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To resect or not to resect? That is the question

Mazen Nouredin¹, David E Kleiner², Ahmed M Gharib³, Gennady Bratslavsky⁴, and Theo Heller¹

¹Liver Diseases Branch, The National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Maryland, USA

²Laboratory of Pathology, National Cancer Institute, National Institutes of Health, Maryland, USA

³Integrative Cardiovascular Imaging Section, The National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Maryland, USA

⁴Urologic Oncology Branch, National Cancer Institute, National Institutes of Health, Maryland, USA

CLINICAL PRESENTATION

A 54-year-old Caucasian female, with a strong family history of renal cell carcinoma, presented with a left kidney lesion. She had a hysterectomy 20 years ago and no significant history of oral contraceptive use. During the current evaluation, she was found to have two hypervascular lesions in the left lobe of the liver in addition to a fatty liver on CT scan as well as MRI (figure 1A-C). The largest lesion was 2 cm in size. She was asymptomatic. Physical examination showed an obese woman with body mass index of 35.6 with an otherwise normal exam. Her laboratory results showed an aspartate aminotransferase of 88 U/l, alanine aminotransferase of 75 U/l, alkaline phosphatase of 93 U/l, total bilirubin of 0.6 mg/dl and direct bilirubin of 0.2 mg/dl. Her albumin, leucocytes, haemoglobin and platelets were all normal. She underwent a left nephrectomy. She also underwent a hepatectomy of segments 2 and 3 due to suspicion of metastatic disease. The pathology from the liver lesions is shown in figures 2 and 3.

QUESTION

What is the diagnosis of the liver lesions?

Correspondence to Dr Mazen Nouredin, Liver Diseases Branch, The National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, 10 Center Drive, Bldg 10 Room 4-5722, Clinical Center, Bethesda, Maryland 20892, USA; noureddinm@niddk.nih.gov.

Competing interests None.

Patient consent Obtained.

Ethics approval This study was conducted with the approval of the NIH IRB.

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ANSWER

Figure 4 shows an irregular distribution of steatotic hepatic parenchyma with dilated vessels embedded within a fibrosis stroma. The surrounding liver showed steatohepatitis and early fibrosis. These are the typical pathological findings of telangiectatic adenoma (TA).

Benign hepatic liver tumours are classically divided into hepatic adenoma (HA) and focal nodular hyperplasia (FNH). Telangiectatic adenomas (TAs) were originally classified as telangiectatic focal nodular hyperplasia. Recent molecular studies have led pathologists to reclassify telangiectatic adenoma as hepatic adenomas.¹ TA is typically associated with obesity and steatosis.² Paradis *et al* showed that 19% of TA had cellular atypia and one of them had hepatocellular carcinoma (HCC). Other reports have also suggested that TA is associated with obesity and may progress to HCC.³ Dokmak *et al* showed that 10% of TAs progressed to HCC. This risk was associated with an adenoma size of larger than 5 cm.⁴ Our patient had TA that was associated with steatohepatitis and obesity. TA should be suspected when mass lesions are discovered in the setting of obese patients with evidence of fatty liver disease. This is particularly important given recent advances in classification of adenomas and evidence for the development of possible malignancy.

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Figure 1.

(A) MRI of the liver showing hyperintensity of one of the lesions (arrow) on T1-weighted gradient echo fat suppressed MR, arterial phase. (B) MRI of the liver showing hyperintensity of one of the lesions (arrow) on T1-weighted gradient echo fat suppressed MR, venous phase. Note the mild washout of the contrast in the lesion compared to the arterial phase image. (C) MRI of the liver on T2-weighted fast spin echo fat suppressed MR, showing strong hyperintensity of the lesion, with absence of central scar.

Figure 2.
Section of one of the hepatic masses showing a portal-like area with arteries and veins but no bile ducts.

Figure 3.
Haemangioma-like area from the mass. The lesion also showed prominent steatosis.

Figure 4. Dilated vessels (big arrow), parenchyma with fibrosis (dotted arrow) and surrounding steatosis (white arrow) in the adenoma. No bile ducts were present within the lesion. There was no central scar and abnormally formed vessels with myxoid changes were not seen.