Antithrombotic management after transcatheter aortic valve implantation: Evolving evidence and simplified strategies in the 2025 European Society of Cardiology era

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SUMMARY

Transcatheter aortic valve implantation (TAVI) has revolutionised the treatment of severe aortic stenosis, extending from inoperable patients to those at low surgical risk. Yet thromboembolic and bleeding events remain major determinants of outcomes. Antithrombotic management after TAVI is complex because of competing ischemic and haemorrhagic risks in elderly, comorbid patients. Over the past decade, randomised evidence and large registries have progressively shifted the paradigm toward treatment simplification. Current recommendations now favour aspirin monotherapy for patients without an indication for long-term oral anticoagulation (OAC), and OAC monotherapy for those requiring anticoagulation, reserving dual antiplatelet therapy (DAPT) only for selected cases such as recent percutaneous coronary intervention. Subclinical leaflet thrombosis, a frequent but often transient phenomenon, remains of uncertain clinical significance. This review integrates current evidence with real-world insights, mechanistic data, and evolving concepts that underpin the contemporary, individualised approach to post-TAVI antithrombotic therapy.

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INTRODUCTION

The exponential expansion of transcatheter aortic valve implantation (TAVI) across all surgical-risk strata has redefined the management of aortic stenosis.[1] With procedural mortality now below 2% in low-risk trials, attention has turned toward optimising long-term outcomes through refined post-procedural pharmacotherapy.[2,3] Despite remarkable procedural advances, thrombotic, valve-related, and bleeding complications remain the leading causes of morbidity and mortality after TAVI.[4] The challenge lies in achieving the delicate balance between ischemic protection and haemorrhagic safety in an elderly, often frail, and comorbid population.[2] From a hematologic and pathophysiologic perspective, TAVI constitutes a perfect storm for thrombogenesis: the trauma of valve deployment, exposure of sub-endothelial tissue factor, turbulent flow within the stent frame, and sustained inflammatory activation collectively create a prothrombotic milieu.[5] This environment not only predisposes to cerebrovascular and systemic ischemic events but also to subclinical or overt valve thrombosis, an increasingly recognised entity with potential long-term impact on valve durability and outcomes.[6]

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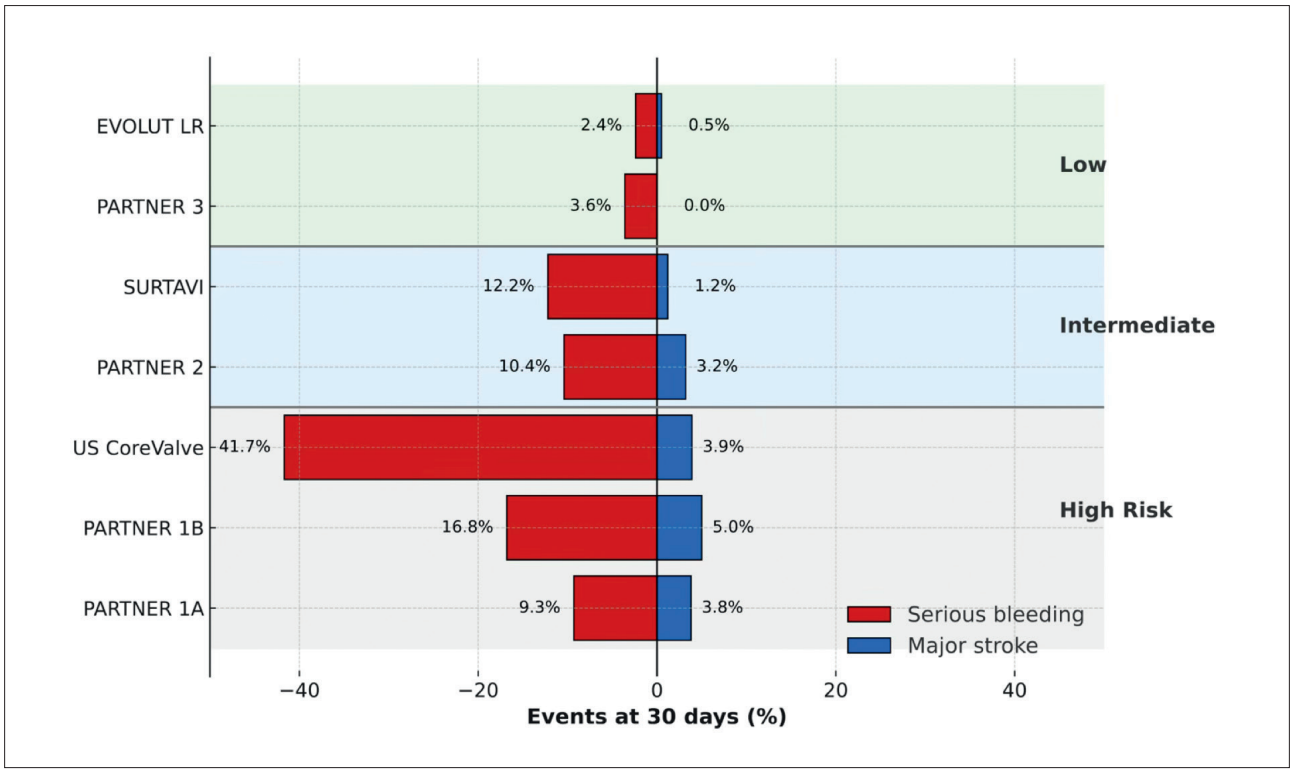

