Paradoxical heart rate response after atropine sulfate administration in total atrioventricular block complicating acute myocardial infarction A case report

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Submission date: 14-Mar-2022 05:24PM (UTC+0800)
Submission ID: 1783933185
File name: block_complicating_acute_myocardial_infarction_A_case_report.pdf (1.53M)
Word count: 1820
Character count: 10370

Case Report

Paradoxical Heart Rate Response After Atropine Sulfate Administration in Total Atrioventricular Block Complicating Acute Myocardial Infarction: A Case Report

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Abstract

Acute myocardial infarction is sometimes complicated by atrioventricular block. Advanced cardiac life support guideline for the treatment of atrioventricular block suggests early use of atropine. Atropine works as a parasympatholytic drug that enhances SA node automaticity and AV node conduction. We report a case of a male patient with inferior myocardial infarction and total atrioventricular block who showed a marked reduction in heart rate after first and second atropine administration, a paradoxical worsening of the block. Atropine has been associated with some adverse consequences, including proarrhythmic effect, worsening of the high-grade atrioventricular block, and worsening of the ischemic situation. In this case of total atrioventricular block caused by acute myocardial infarction, immediate revascularization can be the only required management. Awareness of this potential adverse reaction will help the clinician make a risk/benefit ratio consideration regarding the use of atropine for certain patients.

Keywords: acute myocardial infarction, case report, heart rate, total atrioventricular block

Introduction

Acute myocardial infarction (AMI) is frequently complicated by atrioventricular block (AVB). There is a greater incidence of total AVB in inferior AMI (odd ratio = 2.59) than in anterior or lateral AMI.[1] Advanced Cardiac Life Support (ACLS) guidelines for the treatment of hemodynamically unstable bradycardia or AVB suggest the early use of atropine. Atropine works as a parasympatholytic drug that enhances sinoatrial (SA) node automaticity and atrioventricular (AV) nodes conduction by direct vagolytic action.[2] However, several reports have shown the potential adverse reaction of atropine when used in AVB complicating AMI. We report a case of a patient with inferior myocardial infarction and total atrioventricular block who showed a marked reduction in heart rate after atropine administration, a paradoxical worsening of the block.[3]

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DOI: 10.4103/ijdpdd.ijdpdd_76_21

Case Report

A 67-year-old man patient presented to the emergency room with retrosternal chest pain for 3 h before admission. The pain radiated to the left shoulder and neck. Patient never had chest pain like this before. Prior history was suggestive for risk of cardiovascular disease. Patient had uncontrolled hypertension for 3 years and smoked excessively for 50 years. Physical examination showed irregular heart rate 58 beats/min, respiration rate 22 breaths/min, blood pressure 110/70 mmHg. Other systemic findings were within normal range. Electrocardiography

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Submitted: 10-Jul-2021 **Revised:** 22-Jul-2021
Accepted: 05-Oct-2021 **Published:** 24-Nov-2021

How to cite this article: Rachmi DA, Novira RY, Mulia EP, Andrianto A. Paradoxical heart rate response after atropine sulfate administration in total atrioventricular block complicating acute myocardial infarction: A case report. Bali J Anaesthesiol 2021;5:271-4.

(ECG) at admission showed ST elevation in inferior lead and total AVB (TAVB) with ventricular and junctional escape rhythm [Figure 1]. Posterior and right ventricle ECG did not show any significant findings. Elevated cardiac enzyme marker confirmed the diagnosis of myocardial infarction. Patient was diagnosed with inferior ST-Elevation myocardial infarction (STEMI) with TAVB.

Oxygen, 320mg aspirin, and 300mg clopidogrel were given as initial therapy for acute coronary syndrome. Since the presentation was within the window period of 3h and the hospital did not have cardiac catheterization laboratory, reperfusion by fibrinolytic agent was chosen. Before fibrinolysis, 0.5mg atropine sulfate was injected to treat the bradycardia. The heart rate recorded soon after the first atropine administration was 38 beats/min. His heart rate became even slower, and chest pain worsened after second atropine administration (30 beats/min). We started continuous Dopamine infusion at 5 ug/kg/min, to which he responded well, increasing his heart rate and blood pressure. During and after the administration of 1.5 million IU streptokinase, the patient remained stable. Vital signs were normal during fibrinolysis [Table 1]. ECG post fibrinolytic and the following day showed complete resolution of AVB [Figures 2 and 3]. The patient was discharged on the sixth day of hospitalization.

