Case Report

A Case of Aortic Dissection with Bicuspid Aortopathy for Prosthetic Valve Dysfunction

Ken Nakamura¹, Kouan Orii¹, Takayuki Abe¹, Hirofumi Haida¹, Kazuhiro Hashimoto², and Takashi Kunihara²

¹Department of Cardiac Surgery, Saitama Prefectural Cardiovascular and Respiratory Center ²Department of Cardiovascular Surgery, The Jikei University School of Medicine

ABSTRACT

Patients with bicuspid aortic valves are prone to aortic complications. Here, we present a 72-year-old man for whom prosthetic replacement of a stenotic bicuspid aortic valve had been performed 2004. After surgery, the patient was followed up as an outpatient with yearly echocardiographic and computed tomographic examinations. A recent routine checkup revealed an ascending aortic diameter of 45 mm, mild prosthetic valve regurgitation, moderate-to-severe stenosis, and a peak pressure gradient of 47 mm Hg. Eight months later the patient complained of dyspnea. Echocardiography showed severe aortic regurgitation and stenosis (peak pressure gradient, 58 mm Hg) and an intimal flap in the ascending aorta with jet blood flow towards the posterior aortic wall. Computed tomography showed localized dissection and dilation of the ascending aorta (56 mm in diameter). Emergency aortic valve re-replacement and partial aortic arch replacement were performed. A dissecting entry tear was found during surgery at the posterior aortic wall. This case of structural valve dysfunction with rapidly progress ascending aortic enlargement and aortic regurgitation suggests that aggressive surgical treatment should be considered in such cases.

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Key words: bicuspid aortopathy, aortic dissection, prosthetic valve dysfunction

Introduction

Patients with bicuspid aortic valves are prone to aortic complications. However, still controversial are the effects of the ascending aorta diameter at the time of aortic value replacement surgery and whether the ascending aorta and the aortic valve should be simultaneously replaced. The Japanese Circulation Society states that simultaneous replacement should be performed if the ascending aortic diameter is $\geq 55 \, \mathrm{mm}^1$. Furthermore, a study of patients with bicuspid aortic valve has found that aortopathy is avoided by only

43% of patients with an ascending aortic diameter of ≥ 45 mm². Aortopathy might be caused by the original weakness of the aortic wall; however, additional factors of aortopathy might be other problems of blood flow.

In the present case report, we describe a case of aortic dissection associated with prosthetic valve dysfunction.

CASE REPORT

A 72-year-old man complicated Parkinson's disease presented to our institution with dyspnea. In 2004 the pa-

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中村 賢,織井 恒安,阿部 貴行,灰田 周史,橋本 和弘,國原 孝

Mailing address: Ken Nakamura, Department of Cardiac Surgery, Saitama Prefectural Cardiovascular and Respiratory Center, 1696, Itai, Kumaga-ya-shi, Saitama, 360-0197, Japan

E-mail: cardiacsurgken@ybb.ne.jp, kenkentakota@yahoo.co.jp



Diastole Systole

Fig. 1. Preoperative echocardiography (diastole and systole)

An intimal flap in the ascending aorta was seen during systole with jet blood flow towards the posterior aortic wall.

tient had undergone prosthetic replacement with a 21-mm Carpentier-Edwards Perimount stented valve (Edwards Lifesciences Corp., Irvine CA, USA) of a bicuspid aortic valve that was causing stenosis. At that time the diameter of the ascending aorta was 40 mm. After surgery, the patient was followed up as an outpatient with yearly echocardiographic and computed tomographic (CT) examinations.

At a routine checkup 8 months before dyspnea developed, examinations had revealed an ascending aortic diameter of 45 mm with mild prosthetic valve regurgitation, moderate-to-severe stenosis, and a peak pressure gradient of 47 mm Hg. The patient was asymptomatic at that time. An examination 1 year earlier had found no aortic valve regurgitation.

Because of the dyspnea, the patient was admitted on an emergency basis to our institution and exhibited progression of prosthetic valve dysfunction. Echocardiography showed severe aortic regurgitation and stenosis (peak pressure gradient, 58 mm Hg). An intimal flap in the ascending aorta was noted with jet blood flow towards the posterior aortic wall (Fig. 1). A CT examination within the aorta showed localized dissection of the ascending aorta with a dissecting entry tear at the posterior aortic wall.

