

The mechanisms of octreotide-induced bradycardia are still not known. Octreotide increases systemic vascular resistance, and bradycardia may be a baroreceptor-induced reflexive response to an increase in the systemic blood pressure (7). However, bradycardia developed at the 60<sup>th</sup> hour of infusion of octreotide in our case. Therefore, a direct

action of octreotide is more likely than a reflexive response.

In conclusion, intravenous administration of octreotide may cause significant bradycardia and cardiac conduction defects. Therefore, electrocardiographic monitoring is advisable while octreotide is administered.

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## Fatal hepatitis B reactivation in an immune patient after anti-leukemic chemotherapy

Bağışık olan bir hastada anti-lösemik tedavi sonrası ölümcül hepatit B reaktivasyonu

*To the Editor,*

A 62-year-old female admitted to the outpatient clinic with gum bleeding in April 2007. Pancytopenia was detected. Bone marrow aspiration and flow cytometric analysis revealed acute promyelocytic leukemia. Her hepatitis B markers were as follows: HBsAg(-), anti-HBs(+) and anti-HBc IgG(+). Remission induction and consolidation chemotherapy (idarubicin + cytosine arabinoside and all-trans retinoic acid) was started and remis-

sion was achieved. In September 2007, she complained of weakness and nausea. Her chemotherapeutic agents were ceased. Moderate pancytopenia developed with marked elevation in liver function tests. Admission laboratory tests were as follows: ALT: 1748 IU/L, AST: 1598 IU/L, total bilirubin: 3.87 mg/dl, direct bilirubin: 1.6 mg/dl, ALP: 634 IU/L, GGT: 145 IU/L, albumin: 3 g/dl, international normalized ratio (INR): 1.16, HBsAg(+), an-

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**Manuscript received:** 17.10.2008 **Accepted:** 17.12.2009

doi: 10.4318/tjg.2010.0057

ti-HBs(-), anti-HBc IgM(+), anti-HBc IgG(+), Anti-HBe(+), and polymerase chain reaction (PCR) HBV DNA >110000000 IU/mL. Serological tests for hepatitis B were consistent with reverse seroconversion from anti-HBs to HBsAg. Lamivudine treatment was started at 300 mg per day. INR progressively elevated, and hypoalbuminemia and ascites developed. The patient's consciousness worsened and she died of liver failure 21 days after the treatment was started.

Hepatitis B virus (HBV) is a DNA virus that can be cleared at rates of more than 95% in acute infection in adults. However, it can cause chronic infection in about 5% of adult patients. Immunosuppressive and chemotherapeutic agents can stimulate the replication of the virus. It is well known that reactivation of HBV in subjects receiving cytotoxic treatment for hematological malignancies occurs in 21-53% of chronic HBsAg carriers and in an

unknown number of HBsAg-negative subjects harboring occult HBV infection (1). Immune reconstitution within the weeks and months following recovery from chemotherapy may be associated with a flare of hepatitis B manifested by hepatocellular injury (2). This status can lead to severe hepatitis and fatal liver dysfunction. In order to avoid this potentially fatal complication, it is important to ensure that all patients at risk of chronic HBV infection are screened before commencing immunosuppressive treatment. HBV-negative patients should be immunized. In the patients with prior HBV infection, even with anti-HBs(+), anti-HBc IgG(+) and PCR HBV DNA(-), anti-viral agents should be started before or at least at the same time as chemotherapy. Nucleoside analogues are more acceptable when compared with other agents in these patient groups. Most studies have suggested lamivudine for hepatitis B reactivation (3, 4).

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## Spontaneous intraabdominal hematomas associated with arterial aneurysms in polycythemia vera

Polisitemia veralı olguda arterial anevrizma ile ilişkili spontan intraabdominal hematoma

*To the Editor,*

A 73-year-old male was diagnosed with polycythemia vera five years earlier and treated with perio-

dic phlebotomies. He also suffered from chronic obstructive pulmonary disease for three years. In

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**Manuscript received:** 13.09.2008 **Accepted:** 05.11.2009

doi: 10.4318/tjg.2010.0058