ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Clonidine - Induced Mobitz Type 2 Heart Block.

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ABSTRACT

Clonidine intolerance has been frequently reported in the last decade. However, in recent years, there has been a significant drop in reporting, both in developed as well as developing countries. Clonidine is a commonly used anti-hypertensive drug. It is a centrally acting alpha agonist. There have been reports of clonidine causing symptomatic bradycardia. Mobitz type-2 heart block is an unstable rhythm, which when left untreated, can be fatal. This is a case report of a patient who developed this sinister bradyarrhythmia at regular doses in a setting of mild renal failure.

Keywords: Bradyarrhythmias, Hypertension, Holter monitoring, ECG

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ISSN: 0975-8585

INTRODUCTION

Mobitz type 2 heart block is defined by a fixed PR interval, followed by a missed beat. It is usually a forerunner of advanced cardiac conduction defects. The site is usually the bundle of His or slightly proximal to it [1].

Clonidine, an antihypertensive drug, activates alpha 2 receptors in the brain, thereby causing reduction in the sympathetic outflow. As a result, noradrenaline levels are reduced, resulting in fall in blood pressure. It acts both by reducing peripheral resistance and cardiac output, and may result in postural hypotension. Sinus arrest and symptomatic bradycardia in patients with pre-existing AV node and SA node blocks are cardiac adverse effects associated with clonidine use [2].

Case Report

A 57-year-old male patient, known to be a hypertensive and a type-2 diabetic, presented with persistent hiccups, abdominal discomfort with severe nausea, vomiting and giddiness to our hospital. On examination, he was conscious and alert. He had bradycardia (with a pulse rate of 30 beats per minute) and his blood pressure was 120/86 mm of Hg. All laboratory investigations were within normal limits, except renal function tests, which were deranged.Electrocardiogram (ECG) showed bradycardia with type 2 Mobitz block (as shown in Figure 1). He was treated with antiemetics and IV fluids and his antihypertensive medications were continued. He was on clonidine as antihypertensive medication. He was advised a Holter testing for the assessment of fluctuating heart rate. Holter report indicated that he had bradycardia with long pauses. Echocardiography showed mild left ventricular dysfunction. It was diagnosed as clonidine-induced type 2 heart block, and clonidine was discontinued. His other antihypertensives (a calcium channel blocker and an alpha1 blocker) were continued.

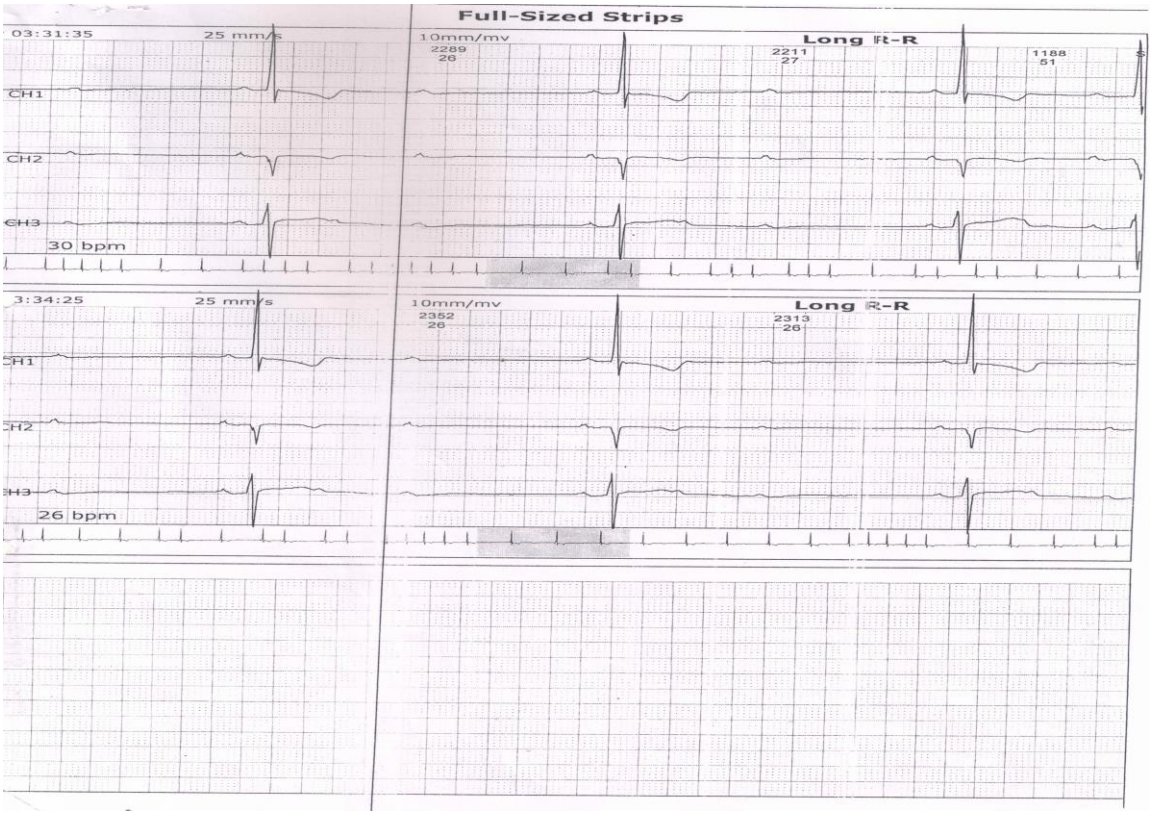

Figure 1: ECG (On Clonidine)

