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Concomitant Replacement of the Aortic Valve with Supracoronary Ascending Aorta in a Young Male Presented with Severe Bicuspid Aortic Stenosis with Ascending Aortic Aneurysm: A Case Report

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Abstract

A bicuspid aortic valve (BAV) is the most common congenital heart valve defect. It is linked to aortic stenosis, aortic insufficiency, and aortopathy like coarctation of the aorta, ascending aortic aneurysm, aortic dissection, and rupture. It occurs in about 1-2% of the population and is more common in males. Transthoracic echocardiography is the gold standard for diagnosis of aortic valve disease with aortopathy. Further investigation, like a CT scan or MRI of the chest, is required to check the size of the aorta for further management. Simultaneous corrective surgery for both the aortic valve and ascending aorta is the main stay of treatment, although it is a challenging procedure.

Keywords

Bicuspid aortic valve, Aortic stenosis, Ascending aortic aneurysm, Aortic valve replacement, Supra-coronary ascending aorta replacement.

Introduction

A bicuspid aortic valve is one of the most common congenital heart valve diseases that occurs in 0.5-2% of the general population and is more common in males, as described by Kong et al. in 2017 and Liu et al. in 2019 [1,2]. In a bicuspid aortic valve (BAV), there are only two functional cusps or leaflets instead of three. Most commonly, this results from fusion of two adjacent cusps during embryologic development. Rarely, a bicuspid aortic valve comprises two unequally sized cusps with no raphe, called "Pure" BAV. The most widely used surgical classification of BAV is "The Sievers and Schmidtke's classification", based on the number of raphes and categorized into (a)Type 0, involving a true bicuspid valve, two symmetric cusps with no raphe; (b) Type 1, in which there is one fusion line (with one raphe) between two cusps; and (c) Type 2, in which two fusion lines (with two raphes) are present. A recent survey found that, 75% of people with bicuspid aortic valves (BAVs) will have aortic stenosis and dilatation of the supra-coronary ascending aorta, 15% with have dilatation of the proximal aortic root and aortic insufficiency, and the prognosis of the other 10% is unknown [3,4]. It is also associated with aortopathy like coarctation of the aorta, aortic dissection, and rupture.

In 2017 Roman et al., also found that in patients with bicuspid aortic valve disease, primarily aortic stenosis (AS), the prevalence of dilatation in the tubular ascending aorta was higher; whereas the prevalence of the diffuse dilatation of the aorta (annulus, aortic root, or tubular ascending aorta) was increased in bicuspid aortic valve patients with aortic regurgitation or insufficiency [5].

Classification of ascending aortic dilatation were categorized into three: (1) Fazel's class (2) Park's class (3) Della Corte's class. Aortic root phenotype (Sinuses of Valsalva/ aortic root is the main site of dilatation and aortic sinuses > ascending aorta) and ascending phenotype (Tubular ascending aorta is the main site of dilatation and ascending aorta > aortic sinuses) are the two primary phenotypes of ascending aorta dilatation proposed by Della Core et al. in 2014. Despite its simplicity, this classification method demonstrated potential predictive utility for aortic dilatation [6].

The majority of patients with bicuspid aortic valve disease may have complicated aortic valve dysfunction and aortic dilatation. Aortic dilatation can progress to aortic aneurysm. If an aortic aneurysm is untreated, it may lead to life-

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threatening events like aortic dissection or rupture. So, timely surgical intervention is the fundamental treatment approach for bicuspid aortic valve stenosis with ascending aortic aneurysm [7].

