

Reduced Iliac TAWSS as a Hemodynamic Correlate of Aortic Stent-Graft Partial Thrombus Formation

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Background: It was hypothesized that post-endovascular aneurysm repair (EVAR) iliac flow disturbances and geometry variation may predispose to thrombus formation within the aortic stent component.

Methods: A retrospective cohort of 52 patients (average age of 72 ± 5.2 years; 88.8% male) treated with infrarenal bifurcated stent graft was analyzed using patient-specific iliac geometry measurement and computational fluid dynamics (CFDs) analysis. Digital computed tomography angiography (CTA) datasets were reconstructed to obtain aortoiliac lumen geometry, and pulsatile flow simulations were performed to compute time-averaged wall shear stress (TAWSS), oscillatory shear index (OSI), and WSS extremes. Iliac geometric parameters (tortuosity, bifurcation angle) were also quantified. Patients were stratified by presence or absence of aortic in-stent thrombus detected during surveillance CTA.

Results: Iliac WSS metrics differed between groups, with the thrombus cohort demonstrating lower mean TAWSS (0.3 Pa vs. 0.77 Pa, $P = 0.006$). Conversely, OSI and geometric parameters showed no significant intergroup differences. Spearman analysis revealed strong inverse correlations between TAWSS and OSI ($\rho = -0.60$ to -0.64 , $P < 0.001$), while tortuosity did not correlate with either WSS metrics. Multivariate analysis adjusting for age and mean hemodynamic parameters did not demonstrate statistically significant associations with aortic thrombus.

Conclusion: Reduced iliac TAWSS magnitude, rather than OSI or iliac tortuosity, appears to characterize post-EVAR hemodynamics in patients who develop aortic in-stent thrombus. These data support a mechanistic role for low-shear outflow conditions in promoting thrombotic complications after EVAR and highlights the potential value of shear-based CFD profiling for risk stratification.

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Data Availability: The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

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INTRODUCTION

Intraluminal or mural thrombus formation within endovascular stent-grafts is an increasingly recognized late finding during post-endovascular aneurysm repair (EVAR) surveillance imaging. Although frequently incidental and hemodynamically insignificant, in-stent thrombus represents a potential substrate for limb thrombosis, distal embolization, and secondary intervention. Contemporary series report mural or intraprosthetic thrombus (IPT) in approximately 20–30% of EVAR recipients, most commonly within the first postoperative year and often without overt clinical sequelae.¹ Device geometry and adverse flow environments, including abrupt caliber transitions, tapered main bodies, and aortouniiliac configurations, have been implicated as structural drivers of thrombus development.¹ Comparative data suggest device-specific susceptibility, with Zenith Alpha limbs demonstrating higher rates of limb graft occlusion compared with Endurant II despite similar IPT incidence.² Current European Society for Vascular Surgery (ESVS) 2024 guidance classifies benign mural thrombus without stenosis or embolic events as a nonthreatening radiologic finding that does not materially increase rupture or occlusion risk, typically warranting observation alone.¹ Conversely, partial thrombotic limb narrowing $\geq 50\%$ secondary to limb kinking or stenosis has been linked to subsequent occlusion and may necessitate relining, thrombectomy, or adjunctive stenting.^{1,2} Evidence is insufficient to recommend a standardized antithrombotic strategy for asymptomatic IPT, although intensified therapy has been proposed in select progressive cases.¹ Given the lack of consensus and the potential for limb-related morbidity, lifelong imaging surveillance remains essential to detect thrombotic progression, device-related mechanical abnormalities, and sac behavior.³

Thrombus formation within aortic stent-grafts following EVAR arises predominantly from disturbed flow related to graft geometry and local luminal configuration rather than systemic hypercoagulability alone. Most current evidence derives from computational fluid dynamics (CFDs) imaging studies and large anatomical outcome cohorts rather than dedicated thrombus registries.^{4,5} Iliac limb tortuosity and sharp angulation generate spiral and recirculatory flow patterns, promoting intraluminal thrombus in 22–31% of bifurcated grafts.⁴ Sharp caliber transitions, including large main bodies tapering to smaller limbs and aortouniiliac configurations, are especially prone to mural thrombus deposition.¹ Branch protrusion into the lumen

alters shear environments, with >5 -mm protrusion producing large abnormal wall shear stress (WSS) regions and inferred thrombotic risk.⁵ Nonstandard renal branch orientations (e.g., “ballerina”) further increase thrombus, compression, and occlusion events compared with standardized geometries (45% vs. 16%).⁶ Hemodynamically, recirculation zones at bifurcations, kinks, and diameter changes favor particulate deposition and simulated thrombus growth.^{4,5} Larger patent luminal volumes and reduced intrasac thrombus are associated with greater complication rates, particularly at the neck seal, likely via increased graft mobility and flow disturbance.^{1,7} Contemporary ESVS 2024 guidance reports mural thrombus rates around 21% and strongly linked to barrel or tapered configurations but generally benign in the absence of symptoms or flow-limiting stenosis.¹ When thrombus becomes symptomatic, progressive, or hemodynamically significant, individualized intervention or intensified antithrombotic therapy is advised.¹

Despite growing recognition of thrombus formation within aortic stent-grafts following EVAR, the mechanistic contribution of distal outflow hemodynamics arising from the iliac limbs remains poorly defined. Prior studies have predominantly focused on local geometric configurations and hemodynamic disturbances within the aortic graft body itself, such as caliber transitions, graft tapering, and intra-aortic flow recirculation.⁸ While these factors are important, they do not fully account for the global hemodynamic coupling within the stent-graft system, in which the iliac limbs function as the principal outflow pathways.

