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An asymptomatic huge hepatocellular carcinoma with intra-atrial tumor thrombus in a patient with chronic hepatitis B viral infection

Kronik B hepatitli bir hastada, intra-atriyal trombüse neden olan dev, asemptomatik hepatosellüler karsinom

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Hepatocellular carcinoma is the most important complication of cirrhosis. All types of cirrhosis predispose to hepatocellular carcinoma, but 10 to 20% of cases may develop in patients without cirrhosis. Here, we present an asymptomatic precirrhotic patient with hepatocellular carcinoma and intra-atrial tumor thrombus.

Key words: Hepatocellular carcinoma, chronic hepatitis B

Hepatoselüler karsinom sirozun en önemli komplikasyonudur. Her tür sirozda hepatosellüler karsinom gelişebilir, fakat vakaların %10-20'sinde siroz yoktur. Bu yazıda, hepatosellüler karsinom ve intra-atriyal trombüs gelişen presirotik dönemde asemptomatik bir hasta sunuldu

Anahtar kelimeler: Hepatoselüler karsinom, kronik hepatitis B

INTRODUCTION

Hepatocellular carcinoma (HCC) accounts for 90% of primary liver neoplasms (1). The major risk factor for HCC is cirrhosis. HCC is a common cause of death among patients with compensated cirrhosis (2). All types of cirrhosis may predispose to HCC, but the incidence is particularly high in patients with chronic hepatitis B and C virus infection (1). Approximately 10 to 20% of cases of HCC develop in patients without cirrhosis. HCC features may also differ related to the presence or absence of underlying cirrhosis (2). Generally, all noncirrhotic patients are symptomatic. In this paper, we present an asymptomatic precirrhotic patient with HCC and intra-atrial tumor thrombus.

CASE REPORT

A 65-year-old man (A-B) admitted to the hospital with cough and sputum. As he was diagnosed with pneumonia, a thoracic tomography was performed. Lower images of computerized thoracic tomography revealed a mass in the liver; there was no metastatic lesion. Abdominal ultrasound also confirmed liver mass (Figure 1). After initiation of antibiotic therapy for pneumonia, he was referred to our center for further investigation. Physical examination revealed a 4 cm hepatomegaly. Laboratory analyses were as follows: erythrocyte sedimentation rate 37 mm/h, leukocyte 4780/mm^3, neutrophil 2310/mm^3, lymphocytes 1580/mm^3, hematocrit 50.9%, hemoglobin 17.2 g/dl, platelets 166000/mm^3, prothrombin time 13 sec (control 10-14.5), glucose 87 mg/dl, aspartate aminotransferase 54 IU/L (5-34), alanine aminotransferase 133 IU/L (0-55), alkaline phosphatase 123 IU/L (40-150), gamma glutamyl transpeptidase (GGT) 207 IU/L (5-85), total bilirubin 0.84 mg/dl (0-1), direct bilirubin 0.33 mg/dl (0-0.5), albumin 3.0 g/dl, gamma-globulin 1.5 g/dl, and alpha-feto protein 6.64 ng/ml. Hepatitis B surface antigen (HBsAg) and antibody to hepatitis e antigen were positive [Hepanostika HBsAg Uni-Form II (Organon Teknika, Italy), Pasteur (France) for anti-HBe]. HBV DNA was positive by hybridization. Hepatitis C virus antibody (anti-HCV) (Organon Teknika, Italy) and

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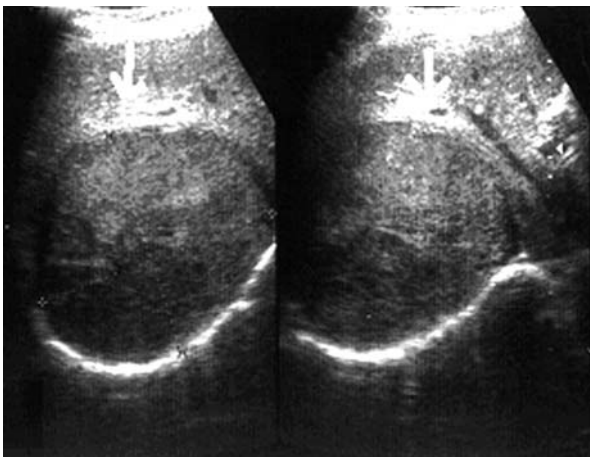

Figure 1. Peripheral capsulated mass on ultrasonography

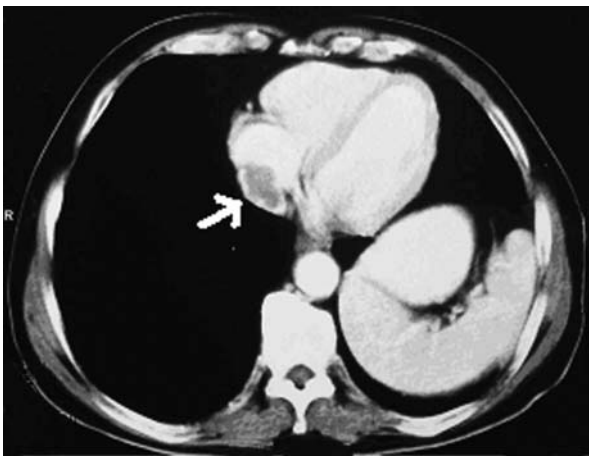

Figure 3. Thrombus in right atrium

hepatitis D virus total antibody (anti-HDV) (Diasorin) were negative.