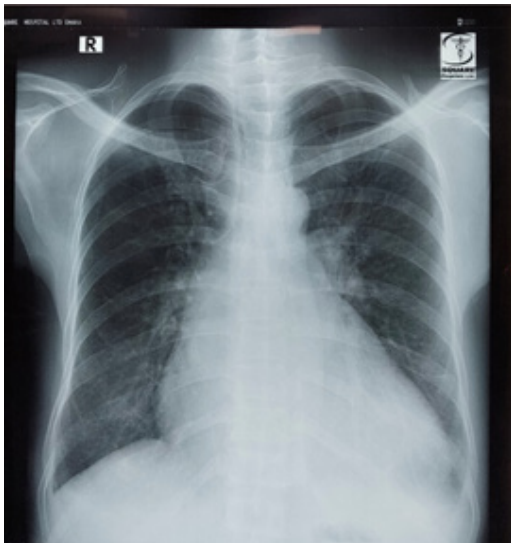
In the 1960s, concurrent ascending aorta and aortic valve replacement was first implemented. Supracoronary ascending aorta replacement accompanied by aortic valve replacement was first documented by Groves et al. in 1964 and is the operation of choice in patients with concomitant aortic valve pathology and ascending aorta aneurysm [8].

Nowadays, bicuspid aortic valve disease and aneurysm of the ascending aorta are sometimes managed concurrently, depending on whether the lesion is repairable or needs to be replaced, although it is a challenging procedure [9]. Herein, we present a case of patients with bicuspid aortic valve stenosis with ascending aortic aneurysm treated with concomitant replacement of the supracoronary ascending aorta and aortic Valve.

Case Report

A 45-year-old male got admitted to our cardiac surgery unit through the outpatient clinic as a known case of severe bicuspid aortic stenosis with aneurysmal dilatation of the ascending aorta. He has had complaints of cough, dyspnea, vomiting, and a feeling of heaviness of the chest for last 3 months. He was normotensive and nondiabetic. He had no history of similar diseases and genetic disorders in his family. Before admission to our hospital with these complaints, he was consulted with a cardiologist, and after proper assessment, he was diagnosed with severe bicuspid aortic stenosis associated with aneurysmal dilatation of the ascending aorta and was advised to have surgery.

On examination after admission: pulse rate-82 beats/min, blood pressure-100/60 mmHg, respiratory rate-16/min, and SpO_2 was of 97% at room air. Cardiac examination revealed a heaving apex beat at the left fifth intercostal space and an ejection systolic murmur over the aortic area, which radiates to the right carotid region. Other systemic examinations revealed no abnormality. Laboratory reports, cardiac enzymes, serum bilirubin, liver function, and renal function tests of the patients revealed normal.



The chest X-ray Posterior-anterior view revealed cardiomegaly.
Figure 1: CXR P/A View showed enlargement of cardiac shadow.

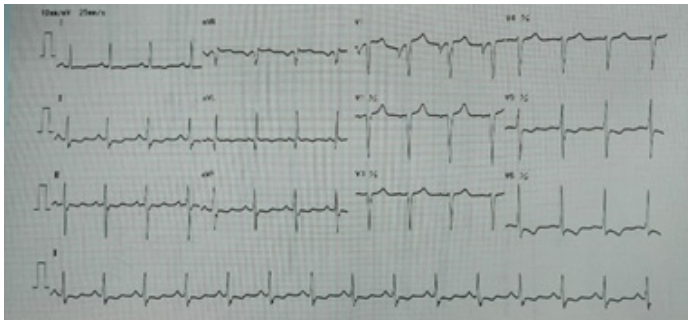


Figure 2: Electrocardiogram showed LVH with a strain pattern and poor R wave progression in precordial leads.

Color Doppler echocardiography revealed a severely stenotic bicuspid aortic valve (AVA-0.3 cm2, MPG-68 mmHg) with moderate AR and PASP-50 mmHg, aortic annulus- 22.0 mm, aortic root- 24.0 mm, STJ-33.0 mm, ascending aorta-47.0 mm. AV cusps are thickened & calcified with severely restricted opening along with aneurysmal dilatation of the ascending aorta from STJ to the proximal arch and severe LV systolic dysfunction (LVEF-25%). Minimal pericardial effusion.

