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When Heyde Syndrome is Not the Whole Story: Severe Aortic Stenosis Revealing Probable Transthyretin Cardiac Amyloidosis

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Abstract | **Case Report**

Background: Heyde syndrome is characterized by the association of severe aortic stenosis, gastrointestinal bleeding from intestinal angiodysplasia, and acquired von Willebrand syndrome. In elderly patients, severe calcific aortic stenosis may also coexist with transthyretin cardiac amyloidosis, a combination associated with advanced myocardial dysfunction and adverse outcomes. ***Case presentation:*** An 80-year-old man with hypertension, insulin-treated type 2 diabetes mellitus, peripheral arterial disease, chronic obstructive pulmonary disease, end-stage renal disease on maintenance hemodialysis, and recurrent gastrointestinal bleeding from previously documented small-bowel and colonic angiodysplasia was admitted for progressive dyspnea, recurrent melena, and symptomatic anemia. Echocardiography demonstrated severe calcific aortic stenosis, concentric biventricular hypertrophy disproportionate to pressure overload, advanced diastolic dysfunction, biatrial enlargement, a granular sparkling myocardial appearance, a small pericardial effusion, and a dilated non-collapsible inferior vena cava. Electrocardiography showed atrial fibrillation with low QRS voltage, creating a voltage-mass discrepancy highly suggestive of cardiac amyloidosis. The patient received two units of packed red blood cells by slow transfusion and was transferred to the cardiac intensive care unit. Despite diuretics, maintenance hemodialysis with controlled ultrafiltration, antibiotic therapy, oxygen supplementation, and escalating inotropic and vasopressor support, he developed refractory cardiogenic shock characterized by severely depressed cardiac output and markedly elevated left ventricular filling pressures. He died before cardiac magnetic resonance imaging, bone scintigraphy, or tissue biopsy could be performed. ***Conclusion:*** This case highlights the need to consider concomitant transthyretin cardiac amyloidosis in elderly patients with severe aortic stenosis and Heyde syndrome when ventricular hypertrophy is disproportionate or accompanied by electrocardiographic and echocardiographic red flags. Early recognition of this high-risk phenotype may improve diagnostic assessment, prognostic stratification, and therapeutic decision-making.

Keywords: Heyde syndrome; severe aortic stenosis; transthyretin cardiac amyloidosis; gastrointestinal angiodysplasia; cardiogenic shock; multimodality imaging.

1. INTRODUCTION

Severe calcific aortic stenosis (AS) is the most prevalent valvular heart disease in older adults and remains a major cause of morbidity and mortality. Among its less frequent but clinically significant complications is Heyde syndrome, a clinical entity characterized by the triad of severe AS, gastrointestinal bleeding due to intestinal angiodysplasia, and acquired von Willebrand syndrome. High shear stress across the stenotic aortic valve promotes ADAMTS13-mediated proteolysis of highmolecular-weight von Willebrand factor multimers, resulting in impaired primary hemostasis and recurrent gastrointestinal bleeding [1–3].

Growing evidence indicates that transthyretin cardiac amyloidosis (ATTR-CA) frequently coexists with severe calcific AS, particularly in elderly patients referred for transcatheter or surgical aortic valve replacement, with a reported prevalence ranging from 8% to 16% [4–6]. Because both conditions share overlapping clinical and echocardiographic features—including increased left ventricular wall thickness, restrictive filling physiology, low-flow states, and heart failure—ATTR-CA often remains underrecognized. Failure to identify this dual pathology may significantly influence therapeutic decision-making and adversely affect prognosis [4–7].

The coexistence of Heyde syndrome and ATTR-CA in patients with severe AS represents an exceptionally uncommon and diagnostically challenging clinical scenario. Although each condition has been increasingly recognized individually, reports describing their simultaneous occurrence remain scarce [5,6,8].