From a fluid-dynamic perspective, the iliac limbs impose critical boundary conditions that govern flow division, pressure gradients, and downstream resistance, all of which may influence upstream aortic flow patterns.⁹ Disturbances within the iliac segments, such as reduced forward flow, recirculation, or low WSS may therefore propagate proximally, promoting flow stagnation, prolonged particle residence time, and thrombus-prone environments within the aortic stent-graft. This downstream-to-upstream interaction has been insufficiently explored in current EVAR literature, in which the iliac arteries are often treated as passive conduits rather than active determinants of graft hemodynamics.

Accordingly, this study aims to investigate whether iliac flow disturbances and geometry-derived hemodynamic alterations are associated with subsequent thrombus formation within the aortic stent-graft. By integrating patient-specific CFD analysis with imaging-based thrombus assessment, we seek to provide a system-level perspective

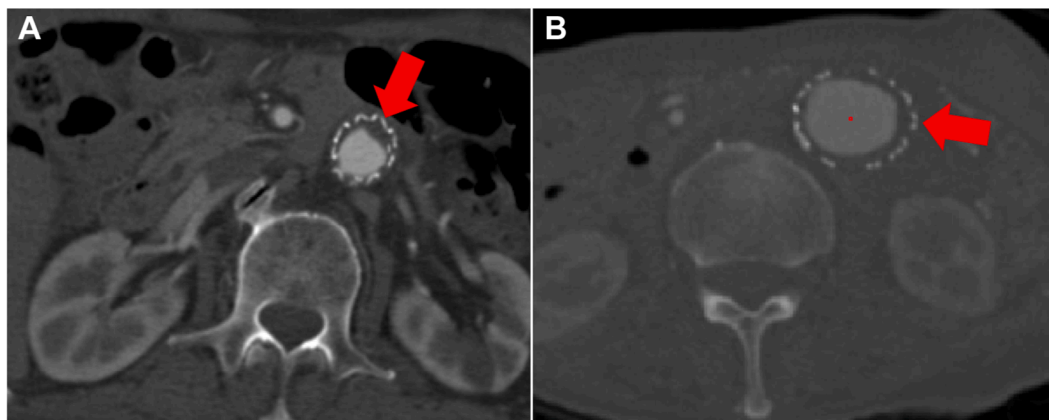


Fig. 1. CTA images of aorta in-stent partial thrombus. Axial CTA images (**A and B**) of representative cases showing circumferential thrombus formation within aorta stent graft (red arrows). CTA, computed tomography angiography.

on EVAR hemodynamics and to identify iliac shear-related metrics as potential markers for thrombotic risk stratification.

MATERIALS AND METHODS

The Hospital's institutional review board reviewed and approved the study protocol. Informed consent was waived due to its retrospective design. All methods complied with institutional standards and with the ethical principles of the Declaration of Helsinki.

Patient Demographic

A single-institution retrospective cohort of EVAR patients treated for infrarenal abdominal aortic aneurysms (from 2015 to 2022) with Endurant stent-grafts (Medtronic Vascular, Minneapolis, MN) was examined. Inclusion required bilateral iliac limb configuration and availability of both early (≤ 30 days) and surveillance computed tomography angiography (CTA). Stent-grafts lacking iliac limbs, poor-quality CTA studies, or absence of follow-up imaging were grounds for exclusion. Fifty-two patients (48 male, average age of 72 ± 5.2 years) satisfied the final criteria: 40 without and 12 with aortic in-stent partial thrombotic changes on subsequent CTA.

CTA Data Acquisition and Interpretation

CTA examinations of the aortoiliac artery were obtained on a dual-source CT platform (SOMATOM Definition Flash, Siemens Healthcare, Forchheim, Germany) using intravenous Ultravist (Bayer Healthcare, Berlin, Germany) contrast injected at

5 mL/s, followed by a 50-mL saline bolus. Acquisition parameters consisted of 260–300 mA tube current with 1.0-mm reconstructions and 5-mm maximum intensity projection images. Two blinded radiologists with 10 and 30 years of vascular imaging experience evaluated all scans. In-stent thrombus was defined as linear or irregular hypoattenuating material within the stent graft lumen exceeding 2 mm in thickness and $>25\%$ circumferential involvement (Fig. 1) as previously described.¹⁰ To ensure uniform assessment, only follow-up CTA studies performed within 3 years post-EVAR were used for grouping into thrombus-negative and thrombus-positive cohorts.

Geometry Measurement

Initial CTA scans without thrombus (within 30 days post-EVAR) were exported in DICOM format and reconstructed in Mimics software (v25.0, Materialise, Leuven, Belgium). Segmentation of the aortoiliac lumen, from 3 cm proximal to the graft to 3 cm distal to the iliac limbs, was performed using thresholding, manual refinement, and smoothing. Geometric measurements were extracted from the resulting three-dimensional (3D) model. Centerlines were automatically generated as the paths of maximally inscribed spheres. The bifurcation angle was taken as the dominant angle between the iliac centerlines, and iliac tortuosity was calculated as the ratio of straight-line distance to centerline length for each limb (Fig. 2).

