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Clinical Image

"Transparent" Liver

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Clinical Image

A 38-year-old female with estrogen receptor-positive breast cancer came to our hospital for routine evaluation. She had undergone mastectomy 18 months ago and received oral tamoxifen (20 mg/day) as adjuvant endocrine therapy for 17 months. She had no diabetes mellitus, hypertension, hyperlipidemia or HBV/HCV infection history, and no drinking habit. Her body mass index was 22.5, and liver function was normal before tamoxifen therapy. Laboratory tests revealed an elevated alanine aminotransferase (ALT) (72 U/L) and glutamyltransferase (115 U/L) (both normal up to 40 U/L); the cholesterol, triglyceride, and aspartate aminotransferase were within normal range. Unenhanced abdominal computed tomography (CT) scan showed diffused liver parenchymal density reduction and intrahepatic vascular density was relatively high with a liver/spleen ratio of -0.13 (Figure 1A, 1B). A diagnosis of tamoxifen-associated severe non-alcoholic steatohepatitis (NASH) was made, although absence of histopathological confirmation. Tamoxifen was discontinued, and letrozole (2.5mg/day) was prescribed. Her aminotransferase levels returned to normal limits 6 months subsequently. A repeat CT one year later showed hepatic steatosis remission with liver/spleen ratio of 1.0. She continued to receive letrozole and remains well after 12 months of follow-up.

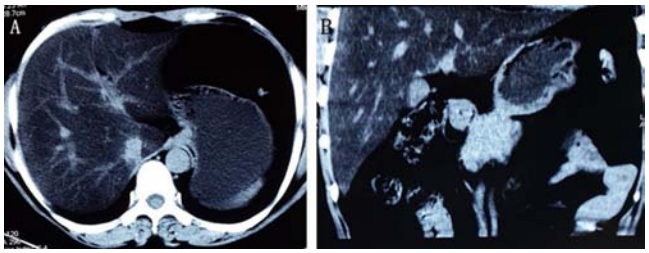

Figure 1: Unenhanced liver computed tomography (CT) scan showed diffused liver parenchymal density reduction and intrahepatic vascular density was relatively high with a liver/spleen ratio of -0.13.

Tamoxifen is a nonsteroidal antioestrogen metabolized in the liver. It has been used for decades as adjuvant treatment of oestrogen receptor-positive breast cancer, as well as a chemopreventive agent.[1] It is associated with an increased risk of endometrial cancer and other adverse reactions, including the development of hepatic steatosis, with an incidence up to 43% [1-3] The predisposing factors of its occurrence include insulin resistance, obesity, hypertriglyceridemia and CYP17 polymorphism [1-3]. Although hepatic steatosis is readily reversed in most cases end of the therapy [1,2] the disease can progress to cirrhosis [4]. If patients develop severe NASH, alternative therapy includes aromatase inhibitor and/or additional administration with Fibrates. Regular CT examination may be needed to monitor fatty changes of the liver and to differentiate steatosis from metastatic liver tumors during tamoxifen treatment [2].

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