The Hidden Danger of Aortic Stenosis—Exercise-Induced Cardiac Arrest in a Young Man with Bicuspid Aortic Valve

Lars Ege Rasmussen, MD, Christian Alcaraz Frederiksen, MD, PhD,
Thomas Krusenstjerna-Hafstrøm, MD, PhD, and Henrik Vase, MD, PhD, *Aarhus, Denmark*

INTRODUCTION

Bicuspid aortic valve (BAoV) is the most common congenital heart defect, with a prevalence of 0.77% in newborns. Bicuspid aortic valve predisposes individuals to valve dysfunction and aortopathy.[1] Routine evaluation of patients with BAoV consists of electrocardiogram, echocardiogram, and clinical examination. When flow conditions are normal, concordant severe aortic stenosis (AS) can be diagnosed when the mean gradient is ≥40 mm Hg or the peak velocity is ≥4.0 m/s and the aortic valve (AoV) area is ≤1.0 cm^2. In rare cases where symptoms do not match echocardiographic findings, patients are often referred for further evaluation.

CASE PRESENTATION

A 19-year-old man with BAoV was admitted following successful resuscitation. During a soccer match with strenuous physical exercise, the patient collapsed with cardiac arrest. Fellow teammates immediately provided cardiopulmonary resuscitation. Defibrillation via an automatic external defibrillator and return of spontaneous circulation were achieved within minutes. The patient was awake and stable before the arrival of the ambulance.

The patient attended regular clinical visits and echocardiography for surveillance of the BAoV. Eighteen months before the cardiac arrest, exercise-induced symptoms were suspected despite low transvalvular pressure gradients on Doppler echocardiography (Figure 1). The patient was referred for exercise treadmill testing, which was interpreted as normal.

During hospitalization, transthoracic echocardiography (TTE) was not able to demonstrate high-gradient AS in spite of multiple experienced sonographers using multiple acoustic windows, including apical and right parasternal, suprasternal, supraclavicular, and subcostal views (Figure 2). As clinical findings were discordant with the TTE findings, severe AS was suspected; hence, cardiovascular magnetic resonance (CMR), and simultaneous invasive left and right heart catheterization at rest were performed.

Right heart catheterization was performed using a 7-Fr triple-lumen pulmonary artery catheter. The catheter was introduced through an 8-Fr sheath in the internal jugular vein and navigated to the pulmonary artery. Left heart catheterization used a 6-Fr sheath inserted into the right radial artery. A 6-Fr diagnostic coronary catheter was placed in the left ventricle (LV) using the standard retrograde technique to cross the AoV. A 0.014″ coronary pressure wire was equalized in the LV at the tip of the catheter. The pressure wire was then advanced to a stable position in the LV, and the catheter was retracted to the ascending aorta.

The test revealed high-gradient valvular AS with elevated left ventricular end-diastolic pressure, normal cardiac output, normal pulmonary pressure, and normal pulmonary vascular resistance (Figure 3). Invasive exercise testing using supine bicycle exercise was omitted

VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE, parasternal long-axis focused view, demonstrates a thickened, noncalcified AoV with systolic doming.

Video 2: Two-dimensional TTE, parasternal short-axis view, demonstrates a congenital BAoV with mobile, thickened, nonrestricted leaflets.

Video 3: Cardiovascular magnetic resonance, balanced steady-state free precession sequence, basal short-axis view, demonstrates a BAoV (right noncusp fusion) with a slit-like opening.

Video 4: Cardiovascular magnetic resonance, balanced steady-state free precession sequence, coronal left ventricular outflow tract view, demonstrates an abnormally bright signal with parallel signal loss along the edges consistent with a high-velocity flow across the BAoV.

Video 5: Cardiovascular magnetic resonance, 3D whole-heart volume-rendered reconstruction, oblique sagittal display with superimposed time-resolved 4D flow, demonstrates the 3D velocity-encoded phase-contrast pattern with the high-velocity flow across the BAoV (*blue*, low velocity; *green*, intermediate velocity; *red*, high velocity).