We report the case of an elderly patient with recurrent gastrointestinal bleeding secondary to documented angiodysplasia in the setting of severe calcific aortic stenosis, consistent with Heyde syndrome. Transthoracic echocardiography revealed several features suggestive of concomitant transthyretin cardiac amyloidosis, including disproportionate biventricular hypertrophy, restrictive filling physiology, biatrial enlargement, and a small pericardial effusion. Despite intensive supportive treatment, the patient rapidly deteriorated and died from refractory cardiogenic shock before confirmatory investigations and definitive therapeutic intervention could be performed. This case underscores the importance of actively screening for cardiac amyloidosis in elderly patients with severe aortic stenosis and Heyde syndrome, particularly when characteristic electrocardiographic and echocardiographic red flags are present, as this association may identify a particularly high-risk clinical phenotype.

2. CASE REPORT

An 80-year-old man with a medical history of hypertension, insulin-treated type 2 diabetes mellitus, peripheral arterial disease treated with aortofemoral bypass surgery in 1997, chronic obstructive pulmonary disease, and end-stage renal disease requiring maintenance hemodialysis for the preceding six months was admitted to our cardiac intensive care unit for progressive dyspnea and acute decompensated heart failure. His history was also notable for recurrent gastrointestinal bleeding secondary to small bowel and colonic angiodysplasia, previously managed with two sessions of argon plasma coagulation (APC), consistent with Heyde syndrome.

Over the previous six months, he experienced progressive exertional dyspnea, worsening from New York Heart Association (NYHA) functional class III to IV, associated with marked fatigue and progressive functional decline. Two months before admission, recurrent melena recurred, requiring repeat gastroenterological evaluation. Although a third APC session was planned, it was postponed because of worsening heart failure, prompting referral to our department. He denied chest pain, syncope, presyncope, or palpitations.

Careful history taking revealed no extracardiac red flags suggestive of transthyretin cardiac amyloidosis, including bilateral carpal tunnel syndrome, lumbar spinal stenosis, spontaneous distal biceps tendon rupture, peripheral or autonomic neuropathy, unexplained orthostatic hypotension, or a family history of amyloidosis or unexplained cardiomyopathy.

On admission, the patient was hypotensive, with a blood pressure of 95/40 mmHg, and was in atrial fibrillation with a ventricular rate of 90 beats/min. Oxygen saturation was 96% on room air. Physical examination demonstrated a harsh late-peaking ejection systolic murmur, best heard at the right upper sternal border and radiating to both carotid arteries, with markedly diminished second heart sound. Signs of congestive heart failure included bilateral basal pulmonary crackles and mild bilateral ankle edema, while no clinical evidence of peripheral hypoperfusion was observed.

Electrocardiography showed atrial fibrillation with a controlled ventricular response (87 beats/min), low QRS voltage in the limb leads despite the subsequently documented increase in left ventricular wall thickness, right-axis deviation, and nonspecific ST-T wave abnormalities. No conduction disturbances or pathological Q waves were present **(Figure 1).**

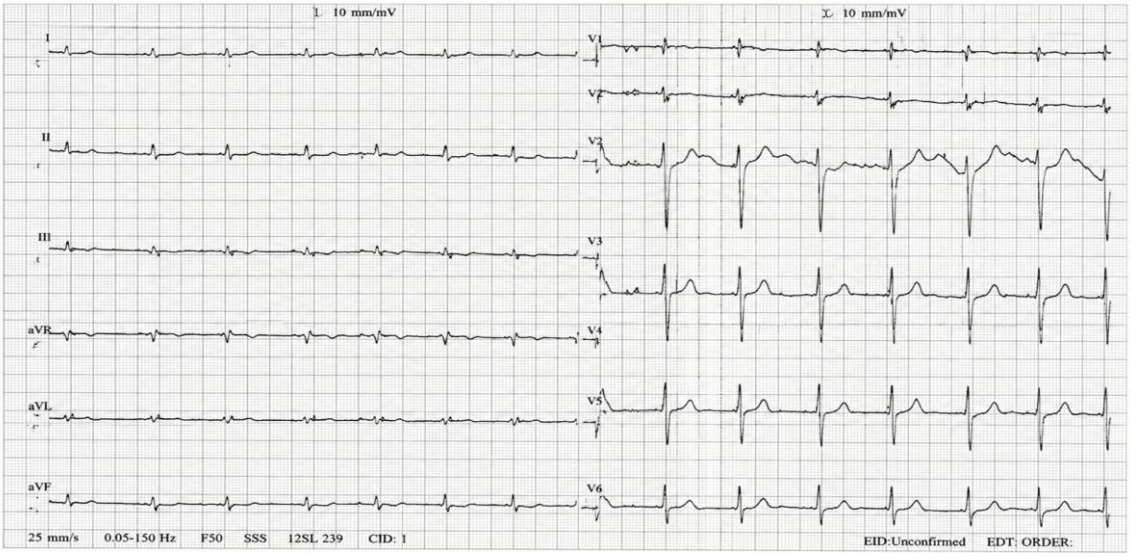

Figure 1: Baseline 12-lead electrocardiogram showing atrial fibrillation with a controlled ventricular response (87 beats/min), low QRS voltage in the limb leads, and right-axis deviation. The combination of low limb-lead QRS voltage with increased left ventricular wall thickness on echocardiography represents a voltage–mass discrepancy suggestive of an infiltrative cardiomyopathy

Chest radiography demonstrated cardiomegaly with bilateral hilar vascular congestion and a right-sided pulmonary infiltrate, raising suspicion of concomitant pneumonia (Figure 2).

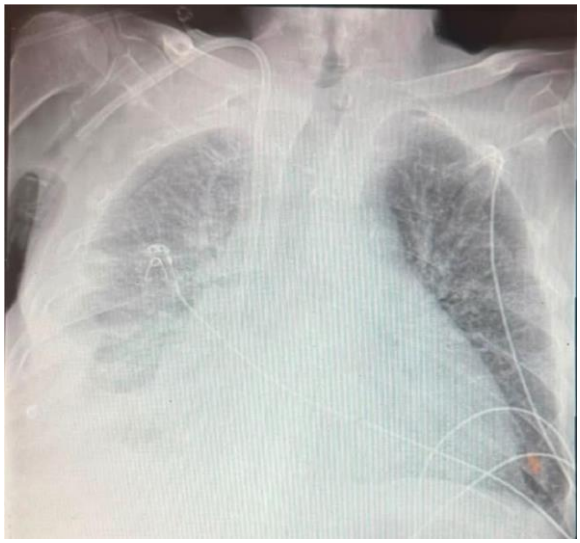

Figure 2: Admission posteroanterior chest radiograph demonstrating cardiomegaly with bilateral pulmonary vascular congestion and a right basal pulmonary opacity suggestive of concomitant pneumonia. A tunneled right internal jugular hemodialysis catheter is also visible

Laboratory investigations revealed microcytic hypochromic anemia (hemoglobin 8.6 g/dL, MCV 70 fL), elevated inflammatory markers (CRP 100 mg/L) with a normal procalcitonin level (0.03 ng/mL), markedly elevated NT-proBNP (>70,000 pg/mL), mildly elevated high-sensitivity cardiac troponin (100 ng/L), and end-stage renal disease requiring maintenance hemodialysis. Serum free light-chain analysis showed elevated κ and λ concentrations with a preserved κ/λ ratio (0.56). Serum protein electrophoresis demonstrated polyclonal hypergammaglobulinemia, while complement levels (C3 and C4) were normal and peripheral blood smear showed no circulating plasma cells. The main laboratory findings are summarized in **Table 1**.

Table 1: Laboratory findings on admission

Parameter	Result	Reference range / Therapeutic target
Complete blood count		
Hemoglobin	**8.6 g/dL**	13.0–17.0 g/dL
Hematocrit	**22%**	40–52%
Mean corpuscular volume (MCV)	**70 fL**	80–100 fL
White blood cell count	10,000/µL	4,000–10,000/µL
Neutrophils	7 ,000/µL (70%)	1,500 –7,500/µL
Inflammatory markers		
C-reactive protein (CRP)	**100 mg/L**	<5 mg/L
Procalcitonin (PCT)	0.03 ng/mL	<0.05 ng/mL
Lactate dehydrogenase (LDH)	200 U/L	120 –250 U/L
Iron studies		
Ferritin	**20 ng/mL**	30 –400 ng/mL
Coagulation profile		
International normalized ratio (INR)	2.0	2.0–3.0
Cardiac biomarkers		
High-sensitivity cardiac troponin	**100 ng/L**	<14 ng/L
NT-proBNP	**>70,000 pg/mL**	<450 pg/mL*
Renal function		
Urea	**1.20 g/L**	0.15–0.45 g/L
Creatinine	**18 mg/dL**	0.70–1.30 mg/dL
Estimated glomerular filtration rate (eGFR)	**7 mL/min/1.73 m²**	>90 mL/min/1.73 m²
Electrolytes		
Sodium	137 mmol/L	135–145 mmol/L
Potassium	4.0 mmol/L	3.5 –5.1 mmol/L

Liver function tests		
Aspartate aminotransferase (AST)	35 U/L	<40 U/L
Alanine aminotransferase (ALT)	35 U/L	<41 U/L
Monoclonal protein screening		
Serum κ free light chains	314	2.37-20.73
Serum λ free light chains	393.92	4.23-27.69
κ/λ ratio	0.56	0.26–1.65
Serum protein electrophoresis	Polyclonal hypergammaglobulinemia	No monoclonal protein
Peripheral blood smear	No circulating plasma cells	Absent
Complement studies		
Complement C3	100 mg/dL	90–180 mg/dL
Complement C4	30 mg/dL	10–40 mg/dL

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; INR, international normalized ratio; LDH, lactate dehydrogenase; MCV, mean corpuscular volume; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PCT, procalcitonin.

Reference value in the general population (age ≥75 years): <450 pg/mL. In patients receiving maintenance hemodialysis, no validated reference range exists because NT-proBNP is chronically elevated; values should be interpreted in the clinical context.

Transthoracic echocardiography **(Figure 3)** demonstrated concentric left ventricular hypertrophy (indexed left ventricular mass 129 g/m²; interventricular septum 17 mm; posterior wall thickness 15 mm) with preserved left ventricular systolic function (LVEF 63%). The aortic valve was severely thickened and calcified, consistent with high-gradient severe aortic stenosis (Vmax 4.36 m/s, mean gradient 47 mmHg, aortic valve area 0.69 cm² [indexed AVA 0.40 cm²/m²]) associated with a low-flow state (stroke volume index 34 mL/m²) despite preserved ejection fraction.

Additional echocardiographic findings included biatrial enlargement, right ventricular hypertrophy, a small circumferential pericardial effusion, and advanced left ventricular diastolic dysfunction with markedly elevated filling pressures. The interventricular septum exhibited a granular ("sparkling") appearance, and pulmonary hypertension was suggested by an estimated pulmonary artery systolic pressure of 68 mmHg, together with a dilated inferior vena cava without inspiratory collapse. Speckle-tracking strain analysis could not be performed because of atrial fibrillation.

