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MULTIFOCAL NODULAR STEATOSIS

T.A. Fassaert, E.R. Ranschaert, M.D.F. de Jong, I. Dubelaar[1]

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Background: A 62-year-old male with no medical history underwent an ultrasonography of the abdomen because of changed bowel habits and abdominal pain. CT-scan of the abdomen was performed, which initially suggested liver metastases. However, no primary tumor could be identified. Further work-up comprised optical colonoscopy which showed diverticulosis but no evidence of malignancy. Tumor markers were within normal limits. US-guided biopsy revealed normal liver parenchyma but the representativity of the biopsy was questioned. PET-CT scan was planned and a new biopsy was requested, but after interdisciplinary discussion it was decided to perform MRI of the liver first.

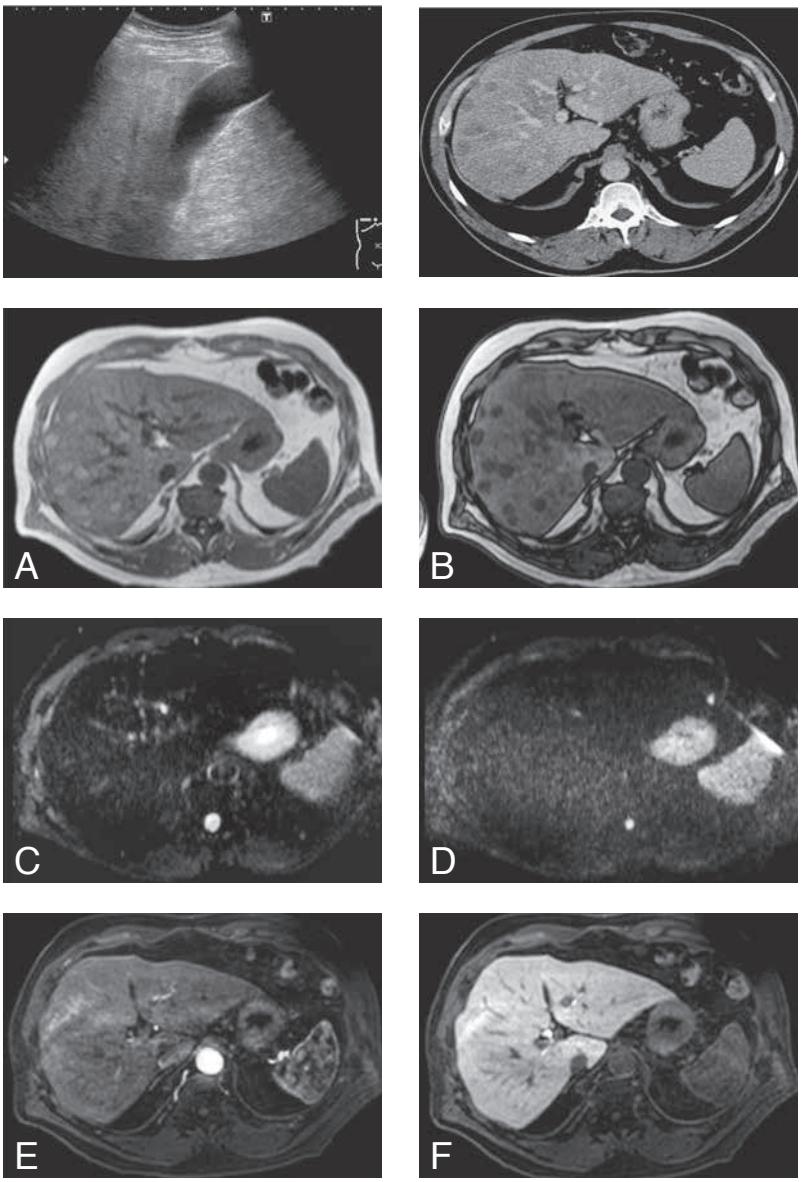

Fig.

