SAGE Open Medical Case Reports
Volume 9: 1–5

DOI: 10.1177/2050313X21996113
journals.sagepub.com/home/sco
SAGE

Dopamine and epinephrine for managing complete atrioventricular block due to nonreperfused acute inferior wall myocardial infarction in a rural hospital: A case report

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Abstract

Inferior wall myocardial infarction occurs in approximately 50% of all myocardial infarctions. The most common conduction disorder of this disease is complete atrioventricular block. Immediate attention must be given to the myocardial infarction patients with conduction block due to the increased mortality rate in these patients. Temporary pacemaker implantation and permanent pacemaker implantation are recommended in complete atrioventricular block cases that do not improve with reperfusion. In this case report, a 64-year-old-female patient came to the emergency department of a rural General Hospital with complaints of epigastric pain, dizziness, nausea, and vomiting for 2 days before admission. She had uncontrolled hypertension without a history of diabetes mellitus, dyslipidemia, smoking, or a family history of heart disease. The electrocardiogram displayed an acute inferior wall myocardial infarction and complete atrioventricular block with escape junctional rhythm with a heart rate of 17 bpm. She was diagnosed with nonreperfused inferior wall myocardial infarction and a complete atrioventricular block. She was successfully treated with only dopamine and epinephrine as the definitive treatment because the patient refused to be referred to a tertiary hospital for percutaneous coronary intervention and pacemaker implantation due to financial reasons. Dopamine and epinephrine may be considered for complete atrioventricular block if transfer to a higher level of care is not feasible and as bridge therapy while waiting for transfer.

Keywords

Complete atrioventricular block, nonreperfused acute inferior wall myocardial infarction, dopamine, epinephrine, rural hospital, case report

Date received: 26 September 2020; accepted: 25 January 2021

Introduction

Inferior wall myocardial infarction (IWMI) occurs in approximately 50% of all myocardial infarction cases. Conduction abnormalities, particularly complete atrioventricular (AV) block, are common in IWMI.[1,2] Immediate attention must be given to the myocardial infarction (MI) patients with conduction block due to the increased mortality rate in these patients.[2] All reversible causes of AV block should be addressed. Reperfusion should not be delayed in patients with acute MI and complete heart block. In patients with heart block secondary to an acute MI, temporary pacing is recommended.[3] However, not every health center is capable of pacemaker implantation, especially hospitals in rural areas. Dopamine and epinephrine may enhance the automaticity of subsidiary pacemakers in a complete AV block due to the positive chronotropic and inotropic effects on heart muscles caused by α_1 and β_1 receptors stimulation through the increase of intracellular cAMP.[4] These drugs may be utilized in certain situations to maintain adequate cardiac output, but not routinely administered to manage complete AV