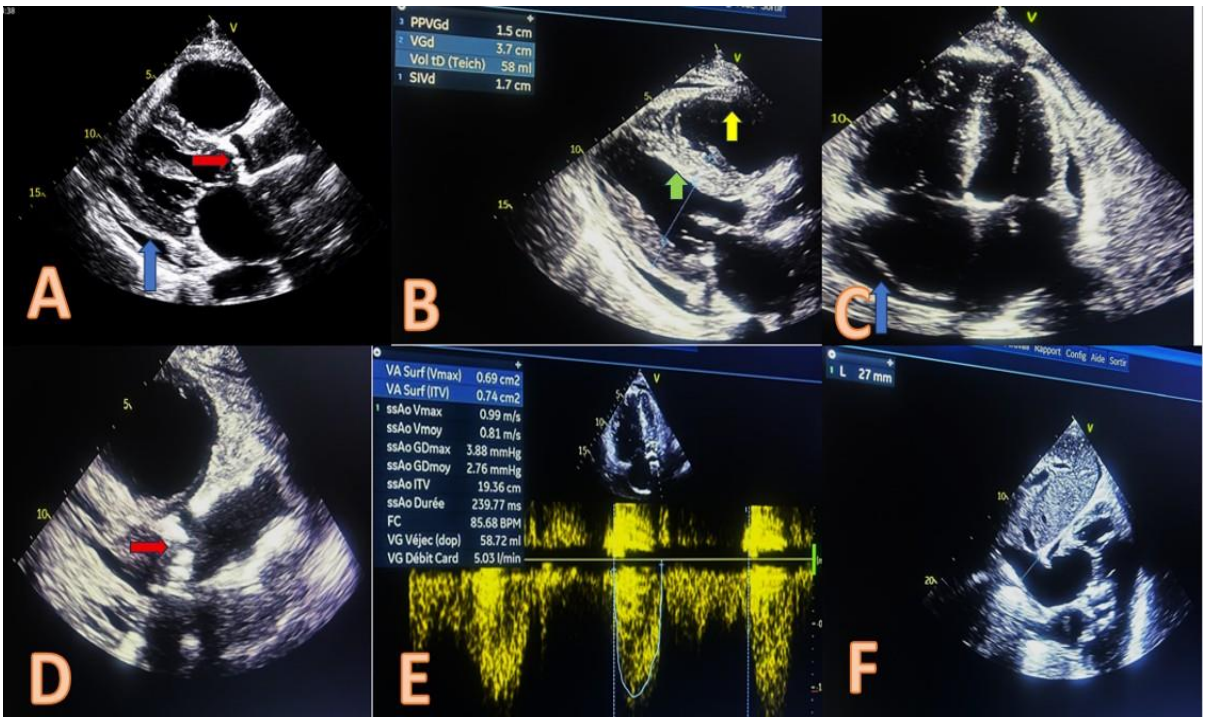

Figure 3: Transthoracic echocardiographic findings of severe calcific aortic stenosis with imaging features highly suggestive of concomitant cardiac amyloidosis

Legend:

A. Parasternal long-axis view demonstrating a heavily calcified aortic valve with severely restricted leaflet opening (red arrow) and a small pericardial effusion adjacent to the left ventricle (blue arrow). (B) Parasternal long-axis view showing concentric left ventricular hypertrophy (green arrow), right ventricular hypertrophy (yellow arrow), a granular sparkling appearance of the interventricular septum, and extensive calcification of the aortic valve and ascending aorta, findings highly suggestive of cardiac amyloidosis.
B. Apical four-chamber view demonstrating biventricular hypertrophy with biatrial enlargement, supporting the suspicion of an infiltrative cardiomyopathy.
C. Zoomed parasternal long-axis view highlighting the markedly calcified aortic valve with severely restricted systolic leaflet excursion (red arrow)
D. Pulsed-wave Doppler of the left ventricular outflow tract velocity–time integral (LVOT VTI) used in the continuity equation to calculate the aortic valve area (AVA = 0.69 cm^2), confirming severe aortic stenosis.
E. Subcostal view demonstrating a dilated inferior vena cava (27 mm) without inspiratory collapse, consistent with elevated right atrial pressure.

Overall, the echocardiographic findings—including severe calcific aortic stenosis, concentric biventricular hypertrophy disproportionate to the degree of pressure overload, a granular sparkling appearance of the interventricular septum, biatrial enlargement, advanced diastolic dysfunction, a small circumferential pericardial effusion, and a dilated non-collapsible inferior vena cava—together with the low-voltage electrocardiographic pattern, raised a strong suspicion of concomitant transthyretin cardiac amyloidosis and prompted further multimodality diagnostic evaluation.

