



## Reversible cardiogenic shock caused by atrioventricular junctional rhythm after percutaneous coronary intervention

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### Abstract

An 82-year-old female patient undergoing cardiogenic shock caused by atrioventricular junctional rhythm immediately after percutaneous coronary intervention (PCI) is described. Pharmacotherapy was invalid, and subsequent application of atrial pacing reversed the cardiogenic shock. PCI-related injury of sinuatrial nodal artery leading to acute atrial contractility loss, accompanied by atrioventricular junctional arrhythmia, was diagnosed. We recommend that preoperative risk evaluation be required for multi-risk patients. Likewise, emergent measures should be established in advance. This case reminds us that atrial pacing can be an optimal management technique once cardiogenic shock has occurred.

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**Keywords:** Cardiogenic shock; Atrioventricular junctional rhythm; Percutaneous coronary intervention; Atrial pacing

### 1 Introduction

The case described herein of cardiogenic shock (CS) in an elderly woman caused by atrioventricular (AV) junctional rhythm after percutaneous coronary intervention (PCI) and its reversal by atrial pacing suggests that adequate compensation of atrial contractility can be vital to hemodynamics. CS during an intervention process, especially in arrhythmia-induced cases, is rare. By reviewing the related literature, we have identified several potential risk predictor variables for CS before intervention performance, including age, heart rate, Killip class, previous acute myocardial infarction (AMI), and sex. In addition, PCI-related injury and myocardial reperfusion injury may be warning signs during the procedure.

### 2 Case Report

An 82-year-old woman with a history of previous extensive anterior myocardial infarction, hypertension, and type 2 diabetes was admitted with a complaint of intermittent, squeezing chest pain over the previous six months. Physical

examination was unremarkable, except for slight, moist rales in the lower lung fields. A 12-lead electrocardiogram showed sinus rhythm with flat or upside down T wave in leads I, V4, V5 and V6 (Figure 1A–D). A Doppler ultrasonic cardiogram (UCG) revealed regional wall motion abnormalities of the left ventricle and borderline ejection fraction.

Coronary angiography revealed complete occlusion in the ostium of the left anterior descending artery (LAD) and subtotal occlusion in the proximal segment of the left circumflex artery (LCX). The thick intermediate branch showed 90% stenosis in the ostium and 50% stenosis in the middle segment (Figure 2A). There was 90% stenosis in the initial segment of the posterior descending artery (PDA) (Figure 2B). LAD and LCX were identified as culprit lesions. After successive predilation, a stent (2.5 × 13 mm, Firebird; MicroPort Co., China) was deployed in the proximal LCX and another (3.5 × 23 mm, Firebird) in the proximal intermediate branch (Figure 2C).

When stenting was completed, the patient's blood pressure (BP) began to decrease progressively, finally reaching 80/55 mmHg. The patient's symptoms (repeated distress, shortness of breath, and dizziness) and signs (altered mental status, oliguria, and cool extremities) indicated insufficient peripheral perfusion. At this time, the sinus rhythm became an AV junctional rhythm, with 45–70 beats per minute (bpm) (Figure 1E). Despite prompt administration of a dopamine intravenous infusion and an atropine bolus, the patient's arterial pressure remained at the level of 90/60 mmHg, with her heart rate

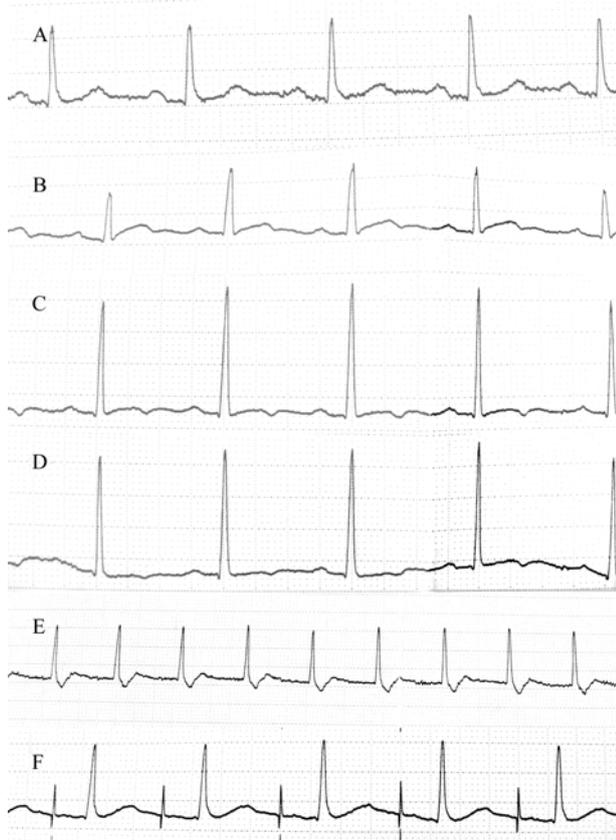
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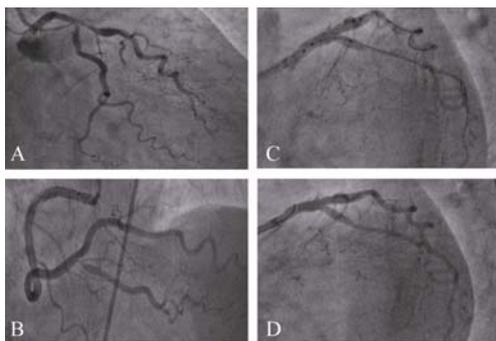
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fluctuating between 60–80 beats/min without rhythm conversion. The technique of crush stenting with kissing balloon post-dilation was carried out (Figure 2C & 2D).



