Clinical utility of percutaneous coronary intervention on left anterior descending stenosis in the setting of third-degree atrioventricular block due to inferior myocardial infarction: a case report

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Abstract

Acute myocardial infarction (MI), particularly inferior MI, may be complicated by the occurrence of third-degree atrioventricular (AV) block. This block is usually temporary, but in some cases it will require a permanent pacemaker (PPM). We report a case of inferior MI and primary percutaneous coronary intervention (PCI) of the right coronary artery (RCA). The third-degree AV block persisted as a result of the no-reflow phenomenon after PCI on the RCA, only to resolve after a second PCI on the left anterior descending (LAD). Improvement in the perfusion of the AV node via the RCA after PCI on the LAD may be able to explain this finding. This case suggests that complete revascularization should be achieved before deciding on the implantation of a PPM.

Keywords

ST-elevation MI • Third-degree atrioventricular block • Primary PCI • No-reflow phenomenon • AV-node physiology • Pacemaker • Case report

Introduction

Third-degree atrioventricular heart block (AVB) due to myocardial infarction (MI) occurs in about 10% of patients with inferior MI and 2.5% of those with anterior MI.1 Third-degree AVB is usually transient; however, when it persists beyond the usual hospital course, permanent pacemaker (PPM) implantation may be indicated.2 Primary percutaneous coronary intervention (PCI) has been more successful that thrombolysis in reducing the rate of AVB in acute MI.3

Learning points

• Atrioventricular (AV) block following inferior myocardial infarction (MI) may occur with delay, even after percutaneous coronary intervention (PCI).
• Improvement in AV nodal perfusion may be achieved after PCI on a stenotic left anterior descending artery (LAD).
• Complete revascularization, even in non-infarct-related arteries should be achieved before decision-making on possible permanent pacemaker (PPM) implantation.
Timeline

| Day | Hour | Event |
|-----|------|-------|
| 0   | 0:00 | Onset of severe chest pain |
| 0   | 8:00 | Arrival at the emergency department and inferior ST-elevation myocardial infarction (STEMI) diagnosis |
| 0   | 8:45 | Percutaneous coronary intervention (PCI) on the right coronary artery (RCA)/no-reflow phenomenon |
| 0   | 9:30 | Occurrence of third-degree atrioventricular heart block (AVB) and hypotension/temporary pacemaker (TPM) insertion |
| 5   | 0:00 | Percutaneous coronary intervention (PCI) on the left anterior descending (LAD) |
| 5   | 0:30 | Onset of intermittent atrioventricular (AV) conduction |
| 5   | 4:00 | Achievement of complete atrioventricular (AV) conduction |

Case

A 73-year-old man with a history of essential hypertension was admitted to the emergency ward of Tehran Heart Center. He complained of severe squeezing chest pain, which had started 8 hours before arrival and was concomitant with diaphoresis and nausea. He smoked 55 pack/year and occasionally abused dried opium latex orally. Physical examination showed a pulse rate of 76 beats/min, blood pressure of 160/90 mmHg, respiratory rate of 20/min, and 96% O₂ saturation. Additionally, S₄ and a systolic murmur (grade II/VI) with maximum intensity in the apical site were audible in auscultation. Other systemic findings were unremarkable. His current medications included losartan (25 mg/BD). Initially, a 12-lead electrocardiogram (ECG) recording was obtained, and it showed ST-elevation in the inferior leads as well as reciprocal ST-depressions in the precordial leads without any AVB (Figure 1A). Additional ECG recording demonstrated further ST-elevation in the right-sided leads. Blood sampling was performed to measure serum troponin levels, although for the next steps we did not wait for the results of the tests (the first troponin-T level was reported to be 997 ng/L with the upper normal limit <24 ng/L).

