CLINICAL IMAGE

Unusual rapid progression of TAVI valve degeneration confirmed on PET-CT scan after the valve-in-valve procedure followed by early valve thrombosis

Danuta Sorysz[1,2], Artur Dziewierz[1,2], Anna Staszczak[3,4], Jarosław Trębacz[5,6], Dariusz Dudek[1,7]

1 Clinical Department of Cardiology and Cardiovascular Interventions, University Hospital, Kraków, Poland
2 Second Department of Cardiology, Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland
3 Nuclear Medicine Unit of the Department of Endocrinology, Oncological Endocrinology and Nuclear Medicine, Jagiellonian University Medical College, Kraków, Poland
4 Nuclear Medicine Unit of the Department of Endocrinology, Oncological Endocrinology and Nuclear Medicine, University Hospital, Kraków, Poland
5 Department of Interventional Cardiology, Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland
6 John Paul II Hospital, Kraków, Poland
7 Digital Medicine & Robotics Center, Jagiellonian University Medical College, Kraków, Poland

Transcatheter aortic valve implantation (TAVI) has become a standard treatment in severe aortic stenosis (AS).[1] However, the major limitations of TAVI remain valve degeneration and thrombosis, which may affect long-term outcomes.[2-4]

An 80-year-old man, 5 years after CoreValve 31 implantation due to severe AS with porcelain aorta, reported exertional dyspnea (New York Heart Association class IV). He had a history of percutaneous coronary intervention in the left main coronary artery, arterial hypertension, pacemaker implantation (complete atrioventricular block after TAVI), and massive obesity (body mass index of 48 kg/m^2). Compared with baseline, transthoracic echocardiography (TTE) revealed a decrease in left ventricular ejection fraction (LVEF) from 65% to 20%, a mean transaortic pressure gradient (MTPG) increase from 9 to 26 mm Hg, and a decrease in the effective orifice area (EOA) after TAVI from 1.9 to 0.8 cm^2 (FIGURE 1A and 1B). Transesophageal echocardiography (TEE) revealed leaflet thickening with slight focal changes and 1 immobile leaflet. Because of a relatively fast (12 months) significant gradient increase, valvular thrombosis was suspected. Anticoagulation treatment was introduced. Due to the decrease in LVEF, a control coronary angiography was performed, and a good outcome of the previous procedure was confirmed. Computed tomography of the heart combined with an ^{18}F-sodium fluoride (^{18}F-NaF) positron emission tomography computed tomography (PET-CT) was performed. An increased ^{18}F-NaF focal uptake within the valve, typical of valve leaflet calcification, was confirmed (FIGURE 1C and 1D). Thus, severe degenerative TAVI valve dysfunction was diagnosed. The Heart Team referred the patient for an urgent valve-in-valve procedure with the brain protection system, and Edwards S3 29 mm valve (Edwards Lifesciences, Irvine, California, United States) was successfully implanted via the transfemoral approach. The control MTPG was 11 mm Hg with EOA of 2 cm^2 (FIGURE 1E). Because of the increased risk of thrombosis due to the valve-in-valve procedure and low LVEF, acetylsalicylic acid 75 mg combined with clopidogrel 75 mg daily were introduced. Six months after the procedure, echocardiography revealed an asymptomatic increase in MTPG to 27 mm Hg with EOA reduction to 1.2 cm^2, and LVEF of 25% (FIGURE 1F). On TEE, decreased leaflet mobility due to thrombi was detected (FIGURE 1G). Apixaban 5 mg twice daily was introduced. As the LVEF remained decreased despite optimal pharmacologic treatment, an implantable cardiac resynchronization therapy defibrillator was implanted. On the follow-up, MTPG was reduced to 12 mm Hg, with EOA of 1.6 cm^2 and LVEF of 40% (FIGURE 1H).

The standard method for diagnosing TAVI dysfunction during follow-up is TTE; however, finding out the etiology usually requires expanding imaging diagnostics with TEE or CT. New methods, such as PET-CT, especially with ^{18}F-NaF as a calcification marker, may be helpful in detecting TAVI degeneration and differentiating other mechanisms of the valve structural deterioration,