Video 6: Two-dimensional TTE, suprasternal long-axis view, demonstrates the distal ascending, arch, and proximal descending thoracic aorta without aneurysm or focal dilation.

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From the Department of Cardiology, Aarhus University Hospital, Aarhus, Denmark.

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Correspondence: Lars Ege Rasmussen, MD, Department of Cardiology, Aarhus University Hospital, Palle Juul-Jensens Boulevard 99, Aarhus N 8200, Denmark (E-mail: *larsra@rm.dk*).

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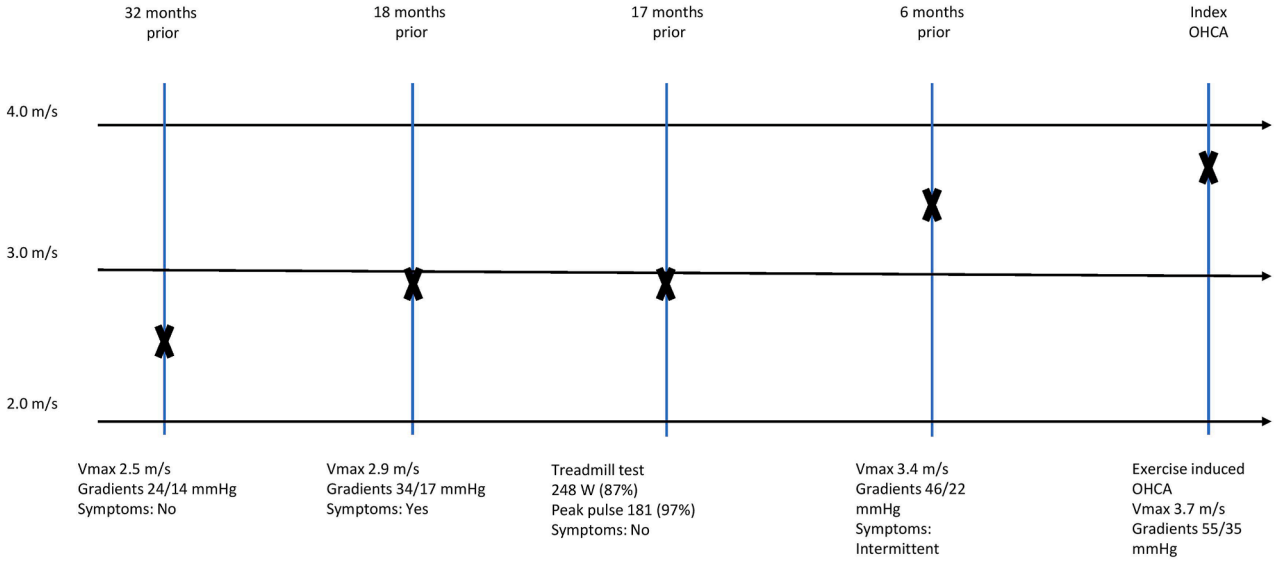


Figure 1 Timeline of serial key echocardiographic findings. *OHCA*, Out-of-hospital cardiac arrest; *Vmax*, peak velocity.

