


CASE REPORT

Multifocal hepatic steatosis mimicking malignancy in two siblings

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Introduction

Hepatic steatosis is a common entity which has been associated with a variety of metabolic disorders including chronic alcohol use, diabetes, obesity, liver disease, protein malabsorption and numerous medications including corticosteroids and chemotherapeutic agents. Described imaging characteristics include diffuse, geographic or occasionally focal patterns.¹ Focal fatty infiltration is preferentially located near the gallbladder fossa or adjacent to the falciform ligament.²

Multifocal hepatic steatosis (MHS) is a rare subtype of hepatic steatosis of unknown prevalence which poses a diagnostic challenge due to difficulty in distinguishing it from malignant disease.³ Steatotic nodules in MHS can vary in size from a few millimeters to several centimeters and may mimic hepatocellular carcinoma or metastases by both ultrasound and computed tomography assessment. Accurate detection of this abnormality is important and may prevent unnecessary investigation and biopsy, as well as anxiety for the patient.

The pathogenesis and natural history of MHS is not well understood and further study as well as reporting of cases are required. Here we present two cases of MHS occurring in adult siblings without identifiable risk factors for hepatic steatosis. The characteristic radiographic appearances of MHS will be described as well as tips provided for accurate detection of this entity. Given the rarity of this entity, the occurrence in two otherwise well adult siblings also raises the possibility of an inherited pathogenesis.

Abstract

Multifocal hepatic steatosis (MHS) is a rare subtype of hepatic steatosis which can pose a diagnostic challenge due to difficulty in distinguishing it from malignant disease. Steatotic nodules in MHS can vary in size from a few millimeters to several centimeters and may mimic hepatocellular carcinoma or metastases by both ultrasound and computed tomography assessment. Accurate detection of this abnormality is important and may prevent unnecessary investigation and biopsy, as well as anxiety for the patient. Here we present two cases of MHS occurring in adult siblings. The characteristic radiographic appearances of MHS will be described as well as tips provided for accurate detection. Given the rarity of this entity, the occurrence in two otherwise well adult siblings also raises the possibility of an inherited pathogenesis.

Case summary

A 49-year-old Caucasian male presented to the emergency department with a 3-day history of central abdominal pain. The patient denied relevant medical or surgical history and received no regular medications. Clinical examination revealed a well appearing afebrile male with a non-surgical abdomen. Computed tomography kidneys, ureter, bladder (CT KUB) was performed and excluded urolithiasis as cause, however, innumerable focal hypodensities present throughout the liver were concerning for hepatic metastases (Fig. 1a). No intralesional enhancement was seen on post-contrast imaging. No primary malignancy was identified. Ultrasound-guided liver biopsy, magnetic resonance imaging (MRI) and positron emission tomography/CT (PET/CT) studies were scheduled.

Directed liver biopsy was canceled following planning ultrasound (Fig. 1b) which showed well-defined, rounded homogeneously hyperechoic lesions with internal vascularity. No hypoechoic halo was evident. The possibility of MHS was raised and the patient proceeded to MRI with gadolinium ethoxybenzyl diethylenetriamine pentaacetic acid (Gd-EOB-DTPA).

Liver MRI demonstrated heterogeneously hyperintense lesions on in-phase chemical shift T1 imaging with signal loss on opposed-phase sequence in keeping with microscopic fat (Fig. 1c,d). T2 imaging revealed multifocal hyperintense lesions which showed complete signal suppression following fat saturation (Fig. 1e,f). No diffusion restriction or contrast enhancement was evident. The findings were in keeping with MHS. Due to ongoing clinical concern, the patient proceeded to deoxy-2-(18) fluoro-D-glucose (FDG) PET/CT imaging (Fig. 1g). The known

