Worsening of Mitral Regurgitation by Balloon Aortic Valvuloplasty for Severe Aortic Stenosis
A Case Report

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Summary
A 91-year-old woman was admitted to our hospital for treatment of congestive heart failure with severe aortic stenosis. After admission, she developed a high fever due to pneumonia and worsened heart failure. We could not perform transcatheter aortic valve implantation (TAVI) because of active infection; therefore, retrograde balloon aortic valvuloplasty (BAV) was urgently performed. A complete atrioventricular block and severe functional mitral regurgitation appeared suddenly after BAV in the absence of mechanical disorders. Her condition improved after several days in our intensive care unit. Pacemaker implantation and TAVI were then performed, and the patient was discharged from our hospital. MR could sometimes exacerbate after BAV in clinical practice; therefore, we set out to report this case.

Key words: Transcatheter aortic valve implantation, Complete atrioventricular block

The use of balloon aortic valvuloplasty (BAV) and transcatheter aortic valve implantation (TAVI) is increasing in Japan. BAV reduces mitral regurgitation (MR) in many cases because of a decrease in afterload that had been elevated by aortic stenosis (AS). Worsening MR is not often mentioned as a serious complication in case reports. However, we experienced a case involving rapid worsening of MR after BAV. We herein describe a patient with severe AS who developed complete atrioventricular (AV) block and worsened MR after BAV.

Case Report
A 91-year-old woman with exertional dyspnea was diagnosed with severe AS. An electrocardiogram showed sinus rhythm with no conduction disturbances. A transthoracic echocardiography showed a severely calcified tricuspid aortic valve with a peak/mean transvalvular pressure gradient of 91/61 mmHg. The left ventricular ejection fraction (LVEF) was mildly reduced (49%) with no regional asynergy, and mild aortic regurgitation (AR) and MR were observed. The right ventricular systolic pressure was 49 mmHg. The left atrial size was 47 mm, and the left atrial volume index was 57 mL/m². Coronary angiography showed no organic stenosis; therefore, we considered that the left ventricular systolic dysfunction was mainly caused by the AS-induced high afterload. The Society of Thoracic Surgeons risk score was 9.69%. The patient agreed to undergo TAVI based on our recommendation. However, 7 days after admission, she developed a fever due to pneumonia and her congestive heart failure worsened. Therefore, we planned a multistage procedure involving BAV to stabilize her condition, followed by TAVI when her condition had stabilized. Transthoracic echocardiography at the time of BAV showed that the LVEF had worsened from 49% to 35% with no regional asynergy. Electrocardiography showed no abnormalities, and no increases in serum biomarkers of myocardial necrosis were observed; therefore, we considered that systemic inflammation and hemodynamic change had exacerbated the left ventricular systolic function. At this point, the MR remained mild. Right heart catheterization showed a mean pulmonary capillary wedge pressure (PCWP) of 19 mmHg. The cardiac index, determined by thermal dilution, was 2.2 L/minute/m². An increase in the PCWP indicated worsening congestive heart failure due to the infection. Simultaneous pressure tracings demonstrated a mean pressure gradient of 37 mmHg across the aortic valve, and the aortic valve area was 0.5 cm² as calculated by the Gorlin formula. Complete AV block appeared when an extra stiff guidewire was introduced into the left ventricle. Transthoracic echocardiography showed that the MR had become moderate. There was no sign of organic dysfunction such as rupture of the chordae tendineae. Wire interaction with the chordae was observed; therefore, the wire was exchanged in the left ventricle. However, the degree of MR did not change. The MR jet blew from the center and the coaptation was shallow; therefore, we determined...
that the etiology of the MR was a hemodynamic disorder (functional MR). We continued the BAV procedure under pacing from a temporary pacemaker. A 12-mm/18-mm balloon catheter was used (Z-MED/Tyshak; B. Braun Interventional Systems) and BAV was performed under rapid pacing (Pacing rate was 180 beats per minute). Transthoracic echocardiography showed that LVEF did not change. The AR had increased from mild to moderate, and the MR had become severe. That is, MR got worse in two phases: just after complete AV block occurred and after balloon inflation under rapid pacing. After BAV, simultaneous pressure tracings showed a mean pressure gradient of 30 mmHg, and the aortic valve area was 0.7 cm². Right heart catheterization after BAV showed a mean PCWP of 25 mmHg and a high V wave in the PCWP recording (Figure 1). The cardiac index, determined by thermal dilution, was 1.8 L/minute/m². A temporary pacemaker was placed in the right ventricle because complete AV block was still present. The patient was transferred to our intensive care unit, where transthoracic echocardiography still showed severe MR and moderate AR (Figure 2 A). Low cardiac output was suspected to be the cause of the patient’s cold skin and oliguria; therefore, an inotropic agent was immediately administered. The inotropic therapy increased her urine output, and the MR gradually decreased (Figure 2B). The patient’s condition gradually improved, and she was moved out of the intensive care unit 6 days later. The AV block did not resolve; therefore, a permanent pacemaker was implanted without any complications. TAVI was performed via the femoral artery without any complications after pacemaker implantation. Intraoperative transesophageal echocardiography also showed trivial MR from the center (Figure 2C, D). The dramatic improvement in MR proved the presence of functional MR. The LVEF improved to 50% with trivial MR, and the patient was discharged from our hospital 16 days after TAVI.

