Focal fatty infiltration and focal fatty sparing of the liver may be confused with multiple liver metastases on both ultrasound and computed tomography (CT) imaging. Radiologists need to be aware of this benign condition so that unnecessary investigations and liver biopsies can be avoided.

**Introduction**

Focal fatty infiltration of the liver is caused by focal deposition of intracellular fat within hepatocytes. Although more commonly detected in adults it has also been described in children.1 The most common cause is alcohol abuse, although diabetes, obesity, certain drugs, toxins, protein energy malnutrition and anoxia are recorded as causing fatty change. At a cellular level there is accumulation of fat in cytoplasmic vacuoles. The significance of fatty change is dependent on the severity and cause. When mild it has no effect on liver function and is usually reversible. Severe fatty change, however, impairs liver function and can cause cell death.2

Focal fatty infiltration can cause difficulty in the interpretation of CT and ultrasound studies of the liver and can easily be confused with liver metastases.1 This is especially true of regions where there is no fatty infiltration in an otherwise diffusely infiltrated liver, called focal fatty sparing.1 This misdiagnosis may lead to unnecessary invasive procedures such as biopsies, and further anxiety on the part of the patient. This diagnostic problem can be avoided by performing an initial unenhanced computed tomography (CT) scan of the liver in all suspected cases and then measuring the density of the affected region in Hounsfield units. Two representative cases in which the correct diagnosis of fatty infiltration was not initially considered are reported.

**Case reports**

**Case 1**

A 60-year-old woman presented with a 3-month history of right upper quadrant pain. Liver function test results were within normal limits. Ultrasound examination demonstrated multiple focal low density regions in both lobes of the liver (Fig. 1a). The contrast-enhanced study demonstrated decreased perfusion in those regions of low density (Fig. 1b). A diagnosis of focal fatty infiltration of the liver was made. The patient was subsequently found to have type 2 diabetes, and her pain settled on oral hypoglycaemic agents.

**Case 2**

A 32-year-old woman known to have type 1 diabetes presented with right upper quadrant pain. Gallstones were suspected on the basis of the clinical symptoms, and an ultrasound examination confirmed their presence. In addition multiple echogenic lesions resembling liver metastases were noted in both lobes. An ultra-
sound-guided percutaneous biopsy of a left lobar lesion demonstrated focal fatty infiltration of otherwise normal hepatocytes. CT of the liver demonstrated multiple areas of low density in both lobes in keeping with diffuse fatty infiltration, with ‘pseudo-lesions’ of foci of normal enhancing liver parenchyma (Figs 2a and 2b). A diagnosis of focal fatty sparing of the liver was made.

Discussion

In both these cases an initial diagnosis of liver metastases was considered on the basis of the ultrasound findings. This is understandable, as the echogenicity of these pseudolesions differs from that of the surrounding parenchyma. In both cases measurement of the parenchymal density of the liver on CT suggested the correct diagnosis. The normal density of the liver on unenhanced CT is between 40 and 50 HU. After contrast injection the density reaches 80–120 HU. A good comparison of normal hepatic enhancement is comparison with the enhanced spleen, which has a similar enhanced density. Fatty change or infiltration will measure anywhere from 10 to 20 HU. An important diagnostic pointer to the correct diagnosis is the fact that normal hepatic venules and portal venules run through these pseudolesions and are not displaced as would be expected by a focal mass.

The true prevalence of focal fatty change or infiltration is unknown in adults, although a recent study in San Francisco demonstrated a prevalence of 25.6% in adolescents aged between 15 and 19 years of age. Although the causation of fatty change or infiltration is well known, the distribution within the liver of focal fatty infiltration and focal fatty sparing is not clearly understood. It is postulated that the distribution of fat deposition is related to regional differences in liver perfusion. Studies using CT portography have demonstrated that regions of fat deposition are associated with decreased perfusion. Yoshimuitsu et al. have demonstrated that these hepatic pseudolesions are due to a ‘third inflow’ of blood to the liver via the cholecystic, parabiliary or epigastric-paraulmbilical veins. The cholecystic vein drains segments IV and V. The parabiliary veins originate in the hepatoduodenal ligament and pancreas to supply the porta hepatitis and segment IV. The epigastric-paraulmbilical veins drain the abdominal wall into the liver in the region of the falciform ligament. The region around the falciform ligament, the porta hepatitis and gallbladder fossa bed are usually affected, while the medial segment of the left lobe, especially segment 4, is often spared.

There appears to be an association between the presence of focal fatty sparing in the medial segment of the left lobe and anomalous gastric venous drainage.

Magnetic resonance imaging (MRI) has been reported to be diagnostic in confirming the presence of fatty infiltration using opposed phase gradient echo sequences or fat saturation sequences.

These two cases demonstrate the importance of complementary imaging investigations in assessing focal liver pathology. Radiologists should always remember to include an unenhanced CT study in their liver imaging protocols and to measure the CT density of focal liver lesions if they are unsure of the diagnosis.

References