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Delayed Stanford Type A Aortic Dissection Following Transcatheter Aortic Valve Implantation in a Patient With Pre-existing Ascending Aortic Dilatation

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Abstract

Acute aortic dissection is an exceptionally rare but potentially fatal complication of transcatheter aortic valve implantation (TAVI). Although most cases are recognized during or shortly after the procedure, delayed presentations following apparently uncomplicated TAVI have also been reported.

An 81-year-old woman with severe symptomatic aortic stenosis and pre-existing ascending aortic dilatation of 45 mm underwent successful transfemoral TAVI with a 26-mm Hydra self-expanding transcatheter heart valve. Seven days later, she presented with sudden severe retrosternal chest pain radiating to the back and neck. Despite hemodynamic stability, a non-ischemic electrocardiogram, and normal serial high-sensitivity cardiac troponin I levels, the clinical presentation raised suspicion of acute aortic syndrome. Transthoracic echocardiography revealed an intimal flap in the ascending aorta. Emergency computed tomography angiography confirmed Stanford type A aortic dissection with associated intramural hematoma. The patient was immediately referred for emergency cardiothoracic surgery but deteriorated and died before definitive repair could be performed.

Delayed aortic dissection should be considered in patients presenting with new severe chest pain after TAVI, even following an apparently uncomplicated procedure and in the presence of initially reassuring hemodynamic, electrocardiographic, and biomarker findings. Pre-existing ascending aortic dilatation may represent a vulnerable anatomical substrate and warrants careful consideration during pre-procedural assessment. A low threshold for urgent multimodality imaging is essential to facilitate early diagnosis and management of this rare but life-threatening complication.

Categories: Cardiology
Keywords: aortic stenosis, computed tomography angiography (cta), emergency echocardiography, transcatheter aortic valve implantation (tavi), type a acute aortic dissection

Introduction

Transcatheter aortic valve implantation (TAVI) has become an established treatment for patients with severe symptomatic aortic stenosis across a broad spectrum of surgical risk profiles. Compared with surgical aortic valve replacement, TAVI offers excellent procedural success rates and favorable long-term clinical outcomes, driving its widespread adoption in contemporary practice [1,2]. Despite ongoing technological advances and increasing operator experience, serious procedural and postprocedural complications may still occur. Among these, acute aortic dissection (AAD) represents one of the rarest but most devastating adverse events because of its rapid clinical progression and high mortality if not recognized promptly [3,4].

The reported incidence of AAD following TAVI is approximately 0.2%, making it an exceptionally uncommon complication [4,5]. Nevertheless, outcomes remain poor, particularly in Stanford type A dissections, which are associated with substantial early mortality despite aggressive treatment strategies [5]. Several procedural mechanisms have been implicated in post-TAVI AAD, including annular injury, guidewire- or catheter-related trauma, and transcatheter heart valve embolization [4,5]. We report a case of delayed Stanford type A aortic dissection occurring seven days after successful implantation of a 26-mm Hydra self-expanding transcatheter heart valve in a patient with pre-existing ascending aortic dilatation.

Case Presentation

An 81-year-old woman presented to the emergency department with severe acute retrosternal chest pain radiating to the back and neck, seven days after undergoing TAVI for severe symptomatic aortic stenosis. Her medical history was notable for dyslipidemia, hypertension, coronary artery disease, and known ascending aortic dilatation measuring 45 mm. Owing to advanced age and increased surgical risk, she underwent successful transfemoral TAVI with implantation of a 26-mm Hydra self-expanding transcatheter heart valve

without immediate procedural complications (Figure 1).