**Figure 1. Electrocardiographic images.** (A–D): Sinus rhythm with flat or upside down T wave leads I, V4, V5 and V6; (E): Atrioventricular junctional rhythm after stenting; (F): Atrial pacing rhythm after pacemaker implantation.



**Figure 2. Angiographic images.** (A): Complete occlusion in the ostium of left anterior descending artery, subtotal occlusion in the proximal left circumflex artery, severe stenosis in the ostium, and moderate stenosis in the middle segment of intermediate branch; (B): Severe stenosis in the initial posterior descending artery; (C): Balloons post-dilation by kissing crush; (D): Post-procedural coronary flow.

Unfortunately, all management efforts failed. Through discussion, the treating physicians reached consensus that atrial systolic function loss spurred by AV rhythm had led to ventricular diastolic volume reduction accounted for the CS. It was determined that rapid restoration of compensation from atrial systole to ventricular volume was the optimal treating procedure. Subsequently, the patient was implanted with a permanent cardiac pacemaker (SS303, AAI Mode; Medtronic, USA). As atrial pacing rhythm became established (>75 beats/min; Figure 1F), the BP increased gradually. The patient proclaimed a remarkable relief in her self-perceived physical status. Two weeks later, the patient was discharged. At six months' follow-up, the patient was in good condition.

### 3 Discussion

CS is a state of impaired end-organ perfusion that results from reduced cardiac output and is characterized by hypotension and impaired tissue perfusion. Very few reports of CS during intervention process are present in the publicly available literature. The case described herein, as an arrhythmia-induced CS that was reversed by atrial pacing, is even more rare.

A predictor scale for CS was developed from the results of the GUSTO-I trial,<sup>[1]</sup> which determined that some of the predictor variables for the development of CS were age, heart rate, Killip class, anterior location of AMI, previous AMI, and sex.<sup>[1,2]</sup> A subsequent study indicated older age and female sex was associated with reduced cardiac power.<sup>[3]</sup> Elderly patients commonly exhibit severe stenosis or massive calcification, both of which impair dilatation or recanalisation and decrease the chances of successfully achieving total revascularization. In the current case report, the woman presented with several risk factors, including previous anterior myocardial infarction, diabetes, hypertension, and heart failure. As such, she was considered high-risk for morbidity. After considering the patient's angiography findings, the treating physicians presented her family with the possibility that coronary artery bypass grafting (CABG) may be the best treatment option. Given the patient's older age and poor systemic condition, however, the family preferred the intervention approach to the CABG surgery.

Generally, compensation of atrial systole is not critical for ventricular filling. Once the atrial contraction of the cardiac cycle is lost, the underlying deteriorated left ventricular systolic function could manifest as remarkably low output. For our patient, the primary reason for cardiac insufficiency was believed to be the loss of atrial auxiliary filling to ventricular diastolic volume that resulted from AV rhythm.

Asakura *et al.*<sup>[4]</sup> previously reported a similar case of severe hypotension during the development of junctional rhythm after anesthesia and suggested that adequate compensation of atrial contractility may be vital to hemodynamics in some cases. Meanwhile, the intra-aortic balloon pump must be used to correct the low cardiac output after PCI, and is frequently applied in cases of coronary defective perfusion. The phenomenon observed in our case where sinus rhythm deletion stimulated simultaneous shock indicated that it was directly concerned with arrhythmia.

Nevertheless, the use of antiarrhythmic drugs is limited by severe hemodynamic impairment, making the arrhythmia unresponsive to these medications. Wong *et al.*<sup>[5]</sup> reported on cardiogenic shock caused by severe coronary spasm immediately after coronary stenting. The authors suggested that although repeated review of angiography findings did not reveal any change, the PCI-related injury of sinoatrial nodal artery (occlusion or stenosis) may have been the major cause of absence of sinus rhythm. In fact, temporary pacing would be effective and economic but insertion and immobilization of the temporary lead in the right atrium is technically difficult to accomplish. Undoubtedly, the embarrassment must delay the critical rescue phase. In addition, elderly patients may have a generally impaired conduction system. For our case, a permanent atrial pacemaker was implanted which promoted atrial contraction, restored atrioventricular rhythm, and facilitated supplementary ventricular filling for sufficient cardiac ejection. The ultimate success of reversing the persistent condition of hypotension verified the originally speculative diagnosis.

Low output becomes enhanced upon PCI-related injury and leads to myonecrosis associated with either stent-related side-branch flow impairment/occlusion or atherosclerotic debris which plugs the downstream coronary microcirculation.<sup>[6]</sup> Thus, the suppressed myocardium itself (indicated by UCG) may account for the transient abnormal myocardial mechanical function. Administration of an inotropic agent and vasoconstrictor are expected to be of therapeutic benefit. However, in our case, large-dose dopamine failed to preserve the patient's BP and additive dobutamine was ineffective. Dopamine is known to increase heart rate or trigger arrhythmias, while dobutamine is also known to produce vasodilation and hypotension.<sup>[7]</sup> We considered noradrenaline as an alternative therapy, but rejected it due to the fact that it may

amplify myocardial oxygen consumption elicited by catecholamine (which was formerly administered) and exacerbate peripheral perfusion due to the patient's comprehensive atherosclerosis. All these elements are capable of aggravating left ventricular contractility, and as a result, we finally decided to perform the atrial pacing technique that provided the eventual resolution of the patient's symptoms.

For multi-risk patients with coronary disease, especially those of advanced age, the complication incidence increases significantly after interventional treatment, and preoperative risk evaluation is necessary for effective treatment and good outcome. The current case serves as a reminder of the possibility of AV junctional arrhythmia inducing acute atrial contractility loss and highlights that once cardiogenic shock has occurred, subsequent atrial pacing can be an optimal management approach.

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