

Original Article

Anatomical and procedural predictors of type Ia and type III endoleaks after EVAR

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Received: June 11, 2026 Accepted: September 21, 2026 Published online: October 20, 2026

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Abstract

Aim: Type Ia and Type III endoleaks are high-pressure failure modes of endovascular aneurysm repair (EVAR) that rapidly repressurize the excluded aneurysm sac but arise through different mechanisms. Type Ia reflects proximal seal-zone failure, whereas Type III reflects modular disconnection, component overlap failure, or graft-fabric failure. This study evaluated distinct risk profiles for these urgent subtypes after elective EVAR.

Material and Methods: This retrospective single-center observational study included 383 consecutive eligible patients who underwent elective EVAR for fusiform infrarenal abdominal aortic aneurysm between 2016 and 2024 and had preoperative and postoperative computed tomography angiography with at least 1 year of follow-up. Morphometric and three-dimensional measurements assessed proximal neck anatomy, aneurysm burden, thrombus burden, iliac morphology, and available procedural-complexity markers.

Results: The cohort included 356 male patients (93.0%) and 27 female patients (7.0%). During 33.2 ± 21.2 months of radiological follow-up, Type Ia endoleak occurred in 16 patients (4.2%) and Type III endoleak in 21 patients (5.5%). Type Ia endoleak was independently associated with conical proximal neck morphology, neck-sac angulation > 60 degrees, neck thrombus, and neck calcification, with adjusted odds ratios of 16.89, 5.72, 4.22, and 21.28, respectively. Type III endoleak was associated with longer aneurysm morphology, greater thrombus burden, higher thrombus-to-aneurysm ratio, iliac tortuosity, and iliac aneurysm involvement. The strongest Type III signals were thrombus-to-aneurysm volume ratio $> 52\%$, aneurysm length > 139 mm, and thrombus volume > 118 cm³, with areas under the receiver operating characteristic curve of 0.880, 0.836, and 0.783, respectively.

Conclusion: Type Ia and Type III endoleaks showed distinct mechanism-specific risk profiles. Type Ia was primarily a hostile proximal-neck phenomenon, whereas Type III was associated with larger and longer aneurysm morphology, thrombus burden, iliac tortuosity, and iliac aneurysm involvement. Because device-specific variables and multivariable Type III modeling were unavailable, Type III findings should be interpreted as hypothesis-generating anatomy-device interaction signals rather than definitive independent predictors. Mechanism-specific assessment may support individualized EVAR planning and surveillance.

Keywords: Aortic Aneurysm, abdominal; endovascular aneurysm repair; endoleak; risk factors; prosthesis failure

INTRODUCTION

Endovascular aneurysm repair (EVAR) is an established treatment for anatomically suitable infrarenal abdominal aortic aneurysm because it reduces early operative burden compared with open repair. Contemporary practice guidelines emphasize

careful anatomical selection, structured imaging follow-up, and prompt recognition of endograft-related complications because late EVAR failure can still lead to secondary intervention, open conversion, or aneurysm rupture [1,2]. Among EVAR-specific complications, endoleaks remain central to long-term durability

CITATION

Altıntaş G, Kulahcioglu E, Ozcelik S, Unal MA, Haksever A, Erkal E, et al. Anatomical and procedural predictors of type Ia and type III endoleaks after EVAR. Turk J Vasc Surg. XXXX;XX(X):XX-XX.



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because they indicate persistent perfusion of the excluded aneurysm sac outside the endograft lumen [3,4].

Although Type Ia and Type III endoleaks arise through different mechanisms, they were evaluated together in this study because both can rapidly reintroduce systemic arterial pressure into the excluded aneurysm sac. Type Ia endoleak represents proximal sealing-zone failure, whereas Type III endoleak results from modular component separation, inadequate overlap, or graft-fabric disruption. In both situations, the protective depressurization achieved by EVAR may be lost, leading to rapid sac pressurization, sac enlargement, and increased rupture risk. Their recognition therefore usually requires prompt or prioritized reintervention rather than prolonged observation [3-6]. This shared high-pressure physiology provides the rationale for evaluating these two entities within the same clinical framework, while their distinct mechanisms justify separate predictor analyses.

Type Ib endoleak was not included in the primary comparison despite its high-pressure physiology because it represents a distal iliac attachment-site failure with a different anatomical substrate, treatment pathway, and prevention strategy. The present analysis was deliberately restricted to the contrast between proximal seal-zone failure (Type Ia) and endograft component or fabric-related failure (Type III). This focus also avoids repeating a general endoleak-subtype analysis and allows a more specific mechanistic hypothesis: Type Ia endoleak is driven predominantly by hostile proximal neck anatomy, whereas Type III endoleak is driven by global aneurysm morphology, iliac configuration, and anatomy-device interaction.

The predictors of Type Ia endoleak have been extensively linked to hostile proximal neck anatomy. Short neck length, conical or reverse-taper morphology, severe neck angulation, large neck diameter, neck thrombus, and circumferential calcification can reduce circumferential apposition and effective sealing [5-13]. These anatomical features should be evaluated together when assessing the suitability of the proximal seal zone [6-13].

Type III endoleak is less common but equally important. Its rarity makes risk stratification more difficult, and many studies have focused on device generation, component separation, fabric tears, or reports from large registries and explant analyses [14-18]. Contemporary evidence indicates that Type III endoleak may be associated with old-generation endografts, non-proprietary extensions, large aneurysm diameter, angulated or calcified necks, component overlap problems, conformational changes, limb kinking, and mechanical stress on modular junctions or graft fabric [14-18]. These observations suggest that Type III endoleak should not be understood solely as a device event; rather, it may represent the interaction between aneurysm geometry, iliac anatomy, endograft configuration, and time-dependent mechanical fatigue.

