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EDITORIAL
Aortic valvular heart disease

Refining stroke risk stratification after TAVR: the contribution of aortic atheroma

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This article refers to 'Association Between Aortic Arch Atheroma and Periprocedural Stroke Following Transcatheter Aortic Valve Implantation' by Suenaga *et al.*,[1] published in this issue of the Journal.[1] Stroke remains a feared complication of transcatheter aortic valve replacement (TAVR), with meaningful consequences for a patient's quality of life, functional status, and longevity.[2] Despite TAVR becoming more prevalent in patients at lower surgical risk and a decline in other TAVR complication rates,[3] post-TAVR stroke rates have remained largely stable,[4] and efforts to mitigate stroke risk have fallen short. Notably, cerebral embolic protection devices were developed to capture debris from native aortic valve expansion during TAVR, but they have not shown clinical benefit in all-comer TAVR populations.[5,6] A risk-model to predict post-TAVR stroke was created from clinical data reported on over 97 000 patients in the STS/ACC TVT Registry, but it had only modest predictive power.[7] Subsequent studies including more detailed clinical and imaging data have found inconsistent results with regards to clinical and procedural factors associated with stroke.[2,8–11]

More recently, efforts to refine stroke risk prediction have increasingly implicated aortic atheroma and plaque as a potential embolic source leading to stroke post-TAVR. Greater aortic plaque thickness and aortic total atheroma burden are associated with periprocedural stroke,[12,13] and severe aortic atheroma is associated with both periprocedural and long-term neurologic events.[14] However, classification of aortic lesions has an impact on study findings. For instance, studies looking at the association between aortic thrombus or atherothrombotic lesions and stroke at 1-month post-TAVR are discordant, albeit with different definitions of what constitutes a severe lesion.[15,16] Notably, many of these studies have used different measures of aortic atheroma and plaque, often consolidating disparate complex lesions into binary classifications for ease of statistical comparison in light of relatively small sample sizes (*Table 1*).

In this issue of *EHJ Valvular and Structural Heart Disease*, Suenaga *et al.*[1] examine the relationship between aortic arch atheroma severity and post-TAVR stroke risk at 1-month in a more nuanced fashion. This study expands upon prior work by leveraging a larger sample to employ an intuitive, arch atheroma grading scale and make comparisons by valve type. Among 1971 patients, aortic arch plaque burden was categorized using a graded scale based on pre-procedural imaging: none/mild (maximum plaque thickness (MPT) <3 mm), moderate (MPT 3–5 mm), severe (MPT >5 without shaggy morphology), or shaggy (MPT >5 with shaggy morphology). The authors find that, compared with mild/no atheroma, moderate atheroma was associated with a 2.9× (95% CI: 1.63–5.28) higher risk of stroke and shaggy atheroma was associated with a 14.6× (95% CI: 6.67–31.87) higher risk of stroke. The association between severe atheroma and stroke risk was not statistically significant but showed a trend towards an increased risk of stroke (aOR 2.81; 95% CI, 0.92–8.61; $P = .07$).

The authors also examined the relationship between aortic arch atheroma and stroke risk separately by valve type. A stepwise increase in periprocedural stroke risk was observed across worsening plaque categories among patients treated with a self-expanding valve (SEV) (2.8% vs 8.2% vs 13.6% vs 33.3%, $P < .001$), whereas stroke risk among balloon-expandable valve (BEV) recipients was only elevated for those with shaggy atheroma (1.1% vs 2.8% vs 1.4% vs 11.9%, $P < .001$). When comparing SEV to BEV directly, SEV were associated with higher adjusted odds of stroke in patients with none/mild atheroma (aOR 2.51; 95% CI 1.01–6.22, $P = .047$), moderate atheroma (aOR 2.91; 95% CI 1.27–6.86, $P = .012$), and severe atheroma (aOR 9.49; 95% CI 1.04–208.12, $P = .046$), while risk of stroke for patients with shaggy plaque was not significantly different for SEV and BEV (aOR 2.63; 95% CI 0.52–12.33, $P = .229$) after adjusting for confounders.