Abdominal ultrasound showed encapsulated heterogenic, hypoechoic solid liver mass of 12x11.5x11 cm in right Couinaud segment 7 (Figure 1). CT revealed a hypodense encapsulated mass without calcification. CT scan obtained after contrast material injection showed heterogeneous involvement with hypodense areas in central zone, which is compatible with necrosis (Figure 2), and tumor thrombus in inferior vena cava extending to the right atrium (Figure 3). Portal system and the other vascular systems were evaluated and there were no thrombi. Portal vein diameter was less than 10 mm. Magnetic resonance imaging (MRI) showed hypointense central scar on T1- and hyperintense on T2-weighted image. Tumor thrombi were seen on T2-weighted image. Liver lesion biopsy revealed well- differentiated macrotrabecular HCC.

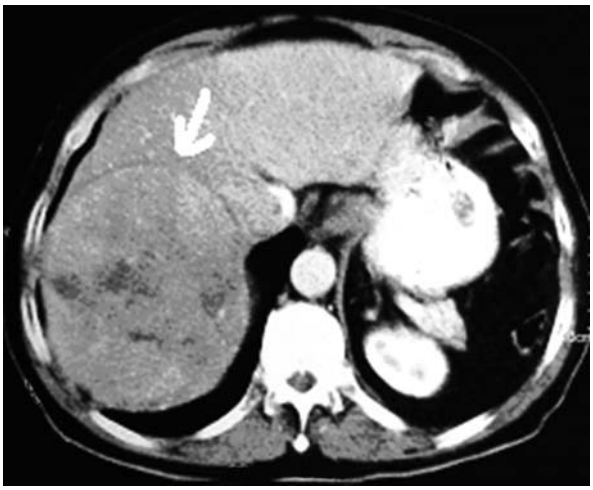

Figure 2. Liver mass in 7th segment and thrombus with partial obstruction in inferior vena cava in contrasted CT scan

The patient was diagnosed with HCC and chronic hepatitis B based on the absence of physical findings compatible with chronic liver disease, noncirrhotic appearance in radiologic images and normal biochemical findings (except aminotransferases and GGT elevation).

DISCUSSION

Worldwide, the majority of patients with HCC have underlying cirrhosis, and it is uncommon to find HCC in a patient without cirrhosis. Among patients with HCC without underlying cirrhosis, HCV infection accounted for 3-54%, HBV infection accounted for 4-29%, and heavy alcohol intake for 0-28% (1). In contrast with cirrhotic patients, HCC is symptomatic in noncirrhotics, and the mean age is older in cirrhotics (3). Sezaki et al. (4) showed that HBsAg-positive older adult patients with HCC are generally in cirrhotic stage, but HCC-developing noncirrhotic patients are young adults. Patients who have single huge mass are more likely to present with symptoms, but our patient was asymptomatic, in precirrhotic stage, and older than expected. In our case, histological findings showed well-differentiated macrotrabecular type HCC. Nzeako et al. (5) showed that trabecular type of HCC is the most common growth pattern in cirrhotic and noncirrhotic patients. We did not need to perform liver parenchymal biopsy because there was no sign of cirrhosis in physical examination, biochemical analyses or radiological images.

Also, no esophageal varices were detected by upper gastrointestinal endoscopy. These findings strongly suggest the patient was in precirrhotic stage.

Hepatocellular carcinoma has a tendency to invade vascular structures (6). Extension to the portal system is common. However, extension into the inferior vena cava or heart without thrombus in the portal system is uncommon. Fukuda et al. (9) reported 4/19 cases with HCC and tumor thrombus only in the inferior vena cava. In our case, we did not consider a prothrombotic factor except HCC because of older age and absence to date of any thrombotic events in his history. Local invasion is seen significantly more often in cirrhotic patients than in precirrhotics. Intra-atrial thrombi were seen in 18 of 439 autopsy cases with HCC (7). In the literature, all HCC cases with tumor thrombus extending to the right atrium were symptomatic and cirrhotic. Interestingly, our case was asymptomatic in spite of a huge mass with intra-atrial tumor thrombus.

In conclusion, this is an interesting case illustrating that a huge encapsulated macrotrabecular HCC with vascular invasion may be seen in an asymptomatic chronic hepatitis B patient.

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