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Title: Complete Heart Block due to Octreotide Infusion in Patient with Cryptogenic Cirrhosis

Running Head: Complete Heart Block due to Octreotid

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Abstract: A 62-year-old man was admitted to the emergency department (ED) with the complaint of intense hematemesis. He was admitted to intensive care unit because of acute esophageal variceal hemorrhage. He underwent sclerotherapy followed by a slow infusion of intravenous octreotide. Complete heart block occured in the patient during octreotide infusion and infusion was stopped. Temporary pacemaker was placed in the patient's heart. Normal sinus rhythm was observed in the follow up one day later and the pacemaker was removed from patient. He was discharged upon recommendation.

Introduction: Octreotide, a synthetic cyclic octapeptide, is a somatostatin analogue. It is used in

acute variceal bleeding in patients with cirrhosis after sclerotherapy [1]. It can also be used in the treatment of acromegaly and carcinoid syndrome [2, 3]. Although rare, octreotide may cause mobitz type 2 block, sinus bradycardia and complete heart block [4]. In this article, we presented the patient who developed a complete heart block due to octreotide infusion after acute variceal hemorrhage.

Case presentation: A 62 years old man was admitted to the emergency department (ED) with complaints of intense hematemesis. Previously, he was followed up medically for cryptogenic cirrhosis. In the physical examination of the patient, tension arterial was measured as 90/60 mmHg, pulse rate 115 / minute, respiratory rate 16/min. In the laboratory findings, hemoglobin was 8.5 mg / dl, and hematocrit was 24.2%. The patient was referred to the gastroenterology in the ED. He was admitted to intensive care unit due to suspected acute gastrointestinal bleeding by gastroenterology. The gastroscopy revealed acute esophageal variceal hemorrhage and he underwent sclerotherapy for treatment, followed by a slow infusion (100 µg/h) of intravenous octreotide. He was followed up as monitorized developed bradycardia (55 / min) and complete heart block was detected at the ECG at the 24th hours of octreotide infusion (figure 1). He was diagnosed with complete heart block and was taken into (coronary intensive care unit (CICU). Temporary pacemaker was placed to patient due to hemodynamic instability. Anamnesis taken from the patient didn't had a cardiac event history and he did not use negative chronotropic drugs before. The complete heart block was ligated to the infusion of ocreotide and infusion was discontinued. It was observed that the patient entered the sinus rhythm (figure 2) on the 1st day of the follow-ups in the CICU. The temporary pacemaker was removed, patient with no additional problems were recommended to be discharged upon recommendation.

Discussion: Our patient was referred to the emergency department because of acute esophageal variceal hemorrhage. The patient had tachycardia initially, but he developed bradycardia after octreotide infusion and a complete heart block was diagnosed. Octreotide can cause bradycardia and heart block with several different mechanisms as dose dependent. In a study, cardiovascular (CV) effect may develop at subcutaneous octreotide dose [4]. The cardiovascular effect appears to be less frequent at 50 µg/h and 100 µg/h 50 [5,6], and more frequent at 250 µg/h doses [7]. Although our patient's dose was relatively less, complete heart

block was observed at 24 hours. The observation of T wave negativity in chest leads on surface ECG after sinus rhythm suggested a possible coronary ischemia in the patient. The lesser dose of octreotides' cardiovascular effect may have become more prominent due to possible coronary ischemia. It may also act directly on acetyl choline receptors as well as on negative chronotropic effects on the heart. In addition, it may increase systemic vascular resistance and create reflex bradycardia on the baroreceptors [8]. Finally, ocreotide suppresses the secretion of vasoactive intestinal peptide (VIP) that can increase the heart rate. Ocreotid may reduce heart rate due to VIP depression [9]. In our patient, we did not consider the possibility of reflex bradycardia because he had hypotension due to acute hemorrhage. We thought it could be caused by mechanisms that could lower the heart rate more directly. There are similar cases in the literature about bradycardia and complete heart block developing after octreotide infusion [10,11]. Bradycardia and cardiac conduction defects may develop during octreotide infusion and the hemodynamics of the patient may be further impaired. These patients should be followed up with closer monitoring.

*The patient was informed and received his approval for this case report.

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