd, arterial phase: No arterial enhancement. **Origin:**

**Description:** T1-WI post Gd, portal venous phase: Faint peripheral enhancing ring. **Origin:**
Description: T1-WI post Gd, delayed phase (5 min): The peripheral enhancement is less conspicuous on this delayed-phase image. Origin: