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Heyde Syndrome Complicated by Pulmonary Embolism Before Transcatheter Aortic Valve Replacement: A Clinical Dilemma Between Bleeding and Thrombosis

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Patient: Female, 70-year-old
Final Diagnosis: Heyde syndrome • pulmonary embolism
Symptoms: Acute dyspnea • hematochezia
Clinical Procedure: Transcatheter aortic valve replacement
Specialty: Cardiology
Objective: Rare disease
Background: Heyde syndrome is an uncommon clinical entity characterized by severe aortic stenosis (AS) and acquired von Willebrand syndrome, typically presenting with recurrent gastrointestinal bleeding secondary to angiodysplasia. Although most reported cases involve isolated gastrointestinal bleeding, the coexistence of thromboembolic events is exceedingly rare and poses a significant therapeutic challenge in balancing hemostatic and anticoagulant strategies.

Case Report: We report a 70-year-old woman who initially presented with massive hematochezia and subsequently developed dyspnea, requiring hospitalization. Physical examination revealed a prominent systolic murmur over the aortic area. Transthoracic echocardiography confirmed severe AS, with an aortic valve area of 0.8 cm² and a mean transvalvular pressure gradient of 71 mm Hg. Together with profound anemia (hemoglobin 44 g/L) and markedly reduced von Willebrand factor ristocetin cofactor activity (vWF: RCo, 25.3%), these findings supported the diagnosis of Heyde syndrome. During the preprocedural evaluation for transcatheter aortic valve replacement (TAVR), acute pulmonary embolism was incidentally identified on computed tomography pulmonary angiography. After hemostatic stabilization, anticoagulant therapy was cautiously initiated, resulting in complete resolution of the pulmonary embolism after 1 month. Given the elevated surgical risk (EuroSCORE II, 8.05%), the patient underwent successful TAVR. Following the procedure, the transvalvular pressure gradient normalized (mean, 13.8 mm Hg), with restoration of normal vWF activity.

Conclusions: This case underscores the therapeutic complexity of simultaneously managing hemorrhagic and thrombotic risks in Heyde syndrome. TAVR remains the definitive treatment for acquired von Willebrand syndrome, while a staged, individualized anticoagulation approach is crucial in patients with concomitant thromboembolic complications. Correcting the underlying AS remains the cornerstone of management.

Keywords: Aortic Valve Stenosis • Gastrointestinal Hemorrhage • Pulmonary Embolism
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Introduction

Severe aortic stenosis (AS) is a common and potentially life-threatening valvular heart disease among older adults. If left untreated, the 2-year mortality rate can approach 50% [1]. The association of severe AS with gastrointestinal bleeding is classically referred to as Heyde syndrome [2]. Severe AS generates chronic high shear stress across the stenotic valve, contributing to progressive cardiac dysfunction and a range of systemic complications [3-5]. Traditionally, the clinical course of Heyde syndrome is dominated by recurrent or refractory gastrointestinal bleeding, whereas concomitant thromboembolic events have rarely been described. We report a patient with Heyde syndrome complicated by acute pulmonary embolism (PE) who was treated at the Department of Cardiology, Tianjin Union Medical Center. The coexistence of severe bleeding and acute thromboembolism posed substantial diagnostic and therapeutic challenges. This report highlights the dilemma of balancing anticoagulation against the risk of recurrent hemorrhage and proposes a practical management approach for similar complex clinical scenarios.

Case Report

A 70-year-old woman presented with massive hematochezia and progressive dyspnea and was subsequently diagnosed with Heyde syndrome complicated by acute PE and bilateral lower-extremity deep vein thrombosis (DVT). We describe the diagnostic evaluation, staged therapeutic management, and 5-month clinical follow-up. Her past medical history included hypertension for 20 years, type 2 diabetes mellitus for 10 years, and longstanding bilateral lower-limb muscle atrophy for approximately 30 years. She had no history of chronic liver disease, chronic gastrointestinal disorders, malignancy, or spontaneous bleeding events. Family history was unremarkable

for hematologic disorders, inherited bleeding tendencies, or other hereditary diseases. Subsequent investigations excluded primary gastrointestinal malignancy as a cause of bleeding.

Four days before admission, the patient developed recurrent episodes of painless, spontaneous bright-red hematochezia, with an estimated blood loss of 200 to 300 mL per episode. One day before transfer, computed tomography (CT) performed at a local hospital demonstrated increased pulmonary vascular markings, bilateral interstitial pulmonary changes, cardiomegaly, irregular gastric morphology, and linear hyperdense lesions within the rectum. Given the persistence of unexplained gastrointestinal bleeding, she was referred to the emergency surgery department of our institution for further evaluation and management. Laboratory investigations on admission revealed profound anemia consistent with acute blood loss, including a red blood cell count of $1.38 \times 10^{12}/L$, hemoglobin of 44 g/L, and hematocrit of 13.7%. The platelet count remained within the normal range ($122 \times 10^9/L$), suggesting that thrombocytopenia was unlikely to have contributed to the bleeding episode.

Emergency upper and lower gastrointestinal endoscopy revealed several potential bleeding-related lesions, including diffusely pale gastric mucosa, a diverticulum in the descending duodenum, multiple colonic diverticula containing blood-stained luminal contents, and tortuous dilated vascular clusters in the ascending colon (Figure 1). Initial management consisted of bowel rest, proton pump inhibitor therapy, intravenous fluid resuscitation, and packed red blood cell transfusion. Despite these interventions, the patient developed sudden-onset dyspnea and orthopnea approximately 12 hours after admission. Laboratory testing demonstrated markedly elevated brain natriuretic peptide levels, supporting a diagnosis of acute decompensated heart failure. Given the coexistence of ongoing gastrointestinal hemorrhage and acute cardiac decompensation, the patient was promptly transferred to

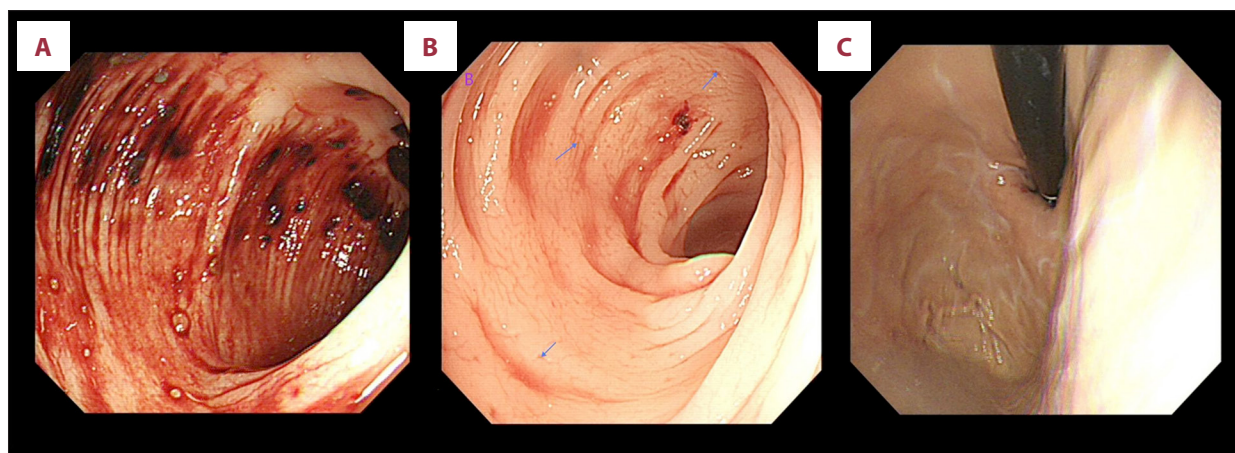


Figure 1. Gastroscopy and colonoscopy findings (A) Intestinal lumen filled with bloody substances. (B) Arrows indicate tortuous and filled venous plexus. (C) "Anemic stomach" appearance.

the coronary care unit (CCU) for intensive hemodynamic monitoring and specialized management.

On admission to the CCU, the patient's vital signs were as follows: temperature, 36.5 °C; heart rate, 93 beats/min; respiratory rate, 17 breaths/min; and blood pressure, 104/63 mm Hg. The patient was alert and oriented, with marked pallor of the conjunctivae, lips, and nail beds. Pulmonary examination revealed coarse breath sounds with bilateral basal crackles, findings consistent with pulmonary congestion. Cardiac examination demonstrated a regular rhythm and a grade 4/6 harsh systolic ejection murmur best heard at the aortic area with radiation to the carotid arteries, a classic finding of severe AS. The abdomen was soft and non-tender, and no peripheral edema was observed. Bilateral calf muscle atrophy was noted on examination.

Routine laboratory investigations revealed no significant abnormalities in liver function, renal function, lipid profile, electrolyte levels, or tumor markers. Persistent fecal occult blood positivity was observed, while N-terminal pro-B-type natriuretic peptide was markedly elevated at 5663.61 pg/mL. Electrocardiography demonstrated diffuse ST-segment depression accompanied by ST-segment elevation in lead aVR, findings that raised suspicion for extensive myocardial ischemia, including possible left main coronary artery disease, multivessel disease, or subendocardial ischemia related to severe AS. Arterial blood gas analysis revealed mild metabolic acidosis without significant impairment of oxygenation. The initial D-dimer level was mildly elevated at 0.99 mg/L. Transthoracic echocardiography demonstrated severe AS, characterized by an aortic valve area of 0.8 cm², peak transvalvular velocity of 4.2 m/s, and mean pressure gradient of 71 mm Hg. Left ventricular systolic function was preserved, with a left ventricular ejection fraction of 55%. These findings established severe AS as the principal cardiac abnormality underlying the patient's clinical presentation.

Given the coexistence of severe AS and recurrent gastrointestinal bleeding, further evaluation of von Willebrand factor (vWF) function was undertaken to investigate the possibility of acquired von Willebrand syndrome. Laboratory testing demonstrated reduced vWF antigen (vWF: Ag) levels of 38.5% (reference range, 50%-160%) and markedly decreased vWF ristocetin cofactor activity (vWF: RCo) of 25.3% (reference range, 48.8%-163.4%), resulting in a vWF: RCo/vWF: Ag ratio of <0.7. In conjunction with endoscopic evidence of vascular malformations and recurrent gastrointestinal bleeding, these findings supported a diagnosis of acquired von Willebrand syndrome and established the diagnosis of Heyde syndrome. Differential diagnoses, including peptic ulcer disease, inflammatory bowel disease, gastrointestinal malignancy, immune thrombocytopenia, and inherited coagulation disorders, such as hemophilia,

were excluded based on endoscopic findings, clinical history, and laboratory investigations. Genetic testing was not performed because of financial limitations. Repeat contrast-enhanced CT revealed no evidence of intrathoracic or intra-abdominal mass lesions or occult malignancy.

A staged and individualized treatment strategy was adopted according to the relative urgency of the patient's competing clinical conditions. Initial management focused on stabilization of active gastrointestinal bleeding through gastric mucosal protection, hemostatic therapy, and blood product replacement, including transfusion of 9 units of packed red blood cells and 400 mL of plasma. These interventions achieved effective bleeding control and increased the hemoglobin concentration to 103 g/L. The patient's EuroSCORE II was calculated at 8.05%, indicating an elevated operative risk for conventional surgical aortic valve replacement (SAVR). Consequently, transcatheter aortic valve replacement (TAVR) was selected as the preferred definitive intervention to reduce procedural risk while addressing the underlying pathophysiological driver of Heyde syndrome.

On hospital day 10, preprocedural aortic and coronary computed tomography angiography (CTA) unexpectedly revealed acute right-sided segmental and subsegmental pulmonary emboli. Retrospective review of clinical data demonstrated persistent mild hypoxemia, with oxygen saturation ranging from 90% to 95% on room air. Repeat laboratory testing showed a marked increase in D-dimer levels to 14.04 mg/L, while ultrasound of the lower limb vessels confirmed bilateral calf DVT.

Risk stratification demonstrated a PE Severity Index (PESI) score of 86 and a Bova score of 3, consistent with an intermediate-risk PE profile. At that time, gastrointestinal bleeding had ceased and hemoglobin levels had stabilized above 90 g/L, allowing consideration of therapeutic anticoagulation despite the recent hemorrhagic event. Although the HAS-BLED score was 3, indicating a moderate bleeding risk, the anticipated benefits of anticoagulation were considered to outweigh the potential risk of recurrent hemorrhage. In accordance with contemporary guideline recommendations [6], a staged anticoagulation strategy was initiated with subcutaneous low-molecular-weight heparin (4000 IU every 12 hours), followed by transition to oral rivaroxaban (15 mg once daily) after 3 days. The patient remained clinically stable without recurrent bleeding and was discharged after a total hospital stay of 16 days.

One month after discharge, follow-up CT pulmonary angiography demonstrated complete resolution of the pulmonary emboli (**Figure 2**). The patient was subsequently readmitted for preprocedural reassessment, during which pulmonary function testing revealed no significant abnormalities. Elective TAVR was successfully performed under general anesthesia using a

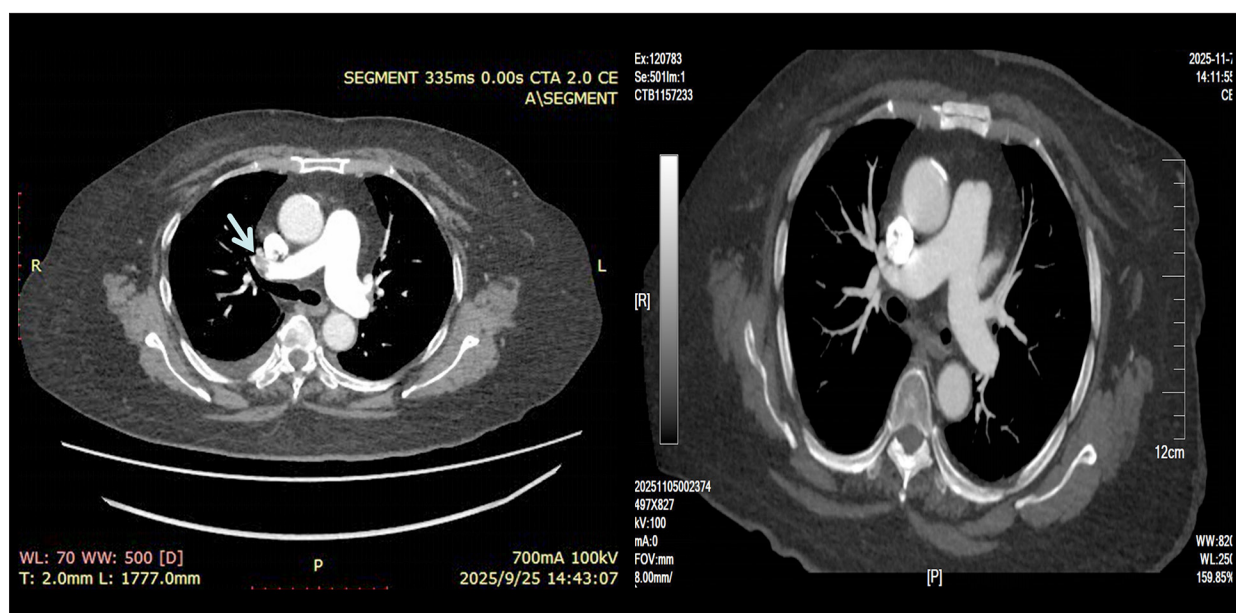


Figure 2. Pulmonary computed tomography angiography imaging. The image on the left is pulmonary embolism; the arrow indicates the thrombus. The image on the right confirms the disappearance of thrombus after anticoagulant therapy.

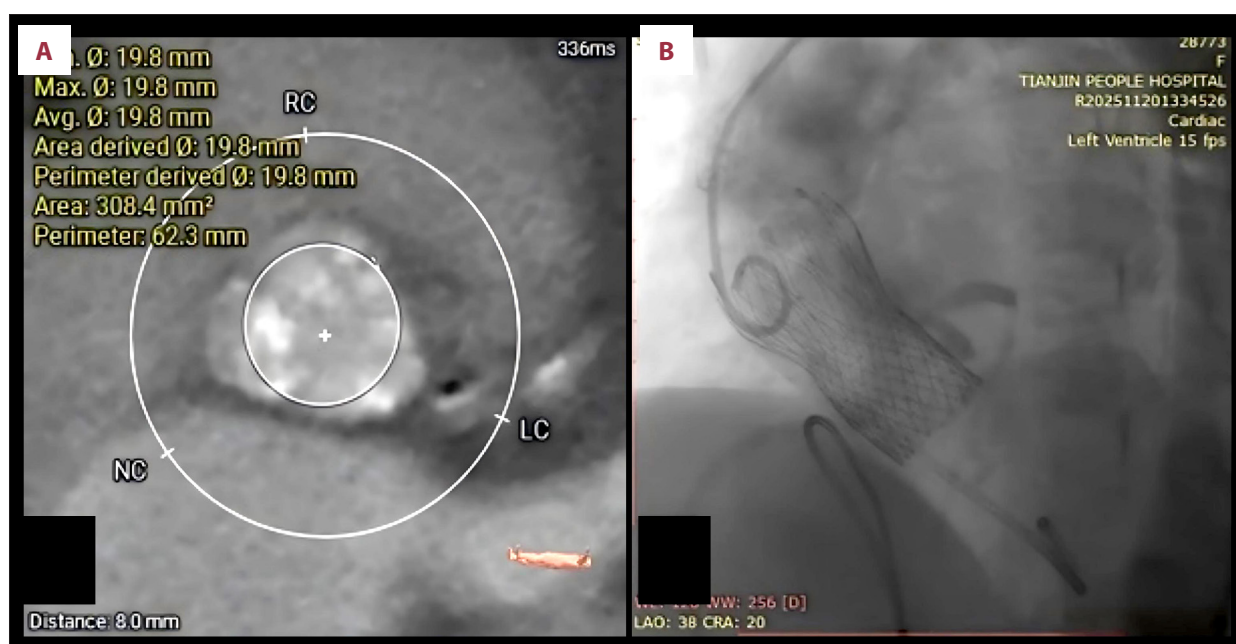


Figure 3. Preoperative transcatheter aortic valve replacement evaluation and postoperative imaging. (A) Preoperative CTA assessment showing a tri-leaflet valve with leaflet calcification, thickening, and partial adhesion. (B) Postoperative imaging showing the implanted valve.

TaurusElite AV21 bioprosthesis valve (Figure 3). Postprocedural transthoracic echocardiography demonstrated marked improvement in aortic valve hemodynamics, with a peak transvalvular velocity of 1.8 m/s and a mean pressure gradient of 13.8 mm Hg (Figure 4). Follow-up laboratory testing demonstrated normalization of von Willebrand factor parameters (vWF: Ag 370.5%, vWF: RCo 269.5%), consistent with correction of acquired von

Willebrand syndrome following relief of valvular obstruction. The patient recovered uneventfully and was discharged in stable condition. A detailed timeline of diagnostic evaluation, therapeutic interventions, and follow-up is provided in Table 1.

At the 4-month follow-up, the patient demonstrated sustained clinical improvement, with no recurrence of dyspnea

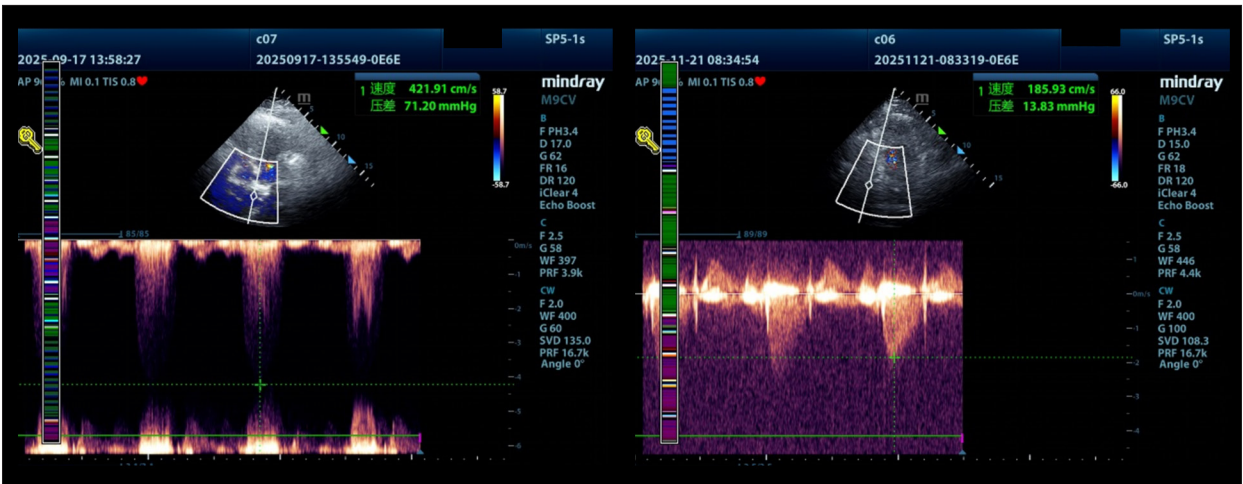


Figure 4. Echocardiographic assessment before and after transcatheter aortic valve replacement. Preoperative assessment on the left image (mean gradient: 71 mm Hg). Postoperative assessment on the right image (mean gradient: 13.8 mm Hg).

Table 1. Diagnosis and treatment timeline.

Time point	Clinical manifestation	Treatment and key findings
4 days before admission	Hematochezia	Patient ignored symptoms
1 day before admission	Fatigue	Emergency blood transfusion; emergency gastroscopy and colonoscopy performed
Hospital day 1	Sudden onset of dyspnea and wheezing; Suspected heart failure	Admitted to CCU; blood transfusion and cardiac function support initiated; physical examination revealed aortic valve murmur
Hospital day 2	—	Cardiac ultrasound confirmed severe aortic valve stenosis
Hospital day 3	—	von Willebrand Factor analysis: vWF antigen 38.5% (reference: 50%-160%); vWF Activity (RCo) 25.3% (reference: 48.8-163.4%)
Hospital day 10	Hematochezia resolved; Hemoglobin rebounded; General condition improved	Incidental finding of acute pulmonary embolism on coronary CTA; anticoagulation initiated; TAVR planned
Hospital day 16	—	Patient discharged
Before second hospitalization (37 days after previous hospitalization)	—	Pulmonary artery CTA showed complete resolution of pulmonary embolism
TAVR performed (50 days after previous hospitalization)	—	TAVR performed under general anesthesia
First follow-up (19 days after TAVR)	No hematochezia recurrence; Good cardiac function	Aortic valve flow velocity: 239 cm/s; LVEF = 62%
Second follow-up (63 days after TAVR; 4 months after initial hospitalization)	No hematochezia recurrence; good cardiac function	Aortic valve flow velocity: 110 cm/s; LVEF = 61%

Abbreviations: CCU, coronary care unit; CTA, computed tomography angiography; LVEF, left ventricular ejection fraction; RCo, ristocetin cofactor; TAVR, transcatheter aortic valve replacement; vWF, von Willebrand factor.

or gastrointestinal bleeding. Follow-up transthoracic echocardiography confirmed normal prosthetic valve function with preserved left ventricular systolic performance. The patient successfully resumed routine daily activities without functional limitation. No recurrent thromboembolic events, heart failure exacerbations, or valve-related complications were observed during follow-up, indicating a favorable mid-term clinical outcome.

Discussion

Heyde syndrome is classically characterized by the triad of severe AS, acquired von Willebrand syndrome, and recurrent gastrointestinal bleeding. In the present case, the diagnosis was supported by characteristic endoscopic findings, severe AS documented by echocardiography, and markedly reduced von Willebrand factor activity. Notably, the unexpected development of acute PE and DVT following stabilization of gastrointestinal bleeding introduced a rare and clinically challenging scenario. The coexistence of hemorrhagic and thromboembolic complications complicated therapeutic decision-making, necessitating a carefully staged management strategy that balanced anticoagulation requirements against the risk of recurrent bleeding.

The electrocardiogram demonstrated diffuse ST-segment depression accompanied by ST-segment elevation in lead aVR, a pattern typically associated with extensive myocardial ischemia, including left main coronary artery disease, multivessel disease, or severe subendocardial ischemia. However, both the preprocedural coronary CTA and intraoperative coronary angiography performed during TAVR excluded significant obstructive coronary artery disease. Consequently, the electrocardiographic abnormalities were considered possibly secondary to severe AS and its associated hemodynamic consequences. Although electrocardiographic findings are generally nonspecific in patients with severe AS, the presence of diffuse ST-segment depression with concomitant aVR elevation may reflect advanced hemodynamic compromise and myocardial oxygen supply-demand mismatch. In severe AS, chronic pressure overload increases myocardial oxygen demand while simultaneously impairing coronary perfusion reserve, thereby predisposing patients to subendocardial ischemia even in the absence of obstructive coronary artery disease. Such electrocardiographic abnormalities have been associated with adverse clinical outcomes and may indicate advanced disease severity [7].

The pathophysiological association between severe AS and recurrent gastrointestinal bleeding in Heyde syndrome is primarily mediated by acquired von Willebrand syndrome [8,9]. In severe AS, excessive transvalvular shear stress induces conformational changes in high-molecular-weight vWF multimers, rendering

them more susceptible to proteolytic cleavage by ADAMTS13 and resulting in functional vWF deficiency [10,12]. The resulting impairment of vascular homeostasis has been proposed to promote VEGF-mediated angiogenesis, facilitating the development of fragile vascular malformations and gastrointestinal angiodysplasia [13,14]. In the present case, the markedly reduced vWF activity and low vWF: RCo/vWF: Ag ratio provided biochemical evidence supporting this pathophysiological mechanism. Furthermore, persistent shear stress may disrupt normal platelet-vWF interactions and contribute to impaired hemostatic function, thereby exacerbating bleeding susceptibility [15]. Reduced cardiac output and impaired mesenteric perfusion associated with severe AS may further contribute to chronic intestinal hypoperfusion and the development of angiodysplastic lesions [16]. Age-related degenerative processes may also represent a shared pathogenic substrate linking AS and gastrointestinal angiodysplasia, potentially explaining the predominance of Heyde syndrome among older adults [17].

ADAMTS13 is a zinc-dependent metalloprotease that plays a central role in the physiological regulation of vWF by cleaving ultra-large and high-molecular-weight vWF multimers. In Heyde syndrome, excessive transvalvular shear stress promotes conformational unfolding of high-molecular-weight vWF multimers, rendering them increasingly susceptible to ADAMTS13-mediated cleavage and leading to acquired vWF deficiency. Although recurrent bleeding represents the hallmark clinical manifestation of Heyde syndrome, emerging evidence suggests that profound disturbances in vWF-mediated hemostasis may occasionally be accompanied by thrombotic complications, highlighting the complexity of the underlying coagulation imbalance [18-20]. Such competing hemorrhagic and thrombotic risks can substantially complicate clinical decision-making, particularly when therapeutic anticoagulation is indicated. In the present case, the mean transvalvular gradient was 71 mm Hg before TAVR, substantially exceeding values previously associated with severe vWF dysfunction and clinically significant bleeding in patients with advanced AS [21].

A notable feature of this case was the occurrence of PE and DVT despite the presence of active bleeding manifestations. Several mechanisms may have contributed to the development of venous thromboembolism in this patient. First, acute gastrointestinal hemorrhage and the subsequent activation of compensatory hemostatic pathways may have created a transient prothrombotic state through enhanced coagulation factor activity and platelet activation [22,23]. Second, prolonged immobilization during hospitalization, together with long-standing bilateral lower-limb muscle atrophy, likely promoted venous stasis, a well-recognized risk factor for venous thromboembolism [24]. The presence of bilateral calf DVT on ultrasonography strongly suggested that the pulmonary emboli originated from thrombus propagation and embolization

from the lower extremities. Third, advanced age, acute illness, and hospitalization-related physiological stress may have further increased thrombotic susceptibility through mechanisms involving endothelial dysfunction and systemic activation of coagulation pathways. Taken together, these factors may explain the unusual coexistence of hemorrhagic and thromboembolic complications in the present patient, highlighting the complexity of hemostatic regulation in selected individuals with Heyde syndrome.