FIGURE 1. The bar charts summarise the 30-day **(A)** and 1-year **(B)** risks for major stroke and serious bleeding in regulatory trials of transcatheter aortic valve replacement across the spectrum of surgical risk. Risk is defined by categories of Society of Thoracic Surgeons Predicted Risk of Mortality score (low risk defined as 4%, intermediate risk defined as 5% - 8%, high risk defined as >8%).

EVOLUT LR: Evolut Low Risk; PARTNER: Placement of Aortic Transcatheter Valve; SURTAVI: Safety and Efficacy Study of the Medtronic CoreValve System in the Treatment of Severe, Symptomatic Aortic Stenosis in Intermediate Risk Subjects Who Need Aortic Valve Replacement.

All this explains the historical reliance on combined antithrombotic therapy, an approach now questioned in light of growing evidence favouring simplified, individualised regimens.[4]

THROMBOTIC AND BLEEDING RISKS AFTER TAVI

Beyond procedural success, thrombotic and bleeding complications remain pivotal determinants of long-term outcomes after TAVI. Both are frequent, often coexistent and influenced by shared risk factors such as advanced age, chronic kidney disease, vascular fragility, and concomitant antithrombotic therapy.[7] However, their temporal pattern and prognostic impact diverge markedly. Thrombotic events after TAVI, including stroke, myocardial infarction, valve thrombosis, and systemic embolism, predominantly occur in the early post-procedural phase but retain prognostic significance throughout follow-up. In the GALILEO analysis, one-year mortality after a thromboembolic event approached 54%, with a median time to death of only 36 days, underscoring their abrupt and devastating clinical impact.[8] Registry data from more than 100,000 patients in the STS/ACC TVT database reported an overall stroke incidence of 2.3%, which remained stable over time, while the occurrence of stroke was associated with a six-fold increase in 30-day mortality.[9] Consistent findings from randomised studies indicate that stroke, the most feared manifestation, occurs in approximately 2–5% of patients in the periprocedural phase and 1–2% annually thereafter (*Figure 1*).[10] The mechanisms underlying these events are multifactorial, encompassing procedural embolisation, systemic inflammation, hypercoagulability, and particularly atrial fibrillation, the most prevalent and clinically impactful contributor to late thromboembolism. Atrial fibrillation is present in up to 40% of TAVI candidates and newly diagnosed in 10–15%, doubling the risk of stroke and death and mandating oral anticoagulation.[11] The timing of stroke onset reflects distinct mechanisms and risk profiles.[9] **Early strokes** are predominantly embolic, resulting from catheter manipulation within the aortic arch, balloon valvuloplasty, or valve crossing of heavily calcified cusps. Large-bore sheaths and balloon post-dilatation can cause

endothelial trauma, activating platelets and the coagulation cascade, while transient hemodynamic compromise during rapid pacing may further reduce cerebral perfusion, especially in watershed territories. **Late strokes**, by contrast, are mainly driven by AF, incomplete endothelialisation of the prosthetic frame, and persistent atherothrombotic burden in elderly and frail patients. By contrast, bleeding events, though more frequent, exert a more delayed but less lethal impact. Major or life-threatening bleeding (BARC 2–3) occurs in up to 10–15% of patients within the first year, yet is associated with a lower one-year mortality rate of approximately 17%, with a median delay to death of 178 days.[12] Early bleeding is often procedural (access-related, anticoagulation-induced), whereas late events are predominantly gastrointestinal or drug-related, reflecting cumulative exposure to oral anticoagulant (OAC) or antiplatelet therapy. This dichotomy underscores that thrombotic events carry a higher early mortality burden, while bleeding complications contribute to long-term morbidity, frailty, and re-hospitalisation. Consequently, post-TAVI management must balance early ischemic protection with later bleeding prevention through phase-adapted, simplified antithrombotic regimens.[4]

TAVI THROMBOSIS AND SUBCLINICAL LEAFLET THROMBOSIS

Clinically overt thrombosis after TAVI remains uncommon, with an incidence of approximately 0.6-1% across large multicentre cohorts.[13] The median time to diagnosis is around six months, and most patients present with dyspnoea or a progressive rise in transvalvular gradient, while up to one-third remain asymptomatic. Echocardiography typically demonstrates a marked increase in mean gradient with leaflet thickening, whereas CT imaging confirms mural or leaflet-level thrombus formation (*Figure 2*).[14] Although infrequent, TAVI thrombosis is clinically relevant because of its association with prosthetic dysfunction, recurrent hospitalisation, and poorer survival.

