Combined Therapy with Percutaneous Coronary Intervention and Percutaneous Aortic Valvuloplasty under Mechanical Support for an Elderly Patient with Cardiogenic Shock

Koji Kato¹, Naoki Sato¹, Nobuhiko Fujita¹, Takeshi Yamamoto¹, Yu-ki Iwasaki¹, Kenji Yodogawa¹, Morimasa Takayama², Keiji Tanaka¹ and Teruo Takano²

¹Intensive and Cardiac Care Unit, Department of Internal Medicine, Nippon Medical School
²Department of Internal Medicine, Nippon Medical School

Abstract

Percutaneous aortic valvuloplasty is reportedly a useful tool for the management of critical and severe aortic stenosis with cardiogenic shock¹. However, early percutaneous coronary intervention for cardiogenic shock is beneficial for elderly patients with acute myocardial infarction²,³. We describe a patient with critical aortic stenosis who presented with severe coronary stenosis of the left main trunk and the ostium of the right coronary artery. We performed percutaneous coronary intervention and percutaneous aortic valvuloplasty under intra-aortic balloon pump and percutaneous cardiopulmonary support. After these procedures, the cardiogenic shock was reversed, and the patient could be weaned from both intra-aortic balloon pump support and percutaneous cardiopulmonary support.

Key words: cardiogenic shock, coronary intervention, aortic valvuloplasty, intra-aortic balloon pump, percutaneous cardiopulmonary support

Case Report

An 84-year-old woman with cardiogenic shock was admitted to Nippon Medical School Hospital. The patient had had dyspnea and chest discomfort on exertion for 3 years, and the symptoms had worsened in the past 2 months. She was diagnosed with diabetes mellitus and hyperlipidemia at the age of 60 years and was given medication by her general practitioner.

On admission, the consciousness was clear, but blood pressure was 80/40 mmHg. A systolic murmur was heard over the third intercostal space on the right side of the sternum (Levine III/VI), and diffuse coarse crackles were audible. Electrocardiography showed a complete right bundle-branch block; ST elevation in leads V1~V2 and aVR; and ST depression in leads I, II, III, aVF, and V3~V6 (Fig. 1).

A chest X-ray film revealed cardiomegaly with severe pulmonary congestion and pleural effusion (Fig. 2). Echocardiography showed severe diffuse hypokinesis of the left ventricle, a low ejection fraction, and a low cardiac output.

Correspondence to Koji Kato, Intensive and Cardiac Care Unit, Department of Internal Medicine, Nippon Medical School, 1-1-5 Sendagi, Bunkyo-ku, Tokyo 113-8603, Japan
E-mail: katokoji@nms.ac.jp
Journal Website (http://www.nms.ac.jp/jnms/)
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**Fig. 1** Electrocardiogram

On admission:
Complete right bundle branch block, ST elevation in leads V1 ~ V2 and aVR, and ST depression in leads I, II, III, aVF, and V3 ~ V6.

After PCI:
Narrow QRS, QS pattern with ST elevation in V1 ~ V3 and inverted T wave in I, aVL, V4 ~ V6.

**Fig. 2** Chest X-ray showed cardiomegaly with pulmonary congestion (cardiothoracic ratio, 66.7%).

Fraction of 21.7%, and aortic stenosis with a transvalvular pressure gradient of 48 mmHg. Laboratory investigation revealed anemia (red blood cells: $285 \times 10^3/ µl$, hemoglobin: 7.3 g/dl), leukocytosis (white blood cells: $147 \times 10^3/ µl$), and normal levels of creatine kinase (CK) and the MB isoenzyme of CK (CK-MB). However, rapid test for cardiac troponin T and human heart-type acid-binding protein were positive. Renal function and electrolyte were normal (creatinine, 0.90 mg/dl; Na, 144 mEq/l; K, 3.9 mEq/l; Cl, 110 mEq/l). Arterial blood gas analysis under oxygen administration at 10 L/min through a reservoir mask showed that the partial pressures of oxygen and carbon dioxide were 63.4 and 32.7 torr, respectively. Soon after admission, the patient became drowsy owing to cardiogenic shock by severe ischemia and aortic stenosis. The blood pressure at that time was 64/48 mmHg. We decided to perform coronary angiography emergently. We introduced intra-aortic balloon pump (IABP) support immediately and performed coronary angiography, which revealed 90% stenosis of the left main trunk (LMT) and the ostium of the right coronary artery (RCA) (**Fig. 3A, 3B**).

In spite of the IABP support, the hemodynamics remained unstable; therefore, we initiated percutaneous cardiopulmonary support (PCPS) and artificial respiration. Under both IABP support and PCPS, we performed percutaneous coronary intervention (PCI) with stenting of the lesions of the LMT and the ostium of the RCA. A 6-F JL guide
Fig. 3 Coronary angiogram before PCI revealed that 90% stenosis of the RCA ostium (panel A), and 90% stenosis of the LMT (panel B); PCI was successfully performed for both lesions (panel C and D).

catheter (Bright-tip, Johnson and Johnson, Miami, USA) was used to engage the left coronary artery. The LMT lesion was crossed with a 0.014-inch guide wire (Run-through, Terumo, Tokyo, Japan), predilatation was performed with a balloon (Sprinter, 2.5/20 mm, Medtronic, Minneapolis, MN, USA), and then a stent (Driver, 4.0/12 mm, Medtronic) was implanted. For the lesion in the RCA, a 6-F JR4 guide catheter (Bright-tip, Johnson and Johnson, Miami, USA) and the previously mentioned wire were used. Predilatation was also performed with the same balloon. The Express II stent (3.0/12 mm, Boston Scimed, Massachusetts, USA) was successfully implanted. Finally, good coronary flow was obtained in both coronary arteries (Fig. 3C, 3D).

