JACC: CASE REPORTS VOL. 4, NO. 11, 2022

MINI-FOCUS ISSUE: PROCEDURAL COMPLICATIONS

ADVANCED

CASE REPORT: CLINICAL CASE SERIES

Aortic Sinus Contrast Retention During TAVR

A Warning Sign Preceding a Potential Thrombotic Complication

Lluis Asmarats, MD, PhD, Chi-Hion Li, MD, Xavier Millán, MD, PhD, Irene Menduiña, MD, Estefanía Fernández-Peregrina, MD, Lola Gutiérrez-Alonso, MD, Mario Torres, MD, Antonio Serra, MD, PhD, Dabit Arzamendi, MD, PhD

ABSTRACT

Sinus contrast material retention after transcatheter aortic valve replacement (TAVR) is a rare phenomenon that may reflect an increased risk for thrombotic complications. We present 3 cases of persistent contrast agent retention in the sinus of Valsalva during the TAVR procedure that portend the occurrence of embolic stroke or bioprosthetic valve thrombosis. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2022;4:666-670) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

INTRODUCTION

Despite the continued expansion of transcatheter aortic valve replacement (TAVR) indications, thrombotic events (stroke and valve thrombosis) following TAVR remain a relevant concern. The incidence of cerebrovascular events post-TAVR ranges from 1% to 11%, with an estimated incidence of subclinical leaflet thrombosis of 10% to 15%.[1,2] Thrombotic events post-TAVR may occur as a combination of changes in the coagulation cascade, along with modification of flow dynamics (blood stasis resulting from stagnant flow in the sinuses of Valsalva [SoVs]). Here we present a series of 3 patients presenting with early thrombotic complications after TAVR, all preceded by dense, persistent contrast material retention within the SoVs during valve implantation.

LEARNING OBJECTIVES

- To recognize the retention of contrast material in the sinus of Valsalva during TAVR.
- To identify this characteristic imaging finding as a warning for potential thrombotic complications.
- To provide close neurovascular surveillance and consider early anticoagulation when observing this rare phenomenon.

PATIENT 1

An 86-year-old woman with previous breast cancer treated with surgery and radiotherapy and severe symptomatic aortic stenosis with a mean gradient of 38 mm Hg was referred for TAVR. Preprocedural computed tomography (CT) confirmed severe calcific aortic stenosis (calcium score, 2,372 AU) extending toward the left ventricular outflow tract, with an aortic annulus area of 402 mm^2, perimeter of 72 mm and mean diameter of 23 mm, large SoVs (32 mm), and a

From the Department of Cardiology, Santa Creu i Sant Pau Hospital, Autonomous University of Barcelona, Barcelona, Spain.
The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received February 23, 2022; accepted March 28, 2022.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2022.03.031

narrow sinotubular junction ([STJ] 22 mm; 13 mm/m^2) (Figures 1A and 1B). Given the potential risk of annulus rupture, transfemoral TAVR with a mechanically expandable valve (Lotus, Boston Scientific) was planned. Unfractionated heparin (70 U/kg) was used in all procedures. After valvuloplasty with a 20-mm balloon, a 23-mm valve was successfully implanted on the first attempt. Before valve release, marked contrast material retention and slow washout were noticed in the left coronary cusp (Figures 1C and 1D, Videos 1 to 3). Transthoracic echocardiography showed a mean gradient of 6 mm Hg and no residual aortic regurgitation. Three hours after the procedure, the patient experienced an acute right sensorimotor syndrome (right-sided hemiparesis and sensory impairment). Brain CT showed no new onset ischemic lesions, and the patient was discharged home with aspirin therapy 9 days after the procedure. At 6-month follow-up, the patient had nearly full recovery of neurologic signs with normal TAVR function.

PATIENT 2

A 73-year-old woman with severe aortic stenosis was referred for aortic valve replacement. She had a history of severe chronic obstructive pulmonary disease, previous ischemic stroke, amyloid angiopathy-related cerebellar hemorrhage, permanent atrial fibrillation (AF) with percutaneous left

ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation

CT = computed tomography

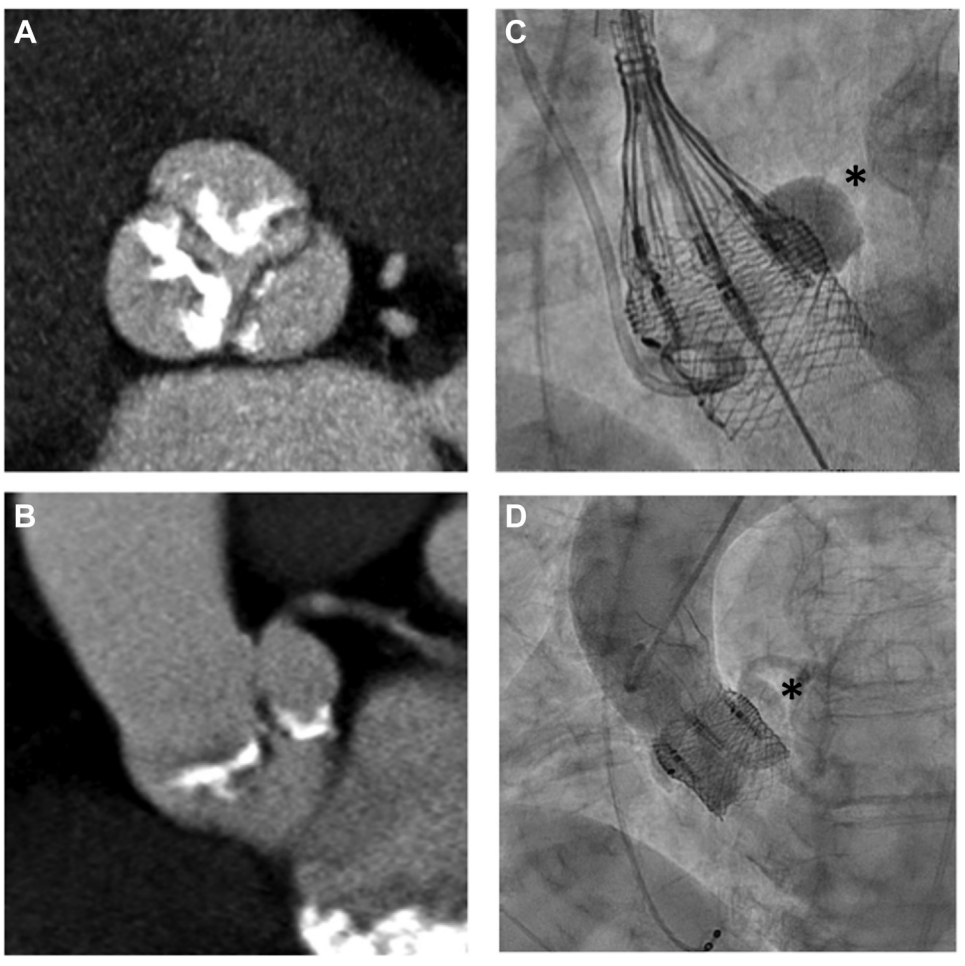
SoV = sinus of Valsalva

STJ = sinotubular junction

STS = Society of Thoracic Surgeons

TAVR = transcatheter aortic valve replacement

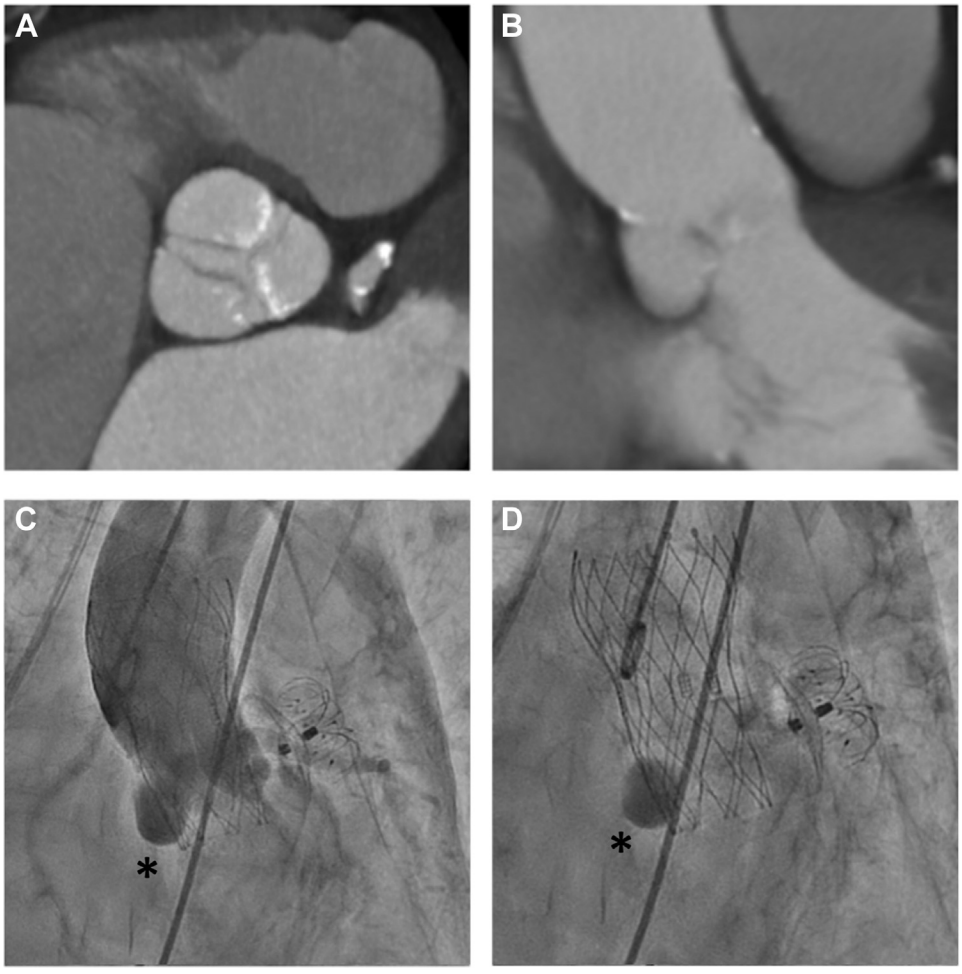
FIGURE 1 Patient 1



(A and B) Pre-transcatheter aortic valve replacement computed tomography scan showing severe valve calcification and a narrow sinotubular junction. **(C and D)** Fluoroscopic view showing the upper stent frame extending