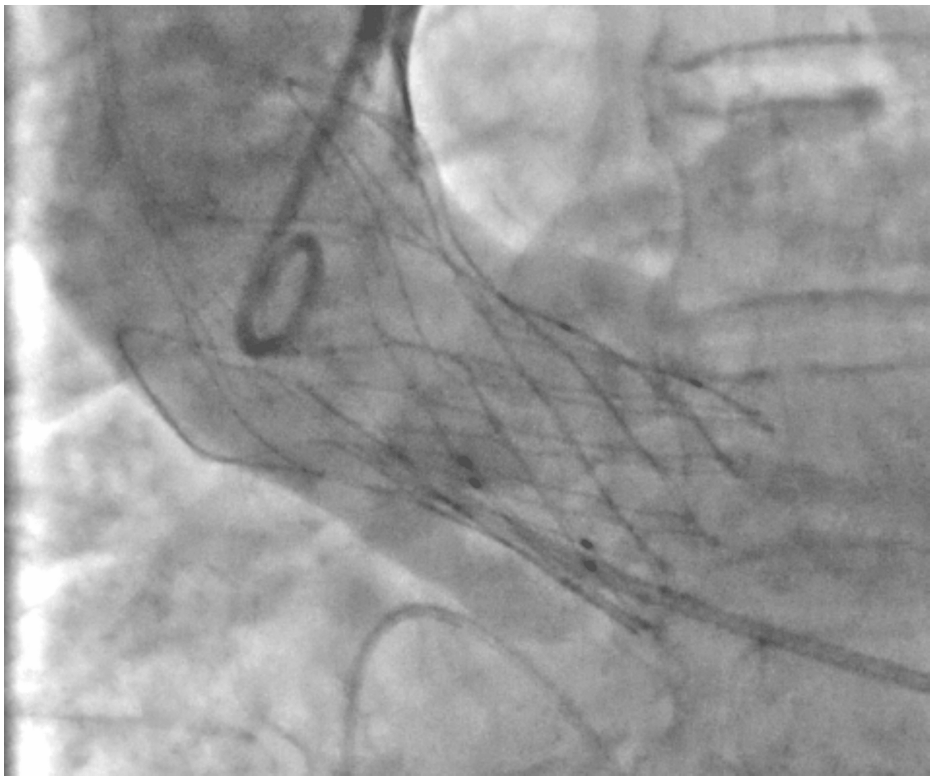


FIGURE 1: Fluoroscopic image

Fluoroscopic image obtained during the TAVI procedure demonstrating successful implantation and appropriate positioning of a 26-mm Hydra self-expanding transcatheter heart valve.

TAVI: transcatheter aortic valve implantation

On presentation, the patient was hemodynamically stable, with a blood pressure of 130/80 mmHg and a heart rate of 72 beats/min. Oxygen saturation was within normal limits, with no signs of cardiogenic shock or peripheral hypoperfusion. Physical examination revealed no focal neurological deficits, although the patient continued to experience severe chest pain.

Electrocardiography demonstrated normal sinus rhythm without ischemic ST-segment or T-wave abnormalities. Laboratory investigations revealed normal serial high-sensitivity cardiac troponin I levels, whereas the D-dimer level was markedly elevated, raising suspicion for acute aortic syndrome. Additional findings included a reduced hematocrit and mildly elevated urea (Table 1).

Laboratory parameters	Reference ranges	Presentation
Creatinine (mg/dL)	0.66-1.44	0.9
Urea (mg/dL)	17-43	44
White blood cells (×10³/μL)	4-11	9.34
Hematocrit (%)	42-54	30.5
Platelets (×10³/μL)	150-400	152
High-sensitivity cardiac troponin I (pg/mL)	<4	First measurement: <4; second measurement: <4
D-dimer (μg/mL)	<0.5	3.2

TABLE 1: Laboratory investigations

Given the severity of the pain and its characteristic radiation, urgent transthoracic echocardiography was performed. Imaging revealed aneurysmal dilatation of the ascending aorta and a mobile intimal flap within the aortic lumen, highly suggestive of acute ascending aortic dissection. No pericardial effusion was observed (Figure *2*).