Discussion

We have herein reported an older patient with functional MR that rapidly progressed from mild to severe after BAV.

MR exacerbation as a complication after BAV is not mentioned in a review though we experience worsening of MR after BAV occasionally in clinical practice. AS is frequently associated with concomitant moderate to severe MR. The rate of concomitant moderate to severe MR with TAVI ranges from 19% to 23%. There are two etiologies of MR: degenerative MR and functional MR. Only a few studies have provided data on the etiology of MR in patients who have undergone TAVI; however, functional MR accounts for approximately 50% of patients with MR among those who have undergone TAVI. In patients with severe AS and concomitant MR who undergo TAVI and BAV, physiological changes occur after aortic flow restoration, which could help to reduce MR severity. The pressure in the left ventricle drops very early after TAVI, and the transmitral pressure gradient can decrease, reducing MR in most patients. However, the se-
Figure 2. Comparison of MR immediately after BAV and before discharge. A: Severe MR immediately after BAV. B: MR reduced dramatically before discharge. C, D: Intraoperative transesophageal echocardiography during TAVI showed trivial MR. MR indicates mitral regurgitation; BAV, balloon aortic valvuloplasty; and TAVI, transcatheter aortic valve implantation.

Table. Cases in Which MR Worsened After BAV or TAVI

| Author         | Year | Age (years) | Sex  | BAV or TAVI | Pre MR degree | Post MR degree | LVEF  | Etiology                          | Therapy                  |
|----------------|------|-------------|------|-------------|---------------|----------------|-------|-----------------------------------|--------------------------|
| Caro-Codon J   | 2016 | 91          | Female | TAVI        | Mild          | Severe         | 50%   | LV systolic dysfunction           | Time course              |
| Leya F         | 2016 | -           | -     | TAVI        | -             | Severe         | > 85% | Suicide left ventricle            | New valve implantation   |
| Hashimoto M    | 2015 | 90          | Female | BAV         | -             | Severe         | Preserved | Rupture of the chordae tendineae | Surgical treatment       |
| Anat-Santos IJ | 2017 | -           | -     | TAVI        | Unknown       | Mitral leaflet perforation due to IE | Surgical therapy |

BAV indicates balloon aortic valvuloplasty; TAVI, transcatheter aortic valve implantation; MR, mitral regurgitation; LVEF, left ventricular ejection fraction; IE, infective endocarditis; Pre, preoperative; Post, postoperative; and LV, left ventricular.

The severity of MR remains unchanged in some cases. Nombela-Franco et al. mentioned that the factors associated with an improvement in MR after TAVI are functional MR, absence of pulmonary hypertension, absence of atrial fibrillation, a normal LVEF, and a normal left atrial size. In our case, the etiology of MR was functional. However, the presence of pulmonary hypertension, a low LVEF, and a large left atrial size can indicate no improvement of MR after aortic valve treatment.