The CTA of the aorta and its branches showed bicuspid aortic valve cusps are thickened & calcified and the dilated ascending aorta. Aortic measurement showed aortic annulus- 22.9 mm, aortic sinus- 38.6 mm, STJ-32.3 mm, proximal ascending aorta-47.0 mm, ascending aorta at MPA bifurcation- 55.4 mm, proximal aortic arch-35.3 mm, mid & distal aortic arch, and descending thoracic aorta showed normal diameter; both coronary arteries are normal in origin and course.

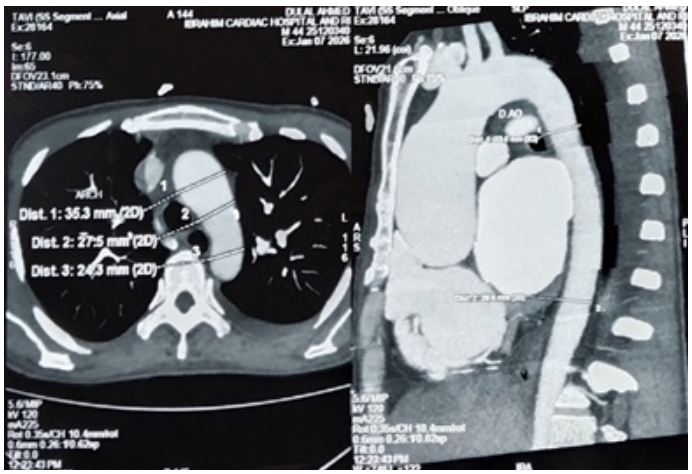


Figure 3: The CTA of the aorta showed the dilatation of tubular ascending aorta.

A coronary angiogram was also done and showed normal epicardial coronary arteries. Therefore, after comprehensive clinical evaluation, anesthetic assessment, and review of the laboratory and imaging findings, the patient was considered fit and ready to undergo surgical intervention under general anesthesia with median sternotomy.

Following median sternotomy, the thymus was dissected. The pericardium was opened. The ascending aorta was found dilated, the PA (pulmonary artery) showed hypertensive, and the LV was also dilated.

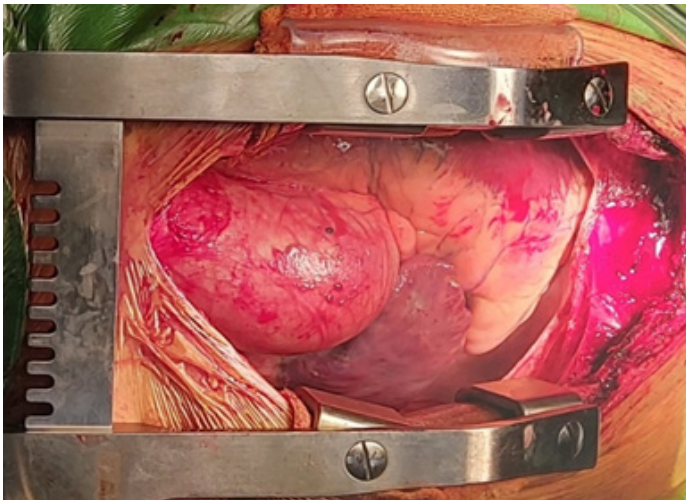


Figure 4: The pericardium was opened after median sternotomy, tubular ascending aorta showed dilated.

After systemic heparinization and achieving target activated clotting time (ACT), standard aortic and two-stage single venous cannulation via the right atrium were performed to establish cardiopulmonary bypass. A left ventricular vent was inserted through the right upper pulmonary vein and positioned across the mitral valve into the left ventricle. Then the ascending aorta was cross-clamped, and myocardial protection was achieved with del-Nido cardioplegia, which was administered antegrade via the aortic root and directly into the coronary ostia to arrest the heart at diastole. Aortotomy was done. The dilated ascending aorta was resected; all diseased tissues were excised. Then the aortic valve was checked; it was found to be severely calcified leaflet, stenotic, and bicuspid in nature.