The modern understanding of subclinical leaflet thrombosis (SLT) emerged in 2015 when Makkar *et al.* first reported the association between reduced leaflet motion (RLM) and stroke in the Portico IDE study.[15] Subsequent systematic imaging analyses revealed that SLT occurs far more frequently than initially suspected, affecting up to 40% of patients in early trials and 10–15% in contemporary real-world registries.[16,17] These observations defined the hypo-attenuated leaflet thickening (HALT)–RLM continuum, reshaping post-TAVI imaging surveillance and mechanistic interpretation.[18] HALT reflects focal thrombus deposition near the leaflet base, while RLM and RELM denote

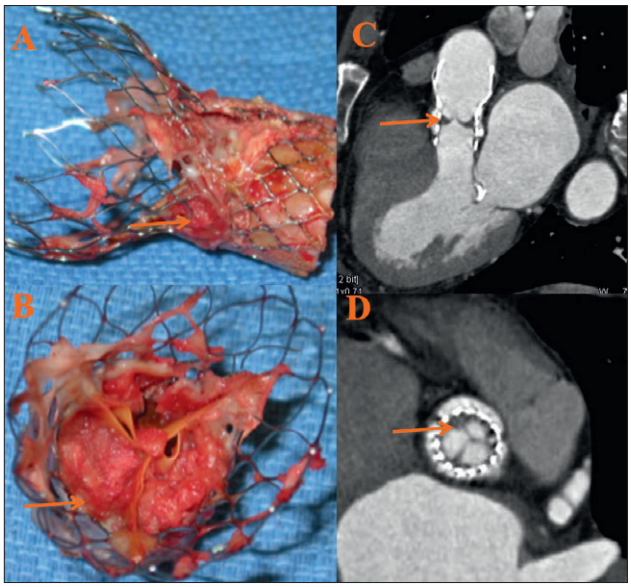

FIGURE 2. Gross and imaging appearance of clinical transcatheter aortic valve thrombosis. **(A, B)** Macroscopic specimens of explanted transcatheter heart valves demonstrate extensive mural and leaflet thrombosis (orange arrows) adherent to both the stent frame and the valve leaflets, resulting in restricted motion and impaired orifice area. **(C, D)** Multidetector computed tomography in longitudinal **(C)** and transverse **(D)** planes reveals hypo-attenuated masses (arrows) consistent with thrombus, with clear evidence of leaflet thickening and reduced leaflet motion.[14]

progressive motion restriction that may or may not translate into elevated gradients.[17] Importantly, CT, not echocardiography, remains the gold standard for diagnosis and grading, as the hemodynamic impact of HALT is often modest and Doppler findings may remain within normal limits.[19] This imaging–clinical dissociation underscores the silent nature of SLT. While early observational data, such as that from Rachid *et al.*, suggested a possible association between HALT and cerebrovascular events, subsequent analyses revealed substantial heterogeneity in imaging definitions and timing.[20] Randomised studies have not confirmed a causal relationship between HALT and stroke, and most lesions remain clinically silent.[21] The prognostic relevance of SLT therefore remains debated, whether it represents a true pathologic substrate predisposing to clinical valve thrombosis or merely a marker of an underlying prothrombotic milieu remains uncertain. The natural history of SLT appears dynamic and variable: approximately half of cases regress spontaneously, one-third remain stable, and a minority (around 10–15%) progress toward hemodynamically significant or clinically overt valve thrombosis.[19,22]

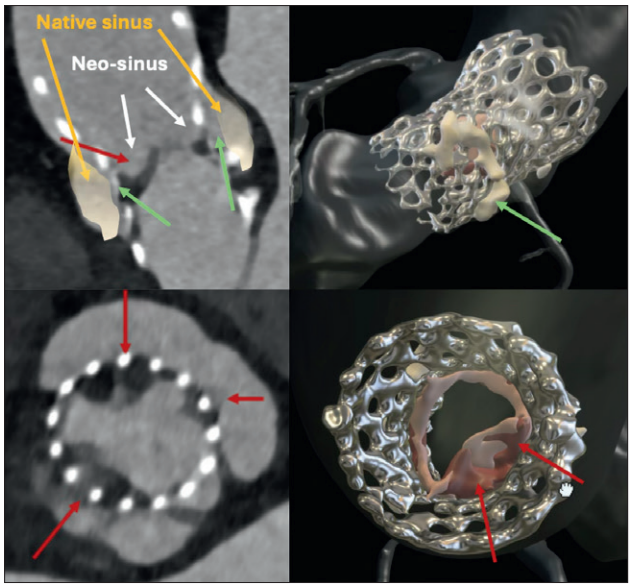

FIGURE 3. This figure demonstrates the spatial relationship between native leaflets, prosthetic stent, and zones most susceptible to thrombotic deposition within the neosinus after TAVI, using corresponding arrows for clarity. Green arrows indicate native calcified leaflet tissue retained within the prosthetic stent frame. Red arrows highlight areas of leaflet thrombosis (HALT), visible by both CT imaging and 3D-rendered anatomical modelling. The left panels correspond to cross-sectional and transverse CT images, while the right panels show 3D reconstructions for spatial orientation of neosinus structures, thrombus, and leaflet positions.