CFD Analysis of Iliac Flow

The reconstructed vascular geometries were imported into COMSOL software (v5.2a, COMSOL

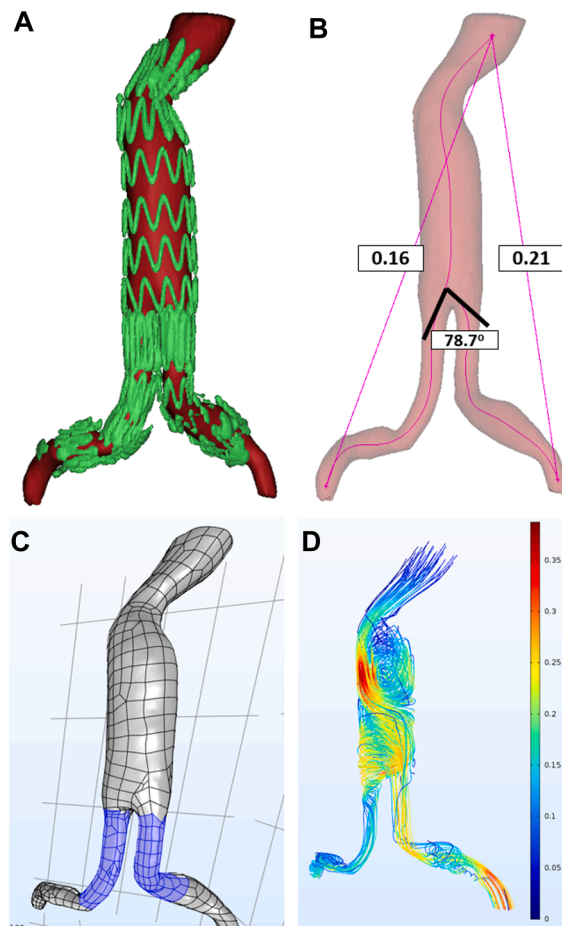


Fig. 2. Study procedure. **(A)** Three-dimensional model of aortoiliac vessel and stent graft created from CT data. **(B)** Measurement of iliac tortuosity and iliac angle. **(C)** Iliac segments for blood-flow postprocessing analysis using computational fluid dynamic. **(D)** Postprocessing result of blood-flow simulation during systolic phase of cardiac cycle. CT, Computed tomography; 3D, three-dimensional.

Inc., Burlington, MA) for meshing and numerical solution of the Navier-Stokes equations. 3D unstructured tetrahedral meshes were generated for each model, consisting of approximately 1.2–2.0 million elements, depending on anatomical complexity. To improve the accuracy of near-wall hemodynamic parameters, localized mesh refinement was applied at the vessel wall and in regions of high velocity gradients, including the aortic bifurcation and proximal iliac segments. Boundary layer elements were incorporated to ensure adequate resolution of WSS. Mesh convergence analysis was performed in a subset of representative cases by progressively refining the mesh density. Time-averaged wall shear stress (TAWSS) and oscillatory shear index (OSI) demonstrated variations of <10% between successive refinements.

Blood flow was modeled as an incompressible, laminar Newtonian fluid with a dynamic viscosity of 0.0035 Pa s and a density of 1066 kg/m³.¹¹ Vessel walls were assumed rigid with a no-slip boundary condition.^{12,13} Patient-specific inlet velocity profiles were derived from published pulsatile flow rates¹⁴ scaled to the measured cross-sectional aortic areas, while outlet boundaries at both iliac arteries were prescribed using a reported pressure waveform.¹⁵ This approach was selected to provide a stable and reproducible representation of downstream hemodynamic loading in the absence of patient-specific flow split measurements. Compared with resistance-based or Windkessel models, pressure-driven outlet conditions offer a computationally efficient and reproducible alternative in vascular CFD simulations, particularly in retrospective imaging studies in which distal vascular resistance and compliance parameters are not directly measurable.^{16,17} However, we acknowledge that this approach does not fully capture patient-specific peripheral impedance and may influence absolute shear stress values. Each simulation covered 3 cardiac cycles with a 0.01-second time increment, and results from the second cycle were used for hemodynamic analysis to minimize transient effects.¹⁸ Computational runs, including postprocessing, required approximately 10–12 hours per case. TAWSS, OSI, and WSS values were calculated using established formulations.¹⁹ Hemodynamic variables were extracted over 2 predefined iliac regions consisting of the proximal 10 cm of each iliac stent segment (Fig. 2). TAWSS describes the mean shear stress during one cardiac period, whereas the maximum and minimum WSS correspond to the extrema within a cycle. OSI characterizes directional changes in shear over the cardiac cycle. Mean values (TAWSS and OSI) represented averaged value of left and right iliac artery.

Statistical Analysis

Normally distributed variables were summarized as mean ± standard deviation, whereas non-normally distributed variables were reported as median with range. Differences between the normal and diseased groups were evaluated using independent *t*-tests for parametric data and Mann–Whitney *U* tests for nonparametric data. Associations between continuous variables were assessed using Spearman rank correlation coefficient (*ρ*). Exploratory multivariate analyses were performed including age and mean hemodynamic variables to account for potential confounding effects. Statistical significance was

Table I. Baseline demographic and clinical characteristics

Variables	Control group (<i>n</i> = 40)	In-stent partial thrombus (<i>n</i> = 12)	<i>P</i> value
Age (year)	72.28 ± 7.16	72.00 ± 5.81	0.91
Male, sex	28 (70%)	8 (66.6%)	0.33
Baseline antithrombotic therapy			
Single antiplatelet therapy	3 (7.5%)	0 (0)	
Dual antiplatelet therapy	4 (10%)	1 (8.3%)	
Oral anticoagulation therapy	4 (10%)	1 (8.3%)	
Outcome			
Reintervention	3 (7.5)	6 (50)	0.02 ^a
Endoleak	4 (10)	1 (8.3%)	0.54
Distal thromboembolic event	1 (2.5)	2 (16.7)	
Migration	1 (2.5)	1 (8.3%)	
Sac growth	5 (12.5)	2 (16.7)	0.29
Graft infection	1 (2.5)	0 (0)	
30-day all-cause mortality	0 (0)	1 (8.3%)	

Independent sample *t*-tests (for continuous variables) and Fisher exact test (for categorical variables) were used to compare differences between normal and diseased groups.