Furthermore, the severity of MR progressed rapidly after BAV in our case. The mechanism of this rapid worsening of MR is not fully understood. We conducted a literature review in PubMed, and English-language case reports were searched using the following terms: “mitral regurgitation,” “TAVI,” and “BAV.” Only four case reports were found, and they are listed in the Table. In these four reports, MR worsened after BAV or TAVI because of rapid pacing, suicide left ventricle (in which the bioprosthesis induces systolic anterior movement and severe MR), rupture of the chordae tendineae, or infectious endocarditis. In our case, the etiology of MR was functional, and no organic disorder was involved. We consider that three conditions might have been involved in our patient’s worsening functional MR. First, complete AV block during BAV exacerbated the MR. This occurred because bradycardia induced an increase in the stroke volume and left ventricular end-diastolic volume, leading to mitral leaflet tethering and MR. In addition, left ventricular dyssyn-
chrony due to right ventricular pacing might have worsened the MR. Second, the AR progressed from mild to moderate, exacerbating mitral leaflet tethering and MR by increasing the left ventricular end-diastolic volume. Finally, we suspect that the progressively decreasing LVEF during the BAV procedure may have increased MR. The patient’s LVEF was at the lower limit of normal upon admission, but decreased further during the BAV procedure from 49% to 35%. This might have caused excess preload or afterload; therefore, mitral leaflet tethering and MR could have easily worsened. If MR worsens after a BAV or TAVI procedure, a mechanical disorder should initially be suspected. After exclusion of a mechanical disorder, such as wire interaction or chordal rupture, worsening MR should be attributed to left ventricular dysfunction and worsening hemodynamics (functional MR), and inotropic therapy should be started immediately.

In this case, complete AV block appeared when a guidewire was introduced into the left ventricle. Conduction disorders sometimes occur in BAV or TAVI procedures. In the literature, AV block with subsequent pacemaker requirement after TAVI occurs in 5.7%-42.5% of cases. The AV node is in close proximity to the subaortic region and the membranous septum of the left ventricular outflow tract, and mechanical stress of the balloon and valve can cause conduction disorders. Possibly, contact of the guidewire with the AV node led to the complete AV block.

TAVI for Asian nonagenarians was reported to be feasible and effective; however, it becomes critical once a major complication occurs. An urgent treatment for a major complication should be performed especially in nonagenarians.

Conclusion

MR usually decreases after a BAV or TAVI procedure, but MR may exacerbate in some cases in clinical practice, and it is a very important complication. If MR worsens after a BAV or TAVI procedure, inotropic therapy should be started immediately to improve left ventricular dysfunction and worsening hemodynamics (functional MR) after excluding the possibility of a mechanical disorder such as wire interaction or chordal rupture.

Disclosures

Conflicts of interest: None.

References

1. Ben-Dor I, Pichard AD, Satler LF, et al. Complications and outcome of balloon aortic valvuloplasty in high-risk or inoperable patients. JACC Cardiovasc Interv 2010; 3: 1150-6.
2. Jabbour RJ, Dick R, Walton AS, et al. Aortic balloon valvuloplasty--review and case series. Heart Lung Circ 2008; 17: S73-81.
3. Leon MB, Smith CR, Mack M, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. N Engl J Med 2010; 363: 1597-607.
4. Smith CR, Leon MB, Mack MJ, et al. Transcatheter versus surgical aortic-valve replacement in high-risk patients. N Engl J Med 2011; 364: 2187-98.
5. Nombela-Franco L, Ribeiro HB, Urena M, et al. Significant mitral regurgitation left untreated at the time of aortic valve replacement: a comprehensive review of a frequent entity in the transcatheter aortic valve replacement era. J Am Coll Cardiol 2014; 63: 2643-58.
6. Caro-codon J, Valbuena-Lopez S, Alvarez-Ortega C, et al. Rapid pacing-induced massive mitral regurgitation during transcatheter aortic valve implantation. Circ J 2016; 80: 748-9.
7. Leya F, Tucheck JM, Coats W. Abnormal distortion of aortic corvalve bioprosthesis with suicide left ventricle, aortic insufficiency, and severe mitral regurgitation during transcatheter aortic valve replacement. Catheter Cardiovasc Interv 2016; 88: 1181-7.
8. Hashimoto M, Fukui T, Takanashi S. Surgical treatment for double-valve destruction after balloon aortic valvuloplasty in a patient with porcelain aorta. Eur J Cardiothorac Surg 2015; 48: 329-31.
9. Amat-Santos IJ, Cortes C, Varela-Falcon LH. Delayed left anterior mitral leaflet perforation and infective endocarditis after transapical aortic valve implantation-case report and systematic review. Catheter Cardiovasc Interv 2017; 89: 951-4.
10. Karyofillis P, Kostopoulou A, Thomopoulou S, et al. Conduction abnormalities after transcatheter aortic valve implantation. J Geriatr Cardiol 2018; 15: 105-12.
11. Miura M, Shirai S, Uemura Y, et al. Early safety and efficacy of transcatheter aortic valve implantation for Asian nonagenarians (from KMH Registry). Int Heart J 2017; 58: 900-7.