CASE REPORT

Imaging appearance of severe subcapsular hepatic steatosis: mimicking hepatic embolic infarcts

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ABSTRACT. We report a case of severe subcapsular hepatic steatosis in a middle-aged woman with diabetic nephropathy requiring continuous ambulatory peritoneal dialysis (CAPD). The CT appearances of the liver were initially suspected to be due to embolic infarcts. However, an accurate diagnosis was made only after a history of intraperitoneal insulin administration was elicited, and this was confirmed on MRI with chemical shift gradient-echo pulse sequences. Haemodialysis was recommenced, and follow-up imaging 4 years later showed complete resolution of subcapsular hepatic steatosis.

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Subcapsular hepatic steatosis is a rare and unique form of non-alcoholic fatty liver, occurring only in individuals with diabetic nephropathy receiving continuous ambulatory peritoneal dialysis (CAPD) with intraperitoneal insulin. Imaging findings are classical, and with the appropriate history of intraperitoneal insulin administration, an accurate diagnosis can be made. Given that this disease entity has only recently been described, we hope to highlight its typical imaging appearances, which may otherwise be misinterpreted as a more insidious finding, thus averting unnecessary intervention.

Case report

A 46-year-old Malay woman with a history of diabetes, end-stage renal failure (ESRF), hyperlipidaemia and hypertension presented acutely with abdominal pain when CAPD was in progress at an outside dialysis centre. There were no localising signs on clinical examination. An abdominal radiograph showed the peritoneal dialysis catheter to be in a satisfactory position but was otherwise unremarkable. CT of the abdomen showed free dialysate fluid in the abdominal cavity. Incidentally, there were multiple well-defined, low-density areas in the liver in the portovenous phase, mainly in the periphery but also in the caudate lobe (Figure 1a). Although these findings were initially suspected to be due to embolic infarcts as she presented with acute abdominal pain, it was noted that the main vessels including the portal veins were patent. Moreover, small patent vessels were observed coursing through these hypoattenuating areas, which were not typical of hepatic infarction (Figure 1a). Her treatment history was obtained from the physician at the dialysis centre and

revealed administration of intraperitoneal insulin during CAPD; therefore, a diagnosis of subcapsular hepatic steatosis was suspected.

MRI of the abdomen was performed for further evaluation. In-phase gradient-echo images showed a similar distribution of hyperintense areas in the periphery of the liver (Figure 1b), corresponding to the hypoattenuating regions present on CT, with corresponding signal loss on the opposed-phase sequences (Figure 1c), indicating the presence of microscopic lipid and cellular water. The diagnosis of subcapsular hepatic steatosis was confirmed. Further review of the history with the dialysis centre that had managed this patient revealed that she was in ESRF initially on haemodialysis, although this was later changed to CAPD about a year previously. The fact that the patient had also been receiving intraperitoneal insulin was not highlighted in the initial referral note.

A urine dipstick test showed elevated red and white blood cells, and microscopy revealed the presence of bacteria. She was treated with antibiotics for a urinary tract infection and was discharged. Unfortunately, she re-presented a few days later complaining of difficulty breathing. This was suspected to be due to a large amount of intra-abdominal fluid causing persistent splinting of the diaphragm and respiratory distress. The decision was made to stop CAPD and recommence haemodialysis. Four years later, the patient had a CT scan performed for another unrelated episode of acute abdominal pain and this showed complete resolution of subcapsular hepatic steatosis (Figure 1d).