Because of recurrent melena associated with symptomatic iron-deficiency anemia, the patient initially received two units of packed red blood cells administered by slow transfusion, together with proton pump inhibitor therapy and close hemodynamic monitoring. Owing to the presence of acute decompensated heart failure, severe symptomatic aortic stenosis, hypotension, and end-stage renal disease requiring maintenance hemodialysis, transfusion was deliberately performed slowly to minimize the risk of volume overload.

The patient was subsequently admitted to the cardiac intensive care unit, where he received comprehensive supportive management, including intravenous loop diuretics, cautious vasodilator therapy as tolerated, maintenance hemodialysis with controlled ultrafiltration, broad-spectrum antibiotic therapy for suspected concomitant pneumonia, oxygen supplementation, and intravenous inotropic support after the onset of hemodynamic deterioration.

Urgent colonoscopy was not performed because the patient was hemodynamically unstable, with acute decompensated heart failure and severe calcific aortic stenosis, making bowel preparation and endoscopic sedation unsafe. In addition, the gastrointestinal bleeding was not massive or uncontrolled at the time of admission and was considered most likely related to previously documented intestinal angiodysplasia. Consequently, priority was given to cardiovascular stabilization, with the intention of repeating endoscopic evaluation once clinical improvement had been achieved.

Despite these measures, the patient's condition deteriorated rapidly, progressing to refractory cardiogenic shock characterized by severely depressed cardiac output and markedly elevated left ventricular filling pressures. Advanced resuscitative measures, including escalation of inotropic and vasopressor support, optimization of preload and afterload, repeated hemodynamic reassessment, and continued renal replacement therapy, failed to restore adequate systemic perfusion. The patient ultimately died from refractory cardiogenic shock with end-stage pump failure.

Consequently, confirmatory investigations for cardiac amyloidosis, including cardiac magnetic resonance imaging, 99mTc-pyrophosphate (or HMDP/DPD, according to local availability) bone scintigraphy, and minor salivary gland biopsy, could not be performed before his death. The diagnosis therefore remained probable transthyretin cardiac amyloidosis, based on the combination of highly suggestive clinical, electrocardiographic, and echocardiographic findings.

3. DISCUSSION

Heyde syndrome is a well-recognized but frequently underdiagnosed association between severe calcific aortic stenosis, gastrointestinal bleeding secondary to intestinal angiodysplasia, and acquired von Willebrand syndrome. High shear stress across the stenotic aortic valve induces conformational changes in circulating von Willebrand factor (vWF), exposing high-molecular-weight multimers to proteolytic cleavage by ADAMTS13. The resulting deficiency of these multimers impairs primary hemostasis and predisposes patients to recurrent bleeding from fragile gastrointestinal

angiodysplastic lesions, particularly in elderly individuals. Restoration of normal valve hemodynamics following surgical or transcatheter aortic valve replacement (TAVR) often corrects the acquired vWF abnormality and substantially reduces recurrent gastrointestinal bleeding, further supporting the causal relationship between severe aortic stenosis and Heyde syndrome [1–4].

Our patient fulfilled the classical clinical triad of Heyde syndrome, presenting with severe symptomatic calcific aortic stenosis, recurrent gastrointestinal

bleeding due to previously documented intestinal angiodysplasia treated by argon plasma coagulation, and chronic irondeficiency anemia requiring repeated blood transfusions. Although recurrent gastrointestinal bleeding is frequently attributed solely to severe aortic stenosis, this case illustrates that additional cardiac pathology may coexist and substantially influence both prognosis and management.

In recent years, increasing evidence has demonstrated that transthyretin cardiac amyloidosis (ATTRCA) frequently coexists with degenerative calcific aortic stenosis in elderly patients. Prospective studies have reported a prevalence ranging from approximately 8% to 16% among patients referred for TAVR, establishing the coexistence of severe aortic stenosis and ATTR-CA (AS-ATTR) as a distinct clinical entity rather than a coincidental finding [5–7]. Patients with AS-ATTR are typically older men with increased ventricular wall thickness, restrictive physiology, conduction abnormalities, elevated natriuretic peptides, and worse clinical outcomes than patients with isolated aortic stenosis. Because both conditions share overlapping manifestations—including heart failure, left ventricular hypertrophy, and diastolic dysfunction—the diagnosis of concomitant cardiac amyloidosis is frequently overlooked unless clinicians actively search for characteristic imaging and electrocardiographic "red flags."