Clinical Decision-Making: Balancing PE and Bleeding Risk

The incidental detection of PE during preprocedural CTA for TAVR created a significant therapeutic dilemma. Risk stratification demonstrated an intermediate-risk PE (PESI grade III, Bova stage II) according to the PESI and Bova scoring systems [26,27], whereas the HAS-BLED score of 3 indicated a moderate risk of recurrent bleeding. Following multidisciplinary discussion, anticoagulation was prioritized over immediate TAVR for several reasons. First, untreated PE carried a substantial risk of clinical deterioration and recurrent thromboembolic events. Second, gastrointestinal bleeding had been effectively controlled, and no further transfusion support was required. Third, heart failure symptoms had stabilized under medical therapy. Accordingly, anticoagulation was initiated with low-molecular-weight heparin 10 days after confirmed hemostasis, followed by transition to rivaroxaban once clinical stability had been achieved. Follow-up CT pulmonary angiography performed 1 month later demonstrated complete resolution of PE without recurrent gastrointestinal bleeding. Rivaroxaban was selected because of its rapid onset of action, predictable pharmacokinetic profile, and absence of a requirement for routine coagulation monitoring [28-30]. The delayed initiation of anticoagulation following confirmation of hemostasis was consistent with contemporary recommendations for balancing thrombotic and bleeding risks after major gastrointestinal hemorrhage [31].

In patients with recent or active gastrointestinal bleeding, prioritizing TAVR before anticoagulation may reduce bleeding-related complications but may also delay treatment of concomitant thromboembolic disease. In the present case, the decision to postpone TAVR and initiate anticoagulation first was based on the presence of intermediate-risk PE, stable hemostasis, and adequately controlled heart failure. Follow-up imaging confirmed complete thrombus resolution without recurrent bleeding, supporting the safety and effectiveness of this staged approach. The sequencing of anticoagulation and valve intervention should therefore be individualized according to thrombotic burden, bleeding risk, and overall clinical status. Should recurrent gastrointestinal bleeding occur during anticoagulant therapy, temporary interruption of anticoagulation, accompanied by gastrointestinal protective measures and endoscopic hemostasis when appropriate, may be

considered, followed by multidisciplinary reassessment of treatment priorities [32].

Aortic valve replacement, either surgical (SAVR) or transcatheter (TAVR), is the definitive treatment for Heyde syndrome as it eliminates the pathological shear stress responsible for acquired von Willebrand syndrome and recurrent gastrointestinal bleeding [33,34]. Given the elevated operative risk reflected by a EuroScore II of 8.05%, TAVR was selected as the preferred treatment strategy in this patient. Following successful anticoagulation and complete resolution of PE, TAVR was performed without complications. Subsequent normalization of vWF parameters, marked improvement in aortic valve hemodynamics, and the absence of recurrent gastrointestinal bleeding during follow-up collectively support successful reversal of the underlying disease process. This case underscores that correction of AS remains the cornerstone of treatment in Heyde syndrome and may facilitate the safe management of coexisting thromboembolic complications through a staged, individualized therapeutic approach [35].

Conclusions

Clinicians should consider underlying high-shear cardiovascular conditions, such as severe AS or hypertrophic obstructive cardiomyopathy, in patients presenting with unexplained gastrointestinal bleeding and should remain vigilant for potential thromboembolic complications. This rare case of Heyde syndrome complicated by acute PE and DVT highlights the therapeutic challenge of balancing hemorrhagic and thrombotic risks. Because severe AS is the primary driver of acquired von Willebrand syndrome, correction of valvular obstruction through TAVR may effectively reverse the underlying hemostatic abnormalities. In the present case, a staged strategy involving bleeding stabilization, carefully timed anticoagulation, multidisciplinary assessment, and subsequent TAVR was feasible and was associated with favorable clinical outcomes. This case expands the recognized clinical spectrum of Heyde syndrome and offers valuable clinical insights into the treatment of patients with coexisting bleeding and thromboembolic complications.

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Generative AI Statement

The author(s) declare that generative AI was not used in the creation of this manuscript.

Ethics Statement

Written and signed consent was obtained from the patient to publish her history, all images, clinical data, and other data included in the manuscript.

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