Discussion

Fatty changes in the liver are usually generalised, occurring in association with alcohol abuse, diabetes, obesity, hyperalimentation, exogenous steroids and

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Figure 1. A 46-year-old Malay woman with end-stage diabetic nephropathy receiving continuous abdominal peritoneal dialysis (CAPD) with intraperitoneal insulin. (a) Axial contrast-enhanced CT scan shows multiple nodular low-attenuation areas in the periphery of the liver. They are more confluent in the caudate lobe (C). The portal vein is patent and its branches course normally through these regions (arrows). (b) Axial breath-hold T_1 weighted spoiled in-phase gradient-echo MRI (repetition time/echo time (TR/TE)=200/4.2) showing a similar distribution of hyperintense areas in the periphery of the liver as well as caudate lobe (arrowheads). (c) Axial T_1 weighted spoiled opposed-phase gradient-echo MRI (TR/TE =200/2.1) showing corresponding regions of signal loss, confirming the presence of subcapsular steatosis (arrowheads). (d) Follow-up contrast-enhanced CT scan 4 years later showing complete resolution of subcapsular hepatic steatosis.

chemotherapy [1]. Fatty changes localised to the subcapsular region (subcapsular steatosis) are a rare and unique form of non-alcoholic fatty liver, occurring only in those individuals receiving CAPD with intraperitoneal insulin therapy [2]. Wanless et al [3] first described this disease in 1989 based on autopsy findings, and Kallio et al [4] first demonstrated it *in vivo* using ultrasound (US).

CAPD is an established method of dialysis in patients with ESRF. For patients with renal failure and insulin-dependent diabetes, administration of insulin into the dialysate obviates the need for subcutaneous injections. Insulin is absorbed by diffusion across the visceral peritoneum into the portal venous system, and maintains the physiological insulin portal/peripheral ratio of ~3:1, exposing the systemic circulation to a lower concentration

of insulin [5]. However, it exposes the subcapsular hepatocytes to a higher concentration of insulin than the remaining liver, as direct absorption through the capsule of the liver also takes place. Insulin blocks the usual oxidation of free fatty acids, leading to preferential esterification into triglycerides in the subcapsular hepatocytes [6]. Steatosis occurs when synthesis exceeds secretion, and the consequence is fatty change limited to the subcapsular area [2]. The severity parallels a longer duration of CAPD, higher dosage of intraperitoneal insulin and greater obesity or higher serum triglycerides [4].

Patients are generally asymptomatic, with a diagnosis made incidentally on US, CT or MRI of the abdomen. Subcapsular steatosis manifests as continuous or discontinuous peripherally echogenic areas on US and as

hypoattenuating regions on CT scan. MRI with chemical shift in-phase and opposed-phase gradient-echo pulse sequences will reveal the microscopic fatty change of the subcapsular steatosis. With the appropriate history, this condition is a recognised entity in imaging literature and should be easily distinguishable from other differentials such as abscesses and primary or secondary tumours, or even liver infarction. This would prevent unnecessary anxiety to the patient and further investigations [6]. US is more sensitive for its detection, with lesions appearing smaller on CT and risk being rendered inconspicuous with contrast enhancement. Given the usually typical appearances on US or CT, MRI should be reserved as a confirmatory test in patients with atypical appearances [6].

One condition that may mimic subcapsular hepatic steatosis is Wilson's disease, in which a perihepatic fat layer is observed on a background of cirrhosis [7]. However, the clinical presentation and associated tests would be helpful in the differential diagnosis. Fatty infiltration is also common in the medial segment of the left hepatic lobe, anterior to the porta hepatis or adjacent to the falciform ligament [8], where there is a mixture of portovenous and systemic blood supply that may cause focal fatty change. There are also other unusual circumstances of focal fatty infiltration, for example infiltration associated with a metastatic insulinoma as reported by Sohn et al [1]. However, in these instances, the lesions are focal and should not be overdiagnosed. Care should also be taken to exclude pseudolesions caused by perfusion defects or beam-hardening artefacts from adjacent ribs [9].

In conclusion, patients on peritoneal dialysis may be investigated for abdominal pain by imaging modalities such as US, CT and MRI. Subcapsular hepatic steatosis may be detected in patients on intraperitoneal insulin and, once diagnosed, subcutaneous insulin therapy

should be recommenced, with haemodialysis considered in severe cases [2].

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