Correspondence to:
Danuta Sorysz, MD, PhD,
Second Department of Cardiology,
Institute of Cardiology, Jagiellonian
University Medical College,
ul. Jakubowskiego 2, 30-688 Kraków,
Poland, phone: +48 12 400 22 50,
email: danuta.sorysz@uj.edu.pl
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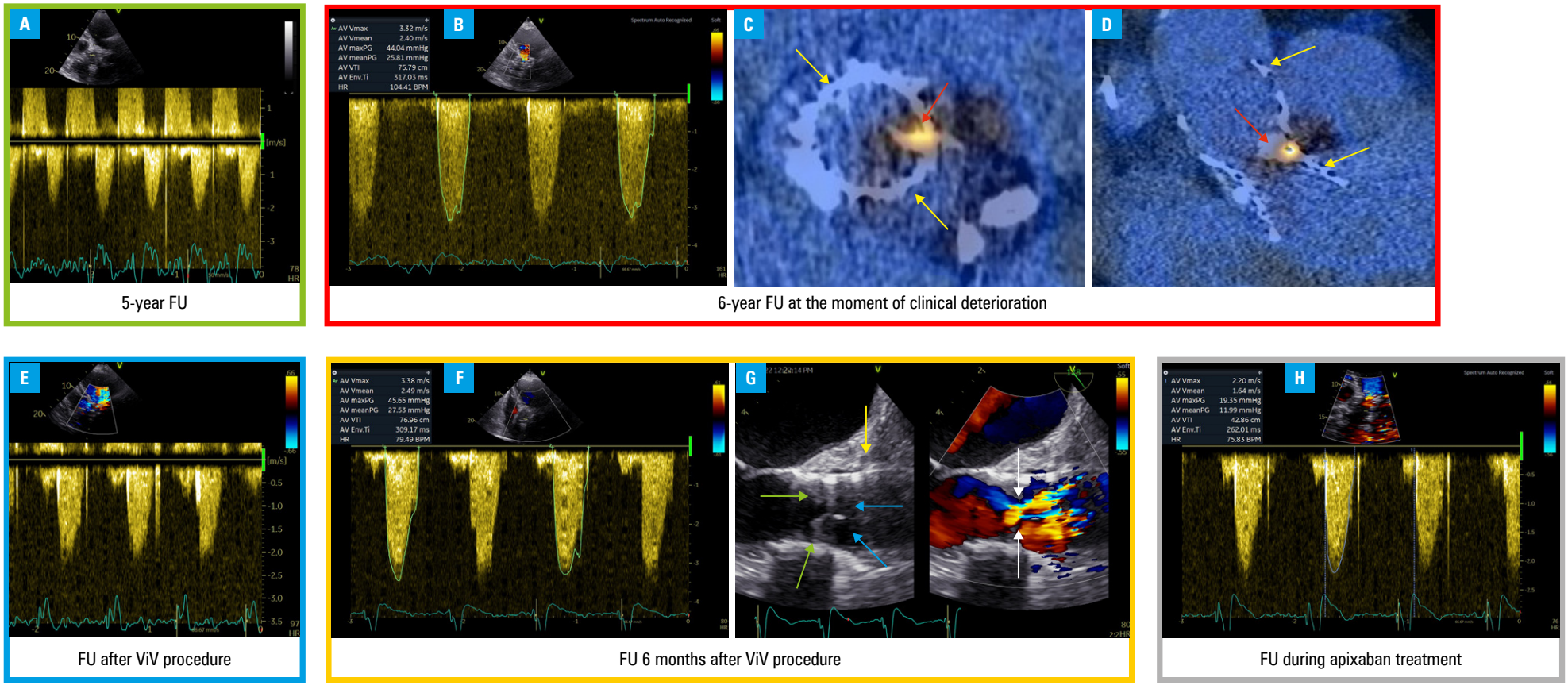

FIGURE 1 **A** – follow-up (FU) 5 years after transcatheter aortic valve implantation (TAVI) and 12 months before occurrence of the symptoms. Transaortic continuous wave (CW) Doppler showing the maximal velocity of 2 m/s, mean transaortic gradient of 9 mm Hg, and calculated effective orifice area (EOA) of 1.9 cm^2; **B**–**D** – follow-up after 6 years at the moment of clinical deterioration (**B** – transaortic CW Doppler showing the rise of maximal velocity and mean transaortic gradient up to 3.3 m/s and 26 mm Hg, respectively, with a decrease in EOA to 0.8 cm^2; **C** – ^{18}F-sodium fluoride [^{18}F-NaF] positron emission tomography computed tomography [PET-CT] short-axis view of the TAVI CoreValve showing focal calcification of the leaflets; **D** – ^{18}F-NaF PET-CT long-axis view of the TAVI CoreValve with focal calcification of the leaflets); **E** – follow-up 1 month after the valve-in-valve procedure, transaortic CW Doppler showing maximal velocity of 2.1 m/s, mean transaortic gradient of 11 mm Hg, and EOA of 2 cm^2; **F**, **G** – follow-up 6 months after the valve-in-valve procedure (**F** – transaortic CW Doppler showing the second rise of maximal velocity and mean transaortic gradient up to 3.4 m/s and 27 mm Hg, respectively, with a decrease in EOA to 1.2 cm^2; **G** – transesophageal echocardiography showing long-axis of the TAVI valve during systolic period. Two-dimensional image on the left shows a reduction of the opening and hypoechogenic thrombus at the base of both leaflets. The image on the right shows color Doppler with narrow flow through the valve); **H** – follow-up during apixaban treatment with transaortic CW Doppler showing a decrease of maximal velocity and transvalvular gradient up to 2.2 m/s and 12 mm Hg, respectively, with calculated EOA of 1.2 cm^2
Yellow arrows indicate the stent of the TAVI valve. Red arrows indicate PET-CT focal uptake of ^{18}F-NaF on the level of the TAVI valve leaflet. Green arrows indicate thrombi on the leaflets. Blue arrows indicate reduced opening of the valve leaflets. White arrows indicate narrow flow through the valve.

including leaflet calcification, leaflet tearing, stent rupture, or stent creep.[4] The treatment of choice in patients with TAVI degeneration remains the valve-in-valve procedure,[5] which, however, may be associated with an increased risk of subsequent valve thrombosis and the need for anticoagulant treatment. At the same time, thrombosis remains a risk factor for embolic events and accelerated valve degeneration.[2,3,5]

ARTICLE INFORMATION

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