Acute Aortic Regurgitation due to Local Avulsion of the Aortic Valve Commissure

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A 69-year-old man was transferred to our hospital because of sudden onset precordial pain and dyspnea. Transesophageal echocardiography revealed massive aortic regurgitation, diastolic separation of the closure line of the aortic cusps and prolapsing motion of the cusps during diastolic toward the left ventricular outflow tract. Aortic valve replacement was successfully performed. During the operation, we found a commissure between the left coronary cusp and the non-coronary cusp that had avulsed from the aortic wall and prolapsed into the left ventricular outflow tract. Valvular cusps were excised and replaced with a mechanical prosthesis. The postoperative course was uneventful and the patient was discharged from the hospital, 25 days after his operation. The histopathological examination showed fibrosis, hyalinosis of the avulsed commissure, and mucoid degeneration of the valve. There was no evidence of pathologic changes, such as aortitis, infective endocarditis, or specific connective tissue disorders.

Keywords: aortic, valve , avulsion, commissure

Introduction

Acute aortic regurgitation is usually caused by aortic dissection, infective endocarditis, or blunt trauma which results in sudden, severe hemodynamic deterioration. Also, it carries a lethal prognosis without an early, correct diagnosis and adequate treatment, including surgical intervention. We herein report a successful case of surgical treatment of acute aortic regurgitation, resulting from avulsion of an atheromatous plaque at one of the commissures of a normal aortic valve.

Case Report

A 69-year-old man was admitted to our hospital because of precordial pain and dyspnea, 3 hours after a sudden onset of symptoms. He had been on medication for hypertension for ten years but had no remarkable history of high fever or trauma. On admission, his blood pressure reached only 70/30 mm Hg and his temperature was 36.8 degrees Celsius. Chest roentgenogram presented severe pulmonary edema with minimal cardiomegaly. A transthoracic echocardiogram demonstrated diastolic prolapse of the non-coronary cusp (NCC) and severe aortic regurgitation (AR). Contrast enhanced computed tomography revealed no evidence of aortic dissection. Emergency cardiac catheterization showed an AR of grade IV, a pulmonary arterial pressure of 56/24 mmHg, pulmonary capillary wedge pressure of 22 mmHg, an aortic pressure of 86/34 mmHg, and a normal coronary angiography.
During cardiac catheterization, he fell into respiratory distress, so he was transferred to the intensive care unit, intubated and ventilated mechanically.

To evaluate more detailed anatomic information, we performed transesophageal echocardiography. It showed that the NCC was prolapsing to the left ventricular outflow tract conjoined by the commissure (Fig. 1). Despite intravenous catecholamine infusion and mechanically ventilation support, his hemodynamic instability did not improve. Surgical treatment was performed 2nd days after the onset of symptoms.

Conventional cardiopulmonary bypass was established, and the aortic valve was explored. It was found that the NCC was prolapsing to the left ventricular outflow tract conjoined by the commissure (Fig. 1). Despite intravenous catecholamine infusion and mechanically ventilation support, his hemodynamic instability did not improve. Surgical treatment was performed 2nd days after the onset of symptoms.

Fig. 1
A: A transesophageal echocardiogram (longitudinal view) indicating prolapse of the noncoronary cusp (arrow).
B: Flow Doppler image of massive aortic regurgitation (arrow head).
LA: left atrium; LV: left ventricle; Ao: aorta

Conventional cardiopulmonary bypass was established, and the aortic valve was explored. It was found that the commissure between the LCC and NCC was avulsed from the aortic wall over a sector of approximately 8 mm and prolapsed down to the left ventricular outflow tract (Fig. 2). There was no evidence of perforation or rupture of the valve leaflet itself or dissections of the aorta. There were many yellowish atheromatous plaques scattered on the aortic intima; therefore, it seems impossible to perform valvuloplasty by suturing the avulsed commissure to the aortic wall at its original position. The valvular cusps were excised and replaced with a 25-mm mechanical prosthesis (St. Jude Medical, St. Paul, MN, USA). Since it was difficult to check the intention of the patient, according to a patient family’s request, prosthetic valve replacement with the mechanical valve was performed.

Histopathological examination revealed fibrosis, hyalinosis of the avulsed commissure, and mucoid degeneration of the aortic valve cusps. There was no evidence of any pathologic changes such as aortitis, infective endocarditis, or specific connective tissue disorders.

The postoperative course was uneventful and the patient was discharged from the hospital, 25 days after his operation. During the two years after the operation, the patient came for follow-up visits and he continued to be in excellent condition.

**Discussion**

Trauma and non-traumatic aortic pathologies such as myxomatous degeneration, rheumatic fever, syphilis, fenestrated aortic valves, infective endocarditis and inherited disorders of connective tissue may occasionally cause AR because of rupture of the aortic valve itself.1 On the other hand, local layer dehiscence of the aortic wall around an aortic commissure is a rare pathophysiologic cause of acute AR. There have been a few reports that degenerative diseases, calcification and rupture of atheromatous plaque at the commissure have been noted as a

Fig. 2 Operative photograph showing the avulsed commissure (arrow).
cause of spontaneous dehiscence of the aortic wall. Among the three cusps, the NCC is estimated to be most commonly involved. Grimball et al. speculated for this fact is that the left and right coronary cusps have coronary arteries that work as pressure buffers against the high hydrostatic pressures. It is well known that atheromatous plaques on the aorta may lead to thinning of the media beneath the plaques, causing weakness in the aortic wall. In our case, there were many yellowish atheromatous plaques scattered on the aortic intima also around the commissures.

The clinical course of spontaneous commissural avulsion is a sudden onset of massive AR that may result in a lethal outcome with deteriorating hemodynamics. Therefore, early precise diagnosis and prompt surgical intervention are mandatory because of the poor clinical results with medical treatment. In our patient, transesophageal echocardiographic observations allow accurate anatomical assessment of aortic valve insufficiency causing from dehiscence of the commissure.

There have been some reports that suggested successful treatment of AR by suturing the detached commissure to the aortic wall. However, we performed aortic valve replacement instead of valvuloplasty, according to the intraoperative findings of the aortic intima, because direct repair of the degenerated and avulsed commissure to the diseased aortic wall may lead to poor long-term result.

Conclusion

We reported a successful treatment case of acute AR caused by local dehiscence of the aortic commissure. In cases of patients presenting with acute AR with aortic cusp prolapse and intact ascending wall, avulsion of the commissure should be taken into consideration, as in our case.

References