Large aneurysm morphology may also increase mechanical instability of the endograft construct. In aneurysms around or above 6 cm, greater sac diameter and altered flow geometry may increase lateral displacement forces on the endograft, particularly at modular junctions and limb segments. However, this diameter signal should be interpreted cautiously as a marker of global aneurysm burden rather than as an independent clinical threshold. Graft configuration and device design may influence Type III risk, especially when large aneurysm morphology coexists with iliac tortuosity, iliac aneurysm involvement, or suboptimal component alignment [14-19].

This manuscript presents an original mechanism-specific investigation within our institutional EVAR experience. Although some cohort-level descriptors and endoleak event counts necessarily overlap with previously cited institutional reports, the present article was conceived to answer a distinct clinical question that was not the objective of those studies: whether two clinically urgent high-pressure endoleak phenotypes with shared sac-pressurization physiology are associated with different anatomical and available procedural risk domains. The manuscript does not re-evaluate volumetric surveillance as a primary endpoint, does not repeat a generalized endoleak management algorithm, and does not analyze Type II endoleak natural history. Its originality lies in restricting the study to Type Ia proximal seal-zone failure and Type III device/component or anatomy-device interaction failure, comparing these mechanisms directly, and translating the findings into mechanism-specific planning and surveillance considerations after elective EVAR.

MATERIAL AND METHODS

Study Design and Population

This retrospective single-center observational study included consecutive eligible patients who underwent elective EVAR for fusiform infrarenal abdominal aortic aneurysm between January 2016 and April 2024. The cohort was derived from patients treated at Türkiye Yüksek İhtisas Training and Research Hospital and Ankara Bilkent City Hospital and included patients with available preoperative and postoperative contrast-enhanced computed tomography angiography (CTA) and a minimum postoperative follow-up duration of 1 year. The study was observational and non-randomized; therefore, no random allocation, allocation concealment, or blinding procedure was used. The sampling strategy was non-probability consecutive sampling of patients meeting final eligibility criteria, and the manuscript was prepared according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) framework.

Inclusion criteria were elective EVAR for fusiform infrarenal abdominal aortic aneurysm, availability of adequate preoperative CTA, at least one adequate postoperative CTA-based surveillance period, and a minimum of 1 year of clinical/radiological follow-

up. Exclusion criteria were ruptured aneurysm, emergency EVAR, saccular aneurysm, juxtarenal aneurysm, complex EVAR, concomitant thoracic endovascular repair, inadequate preoperative or postoperative CTA, and follow-up shorter than 1 year. Saccular aneurysms were excluded because their morphology, rupture-risk assessment, and treatment indication may differ from fusiform degenerative infrarenal abdominal aortic aneurysm, which could confound morphometric predictor analysis. EVAR selection was based on institutional assessment of operative risk, guideline-based aneurysm indication, proximal and distal landing-zone feasibility, iliac access, patient comorbidity, and multidisciplinary judgment; patients with clearly unsuitable anatomy or markedly unfavorable seal conditions were considered for open surgical repair or complex endovascular alternatives when clinically appropriate.

Ethical approval was obtained from the Health Sciences University Ankara Bilkent City Hospital Medical Research Scientific and Ethical Review Board (TABED E1-21-1721; approval date: 14/04/2021). The need for additional written informed consent was waived by the ethics committee because of the retrospective design and use of de-identified institutional data. Because the retrospective database was constructed from patients who already met the final imaging and follow-up eligibility criteria, the exact number of initially screened EVAR patients and the exact number excluded for each criterion could not be reliably reconstructed. Patients who died before completing 1-year imaging, were followed exclusively at other centers, were lost to follow-up, or could not undergo CTA may therefore be under-represented. This selection process is explicitly acknowledged as a methodological limitation.

This institutional EVAR database has supported previous publications addressing different topics. The earlier Type Ia article evaluated hostile neck characteristics in an earlier and smaller subset, whereas the broader article published in the *Journal of Clinical Medicine* focused on overall endoleak distribution, sac dynamics, volumetric surveillance, secondary intervention, and management strategy [7,20]. The present manuscript is an original high-pressure endoleak study with a distinct endpoint hierarchy and interpretive framework: it is restricted to Type Ia and Type III mechanisms, excludes Type Ib/Type II/Type V from the primary mechanistic question, and evaluates proximal seal-zone failure separately from Type III device/component or anatomy-device interaction failure. No prior figure, table, management algorithm, or volumetric-surveillance endpoint is reused as a central element of the present article.

Imaging Acquisition and Anatomical Measurements

CTA images were reviewed using multiplanar and three-dimensional reconstruction. Measurements were performed using 3MENSIO Vascular software (Pie Medical Imaging, Bilthoven, The Netherlands; version 10.6) and the Sarus Workstation/ Advantage Workstation environment connected to the Ankara

City Hospital imaging database (Advantage Workstation 4.2, GE Healthcare Technologies, Chicago, IL, USA). The anatomical variables included maximum aneurysm diameter, aneurysm length, proximal neck diameter at the infrarenal level, proximal neck diameter before the sac, proximal neck length, conical neck morphology, infrarenal neck-sac angulation, neck thrombus, neck calcification, iliac tortuosity, iliac artery aneurysm involvement, total aneurysm sac volume, patent lumen volume, sac thrombus volume, thrombus density, and thrombus-to-aneurysm volume ratio (TR/AO).