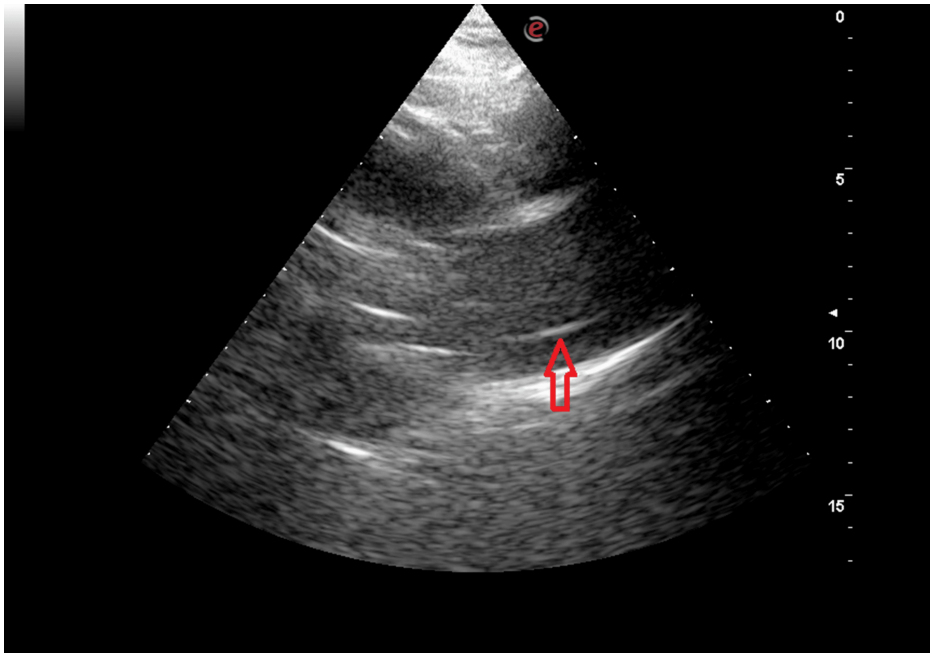


FIGURE 2: Parasternal long-axis view demonstrating the presence of an intimal flap (red arrow) within the lumen of the ascending aorta

Emergency contrast-enhanced computed tomography angiography of the thorax demonstrated acute Stanford type A dissection of the ascending thoracic aorta with associated intramural hematoma (Figures *3*-*4*). The prosthetic aortic valve appeared well positioned, and the dissection flap extended proximally within the ascending aorta.

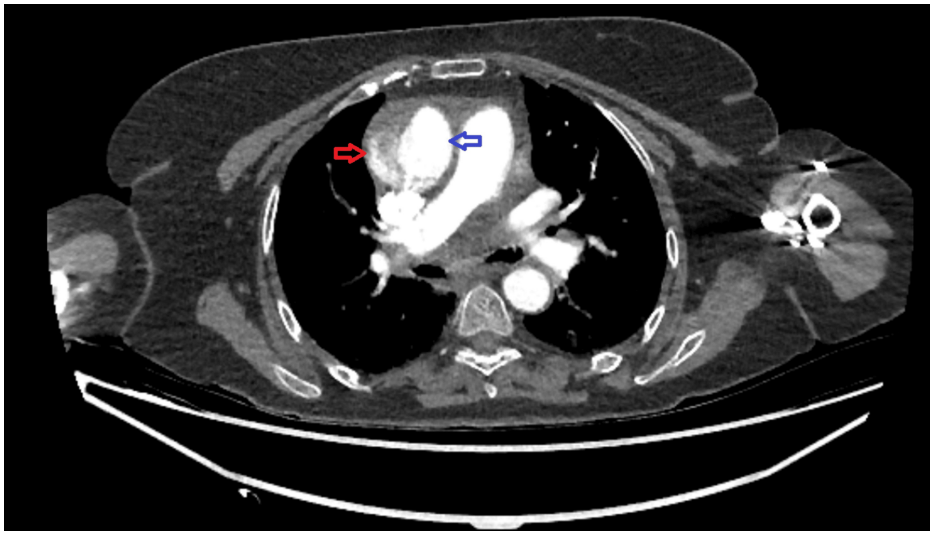


FIGURE 3: Contrast-enhanced computed tomography angiography demonstrating Stanford type A dissection of the ascending thoracic aorta with associated intramural hematoma (red arrow). The true lumen is indicated by the blue arrow