Oral anticoagulation (vitamin K antagonist or direct OAC) usually leads to rapid radiologic resolution within three months, though normalisation of CT findings does not always correlate with improved clinical outcomes.[23] Hence, routine anticoagulation for all TAVI recipients is not justified. Beyond the leaflets, the broader concept of "aortic valve complex thrombosis" encompasses perivalvular thrombi within the sinus of Valsalva and thrombus formation along the stent frame.[17] Integration of multimodal imaging, echocardiography, 4D-CT, and PET/CT, is essential for accurate detection, risk stratification, and longitudinal follow-up.[24,25] These modalities collectively demonstrate that TAVI thrombosis extends beyond a localised leaflet event, representing instead a continuum of thrombo-inflammatory activity involving the entire valve complex, from the neo-sinus to the retained native valve tissue.[17,26]

PATHOPHYSIOLOGY OF AORTIC VALVE COMPLEX THROMBOSIS

Thrombus formation following TAVI results from a complex interplay between prosthesis-related, anatomic, and systemic factors (*Figure 3*).[27] The implantation of a metallic frame with a polymeric sealing skirt inside a heavily calcified native annulus creates a challenging hemodynamic, biological and artificial material interface.[28] Non-endothelialised prosthetic surfaces, residual native leaflets, and exposed calcium promote platelet and coagulation activation, thrombin generation, and fibrin deposition. Regions of stasis and low shear stress, particularly within the newly formed neo-sinuses, further favour coagulation and thrombus accumulation.[29] Procedural and device determinants play a major role in shaping this risk. Valve under expansion, commissural misalignment, and asymmetric deployment disturb sinus flow and delay washout. Supra-annular self-expanding valves, by enlarging the neosinus and altering sinus geometry, are especially prone to flow recirculation.[30] Leaflet design, polymer composition, and stent-frame architecture influence shear gradients and residence time, thereby defining where thrombus develops. A high calcium burden and incomplete prosthesis apposition have consistently emerged as strong predictors of leaflet thrombosis.[5] At the cellular level, both experimental and pathological studies demonstrate the persistence of a pro-thrombotic and inflammatory state. Up-regulation of tissue factor, high rates of residual platelet reactivity on dual antiplatelet therapy (DAPT), and ongoing endothelial injury have been documented throughout the transition from native aortic stenosis to post-TAVI remodeling.[31,32] Histological analysis of explanted valves reveals organised thrombus along leaflet surfaces and peri-stent regions, confirming that valve thrombosis is a dynamic thrombo-inflammatory process rather than a static deposit.[33] Recent evidence has expanded our understanding of the host–prosthesis interaction. Once regarded as biologically inert, the retained calcified native valve is now recognised as an active driver of thrombogenicity after TAVI. ^{18}F-NaF PET/CT imaging demonstrates persistent metabolic activity within the native leaflets months after implantation, independent of mechanical stress.[34] This activity correlates with valve calcium volume and predicts later dysfunction or thrombosis. Histological analyses show continued expression of osteopontin and Runx-2, indicating a sustained osteogenic and inflammatory response.[35] Moreover, the calcified native valve acts as a reservoir of pro-coagulant extracellular vesicles (EVs) released from inflammatory and endothelial cells.[36] These vesicles, rich in tissue factor and adhesion molecules, convert valvular endothelium into a prothrombotic and pro-adhesive phenotype. Patients with severe aortic stenosis exhibit elevated circulating interleukin-1β, interleukin-6, TNF-α, and increased factor Xa activity, which persist transiently after TAVI.[37] This

inflammatory–coagulant crosstalk perpetuates valvular endothelial dysfunction and may maintain local thrombogenic potential even after a technically successful procedure.

ANTITHROMBOTIC MANAGEMENT IN TAVI PATIENTS

After delineating the dual burden of thrombotic and bleeding complications, the next challenge lies in defining how antithrombotic therapy should be applied across the different procedural phases of TAVI.[2] The therapeutic rationale stems from the interaction between prosthesis-related and patient-specific determinants.[27,38]

During the procedure, unfractionated heparin remains the cornerstone to prevent device-related and procedural thrombosis, titrated to maintain an activated clotting time between 250 and 300 seconds. At the end of the intervention, protamine is often administered for heparin reversal, a practice supported by randomised evidence showing reduced life-threatening and major bleeding without excess ischemic events.[39] Whether full reversal may increase periprocedural cerebrovascular events in patients with high baseline stroke risk remains uncertain. In patients with an existing indication for OAC, continuing rather than interrupting oral anticoagulation has been shown to be safe and may even lower thromboembolic risk, simplifying the management of the periprocedural phase.[40]

TABLE 1. Key clinical studies on antithrombotic therapy after TAVI.