Conical proximal neck morphology was operationalized as a Delta A-B diameter difference ≥ 3 mm between the infrarenal neck level and the distal neck before sac expansion. Short neck was evaluated using the proximal neck length variable, and a threshold of ≤ 15 mm was used in regression analysis, while receiver operating characteristic (ROC) analysis identified a cutoff of ≤ 16 mm. Neck-sac angulation was measured on centerline-reconstructed CTA images and was analyzed both as a continuous morphometric parameter and as a categorical variable using > 60 degrees for regression and > 55 degrees for ROC-derived threshold analysis.

For Type III endoleak, emphasis was placed on variables that may affect endograft conformation and modular stress: aneurysm length, maximum sac diameter, total sac volume, thrombus volume, TR/AO ratio, iliac tortuosity, and iliac aneurysm involvement. Type IIIa and Type IIIb subtypes were not consistently distinguishable in the retrospective dataset; therefore, Type III endoleak was analyzed as a unified clinical endpoint.

Procedural Variables and Follow-Up Protocol

Available procedural variables included intraoperative detection of Type Ia endoleak, use of adjunctive endovascular maneuvers when recorded, fluoroscopy time, contrast volume, and procedural-complexity markers such as external iliac artery extension, severe iliac tortuosity, and advanced iliac atherosclerosis. Detailed device- and procedure-specific variables, including endograft manufacturer, endograft generation, modular versus unibody configuration, exact oversizing, component overlap length, proprietary versus non-proprietary extensions, graft migration, limb kinking, and detailed adherence to instructions for use (IFU), were not consistently available in the retrospective dataset. Therefore, the term procedural predictors in this manuscript refers only to the available intraoperative and procedural-complexity markers, and Type III device-specific inference is intentionally interpreted as exploratory.

Surveillance was structured around clinical assessment and imaging at 1, 6, and 12 months after EVAR and annually thereafter whenever feasible. CTA was the primary modality for morphometric analysis. Duplex ultrasonography was used as an adjunctive tool and as an alternative in selected patients

with contraindications to contrast-enhanced CTA, but inclusion in the current morphometric analysis required adequate CTA-based follow-up. Exact counts of imaging examinations at each scheduled interval and the full trajectory of patients followed outside the institution could not be reconstructed reliably from the retrospective database; this limitation may affect endoleak detection timing and observed event rates.

Outcomes and Definitions

The primary endpoint was development of Type Ia or Type III endoleak during postoperative follow-up. Type Ia endoleak was defined as persistent sac perfusion from the proximal attachment site consistent with proximal seal-zone failure. Type III endoleak was defined as persistent sac perfusion attributable to modular component separation, junctional failure, inadequate overlap, or graft fabric defect. Because Type Ia and Type III endoleaks have different mechanisms, predictors were analyzed separately rather than as a single pooled endpoint. No formal a priori sample-size calculation was performed because the study was a retrospective analysis of a fixed eligible cohort; statistical power was therefore limited by the observed numbers of Type Ia ($n = 16$) and Type III ($n = 21$) events.

Secondary endpoints included need for secondary intervention, type of secondary intervention, clinical feasibility of intervention, and available follow-up outcome information. Type Ia endoleaks were considered proximal seal failures and treated when feasible with endovascular measures such as balloon remodeling, proximal cuff placement, extension, adjunctive fixation, or embolization, with open conversion reserved for failure or unsuitable anatomy. Type III endoleaks were treated primarily with endovascular relining or additional stent-graft components when feasible, with open conversion used selectively. Post-intervention technical success, recurrent or persistent endoleak, rupture, and cause-specific mortality after each secondary intervention were not consistently recorded and were therefore not analyzed as formal endpoints.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA) and MedCalc version 15.8 (MedCalc Software Ltd., Ostend, Belgium) under institutional software licenses. Continuous variables were summarized as mean $\pm$ standard deviation or median with range according to distribution. Categorical variables were summarized as frequency and percentage. Normality was evaluated using Kolmogorov-Smirnov testing, skewness-kurtosis assessment, histograms, and quantile-quantile plots. Normally distributed continuous variables were compared using the independent-samples t-test, non-normally distributed variables were compared using the Mann-Whitney U test, and categorical variables were compared using the chi-square test or Fisher exact test when expected cell counts were small.

Univariate logistic regression was used to evaluate candidate predictors. Candidate variables included age, sex, major cardiovascular risk factors, aneurysm diameter, aneurysm length, total sac volume, thrombus volume, TR/AO ratio, proximal neck length, conical neck morphology, neck-sac angulation, neck thrombus, neck calcification, iliac tortuosity, iliac aneurysm involvement, intraoperative Type Ia endoleak, fluoroscopy time, contrast volume, and available procedural-complexity markers. Variables were considered for multivariable modeling when $p < 0.10$ in univariate analysis, when supported by biological plausibility, and when the limited event count allowed inclusion without excessive model instability. For Type Ia endoleak, multivariable logistic regression was performed with a restricted number of proximal neck variables. For Type III endoleak, multivariable regression was not performed because of the limited number of events, strong collinearity among sac-size variables, and absence of key device-specific variables; Type III findings were therefore based on univariate comparisons and ROC analysis. ROC thresholds were selected using the Youden index. Missing data were handled by complete-case analysis, and no multiple imputation was performed. Formal internal validation, calibration analysis, and goodness-of-fit testing were not performed because the subtype-specific models were exploratory and event-limited. Statistical significance was set at $p < 0.05$.