Based on the diagnosis of inferior (i.e. right ventricular) MI, 325 mg of aspirin (ASA), 600 mg of clopidogrel, and 80 mg of atorvastatin were administered and the patient was transferred to the catheterization laboratory for primary PCI (door-to-device time = 45 min). Bedside echocardiography showed an ejection fraction of 35%. Coronary angiography was performed via the right femoral artery. The left anterior descending (LAD) had 90% stenosis in the proximal portion [thrombolysis in myocardial infarction (TIMI) = 3]. The left circumflex artery had no significant stenosis (Figure 2A). The right coronary artery (RCA) was cut-off at the proximal portion with a high

![Figure 1](initial inferior ST-elevation myocardial infarction (A). Complete atrioventricular block development and atrioventricular dissociation after percutaneous coronary intervention of right coronary artery (B). Electrocardiography after temporary pacemaker implantation and paced rhythm (C). Complete resolution of atrioventricular block (D).)
thrombotic burden (Figure 2B). Subsequently, 10 000 units of heparin and 12 mL of epifibatide were infused and a primary PCI for the RCA lesion was performed via two overlapping drug-eluting stents (XIENCE 3 × 18 and Orsiro 3 × 33). Unfortunately, the patient developed the no-reflow phenomenon and exhibited a TIMI of 2 (TIMI frame count = 48) (Figure 2C). Two doses of adenosine (400 μg) were injected intracoronary but all to no avail.

The patient was transferred to the post-catheterization laboratory unit. By this point, he had become lethargic and hypotensive. New physical examination showed bradycardia and a third-degree AVB in ECG (Figure 1B). Consequently, a temporary pacemaker (TPM) was implanted (Figure 1C).

Despite more than 5 days of follow-up, the patient remained pacemaker-dependent. Moreover, his TPM site was unstable, and he became symptomatic during episodes of TPM non-capture. He was, therefore, transferred to the catheterization laboratory for PCI on the LAD lesion and TPM repositioning (or PPM implantation, as needed). The second PCI was performed on the LAD (Ultimaster 3.5 × 12) with excellent results (Figure 2D). Surprisingly, intermittent conduction via the atrioventricular (AV) node was restored within 30 min of PCI on the LAD. Accordingly, PPM implantation was postponed and 4 hour follow-up of the patient showed that the AV node had recovered to complete conduction (Figure 1D). Three days later, the patient was discharged in an acceptable general condition. Echocardiography at discharge showed an ejection fraction of 40%. Now, after 4 months, he is well and receives ASA (80 mg/D), clopidogrel (75 mg/D), pantoprazol (40 mg/D), atorvastatin (80 mg/D), enalapril (5 mg/D), and bisoprolol (2.5 mg/D). He had no new episodes of hospitalization or chest pain.

**Discussion**

Previous reports have predominantly described third-degree AVB in the setting of inferior MI or even severe RCA stenosis without MI. Cases of third-degree AVB secondary to anterior MI have also been described, albeit with different prognoses. Additionally, there have been reports concerning the occurrence of third-degree AVB due to septal occlusion (i.e., jailed septal perforators) as a complication of PCI on the LAD.

We decided to report this case, given the unique course of events leading to the patient’s recovery. In our patient, third-degree AVB was initially triggered by an inferior MI and the associated no-reflow phenomenon in the RCA, but it was later resolved subsequent to PCI on the LAD. Although the dual blood supply of the AV node would probably justify this observation, this has been described mainly via the left circumflex artery and the RCA. We believe that the most likely explanation for our case is that the restoration of the LAD blood flow improves AV nodal perfusion via micro-channels of septal anastomosis from the LAD towards the posterior descending artery (PDA), which in turn serves to fill up the PDA and the posterior left ventricular branch retrogradely and consequently improves the perfusion of the AV nodal branch artery (for instance, consider a patient with a totally cut-off RCA and retrograde filling via the LAD). The chronic presence of significant LAD stenosis can lead to the development of septal collaterals between the LAD and the PDA and supports this hypothesis. This case highlights the significance of distant ischaemia and complete revascularization prior to decision-making on PPM implantation. It remains to be seen whether PCI on a significantly stenosed RCA would confer a comparable dramatic clinical improvement, as was the case in our patient, in patients with third-degree AVB due to anterior MI or in patients complicated by third-degree AVB during elective PCI on the LAD. At this point, further reports are warranted for a more comprehensive picture of this case and patients affected by similar clinical scenarios.

**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** none declared.

**Author Contributions:** B.G. was responsible for performing PCI and the medical management of the patient; he was also responsible for the critical revision of the manuscript. M.S. was responsible for gathering data, drafting, and critically revising the manuscript; he also contributed to the medical management of the patient. M.A. was responsible for drafting and critically revising the manuscript.

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