Taken together, these results paint a more detailed picture of how aortic atheroma can affect post-TAVR stroke risk. Shaggy atheroma emerges as a potent risk factor for post-TAVR stroke, regardless of valve type. The dose-response increase in stroke risk by MPT for SEV, but not for BEV suggests that embolization of aortic atheroma may in fact mediate many post-TAVR strokes, given that SEV interact with the aortic arch much more than BEV. These findings align with the hypothesized mechanism linking aortic atherosclerosis to post-TAVR stroke via embolization of atheromatous debris during catheter manipulation and device advancement through an atherosclerotic aorta.

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Table 1 Association of aortic atheroma, plaque thickness, and atherothrombotic lesions with post-TAVR stroke risk

Study	Sample size	Exposure	Stroke outcome	Association
Kataoka *et al.*[13]	$N = 278$	Total atheroma volume (TAV) measured by volumetric CT analysis of 4 aortic segments. Measured in cm^3 for ascending, descending, and abdominal aorta, as well as aortic arch.	Acute CVE within 24 hours: 16/278 (5.8%).	TAV in ascending aorta > 10.3 cm^3 (OR: 1.81; 95% CI: 1.34–2.21; $P = .008$) and TAV in aortic arch > 2.9 cm^3 (OR: 1.76; 95% CI: 1.29–2.10; $P = .009$) were independent predictors of stroke.
Miyasaka *et al.*[12]	$N = 1023$	Maximal aortic plaque thickness (MPT) in the ascending aorta, the aortic arch, and the descending aorta using CT.	Stroke within 48 hours: 26/1023 (3.1%)	Patients who had strokes were more likely to have greater MPT in the aortic arch (2.4 mm (IQR: 1.3–2.8 mm) vs 1.8 mm (IQR: 1.4–2.2 mm); $P < .01$) and descending aorta (2.9 mm (IQR: 2.1–4.2 mm) vs 2.8 mm (IQR: 2.1–3.6 mm), $P = .049$) compared with patients who did not have stroke.
Bonnet *et al.*[15]	$N = 641$	Presence of severe aortic wall thrombus (AWT) defined by composite score ≥8 (binary). Composite scores were assigned by assessing number of affected segments of aorta, thrombus type, thickness, area, and circumference.	Stroke within 24 hours: 21/641 (3.3%)	Patients with severe AWT had increased chance of stroke compared with moderate or no/mild AWT. (13.7% vs 2.2% vs 1.4%, $P < .001$).
Kikuchi *et al.*[14]	$N = 977$	Presence of severe aortic atheroma (binary) was defined as protruding atheroma of ≥3 mm thickness with protruding components, ulcerated atheroma with ulcer-like intimal disruption, and atheroma of ≥5 mm thickness.	Stroke within 1 month: 43/977 (4.4%)	Patients with severe aortic atheroma had a higher risk of 30-day stroke (adjusted HR: 2.12 (95% CI: 1.15–3.90), $P = .016$) and ischaemic stroke at 2 years (adjusted HR 3.71, 95% CI: 1.35–10.2).
Shirai *et al.*[16]	$N = 278$	Presence of atherothrombotic lesion (binary), defined as: (1) intimal thickening ≥4 mm, OR (2) mobile-looking, ulcerated, or protruding atheroma.	Stroke within 1 month: 4/278 (1.4%)	No difference in stroke risk between patients with and without atherosclerotic lesions (3.5% vs 2.5%, $P = .72$).

This study has several key strengths that make it a valuable addition to the literature on post-TAVR stroke. First, the low loss to follow-up in this study (99.8%) out to 30 days with prospective phone calls to ascertain outcomes provides a high level of confidence in the ability to capture stroke after TAVR. Second, this study has a larger sample than previous studies that examine aortic atheroma and can thus examine impact of differences in gradation of atheroma and interaction between atheroma and valve type in a nuanced fashion. Finally, the intuitive grading scale of aortic arch atheroma used in this study can be readily utilized more broadly, which makes the findings of the study easily replicable by clinicians counselling patients regarding TAVR, particularly given the ubiquitousness of TAVR CT for pre-procedural planning.