RESULTS

Cohort Profile and High-Pressure Endoleak Distribution

The final cohort included 383 elective EVAR patients. The cohort was predominantly male (356/383, 93.0%) with a mean age of 71.8 ± 7.8 years. Cardiovascular risk factors were frequent, including hypertension in 82.0%, smoking history in 70.2%, and coronary artery disease in 53.0%. Baseline aneurysm burden was substantial, with mean maximum aneurysm diameter of 63.5 ± 11.1 mm, mean aneurysm length of 128.0 ± 19.0 mm, mean total sac volume of 236.1 ± 113.8 cm³, and mean thrombus volume of 112.1 ± 70.7 cm³ (Table 1).

The mean radiological follow-up duration was 33.2 ± 21.2 months, with a median of 25 months and a range of 12 to 99 months. Overall endoleak was detected in 100 patients (26.1%). The subtype distribution was Type II in 47 patients (12.3%), Type Ib in 26 patients (6.8%), Type III in 21 patients (5.5%), Type Ia in 16 patients (4.2%), and Type V in 1 patient (0.3%). Because some patients developed more than one subtype during follow-up or had combined patterns requiring treatment in the same session, subtype counts were not treated as mutually exclusive groups. Type Ib, Type II, and Type V were reported for transparency but were not included in the primary Type Ia/Type III mechanistic analysis. The analytical framework and separation of the two endoleak phenotypes are shown in Figure 1.

Table 1. Baseline cohort profile and anatomical context for the Type Ia/Type III analysis	
Variable	Value
Patients	383 elective infrarenal AAA EVAR patients
Male sex	356 (93.0%)
Age	71.8 ± 7.8 years
Hypertension	314 (82.0%)
Smoking history	269 (70.2%)
Coronary artery disease	203 (53.0%)
Maximum aneurysm diameter	63.5 ± 11.1 mm
Aneurysm length	128.0 ± 19.0 mm
Total sac volume	236.1 ± 113.8 cm ³
Thrombus volume	112.1 ± 70.7 cm ³
TR/AO ratio	45.8 ± 11.9%
Proximal neck length	25.7 ± 9.9 mm
Neck diameter level A / level B	23.0 ± 3.4 mm / 23.7 ± 3.8 mm
Conical neck morphology	17.0%
Neck–sac angulation	28.9 ± 23.4 degrees
Neck thrombus / neck calcification	30.8% / 18.3%
Iliac tortuosity	31.6%
Radiological follow-up duration	33.2 ± 21.2 months
Type Ia endoleak	16 (4.2%)
Type III endoleak	21 (5.5%)
Any secondary intervention	54 (14.1%)

Abbreviations: AAA, abdominal aortic aneurysm; EVAR, endovascular aneurysm repair; TR/AO, thrombus-to-aneurysm volume ratio.

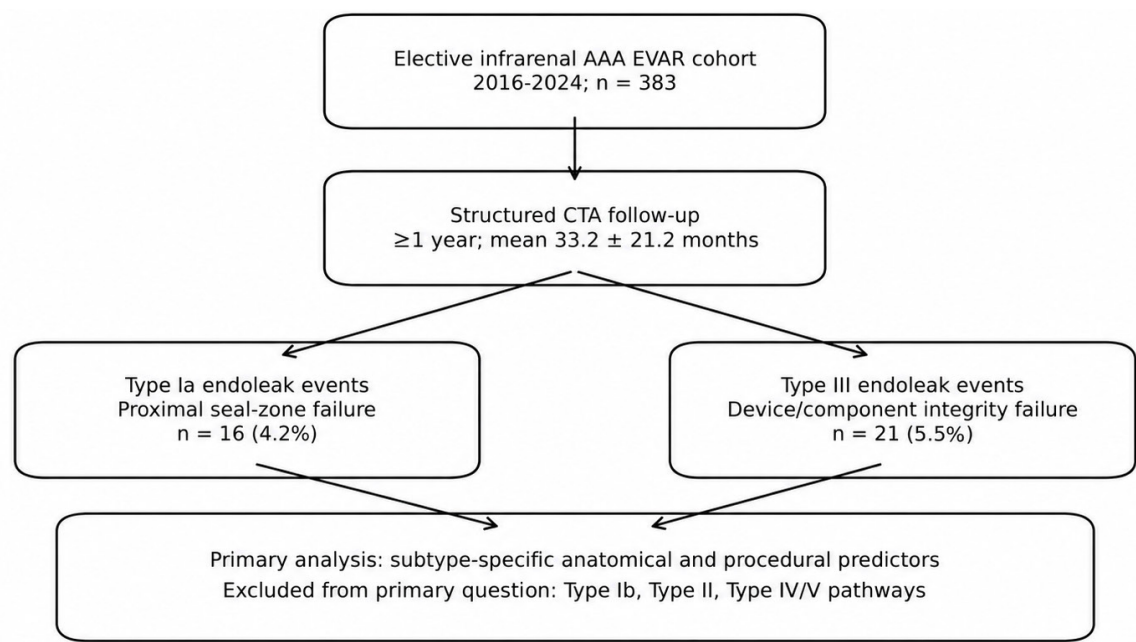


Figure 1. Analytical flow and separation of high-pressure endoleak phenotypes. Counts refer to subtype events within the current cohort; other endoleak types were not included in the primary mechanistic analysis. Abbreviations: AAA, abdominal aortic aneurysm; CTA, computed tomography angiography; EVAR, endovascular aneurysm repair.

