A case of complete heart block induced by octreotide

Oktreotid ile indüklenen bir tam kalp bloğu olgusu

To the Editor,

Acute variceal bleeding is one of the major complications of portal hypertension in cirrhotic patients. The current recommended hemostatic treatment of variceal bleeding is to start a vasoactive drug from admission or upon the patient's transfer to the hospital and associated endoscopic therapy at the time of diagnostic endoscopy (1). The combination of sclerotherapy and octreotide infusion has been advocated in some studies (2). However, it may have effects on cardiac conduction (3).

A 53-year-old female was admitted to our hospital due to overt hematemesis. She had been under treatment for cirrhosis associated with hepatitis B for 26 months. Blood pressure, pulse rate, and hematocrit (Htc) were 80/60 mmHg, 85/min and 32.4%, respectively. After an intravenous bolus of 50 mcg octreotide, 50 mcg/hr octreotide infusion was started. Bradycardia (heart rate 32 bpm) and hypotension (70/40 mmHg) developed at the 60th hour of octreotide infusion. The electrocardiography revealed complete atrioventricular (AV) block (Figure 1A). Although heart rate and blood pressure improved after atropine administration and saline infusion (45-50 bpm), AV block failed to resolve. The patient was monitored, and since hemodynamics were stable, we did not proceed with pacing. Electrolytes were within normal limits. Transthoracic echocardiography was normal. Angiography was performed to eliminate underlying ischemic heart disease but coronary arteries were normal. Six days after cessation of octreotide infusion, the patient was out of complete AV block and in normal sinus rhythm (Figure 1B).

Mild bradycardia has been described after octreotide administration, though not frequent (4). In a small series of patients with acromegaly receiving octreotide subcutaneously, the mean heart rate decreased, and the mean systemic vascular resistance increased (5).

Dilger et al. (3) reported an octreotide-induced third-degree heart block during surgical resection of a carcinoid tumor after a large bolus administration. Herrington et al. (4) described a patient who was receiving octreotide subcutaneously and developed bradycardia (38 bpm) without heart block. In several other reports, octreotide administration did not consistently demonstrate a reduction in heart rate (6).

Figure 1. (A) The ECG revealed complete AV block **(B)** The ECG in normal sinus rhytm

Address for correspondence: Hasan Ali GÜMRÜKÇÜOĞLU
Yüzüncü Yıl Üniversitesi Tıp Fakültesi
Araştırma Hastanesi Kardiyoloji Servisi 65100
Van, Turkey
Phone: + 90 432 215 04 70

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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 60th 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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Mustafa TUNCER[1],
Hasan Ali GÜMRÜKÇÜOĞLU[1], Rafet METE[2],
Yılmaz GÜNEŞ[1], Ünal GÜNTEKIN[1]

Departments of [1]Cardiology and [2]Gastroenterology, Yüzüncü Yıl University, School of Medicine, Van

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 remission 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-

Address for correspondence: Müge USTAOĞLU
Ondokuz Mayis University Faculty of Medicine
TR-55139 Samsun, Turkey
Phone: + 90 362 312 19 19/3572 • Fax: + 90 362 431 66 62
E-mail: ustaoglu.md@gmail.com

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