above the sinotubular junction with contrast agent retention **(asterisks)** in the left coronary sinus during and after valve implantation.

JACC: CASE REPORTS, VOL. 4, NO. 11, 2022
JUNE 1, 2022:666–670

FIGURE 2 Patient 2



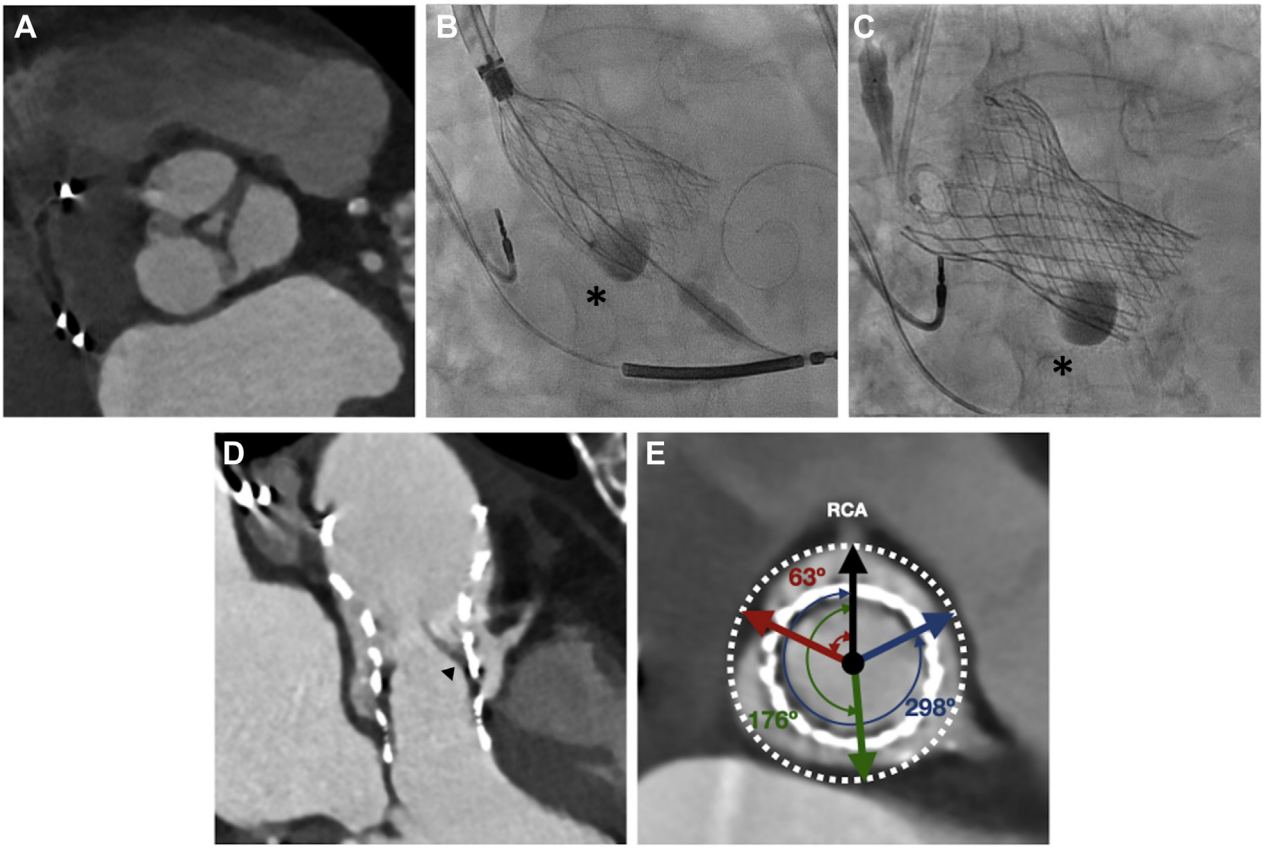
(A and B) Baseline computed tomography showing moderate valve calcification and a calcified sinotubular junction. **(C and D)** Aortography showing a short valve-to-aortic-wall distance above the noncoronary sinus with persistent contrast agent stagnation **(asterisks).**