Type Ia Endoleak: Proximal Seal-Zone Phenotype

Type Ia endoleak was primarily associated with hostile proximal neck features. Conical neck morphology, defined by Delta A-B ≥ 3 mm, had the strongest geometric association with Type Ia endoleak. On univariate analysis, conical morphology was associated with an odds ratio (OR) of 12.75 and a 95% confidence interval (CI) of 4.26 – 38.15 ($p < 0.001$), and it remained independently associated after multivariable adjustment (adjusted OR 16.89; 95% CI 4.05 – 70.48; $p < 0.001$). ROC analysis identified Delta A-B ≥ 3 mm as the optimal threshold for Type Ia endoleak prediction, with an area under the ROC curve (AUC) of 0.787 ($p < 0.001$).

Short proximal neck length was also associated with Type Ia endoleak. Neck length ≤ 15 mm was significant in univariate logistic regression (OR 3.48; 95% CI 1.21 – 9.96; $p = 0.020$), and ROC analysis identified ≤ 16 mm as a diagnostic threshold (AUC 0.792; $p < 0.001$). Although neck length was not retained in the final adjusted model, its clinical relevance remains important because short landing zones reduce the available sealing surface and magnify the effect of conicity, calcification, and thrombus.

Neck-sac angulation was another major risk domain. Patients with Type Ia endoleak had higher preoperative angulation than those without Type Ia endoleak (50.3 ± 26.4 degrees vs. 27.9 ± 22.8 degrees; $p < 0.001$). Angulation > 60 degrees was associated with Type Ia endoleak in univariate analysis (OR 12.11; 95% CI 4.22 – 34.70; $p < 0.001$) and remained significant in multivariable analysis (adjusted OR 5.72; 95% CI 1.36 – 24.03; $p = 0.017$). ROC analysis identified an angulation threshold > 55 degrees

(AUC 0.742; $p < 0.001$).

Neck wall quality was a particularly strong determinant. Neck calcification was present in 81.3% of patients with Type Ia endoleak compared with 15.5% without Type Ia endoleak ($p < 0.001$), corresponding to an adjusted OR of 21.28 (95% CI 4.45 – 101.70; $p < 0.001$). Neck thrombus was present in 62.5% of Type Ia cases compared with 29.4% of non-Type Ia cases ($p = 0.010$), and it remained independently associated with Type Ia endoleak (adjusted OR 4.22; 95% CI 1.03 – 17.22; $p = 0.045$). Intraoperative Type Ia endoleak was observed in 24 patients (6.3%) and was significantly associated with subsequent postoperative Type Ia endoleak ($p = 0.002$), supporting the importance of completion angiography and immediate seal optimization.

The candidate variables screened for Type Ia included clinical factors (age, sex, hypertension, smoking history, coronary artery disease), global aneurysm variables, and proximal neck variables. The final adjusted Type Ia model was restricted to anatomically plausible proximal neck variables to reduce overfitting, and the results should be interpreted as exploratory because only 16 Type Ia events were available.

Risk accumulation was clinically meaningful. Among patients with a single proximal neck risk factor, Type Ia endoleak developed in 12.5%; this increased to 31.2% among those with two risk factors and 56.2% among those with three or more risk factors. The independent Type Ia predictors are displayed in Figure 2, and the mechanistic comparison with Type III predictors is summarized in Table 2.

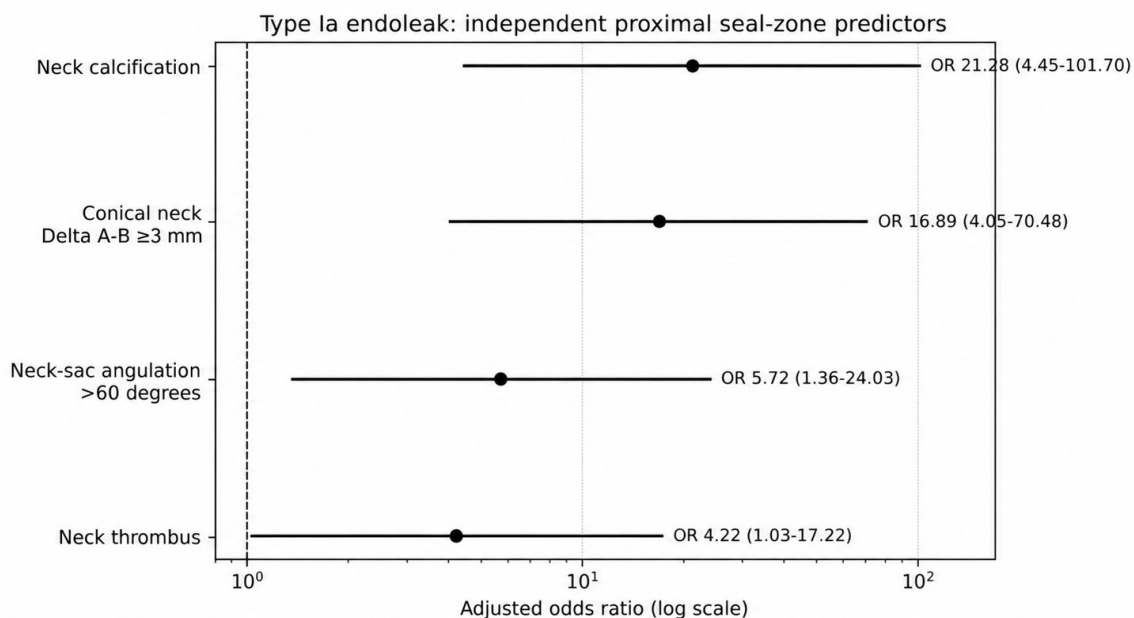


Figure 2. Multivariable predictors of Type Ia endoleak. Forest-style plot showing independent proximal seal-zone variables retained in the adjusted logistic regression model. Points represent adjusted ORs, and horizontal lines represent 95% confidence intervals. Abbreviations: OR, odds ratio.