^aStatistical significance (*P* < 0.05).

defined as *P* < 0.05. All analyses were performed in SPSS (v20.0, IBM, Chicago, IL).

RESULTS

Among the 52 cases evaluated, 12 patients (23.1%) demonstrated thrombotic involvement of the aortic stent-graft on follow-up CTA. The detection occurred at an average of 6.8 months after EVAR, with the time to diagnosis spanning 1 to 13 months.

Iliac Geometry and Hemodynamic Differences Between Control and Aortic Thrombus Group

Table I showed baseline characteristics and clinical information of our sample. Age distribution was similar between those without thrombus and those with in-stent thrombus (72.28 ± 7.16 vs. 72.00 ± 5.81 years, *P* = 0.91). Table II demonstrated hemodynamic and geometry differences between 2 groups. Pronounced differences were found in shear-related hemodynamic metrics. Mean iliac TAWSS was significantly lower in the thrombus group (0.30 Pa (0.24–0.87) versus 0.77 Pa (0.39–1.11), *P* = 0.06), and minimum iliac TAWSS showed a similar reduction (0.18 Pa (0.11–0.34) versus 0.47 Pa (0.15–0.74), *P* = 0.01). Measures of shear oscillation, including mean and maximum OSI, did not differ between groups (all *P* > 0.5), indicating that oscillatory behavior was not associated with thrombotic status in this cohort.

Structural geometric variables, including iliac tortuosity (mean 0.15 vs. 0.15, *P* = 0.77) and

bifurcation angle (72.28° vs. 72.63°, *P* = 0.82), also showed no significant intergroup variation. Figure 3 better illustrated the representative variables in our study.

Taken together, these findings suggest that reduced WSS across the iliac limbs, rather than oscillatory shear or geometric deformation, may constitute the principal hemodynamic correlation of in-stent aortic thrombus formation following EVAR in this patient population.

Exploratory multivariate analysis incorporating age and mean hemodynamic parameters did not reveal statistically significant independent associations with thrombus formation (Supplementary Table 1).

Analysis of Correlation Between Iliac Geometry and Hemodynamic Factors

Spearman rank correlation analysis was performed to evaluate the relationship between post-EVAR iliac WSS, oscillatory index, and geometric parameters. Specifically, correlations between TAWSS and OSI were examined for the right and left iliac arteries (Table III), followed by assessment of the association between iliac tortuosity and TAWSS/OSI metrics (Table IV). A strong inverse correlation was observed between TAWSS and OSI, with right iliac TAWSS demonstrating a significant negative correlation with right iliac OSI (ρ = −0.60, *P* < 0.001) and left iliac TAWSS similarly correlating with left iliac OSI (ρ = −0.61, *P* < 0.001). Mean iliac TAWSS also negatively correlated with mean iliac

