Sclerosing haemangioma mimicking hepatocellular carcinoma

V T W Lee, M Magnaye, H W Tan, C H Thng, L L P J Ooi

ABSTRACT

Sclerosing haemangioma is a rare variant of hepatic haemangioma. The radiological features on computed tomography and magnetic resonance imaging may not be typical for haemangioma and can be confused with hepatocellular carcinoma. We report sclerosing haemangioma occurring in a 65-year-old woman where the radiological features raise the possibility of hepatocellular carcinoma.

Keywords: computed tomography, hepatocellular carcinoma, magnetic resonance imaging, sclerosing haemangioma

Singapore Med J 2005; 46(3):140-143

Department of General Surgery
Singapore General Hospital
Outram Road
Singapore 169608

V T W Lee, MBBS, MMed, MRCS
Registrar

Department of Surgical Oncology
National Cancer Centre
Outram Road
Singapore 169608

M Magnaye, MD, DPBS, FPSGS
Clinical Fellow

L L P J Ooi, MD, FRCS, FAMS
Senior Consultant

Department of Pathology
Singapore General Hospital
Outram Road
Singapore 169608

H W Tan, MBBS, MRCPath
Registrar

Department of Oncologic Imaging
National Cancer Centre

C H Thng, MBBS, FRCR, FAMS
Consultant

Correspondence to:
Dr Victor Lee Tswen Wen
Tel: (65) 6321 4051
Fax: (65) 6220 9323
Email: vicklee@singnet.com.sg

INTRODUCTION

Sclerosing haemangioma is an uncommon lesion of the liver and is the result of extensive hyalinisation of a hepatic haemangioma. The typical imaging features of a haemangioma may not be obvious in the sclerosing variant, and a variety of differential diagnosis may be raised, including primary and even secondary tumours of the liver. Yamashita et al[1] reported a single case of a patient with sclerosing haemangioma mimicking a metastatic liver tumour. In a region endemic for hepatitis B, such as Singapore, the diagnosis of sclerosing haemangioma is difficult and hepatocellular carcinoma (HCC) has to be considered. We report a case of hepatic sclerosing haemangioma mimicking HCC and discuss its management.

CASE REPORT

A 65-year-old Indian woman presented with lower abdominal pain of four days duration. The pain was colicky, unrelated to meals, and associated with nausea and vomiting. She had difficulty in moving her bowels two days prior to admission. There was background history of hypertension, ischaemic heart disease, and hyperlipidemia. She was afebrile. There was mild tenderness in the left iliac fossa. A clinical diagnosis of colonic diverticulitis or constipation colic was made. Contrast-enhanced computed tomography (CT) of the abdomen and pelvis, arranged to exclude diverticulitis, showed no evidence of colonic inflammation. An incidental exophytic 5.3 x 3.1 cm mass in segment VI of the liver was seen with irregular rim enhancement on the portal venous phase (Fig. 1a), with incomplete central filling-in on the delayed phase (Fig. 1b).

Magnetic resonance (MR) imaging of the liver was performed to better delineate the lesion and to rule out the possibility of a haemangioma. This mass was mildly hyperintense on inversion recovery fat-suppressed, moderately T2-weighted images (Fig. 2a) and did not show the typical strong hyperintense appearance of a haemangioma. It was hypointense on T1-weighted images, and showed nodular rim enhancement in the arterial phase. However, there were also patchy nodular areas of central enhancement in the arterial phase which is not a feature of haemangiomas but raises the possibility of HCC (Fig. 2b). These patchy nodular areas of central enhancement did not show the classical mosaic pattern of HCC. The lesion subsequently showed gradual progressive enhancement, becoming predominantly hyperintense, except for central areas of low signal intensity (Fig. 2c). The delayed pattern of enhancement is suggestive of haemangioma or intrahepatic cholangiocarcinoma (Fig. 2d) and is not typical for HCC. There was also atrophy of segments V and VI of the liver. The radiological differential diagnoses were atypical haemangioma, intrahepatic cholangiocarcinoma and HCC.