Table 2. Mechanism-specific predictor domains for type Ia and type III endoleaks

Predictor domain	Type Ia endoleak signal	Type III endoleak signal	Interpretation
Conical proximal neck	Delta A-B ≥ 3 mm: adjusted OR 16.89; AUC 0.787	Not the dominant Type III signal in this cohort	Proximal sealing failure phenotype
Short neck length	≤ 15 mm: univariate OR 3.48; ROC cutoff ≤ 16 mm, AUC 0.792	Not a principal Type III threshold	Reduced landing zone reserve
Neck-sac angulation	> 60 degrees: adjusted OR 5.72; ROC cutoff > 55 degrees, AUC 0.742	Literature links angulated necks to Type III risk; current Type III signal was stronger for sac/iliac burden	Seal failure and device conformation stress
Neck calcification	Adjusted OR 21.28; 81.3% in Type Ia vs. 15.5% without Type Ia	Not the dominant cohort variable for Type III	Impaired circumferential apposition
Neck thrombus	Adjusted OR 4.22; 62.5% in Type Ia vs. 29.4% without Type Ia	Not the dominant cohort variable for Type III	Unstable sealing interface
Aneurysm length	Not central to Type Ia model	149.1 ± 13.7 vs. 126.7 ± 18.5 mm; cutoff > 139 mm, AUC 0.836	Elongated sac increases modular stress
Thrombus burden	Not central to Type Ia model	Thrombus volume > 118 cm ³ , AUC 0.783; TR/AO $> 52\%$, AUC 0.880	High thrombus/sac burden may reflect unstable excluded sac mechanics
Iliac tortuosity	Not a proximal seal-zone determinant	71.4% vs. 29.3%; $p < 0.001$	Device alignment and limb conformation risk
Iliac aneurysm involvement	Not a proximal seal-zone determinant	47.6% vs. 14.6%; $p < 0.001$	Distal extension and modular stress environment
Intraoperative Type Ia endoleak	6.3% of all patients; associated with later Type Ia, $p = 0.002$	Not evaluated as Type III predictor	Completion angiography remains critical

Abbreviations: AUC, area under the ROC curve; OR, odds ratio; ROC, receiver operating characteristic; TR/AO, thrombus-to-aneurysm volume ratio.

Type III Endoleak: Aneurysm Burden, Iliac Complexity, and Mechanical-Stress Phenotype

Type III endoleak showed a different risk pattern. Instead of being defined mainly by the immediate proximal seal zone, Type III endoleak was associated with larger aneurysm burden, greater thrombus burden, iliac tortuosity, and iliac aneurysm involvement. Patients who developed Type III endoleak had significantly longer aneurysms than patients without Type III endoleak (149.1 ± 13.7 mm vs. 126.7 ± 18.5 mm; $p < 0.001$). ROC analysis identified aneurysm length > 139 mm as a strong predictor (AUC 0.836; $p < 0.001$).

Global sac burden was also higher in Type III cases. Maximum aneurysm diameter was 70.8 ± 13.3 mm in Type III cases versus 63.1 ± 10.8 mm in patients without Type III endoleak ($p = 0.002$), with an ROC threshold > 62 mm (AUC 0.693; $p = 0.002$). Total aneurysm sac volume was 305.6 ± 138.6 cm³ versus 232.0 ± 111.1 cm³ ($p = 0.004$), with a threshold > 208 cm³ (AUC 0.691; $p = 0.002$). Although these AUC values were lower than those of aneurysm length and TR/AO ratio, they support the interpretation that larger sacs create a less stable three-dimensional environment for modular endograft components.

Thrombus volume and TR/AO ratio showed strong associations with Type III endoleak. Thrombus volume was 189.7 ± 106.7 cm³ in Type III cases compared with 107.6 ± 65.5 cm³ in non-

Type III cases ($p = 0.002$), with a threshold > 118 cm³ (AUC 0.783; $p < 0.001$). The TR/AO ratio was $59.4 \pm 8.0\%$ in Type III cases compared with $45.0 \pm 11.6\%$ in non-Type III cases ($p < 0.001$), and the threshold TR/AO $> 52\%$ had the highest predictive performance among tested Type III variables (AUC 0.880; $p < 0.001$).

Iliac anatomy strongly distinguished Type III endoleak. Iliac tortuosity was present in 71.4% of Type III cases and 29.3% of non-Type III cases ($p < 0.001$). Iliac aneurysm involvement was present in 47.6% of Type III cases versus 14.6% of non-Type III cases ($p < 0.001$). These findings support a mechanical interpretation: elongated aneurysm sacs, severe iliac tortuosity, and iliac aneurysmal extension may impair device alignment, alter modular overlap geometry, increase conformational stress, and predispose to late component instability or fabric stress.

The ROC-based Type III thresholds are shown in Figure 3. The highest-performing predictors were TR/AO $> 52\%$, aneurysm length > 139 mm, and thrombus volume > 118 cm³, suggesting that a combined assessment of sac length, thrombus burden, and iliac configuration may be more informative than maximum diameter alone.