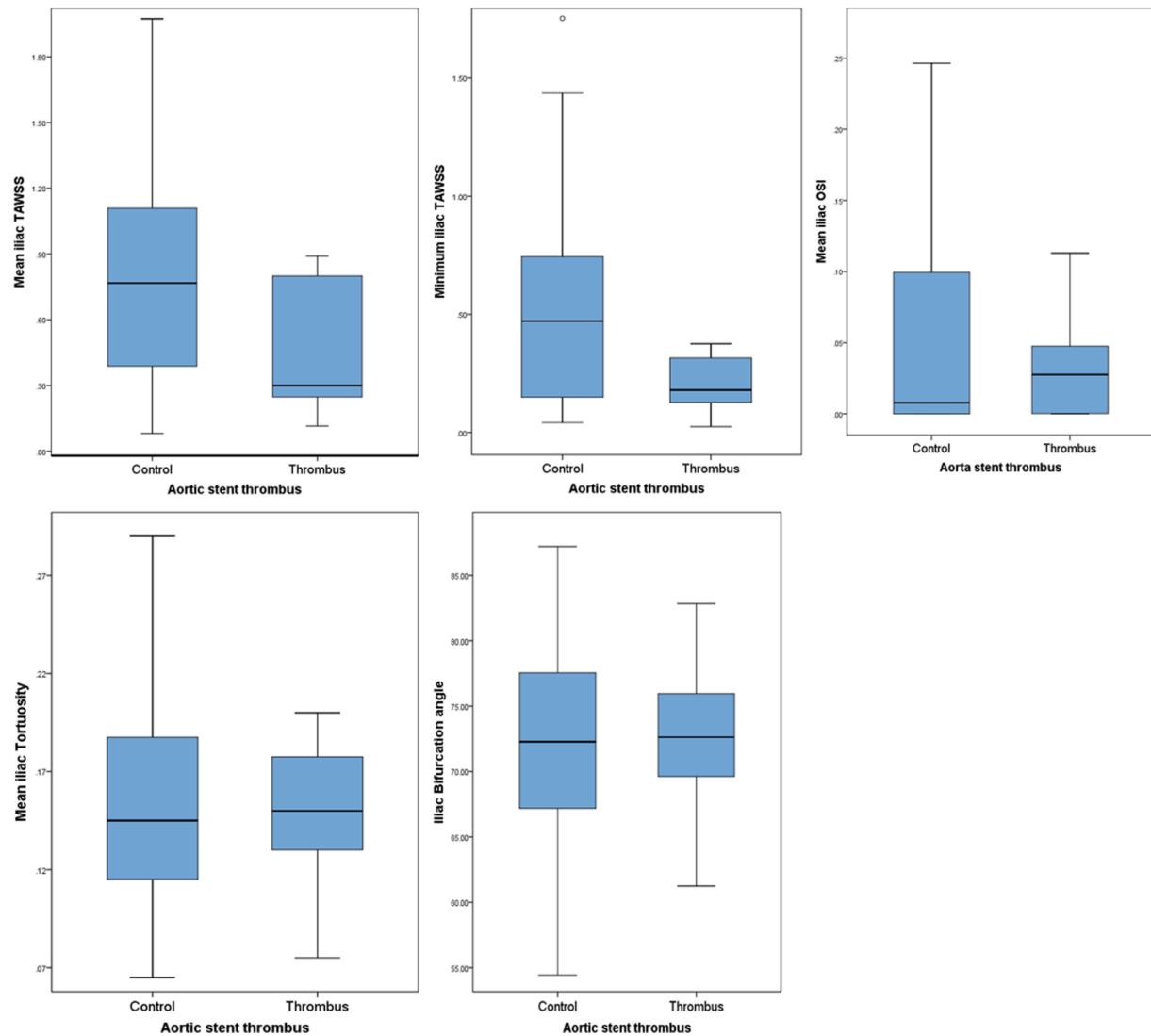


Fig. 3. Box plots demonstrating differences of representative variables between control and thrombus group. TAWSS, time-averaged wall shear stress; OSI, oscillatory shear index.

OSI ($\rho = -0.64$, $P < 0.001$), further supporting a consistent shear-oscillation relationship (Fig. 4).

In contrast, tortuosity showed no meaningful correlation with TAWSS or OSI across any limb, with right iliac tortuosity yielding $\rho = -0.02$ for TAWSS ($P = 0.91$) and $\rho = 0.10$ for OSI ($P = 0.50$), and left iliac tortuosity yielding $\rho = 0.02$ for TAWSS ($P = 0.89$) and $\rho = -0.15$ for OSI ($P = 0.33$). Mean iliac tortuosity also demonstrated no significant association with either TAWSS ($P = 0.99$) or OSI ($P = 0.84$).

Collectively, these results indicate that post-EVAR iliac hemodynamics are characterized by a robust shear-oscillation coupling, whereas iliac

tortuosity within the studied range appears not to influence local shear behavior.

DISCUSSION

In this study, iliac hemodynamics after EVAR differed significantly between patients with and without aortic in-stent thrombus, with the thrombus group displaying markedly reduced WSS magnitude, while oscillatory shear and geometric parameters remained largely unchanged. The minimum iliac TAWSS was significantly lower in the thrombus cohort (0.18 vs. 0.47 Pa, $P = 0.01$), and

Table II. Comparison of iliac blood flow characteristics and geometry between control and aortic thrombus group

Variables	Control group (<i>n</i> = 40)	In-stent partial thrombus (<i>n</i> = 12)	<i>P</i> value
Right iliac TAWSS	0.56 (0.31–0.84) Pa	0.33 (0.11–1.34) Pa	0.23
Left iliac TAWSS	0.83 (0.16–1.31) Pa	0.38 (0.14–1.41) Pa	0.71
Mean iliac TAWSS	0.77 (0.39–1.11) Pa	0.30 (0.24–0.87) Pa	0.006 ^a
Iliac/aortic TAWSS ratio	6.40 (2.18–16.89)	3.92 (1.59–24.06)	0.65
Minimum iliac TAWSS	0.47 (0.15–0.74) Pa	0.18 (0.11–0.34) Pa	0.01 ^a
Right iliac OSI	0 (0–0.13)	0.01 (0–0.09)	0.53
Left iliac OSI	0 (0–0.04)	0.00 (0–0.02)	0.58
Mean iliac OSI	0.01 (0–0.1)	0.03 (0–0.05)	0.95
Maximum iliac OSI	0.02 (0–0.19)	0.05 (0–0.09)	0.95
Right iliac maximum WSS	2.17 (1.27–4.21) Pa	1.21 (0.41–1.34) Pa	0.004
Left iliac maximum WSS	3.70 (1.01–6.07) Pa	1.70 (0.6–7.03) Pa	0.67
Right iliac minimum WSS	0.08 (0.02–0.14) Pa	0.01 (0–0.05) Pa	0.004
Left iliac minimum WSS	0.09 (0.01–0.17) Pa	0.02 (0.01–0.15) Pa	0.29
Right iliac tortuosity	0.12 (0.1–0.16)	0.14 (0.12–0.18)	0.17
Left iliac tortuosity	0.16 (0.13–0.23)	0.15 (0.13–0.17)	0.38
Mean iliac tortuosity	0.15 (0.12–0.19)	0.15 (0.13–0.19)	0.77
Iliac bifurcation angle (degree)	72.28 (67.19–77.56)	72.63 (65.5–78.16)	0.82

P value from Student *t*-test (parametric variables) or Mann–Whitney *U* test (nonparametric variables).

^a*P* value < 0.05.