Several findings in our patient strongly suggested an infiltrative cardiomyopathy beyond pressure overload hypertrophy alone. Echocardiography demonstrated concentric biventricular hypertrophy disproportionate to the severity of pressure overload, a granular sparkling appearance of the interventricular septum, severe biatrial enlargement, advanced diastolic dysfunction, a small circumferential pericardial effusion, and a dilated non-collapsible inferior vena cava.

Electrocardiography revealed atrial fibrillation with low QRS voltage despite markedly increased ventricular wall thickness, producing the classic voltage-mass discrepancy that represents one of the most valuable clues to cardiac amyloidosis. Although none of these findings is individually diagnostic, their coexistence markedly increases the probability of ATTR-CA and should prompt further multimodality evaluation using cardiac magnetic resonance imaging and bone scintigraphy, as recommended by contemporary ESC guidelines [7–10].

Recognizing concomitant ATTR-CA has important prognostic and therapeutic implications. Unlike isolated aortic stenosis, the coexistence of myocardial amyloid infiltration results in progressive restrictive cardiomyopathy characterized by impaired ventricular compliance, elevated filling pressures, reduced myocardial contractile reserve, atrial remodeling, right ventricular involvement, and conduction system disease. Consequently, symptoms may appear disproportionate to the degree of valvular obstruction, and heart failure often progresses despite apparently preserved left ventricular ejection fraction. Furthermore, several studies have demonstrated that concomitant ATTR-CA independently predicts increased mortality, recurrent heart failure hospitalization, and poorer functional recovery after aortic valve replacement. Although TAVR or surgical valve replacement successfully relieves the valvular obstruction, myocardial dysfunction related to amyloid infiltration persists, thereby attenuating the expected clinical benefit of valve intervention. Conversely, early recognition of ATTR-CA may allow initiation of disease-modifying therapy, such as tafamidis, which has been shown to reduce mortality and cardiovascular hospitalizations in patients with transthyretin amyloid cardiomyopathy [6–10].

The hemodynamic deterioration observed in our patient can be explained by the synergistic interaction between severe calcific aortic stenosis and probable transthyretin cardiac amyloidosis. Severe aortic stenosis created a fixed obstruction to left ventricular outflow, severely limiting the ability to increase stroke volume during physiological stress. Simultaneously, diffuse myocardial amyloid infiltration likely resulted in restrictive ventricular physiology, impaired myocardial contractile reserve, elevated left ventricular filling pressures, progressive pulmonary venous congestion, and probable right ventricular dysfunction. This combination produced critically reduced forward cardiac output associated with persistently elevated filling pressures, ultimately leading to systemic hypoperfusion and multiorgan dysfunction. Such a hemodynamic profile is characteristic of refractory cardiogenic shock, in which circulatory collapse persists despite optimal pharmacological support because both valvular and myocardial components contribute to pump failure. This mechanism likely explains the rapid progression from decompensated heart failure to irreversible shock in our patient.

Following admission to the cardiac intensive care unit, the patient underwent aggressive supportive treatment. Two units of packed red blood cells were administered slowly because of symptomatic anemia and the high risk of volume overload related to severe heart failure and end-stage renal disease. He subsequently received intravenous loop diuretics, maintenance hemodialysis with carefully controlled ultrafiltration, oxygen therapy, broad-spectrum antibiotics for suspected concomitant pneumonia, and escalating inotropic and vasopressor support. Urgent colonoscopy was deliberately deferred because the patient remained hemodynamically unstable with acute decompensated heart failure and severe aortic stenosis, rendering bowel preparation and procedural sedation prohibitively high risk. Moreover, the gastrointestinal bleeding was not massive at admission and was considered secondary to

previously documented angiodysplastic lesions. Despite maximal supportive therapy, the patient progressed to refractory cardiogenic shock characterized by profoundly depressed cardiac output and markedly elevated left ventricular filling pressures, culminating in irreversible end-stage pump failure and death.