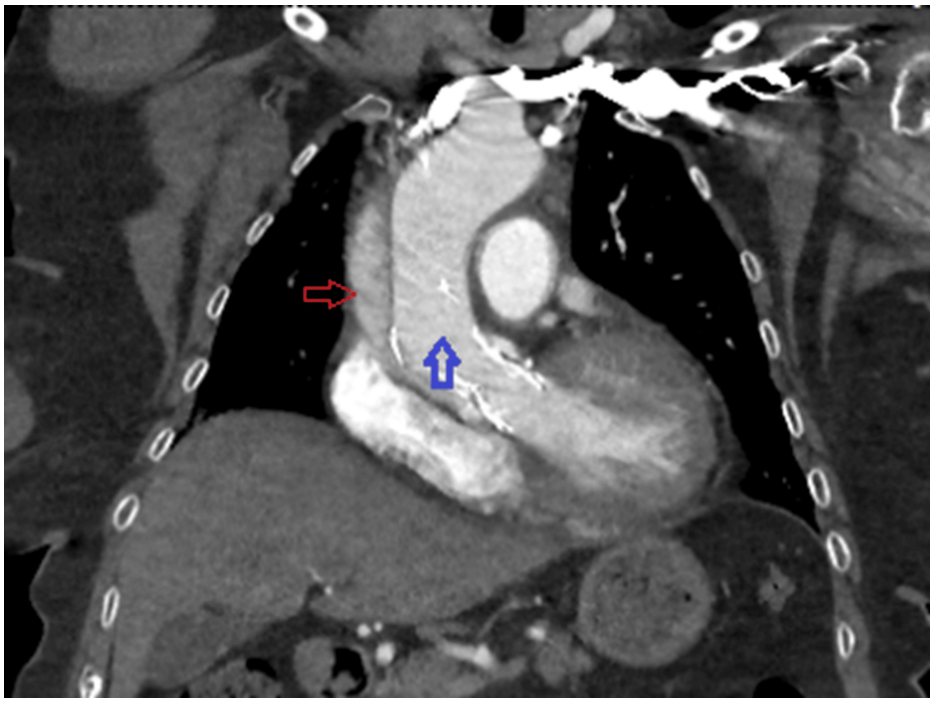


FIGURE 4: Contrast-enhanced computed tomography angiography demonstrating Stanford type A dissection of the ascending thoracic aorta with associated intramural hematoma (red arrow). The true lumen is indicated by the blue arrow

The patient was immediately referred for emergency cardiothoracic surgical intervention. Unfortunately, despite urgent multidisciplinary management and rapid surgical planning, the patient deteriorated clinically and died before definitive operative treatment could be performed.

Discussion

AAD is a rare but potentially fatal complication of TAVI. In a large single-center series of 4,317 TAVI

procedures, nine cases of post-TAVI AAD were identified, corresponding to an incidence of 0.2%, with a reported 30-day mortality of 66.7% [5]. At our center, 588 TAVI procedures were performed over a 2.5-year period, with no other cases of aortic dissection identified.