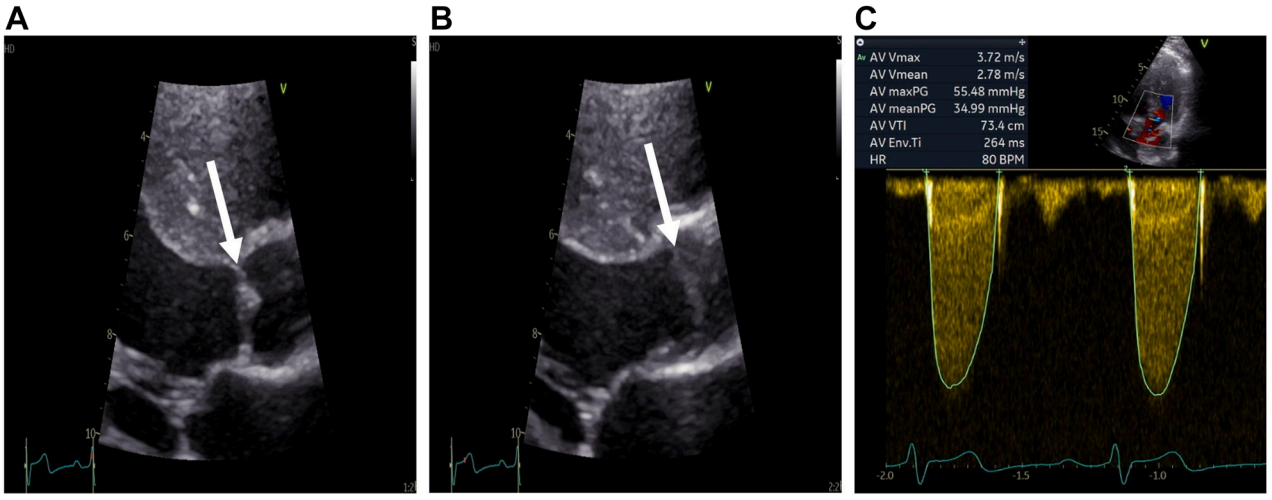


Figure 2 Two-dimensional TTE, parasternal long-axis focused diastolic **(A)** and systolic **(B)** views and apical 4-chamber view with color-flow Doppler-guided continuous-wave spectral Doppler display **(C)**, demonstrates a thickened, noncalcified, AoV with systolic doming *(arrows)* and moderately increased velocity with a spectral profile suggesting moderate AS.

as diagnosis was certain at resting conditions (Table 1). As surgery seemed likely, invasive coronary angiography was omitted and cardiac computed tomography was scheduled to exclude aortopathy and coronary anomaly.

The CMR scan showed a nondilated LV with normal systolic function, normal left ventricular mass, normal T1 and T2 values, no signs of edema, and no late gadolinium enhancement. The CMR scan also demonstrated BAoV with peak velocities of 3.6 m/s and an AoV area of 0.6 cm^2 (Figure 4, Videos 3 and 4). Supplementary 4-dimensional (4D) flow measurements demonstrated high-gradient AS with velocities above 4.0 m/s (Figure 5, Video 5).

The patient underwent surgical AoV replacement on day 8 after the index cardiac arrest. Preoperative cardiac computed tomography demonstrated normal coronary anatomy without obstructive disease and no aortopathy. A 23 mm bioprosthetic AoV was selected to avoid lifelong anticoagulation, in accordance with the patient's wish to continue competitive sports.

The procedure was complicated by complete (third-degree) atrioventricular block, necessitating implantation of a dual-chamber pacemaker before discharge. The patient subsequently returned to their usual lifestyle and resumed competitive sports.

Twelve months after discharge, they once again experienced exercise-induced symptoms. Clinical evaluation showed a normally functioning bioprosthetic AoV (gradients 23/13 mm Hg, peak velocity 2.2 m/s) and normal biventricular function. Symptoms were attributed to automatic mode switching of the pacemaker due to misinterpretation of sinus tachycardia, resulting in relative bradycardia during exercise. Symptoms resolved completely after pacemaker reprogramming.

DISCUSSION

This case highlights that underestimation of the severity of AS can have fatal consequences. In this case, a treadmill exercise test had