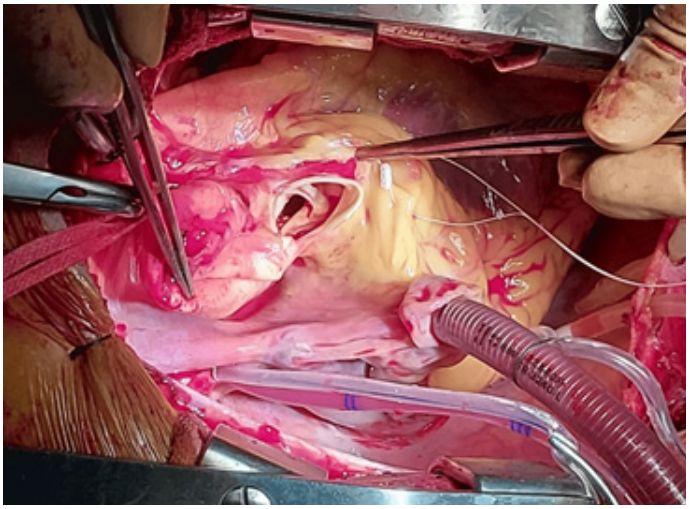


Figure 5: After opening of the ascending aorta, diseased aortic valve showed bicuspid, stenotic and calcified; retraction of fat near aortotomy with valve suture was done for adequate exposure.

Excised the diseased valve, and after sizing the valve orifice, pledgeted sutures were placed in the aortic annulus and replaced with a 23mm SJM Regent mechanical aortic heart valve.

Thereafter, the ascending aorta (supracoronary) was replaced with 30 mm Jotec tube graft.

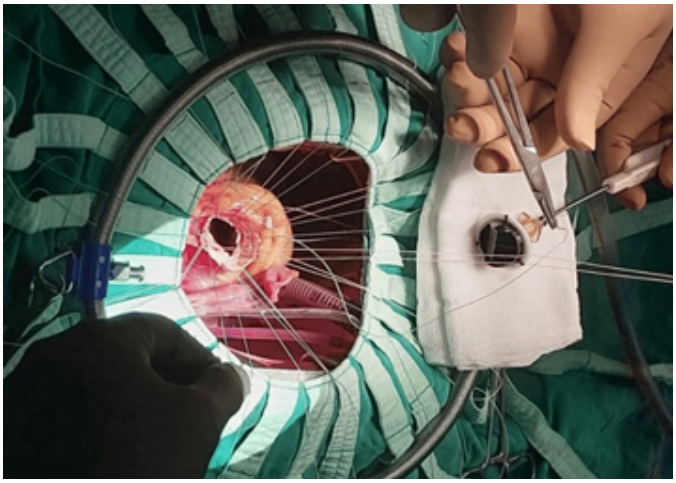


Figure 6: Pledgeted sutures were placed in the aortic annulus after excising the diseased valve for the replacement of an artificial mechanical heart valve.

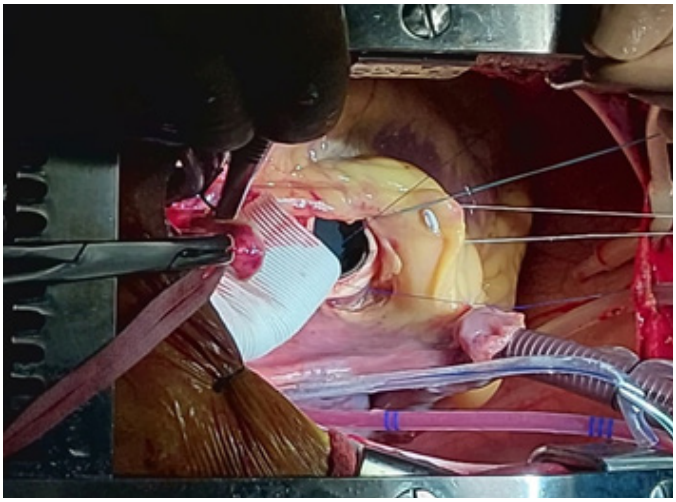


Figure 7: After replacement of the artificial aortic heart valve, the supracoronary ascending aorta was replaced with a synthetic tube graft.