The diameter of the ascending aorta, as shown with CT, was 56 mm and was enlarged much greater than it had been 8 months earlier (Fig. 2). Emergency aortic valve rereplacement and partial aortic arch replacement were performed. Operative examination revealed that the Carpenti-

er-Edwards Perimount valve had been torn at both ends of the noncoronary cusp (Fig. 3). The posterior wall of the ascending aortic had a dissecting entry tear to the false lumen. We concluded that the jet blood flow had been generated via prosthetic valve stenosis and had exacerbated bicuspid aortopathy.

Although rehabilitation was challenging because the patient had Parkinson's disease, he was discharged without complications 2 months later. Based on the patient's presentation and findings, we suggest that despite variation in the recommended timing of surgical intervention, surgeons assessing patients with bicuspid aortopathy might want to consider earlier treatment.

DISCUSSION

Bicuspid aortic valve is the most common congenital heart disease and has a prevalence in the population of 2%³. These valves are associated with a lifetime risk of aortic dissection of 6%, which is 9 times as high as that of the age- and sex-matched general population⁴. Bicuspid aortic valve accounts for 15% of cases of aortic dissection. In addition, an estimated 5% of patients with bicuspid aortic valve die of aortic dissection⁵.

When the present patient underwent prosthetic aortic valve implantation in 2004, the ascending aortic diameter was 40 mm. A study of patients with bicuspid aortic valve and an aortic diameter of 40 to 45 mm found that aortopathy

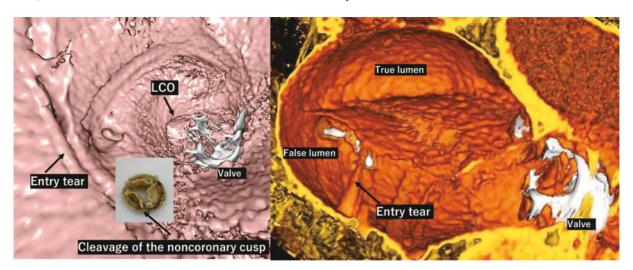


Fig. 2. Three-dimensional computed tomography within the ascending aorta Computed tomography within the ascending aorta showed localized dissection and dilation of the ascending aorta (56 mm in diameter).

LCO; Left Coronary Ostium

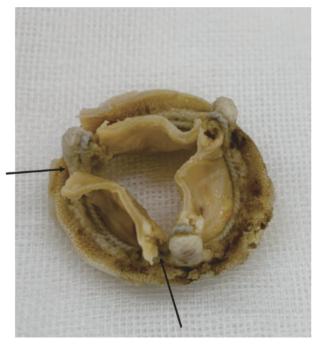


Fig. 3. Photograph of the broken prosthetic valve
Operative examination revealed that the CarpentierEdwards Perimount valve had tears (indicated by arrows) at both ends of the noncoronary cusp.

was avoided by81%². However, another study has concluded that replacing the bicuspid valve alone does not improve aortic disease⁶. Our literature search did not find any studies demonstrating a mechanism by which jet blood flow (as seen in our patient) resulting from structural prosthetic

valve dysfunction (SVD) stimulates the aortic wall to cause aortic dissection. Therefore, we could neither rule in nor rule out this phenomenon as a possible causative factor for the present case of aortic dissection. When the patient was examined 8 months before aortic dissection occurred, aortopathy was not predicted on the basis of diagnostic findings. Furthermore, emergent surgery was not considered necessary at that time because the aortic diameter was 45 mm and the SVD was moderate. It has been commonly reported that 20% of patients have an ascending aortic diameter of less than 45 mm when aortic dissection occurs⁷. Furthermore, in the present case, an echocardiogram 1 year before aortic dissection was diagnosed showed no evidence of aortic valve regurgitation. This suggests that in cases of rapidly progressive prosthetic valve dysfunction, aortopathy can also develop rapidly.

In the present case, It was possible that the patient had moderate prosthetic valve stenosis 8 months before aortic dissection occurred. Later, the noncoronary cusp tears caused severe aortic regurgitation, which increased cardiac output, as well as aortic valve pressure disparity and velocity, which might have contributed to the rapid progression of aortopathy.

Conclusion

We have reported on a patient with rapidly progressive

SVD complicated by aortic dissection. On the basis of the patient's presentation and the findings of echocardiographic and CT examinations, we believe that aggressive surgical treatment should be considered in cases of SVD with rapidly progressive ascending aortic enlargement and aortic regurgitation.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Written informed consent was obtained from the patient for use data for academic publication purposes.

Authors have no conflict of interest.

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