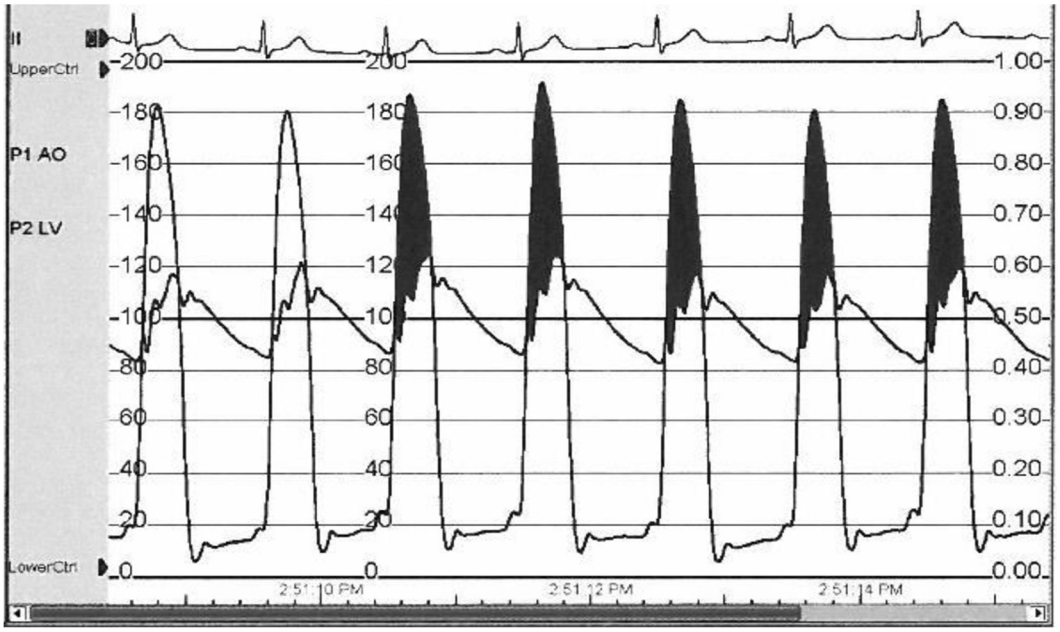


Figure 3 Invasive left heart catheterization at rest with simultaneous catheters in the LV and ascending aorta (AO) demonstrates the pressure curves in mm Hg, with the *shaded area* representing the LV-AO pressure difference.

Table 1 Invasive left and right heart catheterization at rest

Right atrium	(6)
Right ventricle	34 (9)
Pulmonary artery	29 (11)
Pulmonary artery wedge pressure	(13)
Left ventricular end-diastolic pressure	(20)
Transvalvular aortic gradients	(50)
AoV area, cm^2	0.91
Cardiac output, L/min	4.8
Indirect Fick, L/min	5.6
Pulmonary vascular resistance	1.3
Arterial saturation, %	98
Mixed venous saturation, %	73

Gradients are reported as peak (max) gradients in mm Hg with associated mean gradients in mm Hg within parentheses.

been performed 17 months before the episode of exercise-induced cardiac arrest. The patient achieved a maximal workload of 248 W (87% of predicted) and was considered asymptomatic throughout. However, it is reasonable to question whether a standard treadmill test adequately reflects the intensity and physiological demands of competitive sports. Had cardiopulmonary exercise testing been performed instead, it is speculative whether results would have been different. Cardiopulmonary exercise testing provides further information, such as maximal oxygen consumption, ventilatory capacity, and aerobic threshold, and provides the clinician a better opportunity to evaluate whether the patient's exercise capacity has been sufficiently exercised. Patient-reported symptoms, however, do not differ between tests.

Index cardiac arrest as the first symptom of congenital AoV disease has previously been reported.[2,3] This case report is unique and provides serial echocardiographic and stress testing years before the index cardiac arrest. A recent publication of the largest contemporary cardiac autopsy series of patients with BAoV documents that a stenotic BAoV is directly related to the cause of death in 30% of patients with a mean age 37 with sudden cardiac death. Although the study is small ($N = 91$), it underscores the risk of having the severity of this condition undiagnosed or underestimated.[4]

When counseling athletes with BAoV, we typically consider competitive athletics safe when valve disease is nonsevere, aortic dimensions are normal, and no symptoms are present. However, recent American Heart Association/American College of Cardiology guidelines emphasize that moderate AS represents a spectrum of risk, underscoring the importance of individualized risk estimation in competitive athletes with structural heart disease.[5]