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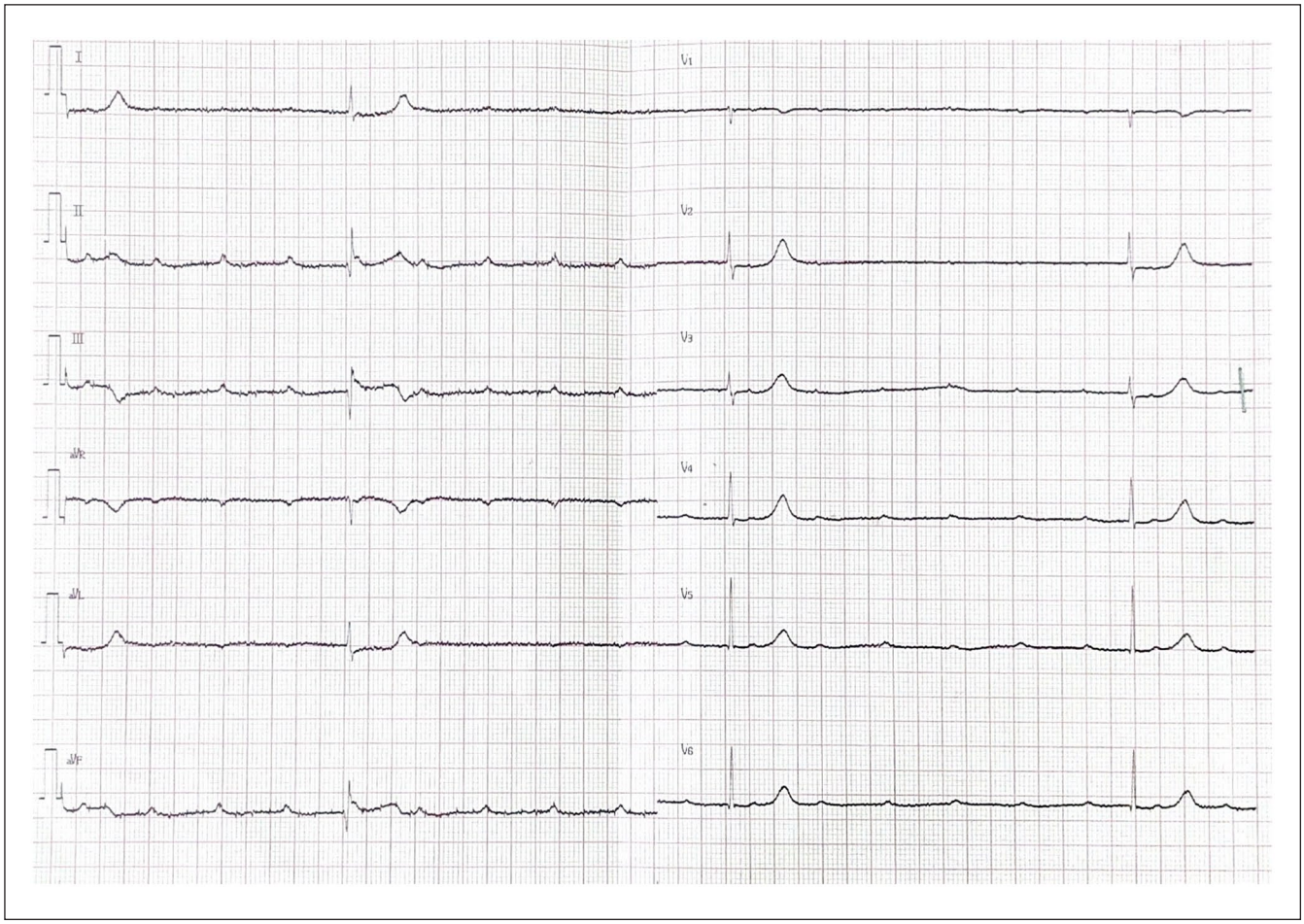

Figure 1. The patient's ECG during the admission indicating acute IWMI and complete AV block.

block in a non-reperfused acute IWMI.[5] In this case report, we aim to illustrate the successful use of dopamine and epinephrine for managing complete AV block in non-reperfused acute IWMI in a rural hospital.

Case presentation

A 64-year-old-female came to the emergency department of *Kota Madiun* General Hospital with complaints of epigastric pain, dizziness, nausea, and vomiting for 2 days before admission. She had uncontrolled hypertension without history of diabetes mellitus, dyslipidemia, smoking, or family history of heart disease. The initial vital signs were normal with blood pressure (BP): 120/80 mmHg, respiratory rate (RR): 20 times/min, and heart rate (HR): 20 beats per minute (bpm). The electrocardiogram (ECG) in Figure 1 displayed an acute IWMI and complete AV block with escape junctional rhythm with a HR of 17 bpm. The laboratory examination indicated mild anemia (Hb = 11 g/dL), azotemia (57 mg/dL), and slight hyperkalemia (5.87 mmol/L) as shown in Table 1. She was diagnosed with acute IWMI with complete AV block, and hyperkalemia, without any signs of cardiogenic shock. The patient refused to be referred to a tertiary hospital percutaneous coronary intervention (PCI) and temporary pacemaker implantation (TPI) due to financial reasons. As TPI and PCI are not possible to be performed in the hospital, she was managed conservatively with NaCl 0.9% 2000 mL/24 h, acetylsalicylic acid 320 mg oral, clopidogrel 300 mg oral, enoxaparin 30 mg IV, dopamine 5–10 mcg/kgBW/min, and epinephrine 6–7 mcg/min. Epinephrine was added to the therapy due to inadequate response from dopamine administration only. The patient was also given 25 mL of D40 injection and 8 units of insulin. On the third day, the patient claimed that she felt better. Dopamine injection was stopped, and the epinephrine dose was reduced to 1.3 mcg/min due to the patient's increased BP (187/61 mmHg). The patient's BP returned to normal on the fourth day (124/87 mmHg). On the seventh day, enoxaparin injection was stopped. There were not any additional symptoms and the ECG in Figure 2 showed IWMI resolution with a first-degree AV block. The echocardiogram showed inferior hypokinesis with a good left ventricular systolic function, as shown in Figure 3. The patient was discharged without any additional symptoms and a stable hemodynamic status.

Table 1. The patient's laboratory results.