The full blood count, urea and electrolytes and liver function tests were normal. Alpha-foetoprotein level was 2.4 μg/L (normal range 1-10), CEA 0.5 μg/L (normal range 0.5-3.5), and CA19-9 3 μ/ml (normal range 3-50). She was hepatitis B antigen positive and hepatitis C antibody negative. Gastroscopy and colonoscopy were both normal. In view of her positive hepatitis B carrier status and the nodular central enhancement in the hepatic arterial phase of the MR images, a diagnosis of HCC had to be excluded.

Surgical extirpation was offered to our patient and she was agreeable. A formal right hemihepatectomy was performed. Intraoperatively, the right lobe of liver was small and shrunken, and the left lobe was hypertrophied. The tumour involved segment VI, with extension to segments V and VII. Operative time was 165 minutes with 200 ml of blood loss. Her

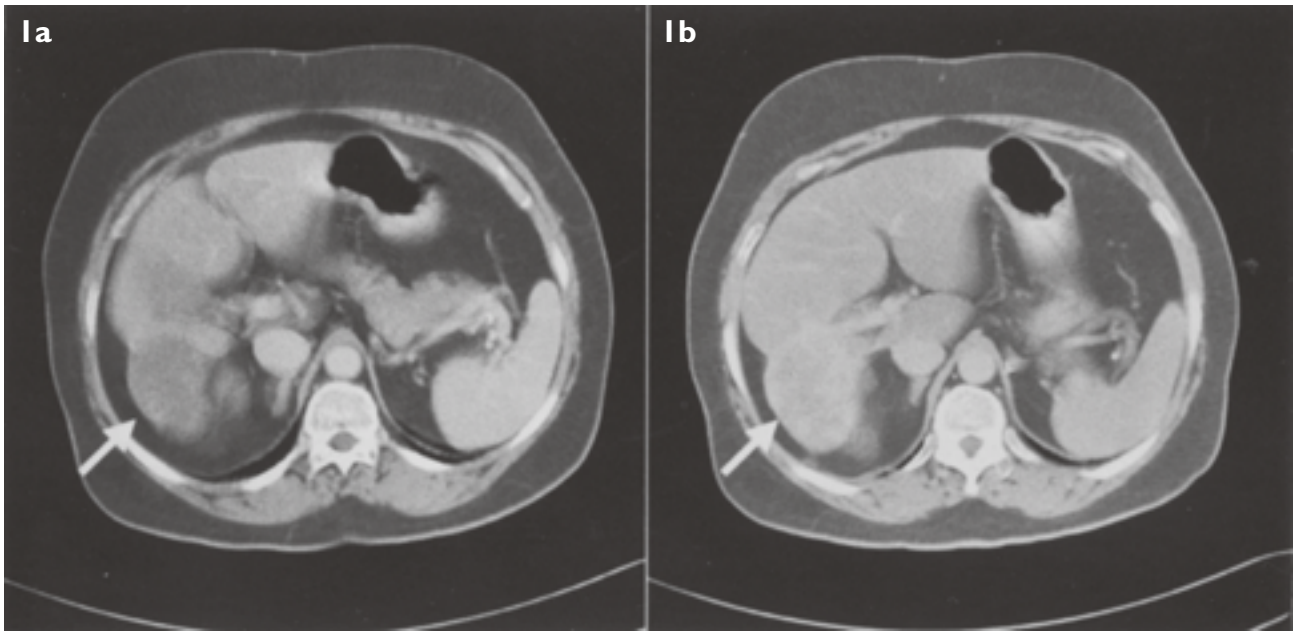

Fig. 1a Enhanced axial CT image (portal venous phase) shows peripheral nodular enhancement and a hypodense centre (arrow),
Fig. 1b Enhanced axial CT image (delayed phase) shows peripheral filling-in (arrow).

