The Case of Asymptomatic Aortic Stenosis: Lessons in Patient Education and Early Symptom Recognition

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Aortic stenosis (AS) is one of the most common and clinically significant valvular heart diseases worldwide (1). Its progression is typically insidious, often reaching critical stages before symptoms become apparent. Patients may be completely asymptomatic for years, with the first clinical manifestation sometimes being sudden cardiac death, heart failure, or syncope (2). This silent progression poses challenges in early detection and timely intervention, often resulting in the need for invasive procedures.

Current guidelines recommend regular monitoring with transthoracic echocardiography and suggest aortic valve replacement (AVR) in symptomatic patients or those with evidence of left ventricular dysfunction (3,4). However, recent literature emphasizes the potential value of proactive surveillance, early symptom recognition, and shared decision-making, especially in high-risk individuals who could have asymptomatic Aortic stenosis (5,6).

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of AS	BAV (or other congenital valve anomaly) Aortic valve sclerosis	Aortic V_{max} <2 m/s with normal leaflet motion	None	None
B	Progressive AS	Mild to moderate leaflet calcification/fibrosis of a bicuspid or trileaflet valve with some reduction in systolic motion or Rheumatic valve changes with commissural fusion	Mild AS: aortic V_{max} 2.0–2.9 m/s or mean ΔP <20 mm Hg Moderate AS: aortic V_{max} 3.0–3.9 m/s or mean ΔP 20–39 mm Hg	Early LV diastolic dysfunction may be present Normal LVEF	None
C: Asymptomatic severe AS					
C1	Asymptomatic severe AS	Severe leaflet calcification/fibrosis or congenital stenosis with severely reduced leaflet opening	Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg AVA typically is ≤1.0 cm^2 (or AVAi 0.6 cm^2/m^2) but not required to define severe AS Very severe AS is an aortic V_{max} ≥5 m/s or mean P ≥60 mm Hg	LV diastolic dysfunction Mild LV hypertrophy Normal LVEF	None Exercise testing is reasonable to confirm symptom status
C2	Asymptomatic severe AS with LV systolic dysfunction	Severe leaflet calcification/fibrosis or congenital stenosis with severely reduced leaflet opening	Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg AVA typically ≤1.0 cm^2 (or AVAi 0.6 cm^2/m^2) but not required to define severe AS	LVEF <50%	None
D: Symptomatic severe AS					
D1	Symptomatic severe high-gradient AS	Severe leaflet calcification/fibrosis or congenital stenosis with severely reduced leaflet opening	Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg AVA typically ≤1.0 cm^2 (or AVAi ≤0.6 cm^2/m^2) but may be larger with mixed AS/AR	LV diastolic dysfunction LV hypertrophy Pulmonary hypertension may be present	Exertional dyspnea, decreased exercise tolerance, or HF Exertional angina Exertional syncope or presyncope
D2	Symptomatic severe low-flow, low-gradient AS with reduced LVEF	Severe leaflet calcification/fibrosis with severely reduced leaflet motion	AVA ≤1.0 cm^2 with resting aortic V_{max} <4 m/s or mean ΔP <40 mm Hg Dobutamine stress echocardiography shows AVA <1.0 cm^2 with V_{max} ≥4 m/s at any flow rate	LV diastolic dysfunction LV hypertrophy LVEF <50%	HF Angina Syncope or presyncope
D3	Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS	Severe leaflet calcification/fibrosis with severely reduced leaflet motion	AVA ≤1.0 cm^2 (indexed AVA ≤0.6 cm^2/m^2) with an aortic V_{max} <4 m/s or mean ΔP <40 mm Hg AND Stroke volume index <35 mL/m^2 Measured when patient is normotensive (systolic blood pressure <140 mm Hg)	Increased LV relative wall thickness Small LV chamber with low stroke volume Restrictive diastolic filling LVEF ≥50%	HF Angina Syncope or presyncope

Incorporating risk stratification tools and biomarkers such as NT-proBNP or global longitudinal strain (GLS) may aid in identifying patients who would benefit from earlier intervention (7,8). Exercise testing plays a crucial role in uncovering symptoms in patients who may underreport them or attribute them to aging or comorbid conditions. In asymptomatic individuals with severe aortic stenosis (AS), findings such as a decline in blood pressure or diminished exercise capacity during testing serve as indicators for proceeding with aortic valve replacement (AVR) (9,10).

During physical examination, auscultation alone has limited sensitivity in detecting valvular heart disease, particularly during its early or moderate stages. Consequently, many cases of asymptomatic AS are uncovered incidentally, often during preoperative assessments, evaluations for unrelated cardiovascular symptoms, or as part of routine echocardiographic screening in populations such as the elderly or those with known cardiovascular comorbidities (11). Current guidelines from the American College of Cardiology and the American Heart Association (ACC/AHA) recommend aortic valve replacement (AVR) in patients without symptoms when the left ventricular ejection fraction (LVEF) drops below 50%, when another cardiac surgery is scheduled, or when exercise testing provokes symptoms or hypotension (11). Beyond these clear-cut indications, AVR may also be appropriate in cases demonstrating rapid hemodynamic progression—defined by an increase in aortic jet velocity exceeding 0.3 m/s per year—or when biomarkers such as B-type natriuretic peptide (BNP) exceed three times the normal limit or peak aortic velocity surpasses 5.0 m/s (11). This is based on the low annual risk of sudden death (<1% per year) in this population and the potential risks associated with valve intervention. There is still an ongoing debate regarding the optimal timing of aortic valve replacement in asymptomatic severe aortic stenosis. The balance between procedural risk and long-term benefit of early AVR is not uniform across all asymptomatic patients, and current evidence does not definitively support a universal early intervention strategy (12).

New-onset atrial fibrillation (AF) is one of the most frequent arrhythmic complications following surgical aortic valve replacement (SAVR), generally arising within 2–4 days after the procedure and affecting roughly one-third to one-half of patients, an incidence higher than that observed after transfemoral transcatheter aortic valve replacement (TAVR) (13,14). The arrhythmia is thought to result from a combination of perioperative sympathetic activation, myocardial and pericardial inflammation due to pericardiotomy, atrial trauma, abrupt postoperative changes in loading conditions, and electrolyte shifts such as diuretic-induced hypokalemia (13,14). Larger preoperative left atrial dimensions and an exaggerated postoperative inflammatory response have been identified as independent predictors, while advanced age and marked atrial enlargement increase the risk of persistent AF beyond the early postoperative period (15,16). Perioperative AF has been linked to greater in-hospital mortality, heightened stroke risk, prolonged hospitalization, higher likelihood of late AF recurrence, and reduced long-term survival (13,16,17). In severe aortic stenosis (AS), the coexistence of AF, either preceding or following valve intervention, is associated with poorer outcomes, reflecting the reciprocal relationship between AF and heart failure (16,18–20).

Review:

This case report illustrates an uncommon yet clinically important scenario: a relatively younger, asymptomatic patient with severe aortic stenosis (AS) discovered incidentally during a routine examination, who later developed postoperative atrial fibrillation (AF) after surgical aortic valve replacement (SAVR). The article does a good job of summarizing current guidelines and emphasizing that AS often progresses without symptoms, which makes timely diagnosis challenging.

The authors reinforce that while auscultation can detect advanced disease, it has limited sensitivity in earlier stages, and confirmatory imaging with echocardiography remains essential. The discussion of biomarkers (NT-proBNP, GLS) and exercise testing provides practical tools for risk stratification in asymptomatic patients. The section on postoperative AF is particularly useful, explaining its causes, risk factors, and clinical impact.

Strengths of the paper include its grounding in evidence-based guidelines, clear linkage between the case and broader clinical principles, and emphasis on preventive detection. A limitation is that it could expand on the rationale for choosing surgical rather than transcatheter AVR in this patient and provide more detail on the AF management strategy used postoperatively.

Case Presentation:

A 58-year-old physically active male with no significant past medical history presented for a routine health checkup at his wife's request. He reported no chest pain, dyspnea, syncope, palpitations, fatigue, or exercise intolerance. His vitals are: HR: 92, BP: 130/78, RR: 16, SpO2: 97% on room air. Physical examination revealed a grade III/VI systolic ejection murmur best heard at the right upper sternal border with radiation to the carotids.

He was referred to cardiology, where a 12-lead ECG demonstrated left ventricular hypertrophy. Transthoracic echocardiography confirmed severe aortic stenosis (aortic valve area <1.0 cm^2, mean transvalvular gradient ≥40 mmHg) and concentric left ventricular hypertrophy with preserved ejection fraction. He subsequently underwent surgical aortic valve replacement. Intraoperatively, he developed atrial flutter, which evolved into permanent atrial fibrillation postoperatively, requiring rate control and anticoagulation.