Parameter	First day	Second day	Units
Complete blood count			
Hemoglobin	11	–	g/Dl
Leukocyte	7.6	–	$\times 10^3$ cells/μL
Hematocrit	33	–	%
Platelets	199	–	$\times 10^3$ cells/μL
Blood chemistry			
Glucose	204	–	mg/dL
Triglyceride	111	–	mg/dL
High-density lipoprotein (HDL)	58	–	mg/dL
Low-density lipoprotein (LDL)	84	–	mg/dL
Blood urea nitrogen (BUN)	57	–	mg/dL
Creatinine	2.62	–	mg/dL
Uric acid	5.2	–	mg/dL
Aspartate transaminase (AST)	188	–	U/L
Alanine aminotransferase (ALT)	89	–	U/L
Albumin	3.5	–	g/dL
Electrolyte			
Sodium	141	144	mmol/L
Potassium	5.87	4.71	mmol/L
Chloride	112	109	mmol/L

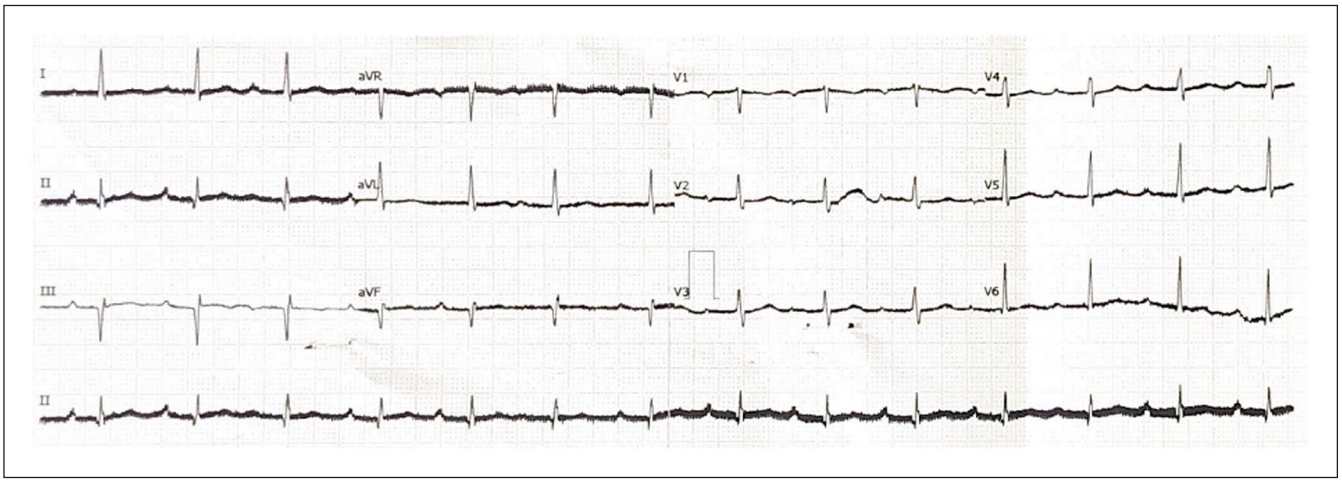

Figure 2. The patient's ECG on the seventh day after the admission showing a first-degree AV block indicating IWMI resolution.

Discussion

In approximately 80% of the population, the myocardial inferior wall is vascularized by right coronary artery (RCA) from the posterior descending artery.[6] The arterial supply of AV node arises from the RCA or left circumflex artery (LCx) and septal perforator branch of left anterior descending artery (LAD). An occlusion in RCA would lead to an IWMI, disrupting the AV node conduction.[7] Up to 90% complete AV block cases in acute IWMI occurred above the His bundles, causing junctional rhythm ($>$ 40 bpm).[8] In addition to RCA occlusion, there are several mechanisms which could lead to a conduction disorder in acute MIs: (1) Increased vagal tone; (2) Systemic hypoxia; (3) Electrolyte disturbance, such as hyperkalemia which occurred in this patient; (4) Increased local adenosine; and (5) Acid-base balance disturbance (metabolic acidosis).[9] In patients with acute inferior infarct secondary to an occluded RCA, immediate restoration of arterial perfusion may lead to improvement of the complete AV block. Reperfusion should not be delayed in patients with acute MI and complete AV block.[3,10] In the era of percutaneous coronary intervention (PCI) as a treatment of choice, the rate of complete AV block in acute MI is 2.5%–3%, whereas in the fibrinolysis era, the rate was 7.3%.[11] Kant et al.[12] reported a patient with complete AV block complicating inferior wall, right ventricular, and anteroseptal MI that

