Ruptured hepatocellular carcinoma in a patient with chronic hepatitis C and a sustained viral response

Kalıcı viral yanıtı olan kronik hepatit C hastasında hepatosellüler kanser rüptürü

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Dear Editor,

Hepatitis C virus (HCV) is one of the most common causes of chronic liver disease in the world (1). The goal of chronic hepatitis C therapy is to prevent cirrhosis of the liver and hepatocellular carcinoma (HCC) by achieving a sustained virologic response (SVR). It has been reported that telaprevir-based triple therapy enabled 60%–70% SVR in genotype 1 (G1) patients (2,3).

The 74-year-old male patient was diagnosed with chronic hepatitis C 6 years ago, and peginterferon (P) + ribavirin (R) therapy was initiated. As HCV-RNA positivity continued in the 24th week of treatment, the patient was considered unresponsive, and his treatment was concluded. With the introduction of telaprevir into the therapy approximately one year later, the G1b patient was started on a triple therapy (telaprevir, PR) for 12 weeks followed by a 36-week course of double therapy (PR); thereby, his treatment was completed in a total of 48 weeks. The patient, who now had SVR, has been followed up in the 4 years since the completion of his therapy by carrying out tests on his HCV-RNA and alpha-fetoprotein (AFP) levels, alongside abdominal ultrasonography (ABD USG) performed every 6 months.

The patient, who presented to the emergency department with a complaint of severe abdominal pain reflecting on his right shoulder about 4 months after his latest follow-up, had a heterogeneous solid mass about 6 cm in size in his eighth liver segment alongside free fluid in the perihepatic and pelvic areas, as revealed by ABD USG. The results of testing showed that the patient had a high AFP level of 36 ng/mL (0–8.1).

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The results of all abdominal computed tomography scans

carried out in view of these findings showed that the

patient's liver was of a normal size with no pronounced

undulation of the contours. A heterogeneous solid mass lesion about $67 \times 73 \times 68$ mm in size growing toward the

subdiaphragmatic space in liver segment 8 was observed

to reveal a hyperdense, possibly arterial contrasting image; the presence of high-density fluid around the mass

in the subdiaphragmatic space was also visualized. Con-

sequently, the rapidly contrasting arterial mass on the he-

patic dome was diagnosed as HCC (?), hepatic adenoma

(?), and a ruptured mass, and it was recommended that

the patient undergo dynamic magnetic resonance (MR).

A T1A hypointense, T2A mildly hyperintense heterogene-

ous solid mass, 63×70 mm in size with fine borders and

irregular contours, was visualized on the dome level of

As a result, the patient was diagnosed with spontaneous

rupture of the mass because of his high AFP levels, the

presence of a solid mass concordant with HCC, contour

irregularity neighboring the superior, and signs of hemo-

peritoneum observed in the perihepatic area. The patient was taken into surgery and died in the perioperative pe-

The risk of HCC development in patients with SVR th-

rough direct-acting antiviral (DAA) has proven to be a

controversial issue (4,5). Patients with SVR through DAA

administration should be followed up every 6 months

with regards to ABD USG results and AFP levels.

the eighth segment of the right lobe (Figure 1).

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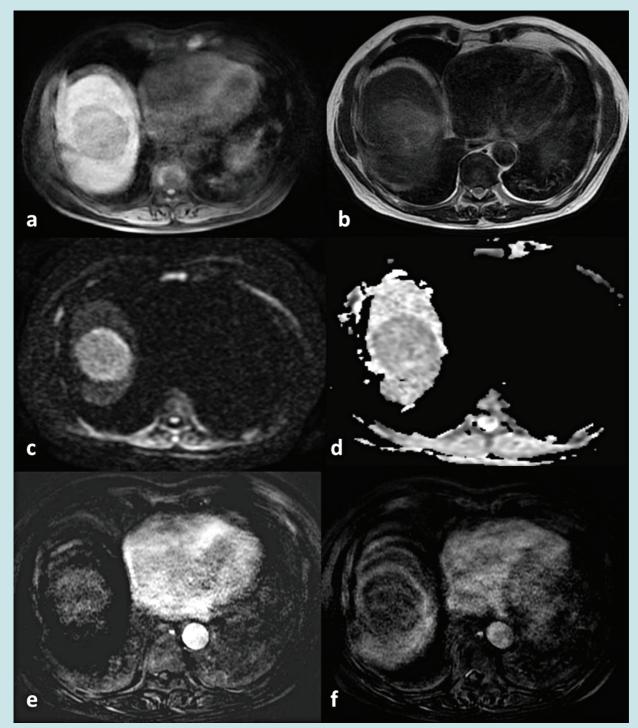


Figure 1. On abdominal MR images, the hepatocellular carcinoma is seen as a T1 hypointense **(a)**, T2 mildly hyperintense **(b)** solid mass in the right liver lobe. It is hyperintense on diffusion-weighted images **(c)** and hypointense on the apparent diffusion coefficient (ADC) map images **(d)**, which is compatible with diffusion restriction. The mass is enhanced in the arterial phase of dynamic contrast-enhanced images **(e)** and is washed out in the portal venous phase **(f)**, which is the typical contrast enhancement pattern of hepatocellular carcinoma.

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