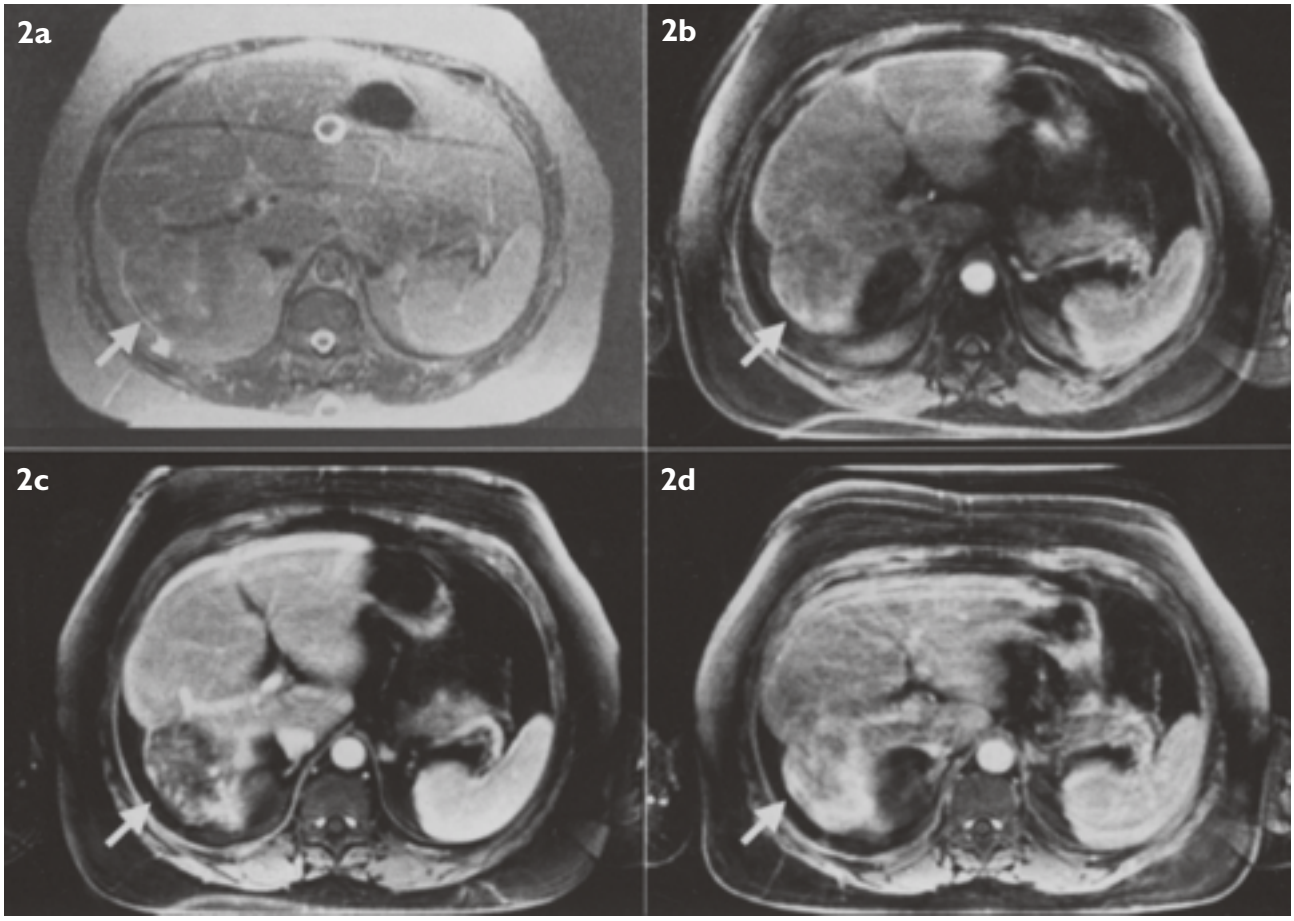

Fig. 2a Axial fast spin-echo inversion recovery fat-suppressed MR image (top left) shows that the lesion is only mildly hyperintense (arrow).
Fig. 2b Gadolinium-enhanced (hepatic arterial phase) axial fat-suppressed gradient echo T1-W MR image (top right) shows peripheral nodular enhancement as well as the central nodular enhancement (arrow).
Fig. 2c Gadolinium-enhanced (portal venous phase) axial fat-suppressed gradient echo T1-W MR image (bottom left) shows progressive filling-in of the lesion. The peripheral nodular enhancing areas are more clearly seen (arrow).
Fig. 2d Gadolinium-enhanced (delayed phase) coronal fat-suppressed gradient echo T1-W MR image (bottom right). Note the incomplete filling-in of the lesion (arrow).

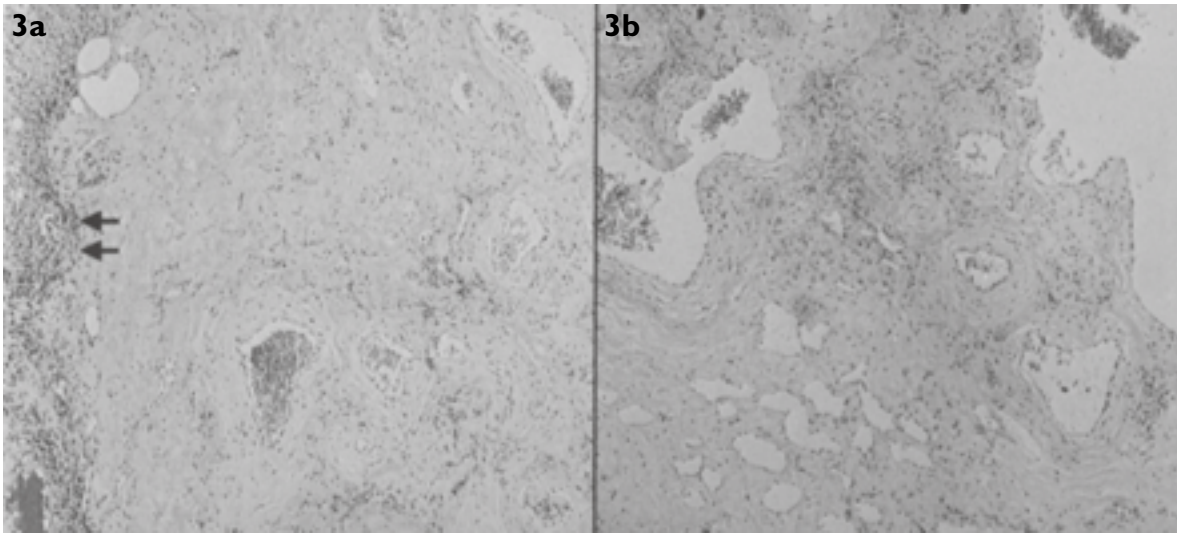

Fig. 3a Photomicrograph shows that the lesion consists of a collection of blood vessels in abundant fibrohyaline matrix. It is well demarcated from the adjacent liver parenchyma, seen on the left of the picture (double arrow). (Haematoxylin & eosin, x50)
Fig. 3b Photomicrograph shows that the blood vessels are of varying sizes. Some are thick-walled while others are slit-like. They are lined by a thin single layer of endothelium. (Haematoxylin & eosin, x50)

postoperative recovery was uneventful and she was discharged on the eighth post-operative day. On review at three months, she was well.

The macroscopic appearance of the tumour was a non-encapsulated, circumscribed, firm lesion measuring 5.5 x 4 x 3.5 cm. The lesion showed a collection of blood vessels within an abundant hyaline matrix (Fig. 3a). There were thick-walled blood vessels with intimal thickening and smaller irregular slit-like vessels lined by thin endothelium within the hyaline matrix (Fig. 3b). The lesion was well-demarcated and distinct from the adjacent normal liver parenchyma. No malignant cells were present.