Trial	Treatment & Control	Timeframe	SLT (HALT/RLM)	MACCE/ Ischemic Events	Bleeding	Advantage
ARTE (N=222)	DAPT vs Aspirin	90 days	NA	Death/MI/ stroke/TIA: 15.3% vs 7.2% (NS)	Major/LT: 10.8% vs 3.6% (P=0.038)	Aspirin monotherapy reduces bleeding
POPULAR TAVI A (N=665)	Aspirin vs DAPT	1 year	NA	CV death/MI/ stroke: 9.7% vs 9.9%	Any: 15.1% vs 26.6% (P=0.001)	Aspirin monotherapy safest
GALILEO (N=1644)	Rivaroxaban+ aspirin vs DAPT	90 days	HALT: 12.4% vs 32.4%; RLM: 2.1% vs 10.9%	Death: 2.6% vs 1.7%; Stroke: 3.5% vs 0.9%	Major/LT: 3.5% vs 0.9%	No net benefit; higher harm with rivaroxaban
ATLANTIS (N=1049)	Apixaban vs antiplatelet ± DAPT	1 year	Thrombosis: 19.2% vs 25%; HALT/ RLM: 8.9% vs 13.0%	Death/MI/ stroke/ Peripheral embolism: 8.6% vs 6.4%	Major/LT: 9.5% vs 6.9%	No outcome benefit; increased mortality in some strata
ADAPT-TAVR (N=229)	Edoxaban vs DAPT	6 months	HALT: 37.3% vs 48.6%; RLM: 2.9% vs 7.3%	Death: 2.7% vs 1.7%; Stroke: 1.8% vs 1.7%	Major/LT: 5.4% vs 3.4%	Edoxaban reduces subclinical thrombosis
POPULAR TAVI B (N=313)	OAC vs OAC+ clopidogrel	1 year	NA	CV death/MI/ stroke: 13.4% vs 17.3%	Any: 21.7% vs 34.6% (P=0.01)	OAC monotherapy safest for AF
ENVISAGE-TAVI AF (N=1426)	Edoxaban vs warfarin	18 months	No valve thrombosis	CV death/MI/ stroke: 8.2 vs 8.1/100 person-years	Major: 9.7 vs 7.0/100 person-years (NS)	Edoxaban non-inferior to VKA; more GI bleeding

DAPT: dual antiplatelet therapy; OAC: oral anticoagulation; HALT: hypo-attenuated leaflet thickening; RLM: restricted leaflet motion; LT: life-threatening; MACCE: major adverse cardiac and cerebrovascular events; AF: atrial fibrillation; NS: not significant; VKA: vitamin K antagonist; GI: gastrointestinal.