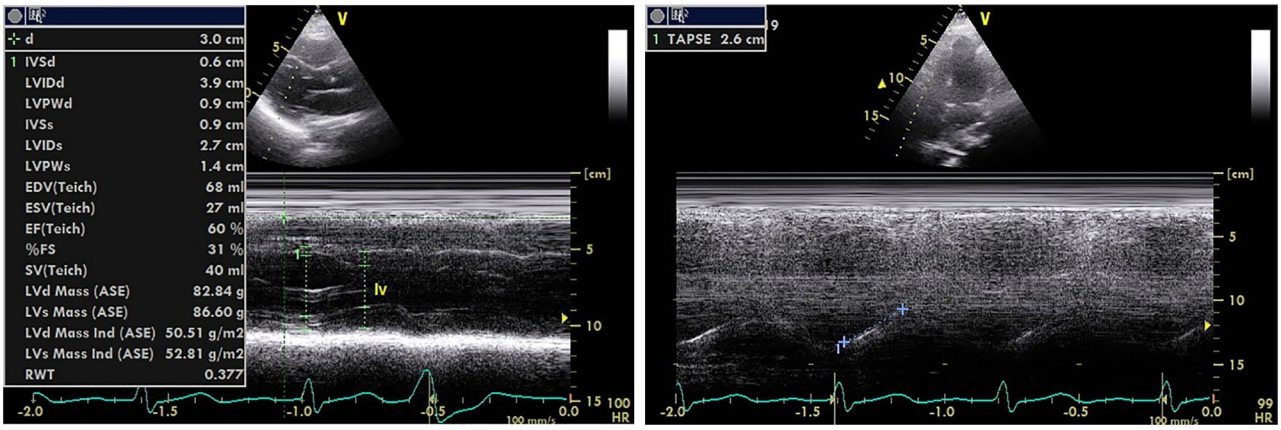

Figure 3. The patient's echocardiogram result on the seventh day after the admission showing inferior hypokinesis with a good left ventricular systolic function.

showed improvement to a normal sinus rhythm in 24 h after being managed by fibrinolysis, atropine, and other acute MI treatments. Unfortunately, there has not been any published randomized clinical trials (RCTs) comparing different conduction disorders managements in acute MIs.[13] ACC/AHA/HRS 2008 recommended cardiac permanent pacemaker implantation in complete AV block as a complication of non-reperfused IWMI. Reperfusion and pacemaker implantation were indicated in the patient; however, she refused to be referred due to financial reasons. In situations like this, which commonly occurs in developing countries, treatment decisions are based on the best available evidence and on the patient's goal of care and preferences. Shared decision-making and patient-centered care are endorsed and emphasized by respecting the patient's decision even if it was not in line with the guideline's recommendation.[5] Tissue plasminogen activator (tPA) was not considered because the patient had already presented with late onset STEMI as indicated by the presence of a pathological Q-wave in the inferior lead. The electrocardiogram result showed a pathological Q-wave in the inferior lead. Therefore, the patient was managed conservatively using dual antiplatelet therapy, anticoagulant, dopamine, and epinephrine. In the 2015 AHA Update, intravenous infusion of chronotropic agents (dopamine or epinephrine) is recommended for symptomatic bradycardia or unstable bradycardia as an equally effective alternative to external pacing when atropine is ineffective.[14] In a study evaluating the feasibility of treatment which compared dopamine with transcutaneous pacing (TCP), there were not any differences between the treatment groups based on patient survival and length of stay.[5] The catecholamines that act on β1 receptor can be used as a treatment for bradycardia. A stimulation in β1 receptor can increase intracellular cAMP concentration which activates Calcium (Ca^{2+}) channels. This would lead to Ca^{2+}-mediated enhanced chronotropic responses and positive inotropy by increasing cardiac contractility through actin-myosin-troponin system. β1 receptor stimulation increases HR, cardiac contractility, AV node conduction, and automaticity of subsidiary pacemakers in complete AV blocks. Thus, this mechanism may be utilized in specific situations to maintain adequate cardiac output.[5,7] Dopamine is an endogenous catecholamine and also norepinephrine precursor. In a moderate dose of 3–10 μg/kg/minutes, dopamine acts on β1 receptor, releasing positive inotropic and chronotropic effects.[7] Clinical efficacy of dopamine was shown to be equivalent to TCP in a small RCT of patients with unstable bradycardia unresponsive to atropine in a prehospital setting.[15] Epinephrine, given at 2–10 μg/min, acts on α1, β1, and β2 receptors, yielding a greater effect than dopamine. Epinephrine is preferred to be administered in patients with hypotensive conditions. Both Dopamine and Epinephrine can be given as a monotherapy or in combination.[7] In this case, the patient was successfully treated primarily with both drugs as indicated by her ECG results in Figure 2 and echocardiography results in Figure 3 during the follow-up. There are several theorized mechanisms explaining the possible mechanisms associated with recovery of conduction in this patient, namely the vagal and ischemia hypothesis. Patients with coronary arterial disease are more susceptible to vagal stimulation. Decades before PCI was available, several publications reported the reversal of 2:1 AV block to 1:1 AV conduction with atropine administration. However, whether cardiac sensitivity to vagal stimuli after revascularization is able to fully reverse complete AV block is difficult to prove and remains to be a rare phenomenon. The findings in this case were more in line with the ischemia hypothesis, which explains that conduction defects which resolve with revascularization occur due to ischemia. This is consistent with complete restoration of 1:1 AV conduction found in this patient. Several cases of complete AV