Discussion:

Aortic stenosis typically presents with symptoms of fatigue, shortness of breath, and exertional dyspnea or even sudden cardiac death. (21) It can also present as insidious chest pain, especially in young adults, and can even be asymptomatic in older individuals, as it presented in the patient who presented to us. (22)

- *Initial diagnostic testing (23):*

Recommendations for Diagnostic Testing: Initial Diagnosis of AS Referenced studies that support the recommendations are summarized in Online Data Supplement 3.		
COR	**LOE**	**Recommendations**
1	A	1. In patients with signs or symptoms of AS or a BAV, TTE is indicated for accurate diagnosis of the cause of AS, assessment of hemodynamic severity, measurement of LV size and systolic function, and determination of prognosis and timing of valve intervention.[1,2]
1	B-NR	2. In patients with suspected low-flow, low-gradient severe AS with normal LVEF (Stage D3), optimization of blood pressure control is recommended before measurement of AS severity by TTE, TEE, cardiac catheterization, or CMR.[3–7]
2a	B-NR	3. In patients with suspected low-flow, low-gradient severe AS with reduced LVEF (Stage D2), low-dose dobutamine stress testing with echocardiographic or invasive hemodynamic measurements is reasonable to further define severity and assess contractile reserve.[8–10]
2a	B-NR	4. In patients with suspected low-flow, low-gradient severe AS with normal or reduced LVEF (Stages D2 and D3), calculation of the ratio of the outflow tract to aortic velocity is reasonable to further define severity.[1,11–13]
2a	B-NR	5. In patients with suspected low-flow, low-gradient severe AS with normal or reduced LVEF (Stages D2 and D3), measurement of aortic valve calcium score by CT imaging is reasonable to further define severity.[14–18]

- *Changing signs or symptoms*

  In patients with known valvular aortic stenosis, a repeat transthoracic echocardiogram is a good idea when physical examination shows an increase in loudness of murmur, murmur peaks later in systole, the A2 component is diminished or absent in the second heart sound, or symptoms occur that may be attributable to Aortic stenosis. Repeat transthoracic echocardiogram should also be done in patients with increased hemodynamic demands, like noncardiac surgery, pregnancy, systemic infection, anemia, or gastrointestinal bleeding. (23)

- *Routine follow-up:*

  It is important to carry out periodic clinical evaluation of asymptomatic patients with severe aortic stenosis, as the symptom onset is insidious and may not be recognized by the patient. (23)

- *Cardiac catheterisation:*

  When data from noninvasive testing are nondiagnostic or if there is a discrepancy between clinical and echocardiographic evaluation, cardiac catheterization for the determination of the severity of AS can be helpful. Transaortic pressure gradient recordings allow measurement of the mean transaortic gradient via simultaneous LV and aortic pressure measurements. The aortic valve area is calculated using the Gorlin formula by using a Fick or thermodilution cardiac output measurement. (24)

- *Exercise testing (23):*

Recommendations for Diagnostic Testing: Exercise Testing in Patients With AS Referenced studies that support the recommendations are summarized in Online Data Supplement 4.		
COR	**LOE**	**Recommendations**
2a	**B-NR**	1. In asymptomatic patients with severe AS (Stage C1), exercise testing is reasonable to assess physiological changes with exercise and to confirm the absence of symptoms.[1–4]
3: Harm	**B-NR**	2. In symptomatic patients with severe AS (Stage D1, aortic velocity ≥4.0 m/s or mean pressure gradient ≥40 mm Hg), exercise testing should not be performed because of the risk of severe hemodynamic compromise.[5]

Treatment of Aortic Stenosis:

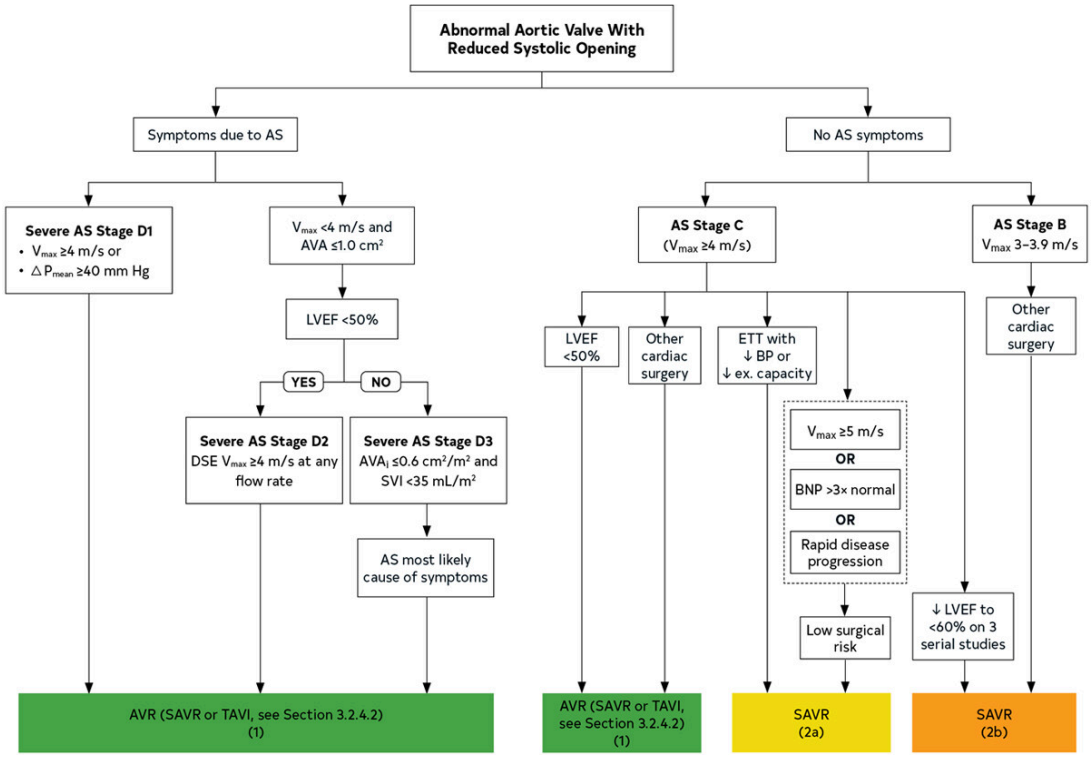
- *Medical therapy(23):*

Recommendations for Medical Therapy of AS Referenced studies that support the recommendations are summarized in Online Data Supplement 5.		
COR	**LOE**	**Recommendations**
1	**B-NR**	1. In patients at risk of developing AS (Stage A) and in patients with asymptomatic AS (Stages B and C), hypertension should be treated according to standard GDMT, started at a low dose, and gradually titrated upward as needed, with appropriate clinical monitoring.[1–3]
1	**A**	2. In all patients with calcific AS, statin therapy is indicated for primary and secondary prevention of atherosclerosis on the basis of standard risk scores.[4–6]
2b	**B-NR**	3. In patients who have undergone TAVI, renin–angiotensin system blocker therapy (ACE inhibitor or ARB) may be considered to reduce the long-term risk of all-cause mortality.[7,8]
3: No Benefit	**A**	4. In patients with calcific AS (Stages B and C), statin therapy is not indicated for prevention of hemodynamic progression of AS.[4–6]