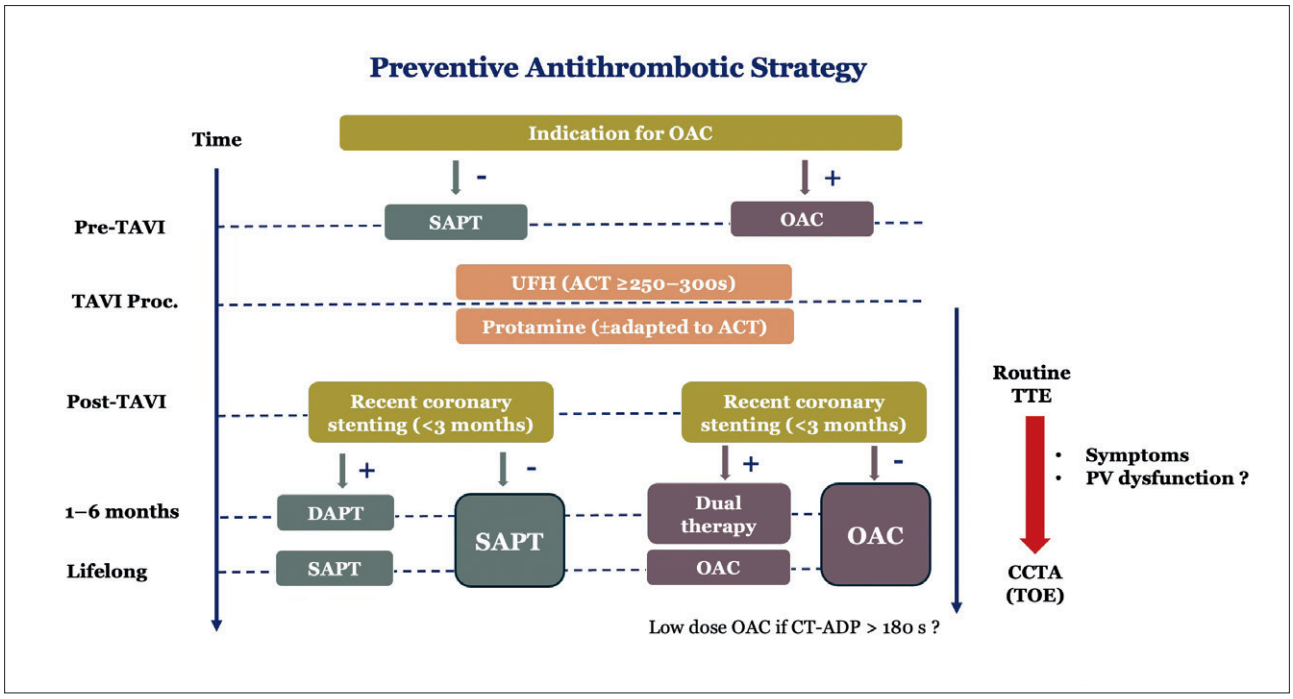

FIGURE 4. Preventive antithrombotic strategy after TAVI.

Algorithm outlining peri-procedural and post-procedural antithrombotic management according to the presence or absence of an indication for oral anticoagulation (OAC). UFH is used during the TAVI procedure with ACT-guided reversal using protamine. Post-TAVI therapy is based on recent coronary stenting status and bleeding risk, with single antiplatelet therapy (SAPT) or OAC as long-term treatment. Routine echocardiographic surveillance (TTE) and CCTA are recommended in patients with symptoms or suspected prosthetic valve dysfunction.

DAPT: Dual antiplatelet therapy.

Beyond the acute phase, a decade of randomised trials has transformed clinical practice from empiricism to evidence-based simplicity (*Table 1*). Early studies such as ARTE and POPular-TAVI established that dual antiplatelet therapy confers no ischemic advantage over aspirin alone but increases major bleeding by approximately 40%.[41,42] Routine anticoagulation in patients without an OAC indication, as tested in GALILEO and ATLANTIS, failed to improve clinical outcomes and raised safety concerns.[8,43] In GALILEO, low-dose rivaroxaban was associated with higher mortality and bleeding, leading to early trial termination. In ATLANTIS, overall results were neutral, but stratum 2, comprising patients without a chronic OAC indication, showed a significantly higher all-cause mortality with apixaban compared with standard antiplatelet therapy, mainly due to non-cardiovascular deaths such as sepsis and renal disease rather than bleeding or ischemic events. The imaging sub-studies of these trials further refined the mechanistic understanding of valve thrombosis. In GALILEO-4D, low-dose rivaroxaban 10 mg daily combined with aspirin for three months markedly reduced HALT and RLM compared with dual antiplatelet therapy, suggesting a dose-dependent anticoagulant effect on subclinical leaflet thrombosis.[18] Similarly, ATLANTIS-4D-CT demonstrated that apixaban 5 mg twice-daily lowered HALT incidence relative to antiplatelet therapy in patients without an OAC indication. However, at 1-year follow-up, neither study showed differences in death, stroke, or myocardial infarction, underscoring that HALT represents an imaging rather than a clinical phenomenon.

The ADAPT-TAVR trial further explored this dissociation between imaging and clinical endpoints. In this study, patients were randomised to edoxaban 60 mg once daily (or 30 mg when indicated), *versus* dual antiplatelet therapy to evaluate the effect on cerebral ischemic lesions and subclinical leaflet thrombosis.[44] At 6 months, HALT was numerically lower with edoxaban (8.5% *vs* 15.3%) but the difference did not reach statistical significance. Similarly, there was no reduction in new cerebral lesions on MRI and no improvement in neurocognitive outcomes. These findings reinforced the concept that preventing HALT does not necessarily translate into measurable clinical benefit and that routine anticoagulation for asymptomatic SLT or silent cerebral lesions cannot be justified.

In patients with atrial fibrillation or another formal indication for anticoagulation, OAC monotherapy remains the optimal strategy. The POPular-TAVI Cohort B trial provided pivotal confirmation that oral anticoagulation alone is sufficient in this setting: compared with OAC plus clopidogrel for 3 months, OAC monotherapy significantly reduced bleeding events without increasing thromboembolic complications. The ENVISAGE-TAVI AF trial compared edoxaban 60 mg daily (or 30 mg when indicated) with vitamin K antagonists and demonstrated non-inferior efficacy for thromboembolic prevention but a significant increase in major bleeding, predominantly gastrointestinal.[45] Conversely, ATLANTIS, using apixaban 5 mg twice daily, found similar efficacy to VKAs with numerically fewer major bleeds, supporting its use in elderly and frail populations.[43] Real-world studies have reinforced these observations. A meta-analysis including more than 25,000 patients showed that DOACs are a safe alternative to VKAs, with similar all-cause mortality, stroke, and major bleeding but a lower incidence of any bleeding.[46] The large SwissTAVI registry confirmed these findings in 2,900 matched patients, showing no difference in the composite endpoint of mortality, stroke, and major bleeding, but a significantly higher 1- and 5-year all-cause mortality among VKA users, along with a lower risk of disabling stroke.[47] Collectively, these data indicate that DOACs offer a more favourable balance between efficacy and safety in the post-TAVI population requiring long-term anticoagulation. The FRAIL-AF randomised controlled trial provided additional nuance in older, frail patients with atrial fibrillation.[48] Switching from well-managed VKA therapy to a DOAC led to an unexpected increase in bleeding complications, mainly driven by clinically relevant non-major bleeding. However, due to design limitations and suboptimal INR control in many elderly patients, DOACs remain a reasonable and often preferable option for older adults, particularly those with unstable INR values or difficulty maintaining therapeutic range.