Complete right bundle-branch block resolved after PCI, and a QS pattern with ST elevation in V1~V3 was observed. (Fig. 1) Maximum CK and CK-MB levels were 2,431 mg/dl and 189 mg/dl, respectively, 16 hours after admission, Anteroseptal acute myocardial infarction (AMI) was diagnosed and was responsible, in part, for the cardiogenic shock.

On the second hospital day, our attempts to wean the patient from PCPS failed because of an uncontrollable lowering of blood pressure when the PCPS flow was reduced. Echocardiography revealed an improvement in ejection fraction to 28.5% and a concomitant increase in the aortic transvalvular pressure gradient from 48 mmHg to 62 mmHg. We then decided that treatment for severe aortic stenosis was necessary to treat the cardiac failure; therefore, valvuloplasty was planned.

We placed 6-F pigtail catheters in both the ascending aorta and the left ventricle for the measurement of the transvalvular pressure gradient, which was found to be 44 mmHg (Fig. 4C). An exchange-length J wire was placed in the left ventricle, and the pigtail catheter was exchanged for a 15 mm Cribier Letac balloon (Boston, Massachusetts, USA), which was positioned across the aortic valve and inflated twice (Fig. 4A, 4B). Simultaneous measurements in the left ventricle and
Fig. 4 Procedure of aortic balloon valvuloplasty was shown. Partial inflation reveals a waist as the stenotic aortic valve indents the dilated balloon (A), and then the balloon was fully inflated (B). Simultaneous pressure records in the ascending aorta and left ventricle before (C) and after (D) aortic valvuloplasty are shown. Before balloon valvuloplasty, blood pressure was 84/60 mmHg and aortic valve gradient was 44 mmHg (C), after balloon valvuloplasty, blood pressure was 100/80 mmHg, which was clearly increased, and aortic valve gradient was decreased by 22 mmHg (D).

the ascending aorta showed that the mean transvalvular pressure gradient after valvuloplasty was 22 mmHg with significant improvement in the systolic blood pressure (Fig. 4D). Hemodynamic measurement under PCPS at a flow rate of 0.5 L/min revealed that the cardiac output had increased from 2.3 to 4.0 L/min and that the aortic valve area, which was calculated with Gorlin’s formula, had also increased from 0.86 to 1.26 cm². Finally, the patient could be weaned from PCPS on the following day, and the IABP support was removed 4 days later. Although the patient’s general condition gradually improved, she died on the 14th hospital day because of unexpected arrhythmia. We could not determine the cause of the arrhythmia, because significant ST-T change, abnormal electrolyte levels, and congestion on X-ray films were not observed until just before the arrhythmia occurred. Although the pressure gradient was slightly increased compared with that just after weaning from PCPS (23 to 36 mmHg) associated with improved left ventricular function by echocardiography, we could not determine the cause of death, because informed consent for autopsy could not be obtained.

Discussion

This patient was admitted to our hospital because of cardiogenic shock and pulmonary edema and was diagnosed with AMI accompanied by severe aortic stenosis. We performed PCI for stenosis of the LMT and the ostium of the RCA under IABP support and PCPS. Although revascularization was performed successfully, the patient could not be weaned from PCPS because of severe aortic stenosis. Subsequently, we performed aortic valvuloplasty, after which the patient could be weaned from both PCPS and IABP support.
The incidence of aortic stenosis in the elderly population is 2% to 9%, whereas aortic valve sclerosis is reportedly more common and is observed in 29% of persons older than 65 years. The most common cause of AS is age-related progression of calcification in the valve cusps. Aortic valve sclerosis is associated with atherosclerotic risk factors, e.g., aging, smoking, male sex, hypertension, and elevated serum levels of low density lipoprotein cholesterol. The combination of AS or aortic valve sclerosis and coronary artery disease in the elderly is expected to occur more frequently in the near future. Furthermore, population-based studies have identified aortic valve sclerosis as an independent predictor of cardiovascular events. Therefore, the management of patients with a condition similar to that described in the present case will be of paramount importance.

According to the American Heart Association/American College of Cardiology guidelines published in 1998 for the management of aortic stenosis, patients with severe aortic stenosis and refractory lung edema or cardiogenic shock may benefit from aortic valvuloplasty as a bridge to valve replacement (Class IIa), but as a Class IIb indication, it is palliative treatment for patients with severe comorbid conditions. However, Agarwal et al. have recently reported that repeat aortic valvuloplasty, which many be one of the strategies for elderly patients who are not candidates for surgery, improves the survival rate as compared with single valvuloplasty. On the other hand, early PCI for cardiogenic shock may be beneficial for elderly patients with AMI; the 30-day mortality rate is less than 50%.

Several reports suggest that in spite of AMI, aortic valvuloplasty and PCI may be safely combined for the treatment of patients not undergoing surgery. McKay et al. have reported that when eight of nine symptomatic patients (average age, 76 years) with severe aortic stenosis, angina, and congestive heart failure were treated with the combined procedures, i.e., aortic valvuloplasty and coronary angioplasty, symptoms improved and discharge was possible.

In conclusion, the present case report demonstrates that elderly patients with AMI and severe aortic stenosis recovered from cardiogenic shock when given combined treatment with PCI and aortic valvuloplasty under mechanical support. In elderly patients not undergoing surgery, a combination of these procedures may be necessary, particularly for critically ill patients with cardiogenic shock caused by severe aortic stenosis and coronary heart disease similar to that in the present case.

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