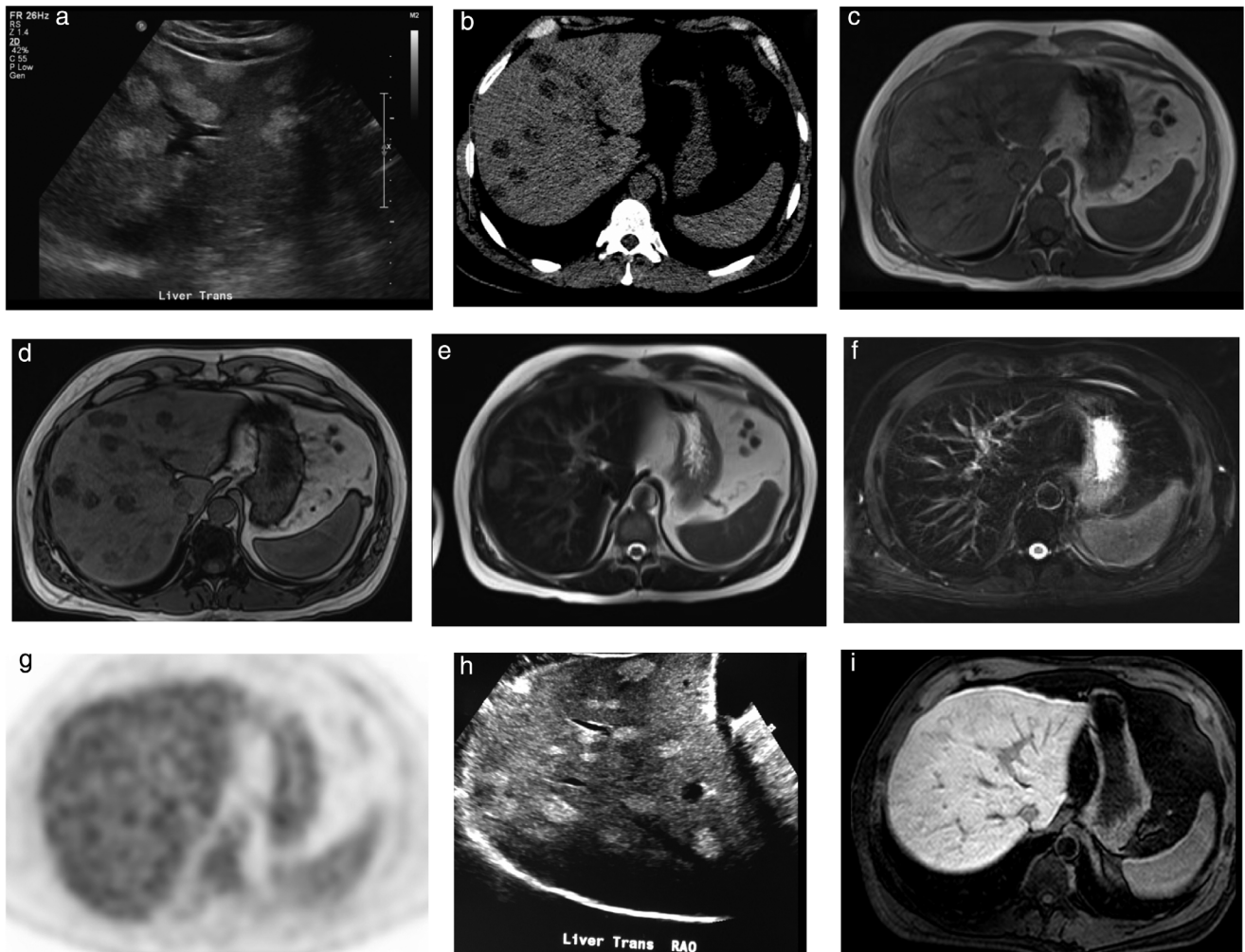


Figure 1 Ultrasound demonstrates numerous well-defined hyperechoic lesions (a) corresponding to multifocal hypodensity on axial non-contrast computed tomography (CT) (b). The lesions appear hyperintense on in-phase chemical shift T1 imaging with signal drop-out on out-of-phase imaging (c,d). Mild hyperintensity is present on T2 imaging (e) and there is complete signal suppression following fat saturation (f). 2-deoxy-2-(18F)fluoro-D-glucose (FDG) positron emission tomography/CT (PET/CT) demonstrates non-avidity (g). Note the lack of perilesional oedema on ultrasound (h), and the homogeneously enhancing liver parenchyma on hepatobiliary phase in keeping with lesions of hepatocellular origin (i).

hepatic lesions were not FDG avid and no abnormal tracer uptake was demonstrated elsewhere.

The combination of findings confirmed the diagnosis of MHS and the patient was spared unnecessary liver biopsy. Ten days later, an otherwise well male sibling of the above patient, aged 40, was referred to an outside radiology practice for investigation of nausea. Abdominal ultrasound revealed innumerable well-defined echogenic lesions throughout the liver (Fig. 1h). Subsequent MRI confirmed MHS (Fig. 1i).

Discussion

Steatosis is one of the most common liver abnormalities demonstrated on cross-sectional imaging, with a general population prevalence of around 15%.² Common manifestations are diffuse, diffuse with focal sparing, geographic and focal patterns. In comparison to the generally straight-forward characterization of these

subtypes, MHS poses a diagnostic challenge due to difficulty in distinguishing it from malignant disease—especially among those with a known history of malignancy. Accurate diagnosis relies on identification of microscopic fat within the lesions and lack of associated enhancing soft tissue. Additional clues to correct diagnosis include temporal stability and lack of mass effect.

Typical sonographic findings are homogenous sharply demarcated hyperechoic foci which often demonstrate posterior acoustic shadowing. The lesions typically lack a peripheral hypoechoic halo, a finding with good negative predictive value for malignancy.⁴ On Doppler assessment, there is absent to minimal flow within the lesions.

The most specific modality for identifying MHS is MRI using fat suppression and chemical shift imaging with opposed-phase sequences.² During the in-phase, lipid and water signals are additive. The opposed-phase signal consists of water less the lipid signal, allowing for detection of small amounts of fat. The

use of Gd-EOB-DTPA shows normal homogenous hepatic enhancement on hepatobiliary (20 min) phase due to the presence of hepatocytes in both affected and unaffected liver (Fig. 1i). The combination of multiple fatty lesions without soft tissue component, and normal uptake on hepatobiliary phase imaging is diagnostic of MHS. Because cellularity is normal in MHS, no diffusion restriction is demonstrated.

Awareness of these imaging findings can avoid further workup and liver biopsy, as well as anxiety to the patient. This paper presents two cases of MHS occurring in adult siblings without identifiable risk factors for hepatic steatosis. Given the rarity of this entity, the occurrence in two otherwise well siblings raises the possibility of an inherited pathogenesis.

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