PICTORIAL MEDICINE

Imaging features of graft-versus-host disease: a rare cause of diffuse liver parenchymal disease

A 15-year-old boy with a history of dyskeratosis congenita and severe aplastic anaemia after haematopoietic stem cell transplantation 10 months earlier, was admitted for jaundice. There was no history of fever or sepsis. He previously had chronic graft-versus-host disease (GVHD) involving the skin and started treatment with prednisolone. On admission, his liver function was deranged (serum bilirubin of 273 μmol/L, alanine aminotransferase of 62 IU/L, and normal alkaline phosphatase). Serology was negative of hepatitis B and C and Epstein Barr virus. His white cell count (WBC) and erythrocyte sedimentation rate (ESR) were within normal limits. An ultrasound of the hepatobiliary system showed moderate hepatosplenomegaly (Fig 1a). There was diffuse increase in liver parenchymal echogenicity (Fig 1b), but the hepatic veins were normal in calibre, and there was no periportal oedema or ascites. Diffuse small bowel thickening was also noted (Fig 2). In the absence of clinical and laboratory evidence of sepsis, these features should not be confused with an infective cause. The overall imaging features were somewhat suggestive of chronic GVHD.

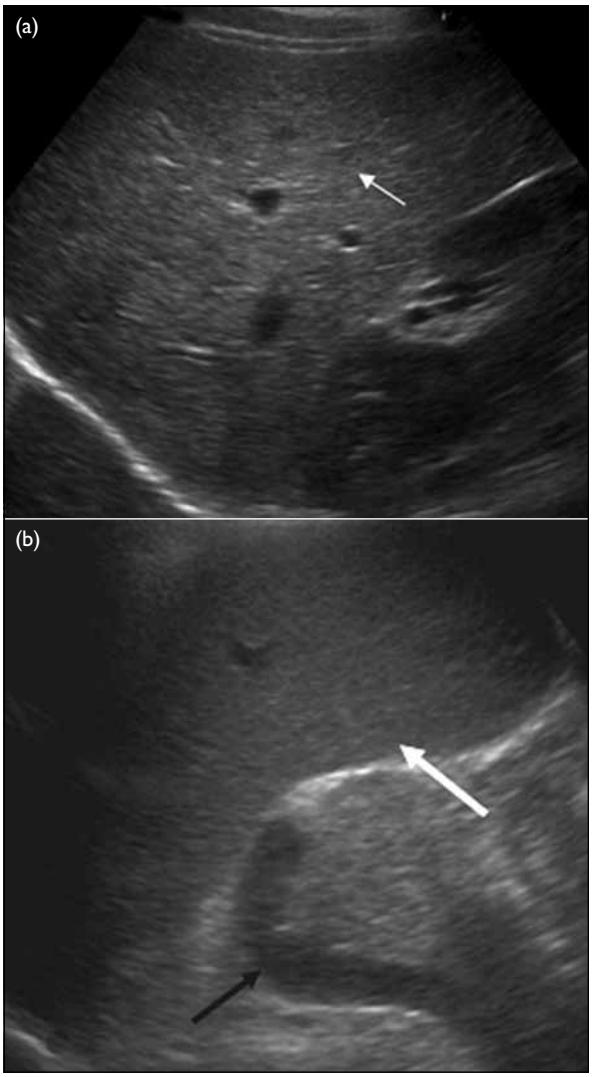

FIG 1. Graft-versus-host disease (GVHD) of the liver
Grey-scale transabdominal ultrasound scans: (a) note the diffuse increase in liver parenchymal echogenicity (arrow). (b) it reveals hepatosplenomegaly (white arrow) and mildly dilated splenic vein (black arrow), which is a commonly associated feature of GVHD

In many haematological disorders and malignancies, haematopoietic stem cell transplantation is now the treatment of choice. After transplantation, hepatic dysfunction is a common complication and affects approximately 80% of patients, and is responsible for up to 5 to 15% of deaths.[1] The common causes of liver dysfunction in post-transplantation patients include infection, drug-/radiation-induced liver injury, hepatic veno-occlusive disease, and GVHD. While the clinical history, microbiology, and awareness of hepatotoxic drugs intake can help in the diagnosis of some of these conditions, the clinical presentations of GVHD are non-specific and often necessitate liver biopsy for confirmation. In this regard, imaging plays a useful role as it is a non-invasive means of arriving at diagnosis, while biopsy should be resorted to only for more difficult cases.

The reported imaging findings in GVHD of the liver include ascites, splenomegaly, periportal

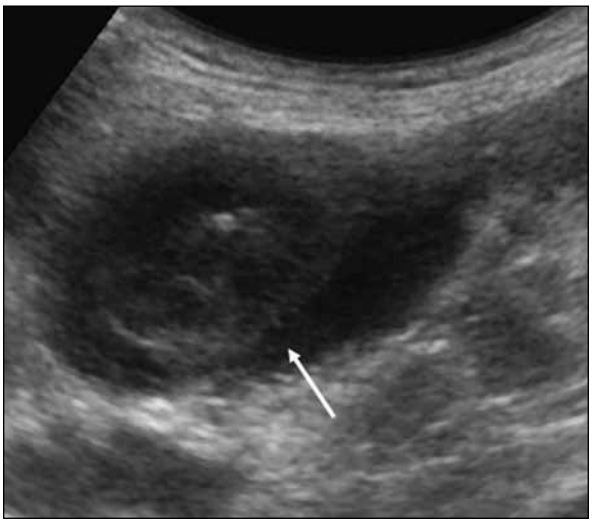

FIG 2. Grey-scale transabdominal ultrasound showing mild diffuse thickening of the small bowel loops (arrow)

oedema, pericholecystic fluid, biliary sludge, as well as gall bladder wall enhancement and thickening. There may be associated intestinal involvement with GVHD, as suggested by bowel wall thickening. Another important differential diagnosis is hepatic veno-occlusive disease. Erturk et al[1] reported that periportal oedema and ascites are more often characteristic of the latter. Moreover, in most patients with veno-occlusive disease, hepatic vein diameters are small, while small bowel wall thickening is predominantly a feature of GVHD.

In our case, absence of sepsis, negative hepatitis serology, as well as a normal WBC and ESR made an infective cause unlikely, and there had been no hepatotoxic drug intake. Thus, in this clinical scenario, the presence of hepatosplenomegaly, diffuse increase in liver parenchymal echogenicity, absence of narrowed hepatic veins, and small bowel thickening (shown on ultrasonography) suggested the diagnosis of GVHD. The patient was subsequently treated with prednisolone and the liver function gradually improved.

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Reference

1. Erturk SM, Mortelé KJ, Binkert CA, et al. CT features of hepatic venoocclusive disease and hepatic graft-versus-host disease in patients after hematopoietic stem cell transplantation. AJR Am J Roentgenol 2006;186:1497-501.