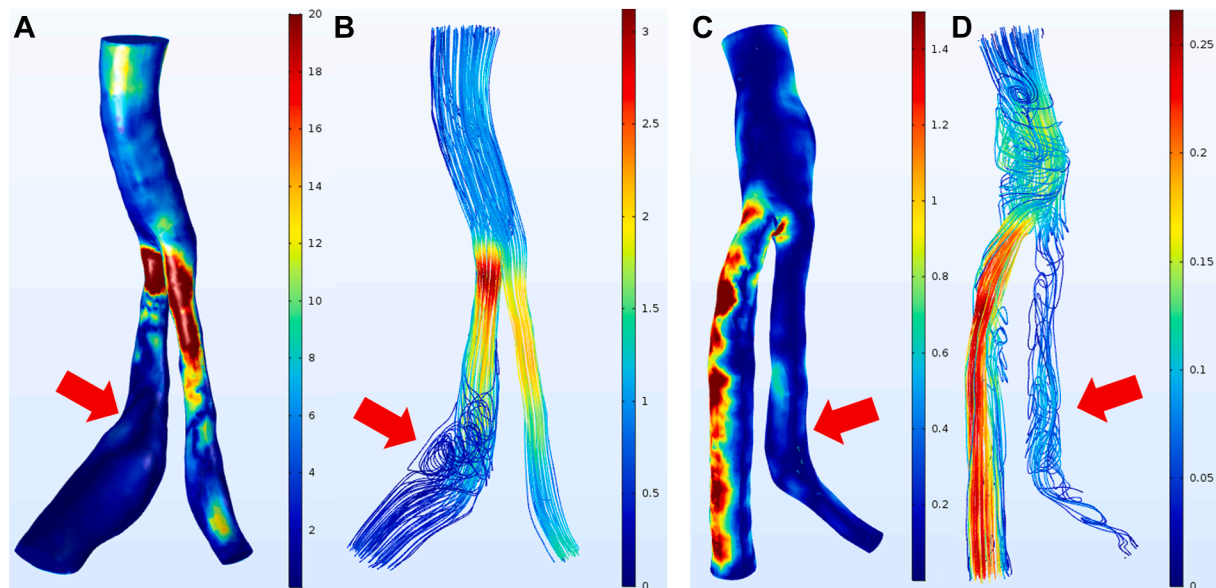
maximum WSS on the right iliac limb was reduced by nearly half (1.21 vs. 2.17 Pa, *P* = 0.004), supporting the interpretation that diminished WSS may contribute to thrombus formation after EVAR. These findings align with established vascular biology showing that chronically low or oscillatory shear at bends and bifurcations promotes endothelial dysfunction, lipid accumulation, and atherosclerotic plaque progression, creating prothrombotic niches.^{20,21} Experimental and clinical studies further demonstrate that low WSS coexists with recirculation zones and prolonged residence time, favoring platelet deposition and fibrin accumulation in aneurysmal or stenotic segments.^{22,23} Although the association between low WSS and thrombus formation is well established in native vascular biology, its applicability to prosthetic stent grafts requires careful interpretation as in our study, thrombus formation happened on prosthetic surface. Unlike native vessels, thrombus formation within endografts occurs on nonendothelialized prosthetic surfaces, in which classical endothelial dysfunction may not be the primary mechanism. Instead, low-shear conditions are more likely to promote thrombogenesis through flow stagnation, increased residence time, and enhanced platelet and coagulation activity within the graft lumen. Therefore, the relationship between reduced shear stress and thrombus formation observed in this study likely reflects hemodynamic rather than endothelial-mediated mechanisms.

Interestingly, oscillatory shear was not associated with thrombotic status in our cohort (all OSI *P* > 0.05), suggesting that post-EVAR iliac flow disturbance may be dominated by reduced forward shear rather than shear oscillation. Iliac tortuosity and bifurcation angle were similarly nondiscriminatory, indicating that within typical EVAR anatomical ranges, geometric deformation may exert less influence on thrombogenesis than flow-mediated shear reduction. Taken together, these results support a mechanistic paradigm in which low post-EVAR iliac shear may transmit prothrombotic hemodynamic conditions to the aortic stent body, consistent with the broader low-shear thrombogenic literature.^{20,21}

In this cohort, iliac hemodynamics after EVAR exhibited a strong inverse relationship between TAWSS and OSI, with right and left iliac TAWSS significantly and negatively correlated with their respective OSI values (ρ = −0.60 and −0.61, both *P* < 0.001), and mean values showing a similar pattern (ρ = −0.64, *P* < 0.001). This shear stress–oscillation coupling aligns with established CFD concepts in which OSI is computed from the time-resolved WSS vector and increases when shear reverses or oscillates throughout the cardiac cycle.²⁴ Prior CFD studies report that regions of low mean or TAWSS typically colocalize with high OSI in disturbed flow environments such as bifurcations and aneurysmal expansions,^{25,26} though the association is not strictly linear nor globally uniform.

Table III. Correlation between post-EVAR iliac TAWSS and OSI

<i>P</i> value (Spearman co-efficient index)	Right iliac OSI	Left iliac OSI	Mean iliac OSI
Right iliac TAWSS	<0.001 (−0.60)	<0.001 (−0.61)	<0.001 (−0.64)
Left iliac TAWSS			
Mean iliac TAWSS			

**Fig. 4.** Representative cases showing negative correlation of WSS and OSI. Systolic-phase WSS map (**A and C**) and flow velocity (**B and D**) of 2 different cases showing

corresponding areas (red arrows) of low WSS and fluctuated blood flow (high OSI). WSS, wall shear stress; OSI, oscillatory shear index.