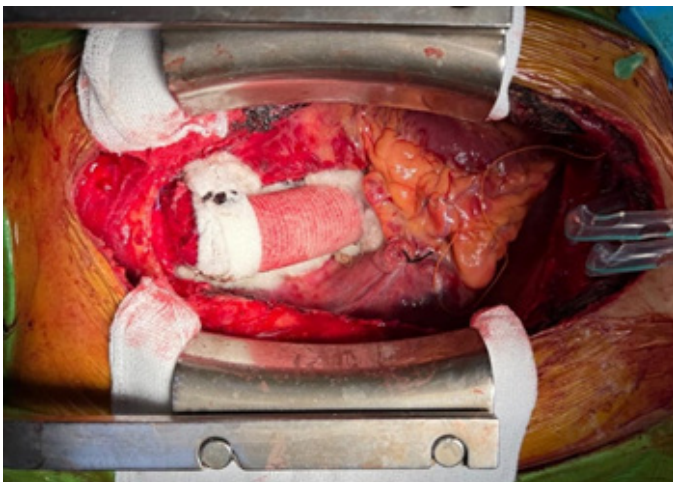


Figure 8: Before closing the chest, the anastomotic site of the replaced synthetic ascending aorta was covered with a hemostatic

fibrillar agent, keeping a temporary epicardial ventricular pacing wire and chest tube drains in the pericardial cavity.

The left heart was carefully de-aired under transesophageal echocardiographic guidance. The aortic cross -clamp was removed, and sinus rhythm resumed spontaneously. Rewarming was completed, and the patient was gradually weaned from CPB with stable hemodynamics. As TEE showed no residual air in the cardiac chambers, decannulation was done. After protaminization and achieving proper hemostasis, the wound was closed in layers, keeping a temporary epicardial ventricular pacing wire and chest tube drains in situ. The patient was transferred to the Cardiothoracic Intensive Care Unit (CT-ICU) with stable hemodynamics. He was extubated once awake, neurologically intact, and meeting respiratory parameters, with satisfactory oxygen saturation on supplemental oxygen, and the subsequent post-operative course was uneventful.

Post-operative color Doppler echocardiography was done and showed a normally functioning prosthetic heart valve in the aortic position with a mean pressure gradient (MPG-7 mmHg), no valvular or paravalvular leakage, no thrombus/vegetation in relation to PHV, trace MR, Gr I TR, PASP-30 mmHg, LVEF-35-40%, RV systolic function is normal, and normal blood flow via the new aorta. The patient was discharged home in stable condition on the 7th day of the postoperative period.

Follow-Up

The patient was doing well and symptom-free at the one-month, three-month, and six-month follow-ups.

Discussion

Hattori et al. in 2017 stated that a bicuspid aortic valve disease is the most common congenital cardiac defect, affecting 0.5-2% of the population [10]. A bicuspid aortic valve has a significant genetic component, most commonly with an autosomal dominant inheritance with variable penetrance and an increased risk of developing aneurysm or dissection. According to a recent survey, 75% of people with bicuspid aortic valves (BAVs) will have aortic valve stenosis and dilatation of the supra-coronary ascending aorta, 15% will have dilatation of the proximal aortic root and aortic insufficiency, and the prognosis of the other 10% is unknown [3,4].

In 2015, Philip et al. also described that the bicuspid aortic valve represents coexisting aspects of a genetic disorder of the aorta and cardiac development, which is characterized at an early stage by asymptomatic dilatation of the ascending aorta with or without valvular lesion and later by frequent susceptibility to aortic aneurysm formation and the most feared complication, aortic dissection [11].

