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## Case Report

# Complete atrioventricular block improved by balloon aortic valvuloplasty for severe aortic stenosis: Usefulness of sheathless technique in the retrograde approach



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#### ABSTRACT

An 86-year-old man was admitted to our hospital for treatment of congestive heart failure with severe aortic stenosis and advanced atrioventricular (AV) block. Despite pharmacological therapy, he developed complete AV block and resultant acute pulmonary edema requiring temporary pacing and tracheal intubation. We urgently performed retrograde balloon aortic valvuloplasty (BAV) with a sheathless technique. The AV block disappeared soon after the procedure; this was probably attributable to the correction of relative myocardial ischemia, obtained by BAV. Thus, he successfully recovered from a critical condition.

<Learning objective: It is widely accepted that balloon aortic valvuloplasty (BAV) dramatically reduces the systolic pressure gradient across the stenotic aortic valve, but the other important hemodynamic point is that it increases the coronary perfusion pressure by raising the diastolic blood pressure, both of which contribute to improving the imbalance between myocardial oxygen demand and supply. And the sheathless technique in retrograde BAV, although not yet popular, is less invasive and may be promising in urgent clinical situations.>

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### Introduction

Balloon aortic valvuloplasty (BAV) for severe aortic stenosis (AS) was recently recognized once again with the advent of transcatheter aortic valve implantation (TAVI) [1]. Atrioventricular (AV) block is a known complication of such interventions targeting the aortic valve [2,3]. In contrast, we herein report a case involving a patient with severe AS in whom BAV improved a pre-existing complete AV block, probably due to correction of relative myocardial ischemia. We also demonstrate the usefulness of the sheathless BAV catheter insertion technique in the retrograde approach, which seems to be less invasive than the conventional approach using a large sheath and is expected to reduce vascular complications.

#### **Case report**

An 86-year-old man with hypertension and a history of heavy smoking was admitted to our hospital with the chief complaints of

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worsening leg edema and dyspnea on effort. His vital signs revealed irregular bradycardia (43 beats/min), high blood pressure (175/64 mmHg), and a low pulse oximetry reading (90% on oxygen at 7 L/min). On auscultation, we detected a small clicking sound in the bilateral lower lungs and a Levine grade III/VI ejection systolic murmur best heard at the apex. Pitting edema was observed in both lower legs.

Twelve-lead electrocardiography demonstrated an advanced AV block with QS pattern and nonspecific T-wave changes in precordial leads V1–V4 (Fig. 1A). A chest radiograph revealed a minor degree of cardiomegaly, pulmonary congestion, and pleural effusion. Blood examination showed a high brain natriuretic peptide level, mild kidney damage, and negativity for troponin I. Transthoracic echocardiography revealed that the left ventricular (LV) ejection fraction was 58%. The aortic valve was tricuspid in nature with mild calcification that apparently limited its opening, and the maximum velocity and mean systolic pressure gradient (PG) across the aortic valve were 5.32 m/s and 56 mmHg, respectively. Then the aortic valve area calculated by the continuity equation was 0.75 cm², resulting in a definitive diagnosis of severe AS. Moderate aortic regurgitation was also present.

We confirmed that no drugs he had been taking were suspected of causing bradycardia. Pharmacological therapy with intravenous administration of furosemide and atrial natriuretic peptide or a

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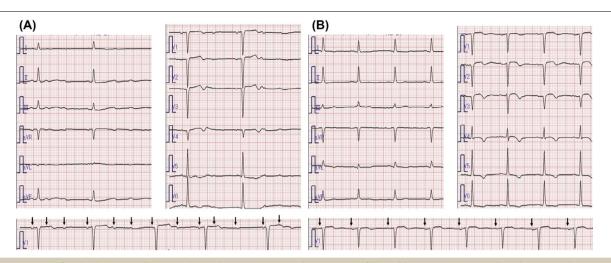


