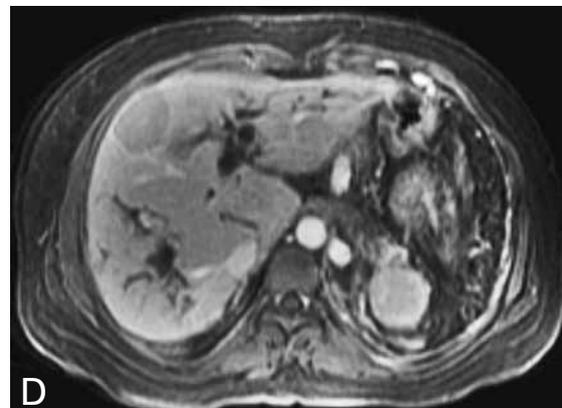
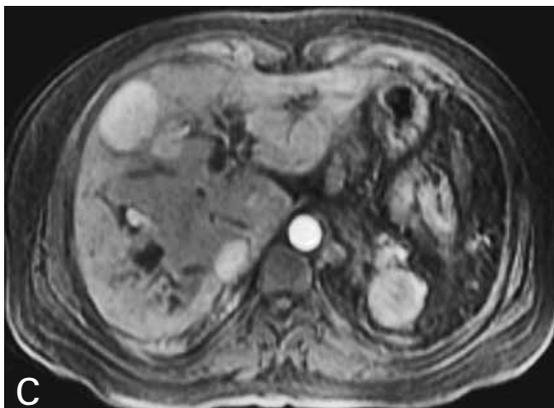
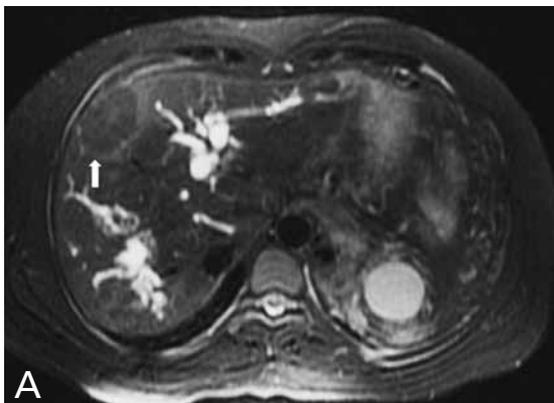
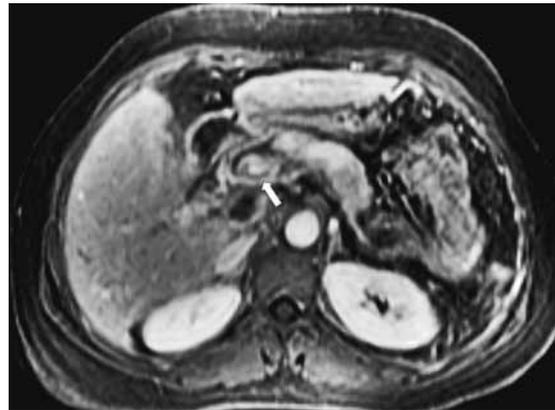


NODULAR REGENERATIVE HYPERPLASIA OF THE LIVER AND PORTAL THROMBOSIS

M. Ruhi Onur, A. Kürsad Poyraz, E. Kocakoç¹

Key-words: Nodular regenerative hyperplasia – MRI, liver

Background: A 35-year-old female presented with upper gastrointestinal tract bleeding. Fifteen years ago, the diagnosis of esophageal varices caused by idiopathic portal venous thrombus was made. Follow-up examinations revealed mild elevation of bilirubin levels and normal alpha-fetoprotein level on blood tests. Splenectomy had been performed 6 years previously due to portal hypertension caused by portal venous thrombus.



1. Department of Radiology, Firat University Hospital Elazig, Turkey

Work-up

On MRI of the liver, Gd-enhanced axial T1-weighted image at the level of the portal vein (Fig. 1), a portal vein thrombus at the confluence level is shown (arrow).

MRI of the liver, at the level of lesion in the right liver lobe (Fig. 2) shows on axial T2-weighted image (A) an isointense mass in segment 7 of right lobe in liver (arrow). Intrahepatic bile ducts are dilated. On axial T1-weighted image (B) the liver mass described in Fig. 2A is hyperintense compared to surrounding liver parenchyma. On axial contrast-enhanced T1-weighted image (following intravenous administration of Gadolinium), arterial phase (C), the lesion is enhanced homogeneously early (arterial phase) after intravenous Gadolinium administration. On axial contrast-enhanced T1-weighted image (following intravenous administration of Gadolinium), venous phase (D), the lesion appears hypointense due to 'wash-out', mimicking hepatocellular carcinoma.

Radiological diagnosis

Histopathologic examination revealed regeneration nodules which were formed by hyperplastic hepatocytes in the absence of parenchymal fibrosis. Based on these findings the diagnosis of *nodular regenerative hyperplasia of the liver* was made.

Discussion

Nodular regenerative hyperplasia (NRH) of liver, is a benign proliferative disease which is characterized by multiple regenerative nodules in liver with varying sizes from 0.1 cm to 15 cm. The pathogenesis of NRH is not well known but it has been associated with a variety of systemic diseases including collagen vascular diseases, lymphoproliferative and myeloproliferative disorders. At present, the nodular transformation in NRH is considered to be a consequence of portal blood flow impairment. According to this vascular hypothesis the basic pathologic injury leading to NRH is obliteration and/or thrombus in the portal venous system. The

central atrophy, produced by decreased blood flow, is compensated for by proliferation of hepatocytes from the portal region that form regenerative nodules.

Clinically, NRH does not cause symptoms and is discovered incidentally unless it is complicated by portal hypertension and its sequelae such as hepatomegaly, splenomegaly, ascites, or esophageal varices. In this patient presence of portal thrombus and portal hypertension was the leading cause of NRH. Ultrasonography commonly reveals large isoechoic hepatic masses with normal echogenicity. On MRI, the nodular lesions appear isointense on T2-WI and hyperintense on T1-weighted images. Lesions enhance early after intravenous contrast administration (arterial phase) with wash-out in venous phase, hereby mimicking hepatocellular carcinoma (HCC). However low AFP values, lack of high signal intensity on T2-WI images and relative homogeneous enhancement allow distinction with HCC. The absence of characteristic high signal scar on the T2-weighted image excludes focal nodular hyperplasia in the differential diagnosis. Adenomas have heterogeneous signal on T1- and T2-weighted images due to fat or hemorrhage contents and were absent in the liver lesions of the presented patient. Since NRH mimicks benign and malignant focal liver lesions, awareness of imaging findings and association between impairment of portal circulation and NRH may be helpful in the diagnosis.

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