Unfortunately, definitive confirmation of ATTR-CA could not be achieved because of the patient's rapidly progressive clinical deterioration. Planned cardiac magnetic resonance imaging, technetiumlabeled bone scintigraphy, and minor salivary gland biopsy could not be performed before death.

Nevertheless, the combination of advanced age, severe calcific aortic stenosis, recurrent gastrointestinal bleeding, concentric biventricular hypertrophy, voltage-mass discrepancy, restrictive physiology, biatrial enlargement, pericardial effusion, and right ventricular involvement strongly supported the diagnosis of probable transthyretin cardiac amyloidosis according to contemporary diagnostic red flags.

This case underscores two important clinical lessons. First, recurrent gastrointestinal bleeding in elderly patients with severe calcific aortic stenosis should immediately raise suspicion for Heyde syndrome. Second, clinicians should avoid attributing all symptoms exclusively to valvular disease. The presence of disproportionate ventricular hypertrophy, low QRS voltage, right ventricular hypertrophy, restrictive physiology, pericardial effusion, or other imaging red flags should systematically prompt evaluation for concomitant transthyretin cardiac amyloidosis. Earlier recognition of this increasingly prevalent association may improve diagnostic accuracy, facilitate appropriate multimodality imaging, optimize patient selection for valve intervention, and identify candidates for disease-modifying therapies, ultimately improving outcomes in this high-risk population.

4. CONCLUSION

This case illustrates that, in elderly patients with severe calcific aortic stenosis and Heyde syndrome, concomitant transthyretin cardiac amyloidosis should be actively suspected whenever ventricular hypertrophy appears disproportionate to pressure overload or is accompanied by characteristic electrocardiographic and echocardiographic red flags. Failure to recognize this increasingly prevalent association may underestimate disease severity, delay appropriate multimodality imaging, and adversely affect therapeutic decision-making. Our patient's fatal course, culminating in refractory cardiogenic shock before definitive diagnostic confirmation could be achieved, underscores the aggressive prognosis of advanced AS-ATTR and highlights the critical importance of early recognition of this underdiagnosed entity.

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Author contributions

M.A.: Conceptualization, patient management, manuscript drafting, and supervision.
H.E.: Literature review, echocardiographic interpretation, data collection, figure preparation, manuscript drafting, and critical revision of the manuscript.
M.M.: Clinical management and critical revision of the manuscript.
I.B.: Clinical management and manuscript revision.
D.B.: Clinical supervision and manuscript review.
H.F.: Patient management and manuscript review.
Z.L.: Critical revision of the manuscript.
A.B.: Supervision, critical revision, and final approval of the manuscript.

All authors read and approved the final manuscript.

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Ethics Approval and Consent to Participate: Ethics approval was not required for this single-patient case report according to the institutional policy of Mohammed V Military Teaching Hospital.

Learning points

- Heyde syndrome should be suspected in elderly patients presenting with severe calcific aortic stenosis and recurrent gastrointestinal bleeding due to intestinal angiodysplasia.
- Concomitant transthyretin cardiac amyloidosis (ATTR-CA) is increasingly recognized in patients with severe aortic stenosis and should be actively considered when imaging or electrocardiographic red flags are present.
- Disproportionate ventricular hypertrophy, low-voltage QRS complexes, restrictive filling physiology, right ventricular involvement, and pericardial effusion should prompt multimodality evaluation for cardiac amyloidosis.
- The coexistence of severe aortic stenosis and ATTR-CA (AS-ATTR) is associated with a poorer prognosis and may contribute to refractory cardiogenic shock and increased mortality.
- Early recognition of this dual pathology may improve risk stratification, optimize the timing of valve intervention, and identify patients eligible for disease-modifying therapies.

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