atrial appendage occlusion, and end-stage renal disease. After heart team review (Society of Thoracic Surgeons [STS] risk score, 8.7%) and given her severe peripheral vascular disease, left transaxillary TAVR was indicated. Preprocedural CT showed moderate aortic valve calcification, with an aortic annulus perimeter of 69 mm and an area of 361 mm^2 and a calcified STJ (**Figures 2A and 2B**). After predilatation with a 20-mm balloon, a 25-mm Portico (Abbott Vascular) valve was implanted with no need for repositioning or recapture. Final angiography showed mild residual aortic regurgitation, with dense contrast agent stagnation at the noncoronary sinus (**Figures 2C and 2D,** Videos 4 and 5). Four hours later, the patient experienced right hemiparesis (4/5) and aphasia. Emergency head CT scan revealed occlusion of the left middle cerebral artery (M2 segment), and the patient underwent emergency thrombectomy with TICI (Thrombolysis In Cerebral Infarction) grade 2b partial recanalization. Unfortunately, the patient developed hemorrhagic transformation and died 3 weeks after the procedure.

PATIENT 3

An 84-year-old woman was admitted with an out-of-hospital cardiac arrest. She had a history of vertebrobasilar insufficiency and nonischemic dilated cardiomyopathy with moderate left ventricular dysfunction. Transthoracic echocardiography showed severe aortic regurgitation with worsening left ventricular systolic function (27%). Cardiac CT showed no

FIGURE 3 **Patient 3**



(A) Baseline computed tomography showing no valve calcification. **(B and C)** Contrast agent stagnation in the noncoronary sinus **(asterisks)** during and after valve implantation. **(D)** Hypoattenuation at the base of the commissure between the right and noncoronary cusps **(arrowhead)** with reduced leaflet motion. **(E)** Angles between each commissure and the right coronary artery (RCA) with no significant commissural misalignment.

coronary artery disease and mild aortic valve calcification (49 AU), with an aortic annulus perimeter of 72 mm and an area of 416 mm^2 (**Figure 3A**). After heart team review, an implantable cardioverter-defibrillator was inserted, and TAVR was indicated (STS risk score, 5.0%). The procedure was performed through transfemoral access, and after 2 recaptures, a 34-mm Evolut Pro (Medtronic) valve was successfully implanted with no residual aortic regurgitation. Given the presence of dense contrast material stagnation in the noncoronary cusp in the aortogram (**Figures 3B and 3C,** Videos 6 and 7), the patient was discharged on low-dose direct oral anticoagulant therapy. At 1-month follow-up, a CT scan showed subclinical leaflet thrombosis between the noncoronary and right coronary cusps (**Figures 3D and 3E**) that resolved with low-molecular-weight heparin treatment.

DISCUSSION

Sinus contrast material retention after TAVR is a rare phenomenon potentially translating to an increased thrombotic risk. The underlying etiologic factors may involve the Virchow triad: endothelial injury or focal sinus dissection (after predilatation, during delivery system advancement, recapturing or repositioning maneuvers, pigtail entrapment), blood stasis (flow stagnation and reduced washout within the neosinus, a narrow or low STJ), and hypercoagulability (older adult patients with cardiovascular risk factors).