TAVI-associated aortic dissection is predominantly a periprocedural complication. In the series by Hiruma et al., five of six Stanford type A dissections (83%) were detected during the TAVI procedure [4]. Similarly, in the series by Ashwat et al., seven of nine dissections (77.8%) were detected intraprocedurally, whereas the two postprocedural cases occurred on days 1 and 3 [5]. However, delayed presentations after apparently uncomplicated TAVI have also been reported. Fujita et al. described an 82-year-old woman with severe aortic stenosis and marked ascending aortic dilatation (57 × 58 mm) who developed ascending aortic dissection on the second postoperative day following an apparently uncomplicated TAVI [6]. Other reports have demonstrated that the interval between TAVI and dissection may be considerably longer. Delayed dissections have been reported several days to weeks after TAVI, including cases occurring 6 and 22 days after the procedure [7], while Al-Attar et al. described aortic dissection and rupture several months after an uneventful TAVI [8]. These observations demonstrate that the absence of evident aortic injury during TAVI does not exclude subsequent aortic dissection and that this complication may become clinically apparent beyond the immediate periprocedural period.

In our patient, Stanford type A dissection occurred seven days after successful TAVI. Importantly, she was hemodynamically stable, with a non-ischemic electrocardiogram and normal serial high-sensitivity cardiac troponin I levels. The combination of delayed presentation and initially reassuring findings could have delayed diagnosis. However, the characteristic severe chest pain prompted bedside transthoracic echocardiography, which demonstrated an intimal flap. Subsequent computed tomography angiography confirmed the diagnosis. Thus, acute aortic syndrome should remain in the differential diagnosis of new-onset severe chest pain after TAVI, even several days after an apparently uncomplicated procedure.

Several mechanisms of TAVI-associated AAD have been described. In the series by Hiruma et al., presumed mechanisms included injury related to the delivery sheath, delivery catheter, valve implantation, stent edge, and pre-dilation ballooning [4]. In another series, posterior annular rupture and transcatheter heart valve embolization or “pop-out” into the ascending aorta were the most frequently identified mechanisms [5]. In our patient, no immediate procedural complication or evidence of aortic injury was identified; therefore, the underlying mechanism of the subsequent dissection remains uncertain.

Pre-existing aortic pathology may represent an important predisposing substrate. Marked ascending aortic dilatation (57 × 58 mm) was present in the delayed case reported by Fujita et al. [6], and substantial ascending aortic dilatation has also been described in other delayed cases [7]. Our patient had a known ascending aortic diameter of 45 mm before TAVI. Although a causal relationship cannot be established, these observations raise the possibility that a dilated ascending aorta may represent a vulnerable anatomical substrate for delayed aortic complications. Further evidence is required to determine whether ascending aortic dimensions independently influence the risk of post-TAVI dissection.

Current European Society of Cardiology (ESC)/European Association for Cardio-Thoracic Surgery (EACTS) guidelines emphasize comprehensive anatomical assessment, including evaluation of the aortic root and ascending aorta, and individualized heart team decision-making when selecting the optimal mode of intervention for severe aortic stenosis [3]. Therefore, the presence of ascending aortic dilatation should be considered in the overall anatomical and clinical assessment when choosing between transcatheter and surgical treatment. In this context, our case highlights the importance of careful pre-procedural anatomical evaluation and continued clinical vigilance after TAVI.

Despite rapid diagnosis and immediate referral for emergency surgery, our patient died before definitive repair could be performed. This case highlights that TAVI-associated aortic dissection may present beyond the immediate procedural period and should be considered in patients presenting with new severe chest pain after TAVI, irrespective of hemodynamic stability, electrocardiographic findings, or cardiac biomarker levels.

Conclusions

Delayed Stanford type A aortic dissection can occur after apparently uncomplicated TAVI and may be fatal despite initially stable hemodynamics and normal cardiac biomarkers. New-onset severe chest pain should prompt urgent aortic imaging, particularly in patients with pre-existing ascending aortic dilatation, to enable timely diagnosis and emergency surgical management.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Critical review of the manuscript for important intellectual content: Andreas D. Kyvetos, Sotirios Xydonas, Athanasios Trikas

Supervision: Athanasios Trikas

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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