1	2
3A	3B
3C	3D
3E	3F

1. Department of Radiology, Jeroen Bosch Hospital, Postbus 90153, 5200 ME 's-Hertogenbosch, The Netherlands

Work-up

Ultrasonography of the liver (Fig. 1) shows multiple hyperechoic nodules throughout the liver parenchyma, some surrounded by a subtle hypoechoic rim.

Contrast-enhanced CT of the abdomen (70 seconds delay after IV injection) (Fig. 2) shows multiple hypodense nodular lesions in left and right liver lobe.

MRI of the liver (Fig. 3) includes axial T1-weighted images, in-phase (A) and opposite phase (B).

The liver lesions are hyperintense on in phase and hypointense on opposite phase images.

Diffusion-MRI DWI images (Fig. 3), with B-values 10 (C) and 400 (D) show no diffusion restriction at the lesions. Dynamic MRI after Gd-EOB-DTPA, shows in the arterial phase (E) (portal phase and venous phase not shown) no abnormal enhancement is seen. In the liver specific phase (20 min after injection of contrast medium) the liver parenchyma enhances homogeneously (F).

Radiological diagnosis

Based on the MRI characteristics and in concordance with the US and CT-findings, the diagnosis of *a multifocal nodular steatosis* was established. Reassessment of the histology that was obtained earlier confirmed hepatic steatosis with a low-grade inflammatory component. At 1 year follow-up, MRI remained unchanged and clinically, the patient did not have any complaints.

Discussion

Multifocal nodular steatosis (MFNS) is a rare subtype of hepatic steatosis of unknown epidemiology and etiology. As with other types of steatosis, the accumulation of triglycerides within hepatocytes can be accompanied with a low grade inflammation. The fatty changes can remain or resolve. There is no difference in cellularity or vascularity compared to normal liver tissue. Steatotic nodules may vary in size from a few millimetres to several centimetres. As was shown in this case, unfamiliarity with this benign entity might result in unnecessary invasive workup and therefore in unwanted risks and anxiety for the patient.

On ultrasonography, the typical findings of MFNS are homogeneous more or less sharply delineated hyperechoic foci, usually with some acoustic shadowing.

On CT, the lesions are round or oval with sharp margins, and are hypodense compared the surrounding liver parenchyma – typically between 20-45 HU- but a large variety in density values is seen. Enhancement patterns follow the normal liver parenchyma. The abnormalities are considered pseudo-lesions, which therefore typically lack mass effect. Frequently however, MFNS lesions are too small to rely on this feature.

While US and CT might fail to differentiate between this benign entity and malignancy, MRI of the liver is highly specific to obtain a correct diagnosis. Phase-shift sequences are very useful in depicting the presence of microscopic fat. On the in-phase images, lesions are iso- or hyper-intense relative to the surrounding tissue. On the opposite phase images, signal intensity drops in fat-containing lesions which therefore appear hypo-intense.

Contrast-enhanced MRI using the liver-specific contrast agent Gd-EOB-DTPA, is reported to have a higher accuracy in the detection of liver metastases, especially for smaller lesions (< 1 cm). In case of MFNS, the liver enhances normally as with an extracellular chelate. In the liver specific phase (20 min after contrast injection) the liver enhances homogeneously because of contrast uptake in the hepatocytes, which are present in both normal parenchyma and steatotic nodules. This enhancement pattern renders malignancy unlikely.

Diffusion-MRI techniques rely on the differences in the motility of water molecules. This is largely influenced by cellular density in tissues: most neoplasms have higher cellularity than liver parenchyma and thus a relatively higher amount of intracellular and lower amount of extracellular water molecules. Diffusion rates are ten times lower intracellularly. So, in comparison to normal liver tissue malignant lesions have restricted diffusion. Diffusion restriction results in high signal intensity with increasing B-values.

Because cellularity is normal in MFNS, no restricted diffusion is shown. Diffusion-MRI has shown to be equally effective to contrast-MRI and PET-CT scan in detecting pathologic liver lesions > 20 mm lesions and superior in detecting lesions < 20 mm.

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