No multivariable Type III model was constructed. Although the Type III event count was slightly higher than the Type Ia count, the strongest Type III variables were highly correlated measures of aneurysm burden, and the most important device-specific

variables for Type III endoleak were not consistently available. A multivariable model using only the available anatomical variables would therefore risk overfitting and could falsely imply that anatomy alone independently predicts Type III failure. The Type III analysis is consequently presented as a descriptive and hypothesis-generating risk-domain analysis.

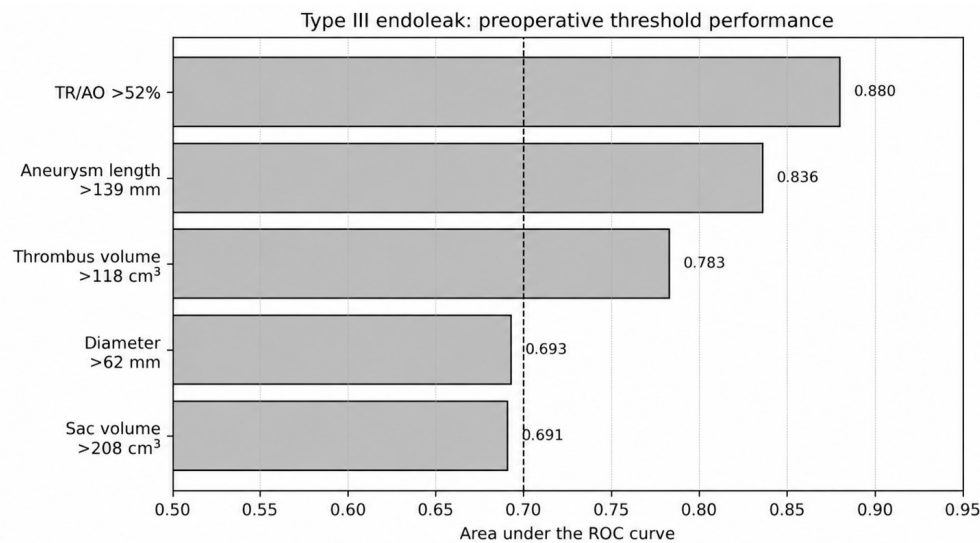


Figure 3. ROC-based threshold performance for Type III endoleak. TR/AO ratio > 52%, aneurysm length > 139 mm, and thrombus volume > 118 cm³ showed the strongest predictive performance. Abbreviations: ROC, receiver operating characteristic; TR/AO, thrombus-to-aneurysm volume ratio.

Secondary Interventions and Treatment Pattern

Secondary intervention was required in 54 patients (14.1%) in the overall cohort, with a mean time to reintervention of 21.7 ± 10 months. Because Type Ia and Type III endoleaks directly pressurize the aneurysm sac, they were generally managed with early intervention when clinically feasible. Treatment details are summarized in Table 3.

Among the 16 patients with Type Ia endoleak, 14 underwent endovascular treatment. Two patients were not treated because of severe comorbidity and intensive care status. Among 21 patients

with Type III endoleak, 16 underwent endovascular intervention and one underwent open surgical conversion. Three patients declined operation, and one patient was followed conservatively because clinical status and anatomical feasibility did not support endovascular treatment. Post-intervention technical success, recurrent endoleak, persistent endoleak, rupture, and endoleak-specific mortality could not be reliably reconstructed for every treated patient and are therefore not reported as formal outcome measures. These patterns nevertheless underscore that high-pressure endoleaks are not merely radiological findings; they frequently translate into treatment decisions and secondary procedures.

Table 3. Treatment implications of Type Ia and Type III endoleaks in the current cohort			
Subtype	Primary failure mechanism	Cohort treatment pattern	Planning implication
Type Ia	Proximal seal-zone failure at infrarenal neck; direct sac pressurization	n = 16; 14 endovascularly treated; 2 untreated because of severe comorbidity/ICU status	Optimize proximal landing zone; consider aggressive ballooning, cuff, adjunctive fixation, embolization, or alternative repair when multiple hostile-neck features coexist
Type III	Modular junction failure, component separation, overlap failure, or fabric defect; direct systemic flow into sac	n = 21; 16 endovascular; 1 open conversion; 3 declined operation; 1 conservative because intervention was not suitable	Assess sac length, thrombus burden, iliac tortuosity, iliac aneurysm, component overlap and long-term device conformation; use relining or open conversion when needed
Shared high-pressure pathway	Direct systemic pressure transmission to the sac	Prompt intervention was favored when feasible	Surveillance should prioritize early classification, sac behavior, and mechanism-specific repair planning

Abbreviation: ICU, intensive care unit.

DISCUSSION

The present study demonstrates that Type Ia and Type III endoleaks should be analyzed as high-pressure EVAR failure modes with different anatomical signatures. Although both subtypes are clinically important because they can directly pressurize the aneurysm sac, the predictors observed in this cohort support two different mechanisms. Type Ia endoleak was dominated by proximal seal-zone variables: conical neck morphology, short neck length, severe neck-sac angulation, neck calcification, and neck thrombus. Type III endoleak, in contrast, was associated with global aneurysm burden, thrombus burden, iliac tortuosity, and iliac aneurysm involvement. The overall endoleak rate of 26.1% is within the range commonly reported after EVAR, often approximately 15-30%, but direct comparisons should account for differences in follow-up duration, CTA-based surveillance intensity, device generation, anatomical complexity, and whether combined endoleak subtypes are counted as mutually exclusive events [3-5].