DISCUSSION

Hepatic haemangioma is the commonest primary liver tumour. Most are asymptomatic. The commonest type of hepatic haemangioma is the cavernous haemangioma. On occasion, this can undergo regressive changes with scarring, areas of thrombosis, calcification and rarely, extensive hyalinisation[2]. Sclerosing haemangioma is a rare type of hepatic haemangioma composed of abundant acellular hyalinised tissue in which small vessels are occasionally seen.

As CT and MR imaging become more commonly used in the investigation of abdominal symptoms, more incidental haemangiomas will be detected. The ability to accurately diagnose these lesions therefore becomes critical. Cavernous haemangiomas are typically strongly hyperintense on T2-weighted MR images, and show peripheral nodular enhancement in the hepatic arterial phase with progressive filling-in. There is a subgroup of haemangiomas which may demonstrate immediate and complete enhancement in the hepatic arterial phase. These rapidly-enhancing haemangiomas are typically small and may be confused with other hypervascular lesions[3].

The imaging appearance of atypical haemangioma has been elegantly reviewed[3,4]. The features include the bright-dot sign, atypical low signal on T2-weighted MR images, a heterogeneous appearance with central scar, presence of calcification, central cystic degeneration, and fluid-fluid levels. They are also associated with adjacent abnormalities such as arterial-portal venous shunt, capsular retraction, and surrounding nodular hyperplasia. As such, atypical haemangioma cannot be readily differentiated from malignant tumour. Sclerosing haemangioma is one variant that gives rise to an atypical imaging appearance. There are few reports of MR imaging of sclerosing haemangioma[3,5,7]. Aibe et al[5] recently described a case of sclerosed haemangioma imaged with CT and MR imaging that had low signal intensity on T1- and T2-weighted MR images. There was no enhancement in the hepatic arterial phase but some enhancement in the delayed phase.

Accurate preoperative imaging evaluation of focal hepatic lesions is essential for patient selection for surgical intervention and for planning extent of surgery. Hepatic angiography has been widely used as an imaging technique for HCC as typical HCCs are hypervascular tumours that have only a hepatic arterial supply. In addition, lipiodol CT has previously been reported to be the most sensitive preoperative imaging modality for HCC. However, lipiodol can also be taken up by a variety of conditions other than HCC. Ngan[8] reported an overall sensitivity of 97% for lipiodol CT but only a specificity of 77%. Nakayama et al[9] have shown that helical CT, which allows scanning of the entire liver during both the arterial and portal venous

phases of contrast enhancement, has superior sensitivity and specificity compared to lipiodol CT. Moreover, both hepatic angiography and lipiodol CT are invasive imaging modalities which require the cannulation of the common hepatic artery.

We favour using multiphasic CT, with MR imaging as a complementary modality in our preoperative evaluation. MR imaging has been shown to detect more lesions and overall smaller lesions compared to helical CT[10], and may yield additional information on characterisation of the lesions. In a recent systematic review of radiological imaging for hepatocellular carcinoma in cirrhotic patients[11], no one imaging technique was shown to be superior. To date, there is inadequate evidence for choosing the best imaging modality for characterising HCC.

In our patient, there were indeterminate imaging features with some imaging features pointing to haemangioma (peripheral nodular enhancement with filling-in), some raising the possibility of HCC (patchy central arterial enhancement), and some pointing to cholangiocarcinoma (progressive central enhancement). Although fine needle biopsy can differentiate a sclerosing haemangioma from HCC, this can potentially lead to rupture or seeding of HCC. In our practice, fine needle biopsy is only performed for confirming an inoperable HCC as seeding of tumour in the needle tract has been reported in 1-3% of cases[12]. We believe that surgical resection of a suspected HCC in our patient is safe, and provides both a diagnosis and solution to the clinical problem. This case report serves to illustrate the atypical imaging appearance of sclerosing haemangioma and the possible confusion it can cause in the diagnosis of HCC, especially in a hepatitis B carrier. Focal liver lesions, which are indeterminate on imaging, as the case illustrated, often require histology for diagnosis, especially when HCC cannot be confidently excluded.

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