This study must also be interpreted in the context of its limitations. Given that this was a retrospective observational study, the possibility of residual confounding exists. Specifically, extensive atheroma arch is likely associated with atherosclerosis in other vascular beds, including those that feed the brain. As such, it is possible that aortic arch atheroma is merely a marker of increased stroke risk, rather than a true mediator of stroke through embolization. Thus, it is possible that newer cerebral embolic protection devices in development that are designed to protect against aortic atheroma embolization may still fall short. Nevertheless, even if aortic atheroma is a marker of stroke risk, the study results can still inform patient counselling. Additionally, the study included a mix of older and newer BEV and SEV, and it is likely underpowered to detect differences in different generations of valve platforms. Notably, newer versions of the Evolut SEV platform have increased flexibility due to multiple spines, which can reduce contact with the aortic arch and theoretically decrease the risk of embolization. Thus, these results on the differential impact of atheroma on valve type may not generalize to the most current-generation of devices.

The findings of this study have important implications that can guide clinicians today. The authors demonstrate that a simple, CT-based quantification of aortic atheroma can provide meaningful data about a patient's risk of post-TAVR stroke and may help inform procedural planning. Patients with more extensive aortic arch atheroma burden should be counselled that they have a higher post-TAVR stroke risk. Additionally, in patients with substantial aortic arch atheroma, a BEV may be preferred over SEV when anatomically feasible to mitigate the risk of stroke, though further study in current-generation devices is needed.

In sum, Suenaga *et al.*[1] move the field beyond blunt, binary descriptors of aortic disease and towards a more clinically actionable understanding of how aortic atheroma burden shapes stroke risk after TAVR. Their work reinforces the central role of the aorta as a potential embolic source, highlights important interactions with valve platform, and underscores the value of careful, pre-procedural CT-based phenotyping. While the possibility of residual confounding and evolving device technology warrant caution, these data provide a pragmatic framework for risk stratification and shared decision-making today. Ultimately, integrating nuanced anatomic risk assessment into procedural planning and testing targeted mitigation strategies in contemporary practice represents a necessary next step if we are to finally bend the stubborn curve of post-TAVR stroke.

Author contributions

Vijay A. Joshi (BS (Conceptualization [supporting]; Writing—original draft [lead]; Writing—review & editing [supporting])), and Neel M. Butala (Conceptualization [lead]; Writing—original draft [supporting]; Writing—review & editing [lead])