Notably, OSI highlights directional shear fluctuations, while TAWSS captures magnitude, and high WSS jets can coexist adjacent to recirculation zones with high OSI but low WSS, underscoring the distinct biophysical interpretation of each parameter.²⁷ Our findings support these conceptual frameworks, demonstrating that iliac flow post-EVAR conforms to the expected CFD behavior of linked but nonidentical WSS and OSI domains. In contrast, iliac tortuosity showed no significant correlation with either TAWSS or OSI (all $P > 0.3$), suggesting that geometric deformation within the studied range did not substantially alter local shear behavior. Sensitivity analyses in prior CFD literature emphasize that OSI is influenced not only by geometry but also by inlet waveforms and outflow split, often responding more strongly to waveform perturbations than WSS itself.²⁸ Moreover, comparisons across rigid-wall CFD and fluid–structure interaction frameworks show robust spatial consistency in OSI-TAWSS patterns despite modest absolute differences.²⁹ Taken together, these data suggest that post-EVAR iliac flow remains governed

by shear–oscillation relationships seen in arterial CFD, and that OSI and TAWSS should be interpreted jointly rather than in isolation when evaluating disturbed flow physiology.

In the present study, hemodynamic assessment was based on region-averaged indices (e.g., TAWSS) rather than detailed spatial characterization of shear stress distributions. Although surface area–based thresholds or histogram analyses of WSS/OSI may provide a more comprehensive representation of abnormal shear environments, such approaches are highly sensitive to mesh resolution, segmentation variability, and geometric smoothing. Given the retrospective nature of this study and interpatient anatomical heterogeneity, standardized implementation of these high-resolution analyses could not be reliably ensured. Therefore, we focused on global hemodynamic metrics to facilitate robust interpatient comparison. Future studies incorporating standardized meshing strategies and advanced postprocessing techniques may further refine spatial characterization of thrombogenic shear patterns.

Table IV. Correlation between post-EVAR iliac tortuosity and TAWSS/OSI

P value (Spearman co-efficient index)	Right iliac TAWSS	Left iliac TAWSS	Mean iliac TAWSS	Right iliac OSI	Left iliac OSI	Mean iliac OSI
Right iliac tortuosity	0.91 (−0.02)			0.50 (0.10)		
Left iliac tortuosity		0.89 (0.02)			0.33 (−0.15)	
Mean iliac tortuosity			0.99 (0.003)			0.84 (−0.03)

Multivariate analysis was explored but not reported due to instability related to the small number of thrombus-positive cases ($n = 12$), which increased the risk of overfitting and yielded inconsistent estimates. Similarly, sensitivity analysis for TAWSS threshold discrimination demonstrated limited performance (low area under the curve) and was therefore not included. In addition, key graft-related structural parameters, including endograft diameter, iliac limb diameter, and graft oversizing, were not consistently available and could not be incorporated into the analysis. As these factors may influence post-EVAR hemodynamics, their omission may limit interpretation of geometry–shear relationships. Accordingly, the present findings should be considered exploratory, and larger studies integrating detailed device parameters are needed to enable robust multivariate modeling and clinically meaningful risk stratification.

In summary, post-EVAR CFD analysis identified reduced iliac TAWSS magnitude as the hemodynamic feature most strongly associated with subsequent aortic in-stent thrombus, whereas oscillatory shear and geometry showed no meaningful contribution. In addition, OSI and TAWSS in post-EVAR iliac artery also showed significant negative correlation, emphasizing their co-effect in iliac blood flow. These findings suggest that low-shear outflow conditions may underpin late thrombotic complications after EVAR.

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work the authors used ChatGPT 5.2 to improve readability and language of the manuscript. After using this tool, the authors reviewed and edited the work and took full responsibility for the content of the publication.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Dac Hong An Ngo: Writing – review & editing, Writing – original draft, Visualization, Validation,

Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Kyeong Ho Lee:** Software, Methodology, Investigation, Formal analysis, Data curation. **Jeong In Lee:** Software, Methodology, Formal analysis, Data curation. **Hong Pil Hwang:** Writing – review & editing, Visualization, Validation, Project administration, Methodology, Investigation. **Young Min Han:** Writing – review & editing, Validation, Supervision, Project administration, Conceptualization. **Hyo Sung Kwak:** Writing – review & editing, Writing – original draft, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.avsg.2026.04.049>.

REFERENCES

1. Wanhainen A, Van Herzele I, Gonçalves FB, et al. Clinical Practice Guidelines on the management of abdominal aorto-iliac artery aneurysms. *Eur J Vasc Endovasc Surg* 2024;67:192–331.
2. Ulsaker H, Lam M, Herje M, et al. A retrospective evaluation of intraprostatic thrombus Formation after endovascular aortic repair in cook Zenith alpha and medtronic endurant II patients. *Eur J Vasc Endovasc Surg* 2023;66:644–51.
3. Teijink J, Power A, Böckler D, et al. Five year outcomes of the endurant stent graft for endovascular abdominal Aortic aneurysm repair in the ENGAGE registry. *Eur J Vasc Endovasc Surg* 2019;58:175–81.
4. Polańczyk A, Piechota-Polańczyk A, Stefanczyk L. A new approach for the pre-clinical optimization of a spatial configuration of bifurcated endovascular prosthesis placed in abdominal aortic aneurysms. *PLoS One* 2017;12:e0182717.
5. Tricarico R, Tran-Son-Tay R, Laquian L, et al. Haemodynamics of different configurations of a left Subclavian artery stent graft for thoracic endovascular aortic repair. *Eur J Vasc Endovasc Surg* 2019;59:7–15.
6. Dean A, Hassan S, Yoon W, et al. Outcomes associated with non-standard configuration of renal branches in aortic endografts. *Ann Vasc Surg* 2025;116:103–8.
7. Oliveira-Pinto J, Ferreira R, Oliveira N, et al. Total luminal volume predicts risk after endovascular aneurysm repair. *Eur J Vasc Endovasc Surg* 2020;59:918–27.
8. Ngo DHA, Lee KH, Lee JI, et al. Impact of aorto-iliac hemodynamics and geometry on thrombotic complications