Bicuspid aortic valve can manifest in a variety of ways. Adulthood is usually when the hallmark symptoms appear. The primary aortic valve-related clinical manifestations are stenosis and incompetence. Other complications include endocarditis, dilated aortopathy, dissection, and rupture [12].

Ascending aortic aneurysms or dilatation and aortic valve diseases often have distinct causes. Ascending aorta dilatation is known to be caused by intrinsic genetic and physiological abnormalities of the aortic wall (e.g., Marfan syndrome, bicuspid aortic valve). Other factors that are associated with ascending aortic dilatation or aneurysm include aging, high blood pressure, smoking, and atherosclerosis.

The most widely used imaging technique for diagnosis of bicuspid aortic valve disease is 2D-TTE.The proximal segment of the ascending aorta, the aortic root (aortic annulus, sinuses of Valsalva, and sinotubular junction), and the aortic valve anatomy can all be fully displayed by 2D-TTE. In 2018, Borger et al. described that a thorough evaluation of the entire thoracic aorta, cardiac MRI or electrocardiographically gated computed tomographic angiography (CTA) is required when 2D-TTE is unable to sufficiently display any aortic segments. Surgical intervention is the fundamental treatment strategy for bicuspid aortic valve stenosis with ascending aortic aneurysm.

Concomitant aortic surgeries, which include aortic valve and ascending aorta replacement, are complex and have high morbidity and mortality rates. It may increase the operative risks like paraplegia, stroke, bleeding, and death but protect patients from long-term complications. Replacing the ascending aorta due to an aneurysm during aortic valve replacement (AVR) is regarded as a standard treatment strategy, although there is added risk because AVR by itself resolves hemodynamic problems but not aortopathy. In clinical practice, patients with bicuspid aortic valve stenosis associated with dilatation of the ascending aorta, whether selective AVR or elective ascending aortic replacement as a concurrent procedure, can reduce the risk of adverse effects in the future.

In 2016, Vendramin et al. found no progressive dilatation of the native aortic root in patients undergoing AVR and supracoronary ascending aorta replacement (AVR-SCAAR), regardless of whether the preoperative root diameter was <40 mm or >40 mm [13].

The reasons for replacing the ascending aorta in patients undergoing aortic valve replacement (AVR) are now well-defined by updated guidelines, especially in the presence of a bicuspid aortic valve disease. The decision criterion for ascending aortic replacement is still up for dispute. Vahanian et al. in 2022 described that the current guidelines [14] recommend replacement at a diameter of ascending aorta of ≥50 mm in the absence of additional risk factors, some experts support for a lower threshold of 45 mm, particularly when concomitant valve surgery is planned or when additional risk factors (e.g., family history, asymmetric dilation or rapid growth) are present. Some institutions or centers take a more aggressive approach in the setting of BAV, replacing the ascending aorta at ≥45 because aortic wall disease may contribute to earlier dissection or rupture [14]. The 2024 ESC guideline update recommends supracoronary ascending aorta replacement at a diameter of ≥45 mm, with dilatation limited to the tubular ascending aorta but with a normal root (< 45mm) in patients with a bicuspid aortic valve undergoing valve surgery.

Conclusion

Supracoronary ascending aorta replacement combined with aortic valve replacement is a safe and effective surgical approach for severely stenotic bicuspid aortic valves accompanied by a dilated ascending aorta in short-term results. Clinical outcomes generally demonstrate low operative mortality and good long-term survival when performed in appropriately selected patients, with the surgical approach guided by current guidelines and variability in technical expertise, decision-making, intraoperative judgment, and experience of the surgical team. Overall, this treatment strategy offers a durable solution with favorable functional and structural results, making a recommended treatment option.

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Not Applicable

Conflicts of interest
The authors declare that there are no conflicts of interest.

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Ethics
Written informed consent was obtained from the patient.

Consent to Participate
The work was approved by all the authors for participation.

Consent to Publications
The work was approved by all the authors for participation.

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