Declarations

Disclosure of Interest

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References

1. Suenaga TIK, Shirai S, Yoshida S, Wakugawa H, Miyamoto K, Miyahara K, *et al.* Association between aortic arch atheroma and periprocedural stroke following transcatheter aortic valve implantation. *EHJ Valvular Struct Heart Dis* 2026.
2. Patel KP, Rathod KS, Lansky AJ, Prendergast B, Kharbanda RK, Mathur A, *et al.* Cerebrovascular events in patients undergoing transcatheter aortic valve replacement: a review. *Stroke* 2024;**55**:2754–64. https://doi.org/10.1161/STROKEAHA.124.047149
3. Carroll JD, Mack MJ, Vemulapalli S, Herrmann HC, Gleason TG, Hanzel G, *et al.* STS-ACC TVT registry of transcatheter aortic valve replacement. *Ann Thorac Surg* 2021;**111**:701–22. https://doi.org/10.1016/j.athoracsur.2020.09.002
4. Huded CP, Tuzcu EM, Krishnaswamy A, Mick SL, Kleiman NS, Svensson LG, *et al.* Association between transcatheter aortic valve replacement and early postprocedural stroke. *JAMA* 2019;**321**:2306–15. https://doi.org/10.1001/jama.2019.7525
5. Kapadia SR, Makkar R, Leon M, Abdel-Wahab M, Waggoner T, Massberg S, *et al.* Cerebral embolic protection during transcatheter aortic-valve replacement. *N Engl J Med* 2022;**387**:1253–63. https://doi.org/10.1056/NEJMoa2204961
6. Kharbanda RK, Kennedy J, Jamal Z, Dodd M, Evans R, Bal KK *et al.* Routine cerebral embolic protection during transcatheter aortic-valve implantation. *N Eng J Med* 2025;**392**:2403–12. https://doi.org/10.1056/NEJMoa2415120
7. Thourani VH, O'Brien SM, Kelly JJ, Cohen DJ, Peterson ED, Mack MJ, *et al.* Development and application of a risk prediction model for in-hospital stroke after transcatheter aortic valve replacement: a report from the society of thoracic surgeons/American College of Cardiology transcatheter valve therapy registry. *Ann Thorac Surg* 2019;**107**:1097–103. https://doi.org/10.1016/j.athoracsur.2018.11.013
8. Saeed Al-Asad K, Martinez Salazar A, Radwan Y, Wang E, Salam MF, Sabanci R, *et al.* Transcatheter aortic valve replacement in bicuspid versus tricuspid aortic valve stenosis: meta-analysis and systemic review. *Am J Cardiol* 2023;**203**:105–12. https://doi.org/10.1016/j.amjcard.2023.06.120
9. Vlastra W, Jimenez-Quevedo P, Tchetche D, Chandrasekhar J, de Brito FS Jr., Barbanti M, *et al.* Predictors, incidence, and outcomes of patients undergoing transfemoral transcatheter aortic valve implantation complicated by stroke. *Circ Cardiovasc Interv* 2019;**12**:e007546. https://doi.org/10.1161/CIRCINTERVENTIONS.118.007546
10. Gorla R, Tua L, D'Errigo P, Barbanti M, Biancari F, Tarantini G, *et al.* Incidence and predictors of 30-day and 6-month stroke after TAVR: insights from the multicenter OBSERVANT II study. *Catheter Cardiovasc Interv* 2023;**102**:1122–31. https://doi.org/10.1002/ccd.30848
11. Cheikh-Ibrahim M, de Pontes VB, Alachkar N, Jaramillo S, Esteves IM, Lauten A. Balloon postdilation after transcatheter aortic valve implantation (TAVI) among self- and balloon-expandable valves: a systematic review and meta-analysis. *Am J Cardiol* 2026;**260**:61–70. https://doi.org/10.1016/j.amjcard.2025.11.012
12. Miyasaka M, Sharma RP, Maeno Y, Taguri M, Yoon SH, Kawamori H, *et al.* Investigation of computed-tomography based predictors of acute stroke related to transcatheter aortic valve replacement: aortic wall plaque thickness might be a predictive parameter of stroke. *J Invasive Cardiol* 2020;**32**:E18–26. https://doi.org/10.25270/jic/19.00263
13. Kataoka Y, Puri R, Pisaniello AD, Hammadah M, Qintar M, Uno K, *et al.* Aortic atheroma burden predicts acute cerebrovascular events after transcatheter aortic valve implantation: insights from volumetric multislice computed tomography analysis. *EuroIntervention* 2016;**12**:783–9. https://doi.org/10.4244/eijv12i6a127
14. Kikuchi S, Trimaille A, Carmona A, Truong DP, Matsushita K, Marchandot B, *et al.* Long-term outcomes following transcatheter aortic valve replacement in patients with severe aortic atheroma. *Am J Cardiol* 2025;**256**:23–30. https://doi.org/10.1016/j.amjcard.2025.07.009
15. Bonnet M, Maxo L, Lohse T, Mangin L, Courand PY, Ricard C, *et al.* Association between aortic wall thrombus and thromboembolic events after transfemoral transcatheter aortic valve replacement. *JACC Cardiovasc Interv* 2024;**17**:1680–90. https://doi.org/10.1016/j.jcin.2024.05.033
16. Shirai Y, Tanaka A, Tokuda Y, Kondo H, Mizutani K, Uemura T, *et al.* Impact of aortic atherosclerosis on clinical outcomes after transfemoral transcatheter aortic valve replacement. *Catheter Cardiovasc Interv* 2025;**106**:2661–7. https://doi.org/10.1002/ccd.70125