Discussion

The pathophysiologic mechanisms that explain AVB events in AMI patients involve reversible ischemic injury of AV nodes and increased parasympathetic influence. If AVB is caused by reversible ischemia injury on AV nodes, the patient tends to respond poorly to atropine therapy. Nevertheless, AVB caused by increased parasympathetic influence will be more likely to respond rapidly to atropine therapy. In most cases, after atropine administration, there would be either no alteration or improvement of clinical condition.[3]

In our case, 0.5 mg dose of atropine was given twice. Instead of increasing, the heart rate became slower consistently soon after each administration. Initially, the heart rate was 58 beats/min and later became 30 beats/min after a total of 1 mg atropine injection. A case reported that 1 mg atropine was given to a patient with sinus rhythm and heart rate of 68 beats/min, and 30 min later, the patient developed 2:1 AV block.[4] Another reported a similar event where such intensification of AVB occurred after atropine administration in AMI patient.[5]

The occurrence of adverse events after atropine administration was quite rare. The adverse events ever documented so far other than worsening of AVB were proarrhythmic effects. Ventricular fibrillation and ventricular tachycardia were among arrhythmic complications that developed after atropine administration.[5] Another adverse event of atropine was potentiation of ischemic condition, which most likely happened in our case as the escape rhythm slowed down after atropine administration. A case reported by Brady

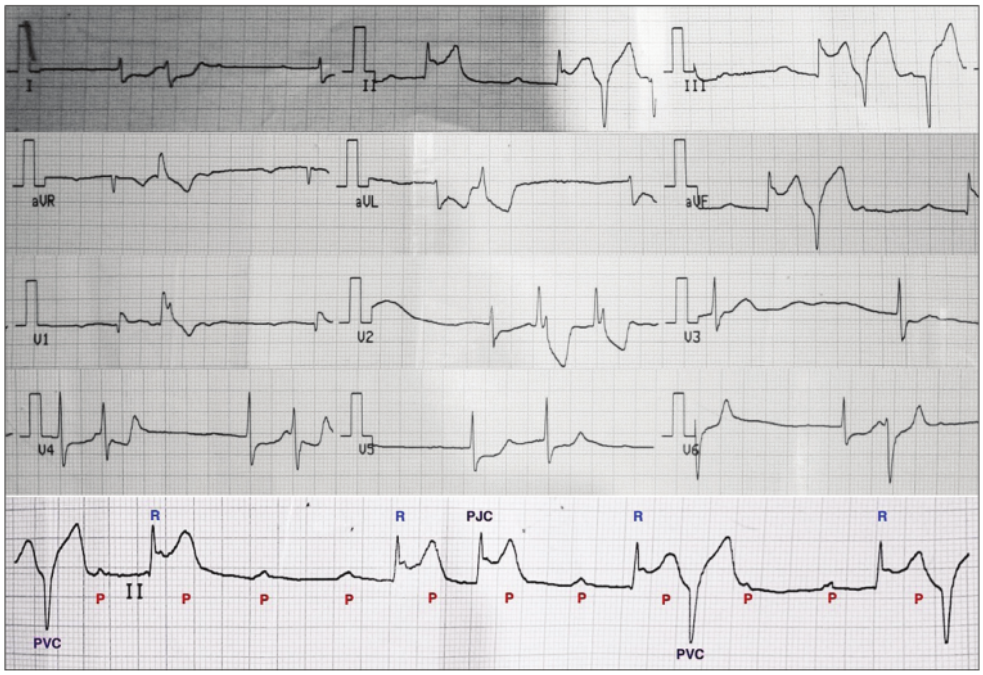

Figure 1: 12-lead ECG at arrival showed inferior myocardial infarction with total AV block with junctional escape rhythm and supraventricular and ventricular extrasystole

Table 1: Vital signs during treatment in emergency department (ED)

Time	Medication	BP (mmHg)	HR (beats/min)	RR (breaths/min)
12.30	Oxygen, IV access 12 lead ECG	110/70	58	20
13.00	320 mg aspirin PO 300 mg clopidogrel PO	110/70	56	20
13.15	0.5 mg sulfas atropine IV (I)	90/70	38	24
13.30	0.5 mg sulfas atropine IV (II)	90/60	30	24
15.00	Fibrinolytic by 1.500.000 IU Streptokinase IV	120/70	65	24

BP: blood pressure, HR: heart rate, PO: peroral, IV: intravenous, RR: respiration rate.