block associated with IWMI are transient and would resolve spontaneously or with revascularization.[7] Other cases would absolutely require a permanent pacemaker implantation due to permanent damage to the AV conduction tissue due to lack of vascularization.[16]

Conclusion

Dopamine and epinephrine may be considered for complete AV block if transfer to a higher level of care is not feasible and as a bridge therapy while waiting for transfer.

Acknowledgements

The authors thank the medical record staff of *Kota Madiun* General Hospital.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

Ethical approval to report this case was obtained from the Ethical Committee of Kota Madiun General Hospital.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

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References

1. Ullah I, Ahmad F, Igbal MA, et al. Comparison of conduction abnormalities between anterior and inferior myocardial infarction. *Rawal Med J* 2005; 40: 48–51.
2. Ram R, Devi KB, Chanu KJ, et al. Study of conduction blocks in acute myocardial infarction. *J Med Soc* 2016; 30: 149.
3. Knabben V, Chhabra L and Slane M. *Third-degree atrioventricular block*. Treasure Island, FL: Statpearls Publishing, 2019.
4. Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhyth. *J Am Coll Cardiol* 2019; 74: e51–e156.
5. Neumar RW, Otto CW, Link MS, et al. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 2010; 122: S729–S767.
6. Bouhuijzen LJ and Stoel MG. Inferior acute myocardial infarction with anterior ST-segment elevations. *Neth Heart J* 2018; 26(10): 515–516.
7. Cardoso R, Alfonso CE and Coffey JO. Reversibility of high-grade atrioventricular block with revascularization in coronary artery disease without infarction: a literature review. *Case Rep Cardiol* 2016; 2016: 1971803.
8. Mehreen S, Ahmed A and Tahir M. Frequency of high degree AV blocks in acute inferior myocardial infarction and their impact on clinical outcomes. *Pakistan Armed Forces Med J* 2016; 66: 281–284.
9. Lim Y, Singh D and Poh KK. High-grade atrioventricular block. *Singapore Med J* 2018; 59: 346.
10. Damay V. Third-degree atrioventricular block in clinically "Silent" acute myocardial infarction. *Medicinus* 2018; 5: V5i3.
11. Aguiar Rosa S, Timóteo AT, Ferreira L, et al. Complete atrioventricular block in acute coronary syndrome: prevalence, characterisation and implication on outcome. *Eur Heart J Acute Cardiovasc Care* 2018; 7(3): 218–223.
12. Kant P, Singh PS, Kumar G, et al. A case of resolution of inferior wall myocardial infarction and varying degrees of atrioventricular block: a case report. *Int J Res Med Sci* 2015; 3: 2492.
13. Overgaard CB and Dzavík V. Inotropes and vasopressors: review of physiology and clinical use in cardiovascular disease. *Circulation* 2008; 118: 1047–1056.
14. Epstein AE, DiMarco JP, Ellenbogen KA, et al. 2012 ACCF/AHA/HRS focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American college of cardiology foundation/American heart association task force on practice guide. *Circulation* 2013; 127: e283–e352.
15. Morrison LJ, Long J, Vermeulen M, et al. A randomized controlled feasibility trial comparing safety and effectiveness of prehospital pacing versus conventional treatment: 'PrePACE.' *Resuscitation* 2008; 76: 341–349.
16. Gang UJO, Hvelplund A, Pedersen S, et al. High-degree atrioventricular block complicating ST-segment elevation myocardial infarction in the era of primary percutaneous coronary intervention. *Europace* 2012; 14: 1639–1645.