Invasive left heart catheterization was historically the reference standard for the diagnostic evaluation of the severity of AS. With the evolution of ultrasound technology, this noninvasive approach has been the primary diagnostic workup since its validation in the 1980s.[6]

Left ventricular fibrosis assessed by late gadolinium enhancement on CMR has been shown to predict the need for AoV replacement within 1 year in patients with BAoV.[7] In the present case, novel 4D flow measurements did yield relevant diagnostic information and could have a role in future studies. Interestingly, a newly published comparison between 4D flow CMR and continuous-wave Doppler (CWD) echocardiography documents complementary diagnostic value for 4D flow CMR for the estimation of AS.[8] The trial, however, is small ($N = 30$), and further studies are needed.

Underestimation of aortic transvalvular pressure gradients with Doppler ultrasound is primarily due to the challenges and pitfalls of performing adult TTE.[9] One common error is the failure to align the CWD beam to the direction of the stenotic jet, thus creating an angulation error whereby CWD gradients are underestimated. The European Association of Cardiovascular Imaging/American Society

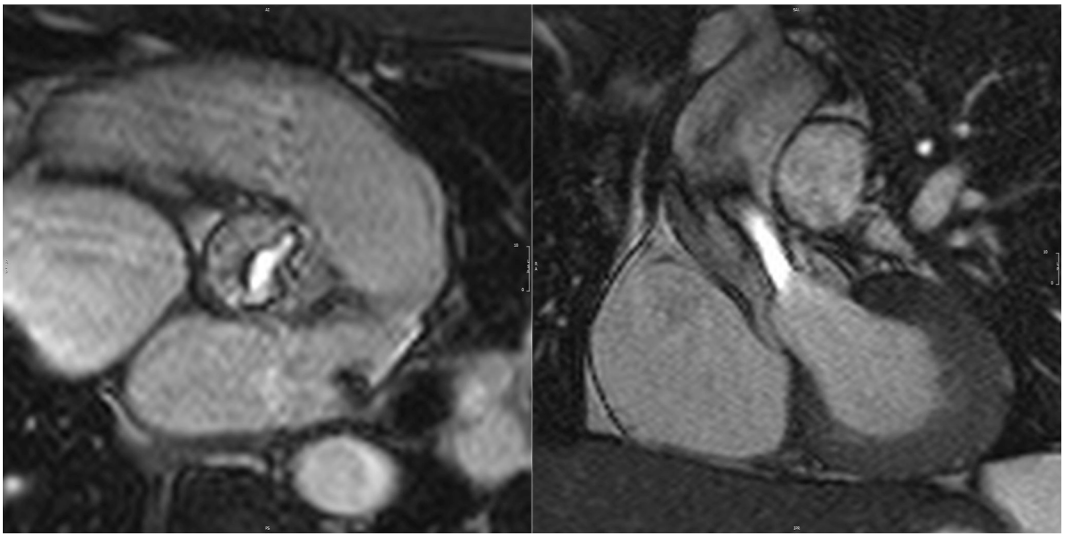


Figure 4 Cardiovascular magnetic resonance, balanced steady-state free processing sequence, basal short-axis systolic view at the level of the BAoV *(left)* and coronary left ventricular outflow tract systolic view *(right)*, demonstrates high-velocity flow across the stenotic BAoV.