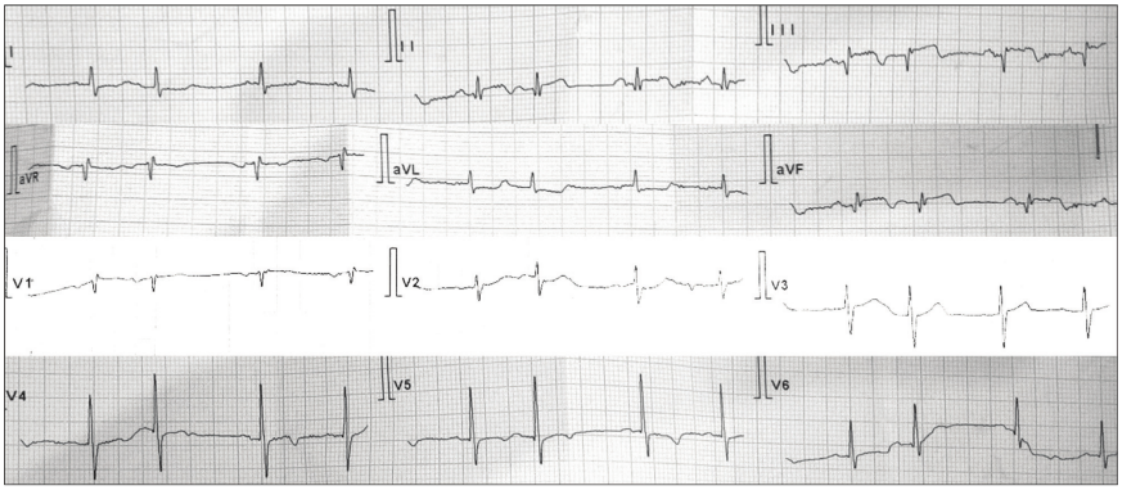

Figure 2: ECG recorded 1 h after fibrinolytic showed a reduction of ST-elevation and resolution of atrioventricular block and occasional supraventricular extrasystole

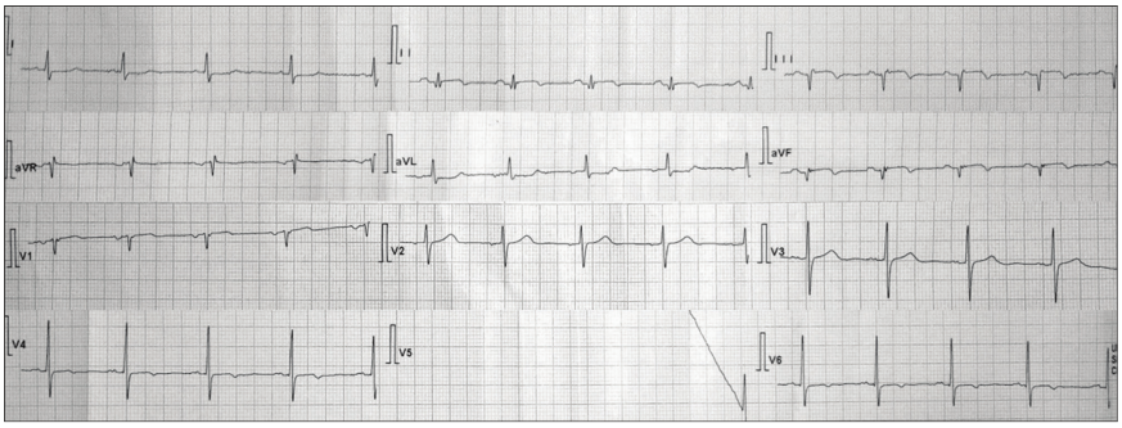

Figure 3: ECG obtained following days after fibrinolytic showed complete resolution of total AVB

also showed possible conversion of acute ischemia to AMI related to atropine administration.[5]

Recommended indications for atropine therapy include: (1) sinus bradycardia with hypoperfusion; (2) symptomatic type 1 second-degree AVB; (3) bradycardia and hypotension after administration of nitroglycerin; and (4) complete heart block at or above the level of AV nodes, i.e., a narrow QRS complex.[3] In our case, the escape rhythm was a narrow QRS complex suggesting the block at or above the level of AV nodes. Paradoxical slowing of the heart rate after administration of atropine has been found rarely in patients with infranodal AV block—Mobitz Type II second-degree AV block and TAVB with a wide QRS complex.[3]

Conclusion

Atropine has been associated with a marked reduction in heart rate that shows paradoxical worsening of atrioventricular block. This case may involve such a patient. Therefore, atropine is indicated only if the patient showed signs of hypoperfusion or clinical deterioration. In our case of TAVB caused by acute myocardial infarction, immediate revascularization can be the only definitive management. An awareness of this potential adverse reaction will help the clinician to make a risk/benefit ratio consideration regarding the use of atropine in a particular patient.

Authors' contributions

DAR and RYN equally contributed to data collection, analysis, and interpretation, and revised manuscript. EPBM contributed to data interpretation, literature review of the case, and manuscript drafting. AA contributed to the literature review of the case and manuscript drafting. All authors have read and approved the manuscript.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Availability of supporting data

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Financial support and sponsorship

The authors received no financial support for the research, authorship, and/or publication of this article.

Conflicts of interest

There are no conflicts of interest.

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