GUIDELINE EVOLUTION AND CONTEMPORARY RECOMMENDATIONS

The 2025 ESC/EACTS Guidelines now recommend lifelong single antiplatelet therapy with aspirin (75–100 mg daily) for most patients after TAVI, as dual therapy offers

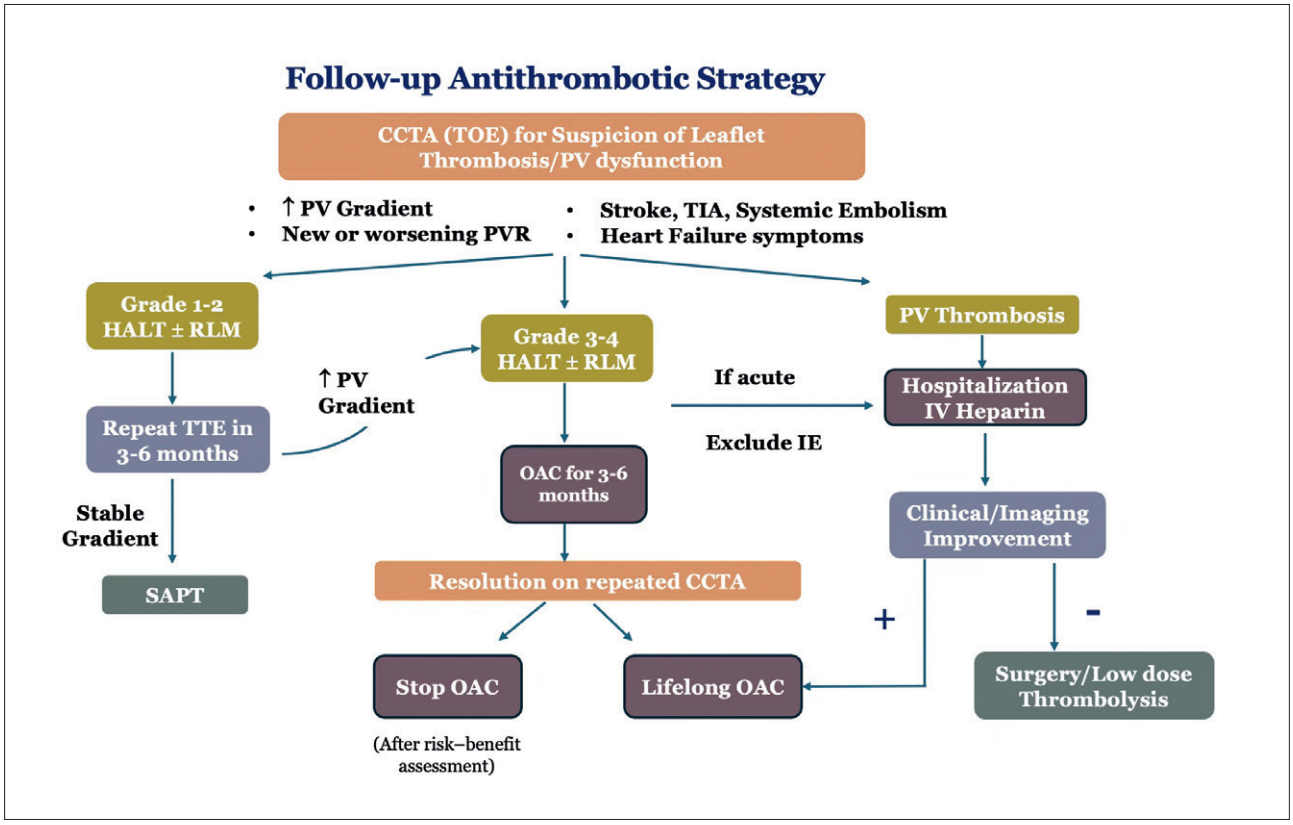

FIGURE 5. Follow-up antithrombotic strategy after TAVI.
Algorithm outlining the management of suspected leaflet thrombosis or prosthetic valve (PV) dysfunction.
CCTA: Cardiac CT angiography; HALT: hypo-attenuated leaflet thickening; OAC: Oral anticoagulation RLM: Reduced leaflet motion; TOE: Transoesophageal echocardiography.