- *Timing of intervention (23):*

Recommendations for Timing of Intervention of AS Referenced studies that support the recommendations are summarized in Online Data Supplements 4 and 5 to 10.		
COR	**LOE**	**Recommendations**
1	A	1. In adults with severe high-gradient AS (Stage D1) and symptoms of exertional dyspnea, HF, angina, syncope, or presyncope by history or on exercise testing, AVR is indicated.[1–7]
1	B-NR	2. In asymptomatic patients with severe AS and an LVEF <50% (Stage C2), AVR is indicated.[8–11]
1	B-NR	3. In asymptomatic patients with severe AS (Stage C1) who are undergoing cardiac surgery for other indications, AVR is indicated.[12–16]
1	B-NR	4. In symptomatic patients with low-flow, low-gradient severe AS with reduced LVEF (Stage D2), AVR is recommended.[17–24]
1	B-NR	5. In symptomatic patients with low-flow, low-gradient severe AS with normal LVEF (Stage D3), AVR is recommended if AS is the most likely cause of symptoms.[25–27]
2a	B-NR	6. In apparently asymptomatic patients with severe AS (Stage C1) and low surgical risk, AVR is reasonable when an exercise test demonstrates decreased exercise tolerance (normalized for age and sex) or a fall in systolic blood pressure of ≥10 mm Hg from baseline to peak exercise.[13,28–30]
2a	B-R	7. In asymptomatic patients with very severe AS (defined as an aortic velocity of ≥5 m/s) and low surgical risk, AVR is reasonable.[15,31–35]
2a	B-NR	8. In apparently asymptomatic patients with severe AS (Stage C1) and low surgical risk, AVR is reasonable when the serum B-type natriuretic peptide (BNP) level is >3 times normal.[32,36–38]
2a	B-NR	9. In asymptomatic patients with high-gradient severe AS (Stage C1) and low surgical risk, AVR is reasonable when serial testing shows an increase in aortic velocity ≥0.3 m/s per year.[39,40]
2b	B-NR	10. In asymptomatic patients with severe high-gradient AS (Stage C1) and a progressive decrease in LVEF on at least 3 serial imaging studies to <60%, AVR may be considered.[8–11,33]
2b	C-EO	11. In patients with moderate AS (Stage B) who are undergoing cardiac surgery for other indications, AVR may be considered.

The table above shows what interventions need to be carried out at what degree of aortic stenosis. The choice of treatment is Transcatheter aortic valve replacement/implantation or Surgical Aortic valve replacement. Surgical aortic valve replacement is more invasive and can be carried out in patients with Vmax more than 5 or left ventricular ejection fraction less than 60% on 3 serial studies. (24)



- *Mechanical or bi-prosthetic Aortic valve replacement(23):*

Recommendations for Choice of Mechanical Versus Bioprosthetic AVR Referenced studies that support the recommendations are summarized in Online Data Supplements 11 and 12.		
COR	**LOE**	**Recommendations**
1	C-EO	1. In patients with an indication for AVR, the choice of prosthetic valve should be based on a shared decision-making process that accounts for the patient's values and preferences and includes discussion of the indications for and risks of anticoagulant therapy and the potential need for and risks associated with valve reintervention.
1	C-EO	2. For patients of any age requiring AVR for whom VKA anticoagulant therapy is contraindicated, cannot be managed appropriately, or is not desired, a bioprosthetic AVR is recommended.
2a	B-R	3. For patients <50 years of age who do not have a contraindication to anticoagulation and require AVR, it is reasonable to choose a mechanical aortic prosthesis over a bioprosthetic valve.[1]
2a	B-NR	4. For patients 50 to 65 years of age who require AVR and who do not have a contraindication to anticoagulation, it is reasonable to individualize the choice of either a mechanical or bioprosthetic AVR with consideration of individual patient factors and after informed shared decision-making.[1–10]
2a	B-R	5. In patients >65 years of age who require AVR, it is reasonable to choose a bioprosthesis over a mechanical valve.[1]
2b	B-NR	6. In patients <50 years of age who prefer a bioprosthetic AVR and have appropriate anatomy, replacement of the aortic valve by a pulmonic autograft (the Ross procedure) may be considered at a Comprehensive Valve Center.[11–13]

What makes this case unique is that this patient was incidentally found to have severe Aortic stenosis, which led to him undergoing surgical aortic valve replacement. Had he not been diagnosed in time and undergone intervention, there could have been dire consequences as serious as sudden cardiac death(22). This also shows the importance of a thorough routine physical examination and how it can help in the prevention of disease.

Conclusion:

This case emphasizes that severe aortic stenosis may remain clinically silent until advanced stages, reinforcing the necessity of systematic cardiovascular evaluation in at-risk or even ostensibly low-risk populations. Routine clinical examination, when coupled with confirmatory imaging, can facilitate early diagnosis and timely intervention, potentially avoiding catastrophic outcomes such as sudden cardiac death.

The development of postoperative atrial fibrillation in this patient highlights its role as a major complication of surgical aortic valve replacement, carrying serious consequences both in the immediate recovery period and over the long term. Identifying risk factors before surgery, applying effective preventive strategies, and maintaining structured postoperative follow-up are crucial to improving patient outcomes. Overall, this case demonstrates the value of a proactive and individualized strategy for diagnosing and managing severe AS; one that combines guideline-based treatment with decisions tailored to each patient's needs and circumstances.

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