- following abdominal aortic endovascular aneurysm repair. *Eur J Radiol* 2025;187:112116.
9. Zhang Y, Zhao D, Si X, et al. Endograft-specific hemodynamics after endovascular aneurysm repair: a CFD analysis. *Sci Rep* 2024;14:31512.
 10. Oliveira NFG, Bastos Gonçalves FM, Hoeks SE, et al. Clinical outcome and morphologic determinants of mural thrombus in abdominal aortic endografts. *J Vasc Surg* 2015;61:1391–8.
 11. Dai Y, Qian Y, Zhang M, et al. Associations between local haemodynamics and carotid intraplaque haemorrhage with different stenosis severities: a preliminary study based on MRI and CFD. *J Clin Neurosci* 2019;66:220–5.
 12. Dai Y, Lv P, Javadzadegan A, et al. Hemodynamic analysis of carotid artery after endarterectomy: a preliminary and quantitative imaging study based on computational fluid dynamics and magnetic resonance angiography. *Quantitative Imaging Med Surg* 2018;8:399–409.
 13. Ngo MT, Kim CI, Jung J, et al. Four-Dimensional flow magnetic resonance imaging for assessment of velocity magnitudes and flow patterns in the human carotid artery bifurcation: Comparison with computational fluid dynamics. *Diagnostics* 2019;9:223.
 14. Les AS, Yeung JJ, Schultz GM, et al. Supraceliac and infrarenal aortic flow in patients with abdominal aortic aneurysms: mean flows, waveforms, and allometric scaling relationships. *Cardiovasc Eng Technol* 2010;1:39–51.
 15. Xiao N, Humphrey JD, Figueroa CA. Multi-scale computational model of three-dimensional hemodynamics within a deformable full-body arterial network. *J Comput Phys* 2013;244:22–40.
 16. Kizhisseri M, Gharai S, Schluter J. An analytical method informed by clinical imaging data for estimating outlet boundary conditions in computational fluid dynamics analysis of carotid artery blood flow. *Sci Rep* 2023;13:14973.
 17. Madhavan S, Kemmerling EMC. The effect of inlet and outlet boundary conditions in image-based CFD modeling of aortic flow. *Biomed Eng Online* 2018;17:66.
 18. Compagne KC, Dilba K, Postema EJ, et al. Flow patterns in carotid webs: a patient-based computational fluid dynamics study. *Am J Neuroradiol* 2019;40:703–8.
 19. Lee UY, Kwak HS. Analysis of morphological-hemodynamic risk factors for aneurysm rupture including a newly introduced total volume ratio. *J Personalized Med* 2021;11:744.
 20. Chiu J, Chien S. Effects of disturbed flow on vascular endothelium: pathophysiological basis and clinical perspectives. *Physiol Rev* 2011;91:327–87.
 21. Cecchi E, Giglioli C, Valente S, et al. Role of hemodynamic shear stress in cardiovascular disease. *Atherosclerosis* 2011;214:249–56.
 22. Owen D, De Oliveira D, Neale E, et al. Numerical modelling of blood rheology and platelet activation through a stenosed left coronary artery bifurcation. *PLoS One* 2021;16:e0259196.
 23. Gutierrez NG, Mathew M, McCrindle B, et al. Hemodynamic variables in aneurysms are associated with thrombotic risk in children with Kawasaki disease. *Int J Cardiol* 2019;281:15–21.
 24. Nordgaard H, Swillens A, Nordhaug D, et al. Impact of competitive flow on wall shear stress in coronary surgery: computational fluid dynamics of a LIMA-LAD model. *Cardiovasc Res* 2010;88:512–9.
 25. Numata S, Itatani K, Kanda K, et al. Blood flow analysis of the aortic arch using computational fluid dynamics. *Eur J Cardiothorac Surg* 2016;49:1578–85.
 26. Youssefi P, Gomez A, He T, et al. Patient-specific computational fluid dynamics—assessment of aortic hemodynamics in a spectrum of aortic valve pathologies. *J Thorac Cardiovasc Surg* 2017;153:8–20.e3.
 27. Chakraborty S, Mantripragada VT, Chakravarty A, et al. Unraveling the complex interplay between abnormal hemorheology and shape asymmetry in flow through stenotic arteries. *Comput Methods Programs Biomed* 2024;257:108437.
 28. Rajabzadeh-Oghaz H, Ooij P, Veeturi S, et al. Inter-patient variations in flow boundary conditions at middle cerebral artery from 7T PC-MRI and influence on Computational Fluid Dynamics of intracranial aneurysms. *Comput Biol Med* 2020;120:103759.
 29. Balasubramanya A, Maes L, Rega F, et al. Hemodynamics and wall shear metrics in a pulmonary autograft: comparing a fluid-structure interaction and computational fluid dynamics approach. *Comput Biol Med* 2024;176:108604.