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

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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.

Keywords: Ocreotide, heart block, bleeding.

Introduction

Octreotide, which is a synthetic cyclic octapeptide, is a somatostatin analogue. It is used for tretaing 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. In the present study, we present a patient who developed a complete heart block beause of octreotide infusion after acute variceal hemorrhage. Informed consent was obtained from the patient.

Case Presentation

A 62-year-old man was admitted to the emergency department (ED) with the complaint of intense hematemesis. Previously, he was medically followed up for cryptogenic cirrhosis. On physical examination, tension arterial was measured as 90/60 mmHg, pulse rate 115/min, and respiratory rate 16/min. Laboratory findings reaveled that 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. Gastroscopy findings revealed acute esophageal variceal hemorrhage and he underwent sclerotherapy, 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 hour of octreotide infusion (Figure 1). He was diagnosed with complete heart block and was taken into (coronary intensive care unit [CICU]). The temporary pacemaker was placed in the patient's heart owing to hemodynamic instability. Anamnesis obtained from the patient did not include a history of any cardiac event and negative chronotropic drug use. 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 on the first day of the follow-ups in the CICU (Figure 2). The temporary pacemaker was removed, and patient had no additional problems and was recommended to be discharged upon recommendation.

Discussion

The patient in this study was referred to the emergency department because of acute esophageal variceal hemorrhage. The patient initially had tachycardia, but he developed bra-

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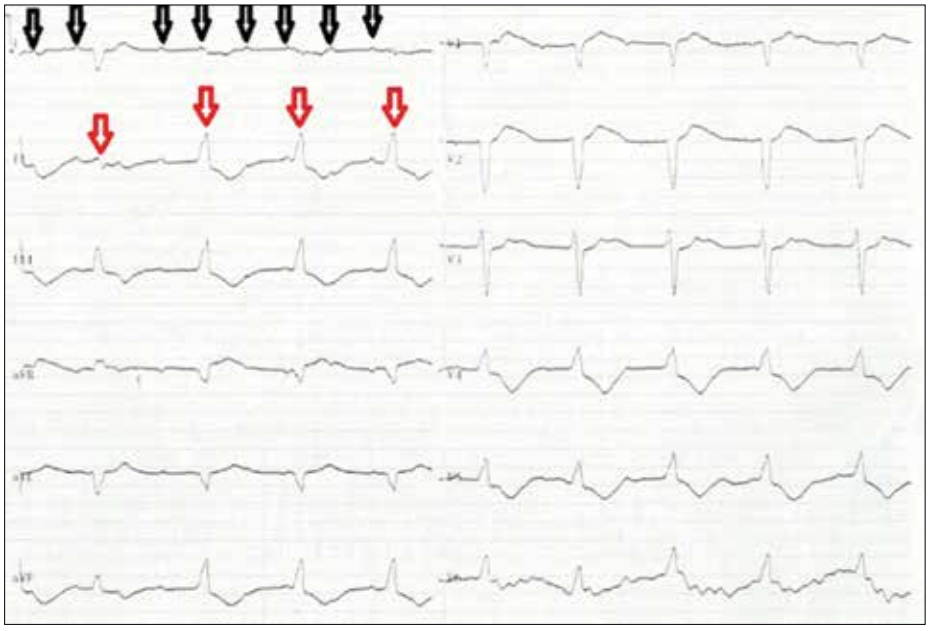

Figure 1. Demonstration of complete heart block on 12 lead surface electrocardiogram. P waves were shown with black arrows, QRS with red arrows.

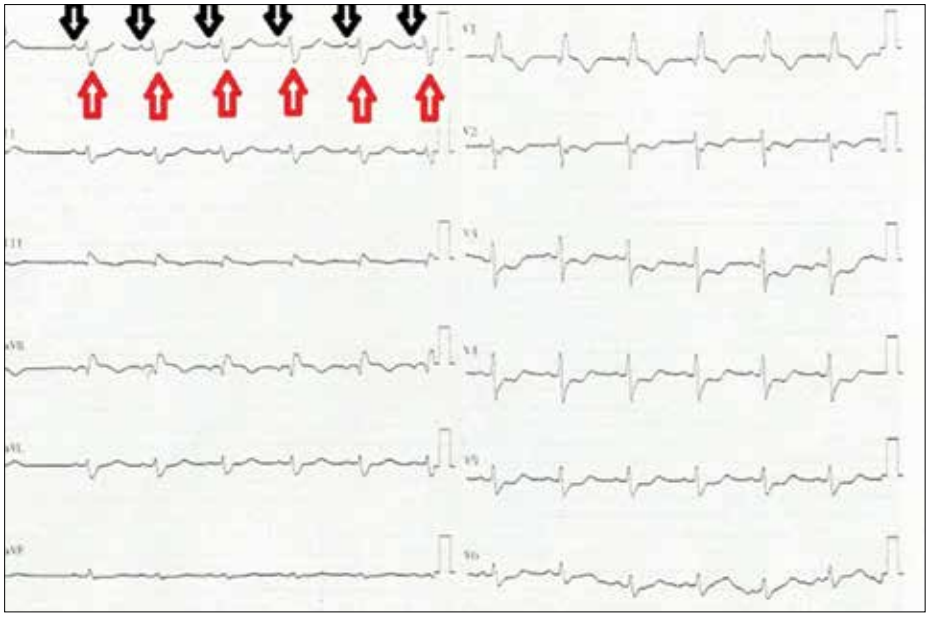

Figure 2. Demonstration of normal sinus rhythm after ocreotid infusion stopped.

dycardia after octreotide infusion and a complete heart block was diagnosed. Octreotide can cause bradycardia and heart block with several different mechanisms depending on its level in the blood. In a study, cardiovascular (CV) effect may develop at subcutaneous octreotide dose [4]. The cardiovascular effect appears to occur less frequently at 50 µg/h and 100 µg/h 50 µg/h doses [5, 6], and more frequently at 250 µg/h doses [7]. Although our patient's dose was relatively less, complete heart block was observed at 24 h. The observation of T wave negativity in chest leads on surface ECG after sinus rhythm suggested a possible coronary ischemia in the patient. The Lower level of octreotides' cardiovascular effect may have become more prominent due to possible coronary ischemia. It may also directly act on acetyl choline receptors and have negative chronotropic effects on the heart. In addition, it may increase systemic vascular resistance and create reflex bradycardia on the baroreceptors [8]. Lastly, ocreotide suppresses the secretion of vasoactive intestinal peptide (VIP) that can increase the heart rate. Ocreotide 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 caused by acute hemorrhage. We believed it could be caused by mechanisms that could lower the heart rate more directly. There are similar cases in the literature regarding bradycardia and complete heart block development 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.

Informed Consent: Informed consent was obtained from the patient.

Peer-review: Externally peer-reviewed.

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