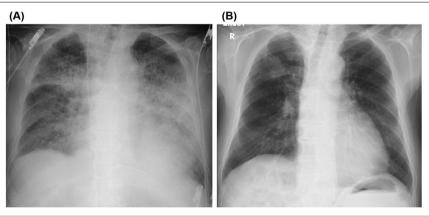
Fig. 1. Time course of 12-lead electrocardiogram. (A) On admission, arrows indicate P waves followed by intermittently conducted QRS complexes, representing advanced atrioventricular block. QS pattern and nonspecific T-wave changes were also present in precordial leads V1–V4. (B) After balloon aortic valvuloplasty (on day 15), sinus rhythm was steadily maintained. Furthermore, QS pattern in V4 disappeared and terminal T-wave inversion became evident in V2–V5.

small dose of dobutamine was minimally effective. The preexisting advanced AV block progressed to a complete AV block on day 6, and emergent temporary pacing was thus introduced. Because a chest radiograph revealed bilateral pulmonary congestion (Fig. 2A) and the patient's respiratory condition was exacerbated, we performed tracheal intubation and introduced mechanical ventilation on day 7. Persistent low-grade fever and inflammatory reaction shown by blood examination made us conceive some concomitant pulmonary infection or noninfectious lung injury, so we initiated antibiotics and steroid mini-pulse therapy as well. Unfortunately, these treatments failed to improve his critical condition. We then concluded that BAV would be the only possible intervention to bail him out of this crisis, and he underwent urgent retrograde BAV on day 9.

In the catheterization room, we manipulated the temporary pacemaker to lower the pacing rate from 70 to 30 paced pulses per minute (ppm), but his own beats were infrequently observed. Emergency treatment, including percutaneous cardiopulmonary support, was prepared in case of possible severe complications. We performed transesophageal echocardiography to reassess and precisely measure the aortic valve and its surrounding structures. Then we inserted a 6-Fr sheath into the right femoral artery for retrograde BAV. The simultaneously measured peak-to-peak PG between the left ventricle and ascending aorta was 30 mmHg (Fig. 3A). A 0.035-in. Amplatz Extra Stiff guide wire (Cook Medical,

Tokyo, Japan) was positioned appropriately at the LV apex. We removed the primary 6-Fr sheath over the stiff guide wire and exchanged it with a BAV catheter (Z-MED II,  $20~\text{mm} \times 40~\text{mm}$ ; NuMED, Inc., Denton, TX, USA) by means of the sheathless technique. Shortly after beginning ventricular burst pacing at 180 ppm, we strongly inflated the balloon in the aortic valve for about 2 s (Fig. 3C) and then immediately deflated it. A second ballooning was performed in the same manner. We exchanged the BAV catheter for a 7-Fr sheath. Comparison of the pre- and post-BAV hemodynamic status revealed that the peak-to-peak PG was clearly decreased from 30 to 10 mmHg (Fig. 3B). No evidence of aortic regurgitation exacerbation or other fatal complications were detected. The 7-Fr sheath was removed with the aid of an 8-Fr Angio-Seal STS Plus (St. Jude Medical, Minnetonka, MN, USA) with no vascular complications.

About 3 h after BAV, electrocardiography revealed that the patient's own P waves followed by conducted QRS complexes were emerging under the backup pacing. Furthermore, sinus rhythm without AV block was maintained subsequently, QS pattern in V4 disappeared, and terminal T-wave inversion became evident in V2–V5 (Fig. 1B). His respiratory condition improved after BAV, and he was successfully extubated on day 12. Coronary angiography on day 30 demonstrated hypoplasty of the right coronary artery, from which the nonstenotic but very thin branch supplying the AV node was derived, and significant atherosclerotic stenosis at segments



Time course of chest radiograph. (A) Before BAV (on day 7), bilateral pulmonary congestion was present. (B) After BAV (on day 38), the lung fields became clear. BAV, balloon aortic valvuloplasty.

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