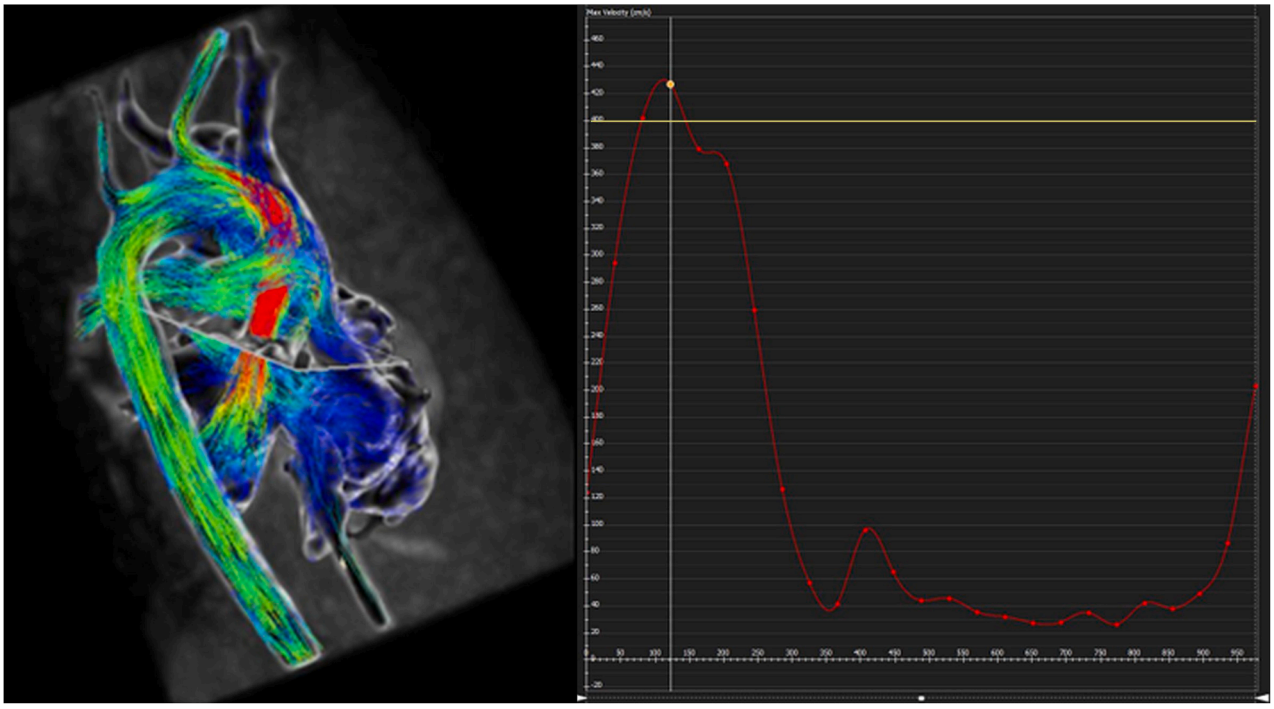


Figure 5 Cardiovascular magnetic resonance, 3-dimensional (3D) whole-heart volume-rendered reconstruction, oblique sagittal systolic display with superimposed time-resolved 4D flow *(left)* and time-velocity flow curve *(right)*, demonstrates the 3D velocity-encoded phase-contrast pattern with the high-velocity flow (*yellow line*; >4.0 m/s) across the BAoV (*blue*, low velocity; *green*, intermediate velocity; *red*, high velocity).

of Echocardiography 2017 guidelines[10] recommend the use of a dedicated small dual-crystal CWD transducer, especially when right parasternal and suprasternal views are used. One could speculate that using such a transducer could have revealed concordant high-gradient valvular AS in the case presented. However, using the nonimaging probe also comes with pitfalls as illustrated in a case report where gradients of a brachiocephalic stenosis were found mimicking severe structural valve deterioration.[11] Furthermore, both parasternal and suprasternal acoustic windows, in the present case, were adequate to make a good CWD estimation of gradients over the BAoV. We propose that the most plausible mechanism is that the BAoV jet was directed away from conventional apical and nonapical TTE acoustic

windows (e.g., toward the sternum), resulting in persistently low CWD gradients despite severe valve disease.

CONCLUSION

Underestimation of severe AS using Doppler ultrasound can have fatal consequences. Further diagnostic evaluation is warranted when discrepancies exist between clinical symptoms and reported severity of AS.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

FUNDING STATEMENT

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DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work the authors used google Co-pilot for language optimization. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

DISCLOSURE STATEMENT

The authors reported no actual or potential conflicts of interest relative to this document.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2026.06.006.

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