ISSN: 0975-8585

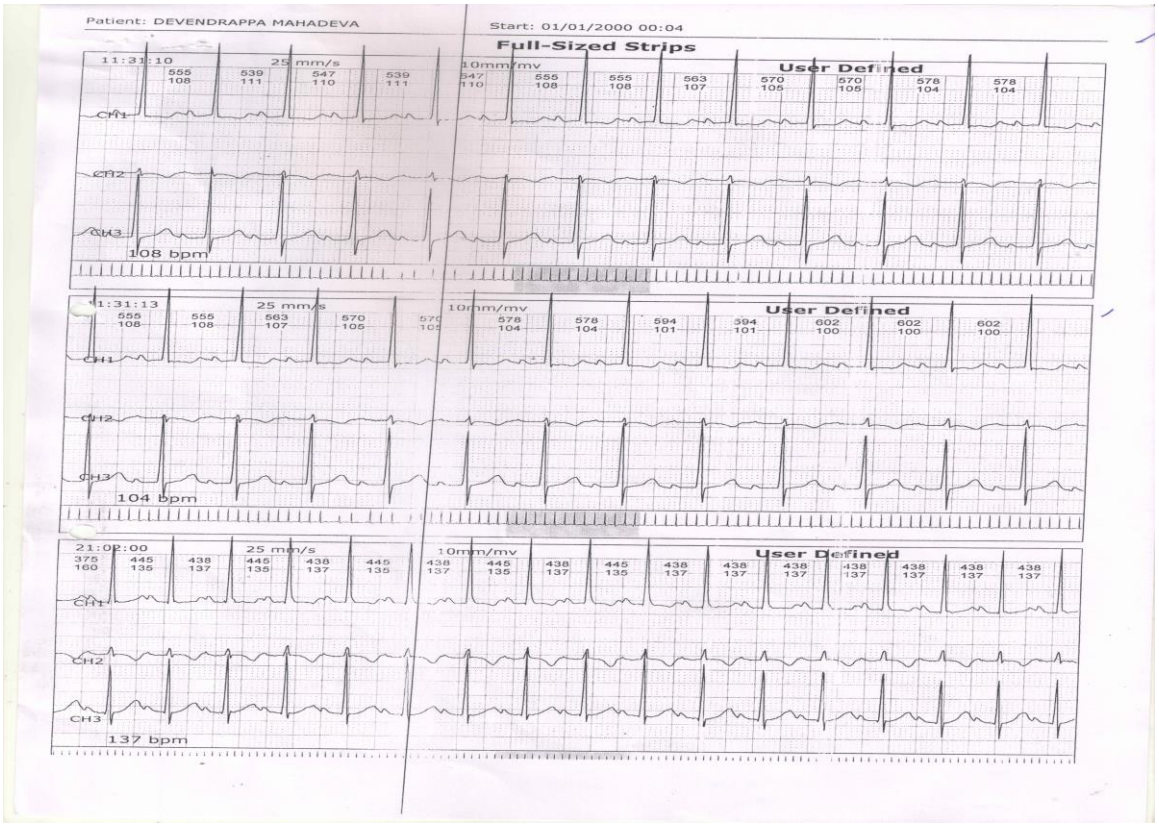

Figure 2: ECG (Off Clonidine)

On discontinuing clonidine, the patient improved. Repeat ECG (as shown in Figure 2) and Holter were normal. The patient was discharged and advised regular follow up.

DISCUSSION

The above-mentioned patient had symptomatic AV block, which resolved on stopping clonidine and there was no other concomitant medication that could have caused the same. The incidence of clonidine-induced bradyarrhythmia is low (0.3%). The risk factor for the symptomatic Mobitz type 2 block in this case can be renal failure [3]. Clonidine causes AV block in a concentration-dependent manner. It took three days for normalization of ECG and Holter recordings.

The exact mechanism for clonidine causing AV block is not known [3]. Most of the cases reported causing AV block so far are due to overdosing or had pre-existing disease of the myocardium and the conduction system [4]. In the above case, the patient neither had a pre-existing damaged myocardium nor was he overdosed.

The causality assessment score based on Naranjo's algorithm was 5. Therefore, it is a "probable" adverse drug reaction.

CONCLUSION

Initiating clonidine as an anti-hypertensive in a setting of renal failure can be dangerous. Hence, proper cardiovascular monitoring and follow up are necessary to prevent and manage cardiac complications like bradyarrhythmias, which may turn out to be life-threatening or even fatal.

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