In patients undergoing percutaneous left atrial appendage occlusion, postangiography retention of contrast material in the left atrial appendage has been associated with an increased risk of device-related thrombosis and a higher risk of thromboembolic stroke.[3] Of note, thrombosis is most likely to

JACC: CASE REPORTS, VOL. 4, NO. 11, 2022
JUNE 1, 2022:666–670

occur in low-flow regions with long blood residence time and poor washout.[4] In particular, sinus sequestration, which is most commonly seen in patients with a low and narrow STJ who are receiving taller-frame devices, may impair blood flow to the SoVs and coronary arteries and in turn may predispose patients to thrombosis. STJ diameter and height are relevant to identify anatomical features at risk of sinus sequestration in redo TAVR, as well as in native valves. Both patients 1 and 2 had narrow STJs (22 and 26 mm, respectively), the former with a low STJ (18 mm; 1 mm shorter than the 19-mm prosthesis height of the mechanically expandable valve) and the latter with STJ calcification. Hence, a short residual distance between the valve and the STJ in patients with small anatomical features may create a cul de sac in the SoVs that contributes to thrombus formation, akin to the phenomenon observed in patients with incomplete sealing of the left atrial appendage. In other patients, sequestration may be functional rather than anatomical, as a combination of reduced sinus washout, stasis, and proneness to thrombosis, and thus more difficult to predict by CT. Indeed, it has been speculated that blood flow stagnation in large SoVs could also increase the risk of hypoattenuated leaflet thickening after TAVR.[5] This was the case in patient 3, who, despite large SoVs and STJ, had follow-up CT that showed isolated thrombosis of the noncoronary cusp. There is also evidence that native-to-prosthetic commissural misalignment may reduce upper sinus flow by 40% which may increase the likelihood of thrombus formation, although this hypothesis could not be explored in our patients.[6]

Previous stroke and new onset AF infer a higher risk for stroke post-TAVR. In the 2 patients experiencing a stroke, only 1 had a history of stroke, and none of the thrombotic events were related to carotid artery disease or new onset AF (chronic AF in 1 patient with left atrial appendage occlusion and no device-related thrombus or patency). Of note, both strokes occurred immediately after the procedure, thus suggesting a procedure-related embolic origin, with most acute (≤24 hours) cerebrovascular events generally related to the procedure (thrombus, calcific embolization, endothelium denudation) rather than to pre-existing comorbidities.[1]

Low-dose aspirin has become the preferred antithrombotic therapy (over dual antiplatelet therapy) post-TAVR in patients without an indication for oral anticoagulant therapy. However, observation of persistent contrast agent retention immediately after TAVR may prompt close neurovascular monitoring and consideration for early anticoagulation and surveillance imaging to enable early recognition and management of ischemic complications. However, given the limited number of patients and other potential sources for cerebral emboli, these findings should be interpreted with caution and remain hypothesis generating.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Drs Asmarats, Millan, and Arzamendi have received proctoring and speaker fees from Abbott. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Dabit Arzamendi, Interventional Cardiology Unit, Hospital de la Santa Creu i Sant Pau, Sant Antoni M. Claret, 167, 08025 Barcelona, Spain. E-mail: darzamendi@santpau.cat.

REFERENCES

1. Armijo G, Nombela-Franco L, Tirado-Conte G. Cerebrovascular events after transcatheter aortic valve implantation. *Front Cardiovasc Med*. 2018;5:104.

2. Cahill TJ, Khalique OK, George I, Kodali S. Valve thrombosis after transcatheter and surgical aortic valve replacement: incidence and outcomes. *J Thorac Cardiovasc Surg*. 2022;163(4):1309–1315.

3. Lu X, Chen T, Liu G, et al. Relations between left atrial appendage contrast retention and thromboembolic risk in patients with atrial fibrillation. *J Thromb Thrombolysis*. 2022;53(1):191–201.

4. Vahidkhah K, Barakat M, Abbasi M, et al. Valve thrombosis following transcatheter aortic valve replacement: significance of blood stasis on the leaflets. *Eur J Cardiothorac Surg*. 2017;51:927–935.

5. Yanagisawa R, Hayashida K, Yamada Y, et al. Incidence, predictors, and mid-term outcomes of possible leaflet thrombosis after TAVR. *J Am Coll Cardiol Img*. 2017;10(1):1–11.

6. Salmonsmith JA, Ducci A, Burriesci G. Does transcatheter aortic valve alignment matter? *Open Heart*. 2019;6:e001132.

KEY WORDS stroke, thrombosis, transcatheter aortic valve replacement

APPENDIX For supplemental videos, please see the online version of this article.