KEY MESSAGES FOR CLINICAL PRACTICE

1. **Single antiplatelet therapy** is now the preferred long-term strategy after TAVI in patients without an indication for oral anticoagulation, reducing bleeding without increasing ischemic events.
2. **Oral anticoagulation alone** (preferably a DOAC) is the optimal approach in TAVI patients with atrial fibrillation or another clear indication for anticoagulation, as combination therapy increases bleeding risk.
3. **Subclinical leaflet thrombosis** (HALT/RLM) is frequent but often transient and usually not associated with clinical events; routine anticoagulation or systematic CT screening is not recommended.
4. **Subclinical leaflet thrombosis:** Anticoagulation generally NOT recommended unless hemodynamically relevant.

no additional ischemic protection but increases bleeding risk (*Figure 4*).[49] In patients with atrial fibrillation or another indication for anticoagulation, oral anticoagulation, preferably with a DOAC (vitamin K antagonists reserved for specific indications), is recommended alone. Combination therapy (aspirin + clopidogrel + either DOAC or vitamin K antagonist) should be restricted to 1–6 months in selected cases with recent PCI and low bleeding risk. Routine anticoagulation in asymptomatic patients with normal valve haemodynamics is not supported.

For suspected valve thrombosis, prompt CCTA and TTE are advised. Patients with disabling stroke or persistent symptoms suggesting prosthetic valve dysfunction should be hospitalised, with infective endocarditis excluded before initiating IV heparin (*Figure 5*). In severe acute ischemic stroke, intravenous unfractionated heparin (UFH) remains the preferred option because of its rapid titration and complete reversibility, whereas LMWH has no validated role in the hyper acute phase and is generally avoided. Surgical intervention or low-dose thrombolytic therapy may be considered in hemodynamically unstable patients or in those with a large thrombus burden (grade 3–4 HALT) unresponsive to medical treatment.[50] Stable patients are treated with OAC (preferably DOAC, or warfarin if contraindicated) for 3–6 months, with repeat CCTA after 3 months to confirm resolution. Treatment failure on DOAC warrants conversion to warfarin (target INR ≈ 3.0), and recurrent events may justify lifelong OAC.

Routine CCTA surveillance for subclinical leaflet thrombosis is not recommended, as the bleeding risk of empirical OAC outweighs potential benefit. Instead, regular TTE follow-up remains standard, with CCTA reserved for patients showing either transvalvular gradients > 20 mm Hg or an increase ≥ 10 mm Hg compared with baseline.[51,52] For advanced HALT or reduced leaflet motion (RLM), 3–6 months of OAC with imaging follow-up is appropriate, while mild cases can be managed conservatively with repeat TTE at 6 months. Anticoagulation should always be individualised according to thrombotic and bleeding risk.

CLINICAL IMPLICATIONS AND FUTURE PERSPECTIVES

The management of TAVI has entered a phase of maturity, where the focus progressively shifts from procedural safety to long-term optimisation. The evolving understanding of the interplay between thrombosis and bleeding has established the principle of selective anticoagulation and reinforced the need for individualised therapy. In daily practice, follow-up now prioritises hemodynamic surveillance and patient-tailored imaging rather than systematic anticoagulation or routine CT screening. Ongoing studies such as CREATE, SCOPE-3, and ACASA-TAVI are redefining post-TAVI pharmacotherapy by testing dynamic, imaging-guided adjustment of antithrombotic intensity. Parallel advances in laboratory diagnostics, including platelet function assays and ADP closure time, may further refine bleeding risk stratification.[53,54] Meanwhile, novel anticoagulant classes targeting FXI or FXIa aim to dissociate thrombotic protection from haemorrhagic risk, heralding a potential paradigm shift. Beyond pharmacology, innovation in imaging and device design continues to shape outcomes. ^{18}F-NaF PET allows early identification of structural valve degeneration, while artificial intelligence models integrating hematologic markers, valve geometry, and frailty indices may soon enable fully personalised antithrombotic regimens. Simultaneously, next-generation

valves with optimised flow dynamics and reduced neosinus stasis are being engineered to minimise thrombogenicity and potentially shorten anticoagulation duration.

CONCLUSION

Antithrombotic therapy after TAVI has transitioned from empirical dual or triple combinations to a rational, evidence-driven approach grounded in simplification and patient selection. Single antiplatelet therapy with aspirin has become the standard for most patients, while oral anticoagulation alone is reserved for those with clear indications such as atrial fibrillation. Clinical vigilance remains essential for identifying patients at risk of thromboembolic or haemorrhagic events and for detecting subclinical valve dysfunction when clinically relevant. As procedural outcomes continue to improve, the focus is shifting toward precision-guided antithrombotic management, leveraging imaging, biomarkers, and artificial intelligence to tailor therapy to individual patient profiles. The ultimate goal is to ensure durable valve function and optimise long-term outcomes through a strategy that balances efficacy, safety, and personalisation.

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