The Type Ia findings are consistent with the hostile neck concept. Proximal seal depends on circumferential wall apposition, sufficient landing length, favorable neck geometry, and a stable interface between the endograft and the aortic wall. Conical neck morphology may generate a distal-to-proximal mismatch that reduces radial sealing at the proximal landing zone. Severe angulation can produce eccentric apposition and persistent gutters, while calcification and thrombus can prevent full contact between the graft fabric and the wall. In the present cohort, these mechanisms were reflected by strong adjusted ORs for conical neck morphology, neck calcification, neck thrombus, and neck-sac angulation.

A particularly important finding was risk-factor accumulation. Type Ia endoleak increased progressively as proximal neck risk factors accumulated, reaching 56.2% among patients with three or more unfavorable proximal neck characteristics. This finding supports a cumulative planning model. A single hostile neck characteristic may be manageable with careful sizing and deployment, but multiple hostile features should prompt reconsideration of standard EVAR suitability, enhanced proximal fixation, planned adjunctive maneuvers, fenestrated or branched alternatives when available, or open repair in appropriate patients. The association between intraoperative and postoperative Type Ia endoleak further supports the need for meticulous completion angiography and immediate seal optimization.

The findings should also be interpreted in the context of treatment selection. Standard infrarenal EVAR may remain reasonable in selected patients with one manageable hostile feature, especially when operative risk is high, but markedly hostile neck anatomy or anatomy outside device-specific IFU should prompt serious consideration of alternatives. These alternatives may include

fenestrated or branched endovascular strategies where available, planned adjunctive fixation, or open surgical repair in patients with acceptable physiological reserve. Open repair remains an important option when durable proximal or distal sealing cannot be achieved safely with standard EVAR, and this should be emphasized when multiple hostile neck features coexist.

Type III endoleak showed a different phenotype. The strongest Type III threshold was TR/AO > 52%, followed by aneurysm length > 139 mm and thrombus volume > 118 cm³, whereas the maximum diameter threshold > 62 mm had weaker discriminatory performance. These findings should not be interpreted as thrombus burden or diameter independently causing component failure. Instead, these variables may be surrogate markers of a large, elongated, mechanically complex aneurysm sac in which the endograft has more space to change conformation over time. In a long sac with substantial thrombus and severe iliac tortuosity, modular overlap may be exposed to longitudinal, torsional, and bending forces. This mechanism remains hypothetical because no biomechanical modeling was performed, but it provides a plausible explanation for the observed association with component separation, limb kinking, fabric abrasion, or late material fatigue.

The observed Type III associations align with contemporary literature. Gennai et al. reported that old-generation endografts, non-proprietary extensions, large aneurysms, and angulated or calcified necks were risk factors for Type III endoleak and that recurrent Type III endoleak is clinically relevant [14]. Grandhomme et al. emphasized that Type III reports remain scarce and that mechanisms include modular disconnection, trunk-limb overlap separation, fabric lesions, suture disruption, and fabric fatigue from kinking [15]. Aras et al. similarly highlighted that unfavorable endograft configuration, redundancy, migration, larger initial aneurysm diameter, and physical forces may exceed design limits and contribute to Type IIb endoleak [16]. Our cohort supports this framework by showing that iliac tortuosity and iliac aneurysm involvement are part of the anatomical stress environment associated with Type III failure. The 6-cm diameter concept is therefore best understood as a practical marker of large aneurysm morphology and potential lateral-force exposure, not as an independent or definitive risk threshold. This interpretation is consistent with the long-term unibody bifurcation endograft experience reported by Iscan et al., in which device architecture was discussed in relation to migration, limb dislocation, and durability in elective EVAR [19].

The originality of this manuscript lies in its mechanism-specific research question, endpoint selection, and clinical interpretation. Although the same institutional EVAR experience has been cited in previous publications, those articles addressed different objectives: an earlier Type Ia-focused hostile-neck analysis in a smaller subset and a broader article published in the Journal

of Clinical Medicine on global endoleak distribution, sac dynamics, volumetric surveillance, secondary intervention, and management strategy [7,20]. The present article does not reanalyze volumetric surveillance as a primary outcome and does not reproduce a global endoleak algorithm. It evaluates Type Ia and Type III endoleaks as two urgent high-pressure EVAR failure phenotypes, contrasts proximal seal-zone failure with Type III anatomy-device interaction, explains why Type Ib is outside the primary mechanistic comparison, and emphasizes different predictor domains for these two complications. The analytical framework, figures, tables, and conclusions were revised to make this originality explicit while maintaining transparent citation of related institutional work.

The procedural implications are practical. For Type Ia risk, preoperative planning should focus on proximal seal suitability: neck length, neck taper, diameter mismatch, angulation, thrombus, and calcification. In borderline cases, the operative plan should anticipate balloon remodeling, proximal cuff placement, active fixation, parallel graft or fenestrated strategies, or alternative repair. For Type III risk, the planning focus should shift to device conformation: sac length, iliac tortuosity, iliac aneurysm involvement, limb trajectory, component overlap, and long-term mechanical stability. In patients with large, elongated aneurysms and marked iliac tortuosity, follow-up should include careful assessment of modular overlap, limb configuration, component separation, kinking, migration, and sac behavior [19,21-25].

The study also highlights limitations in routine datasets. Exact component overlap length, device-specific sizing, proprietary versus non-proprietary extension use, and clear distinction between Type IIIa and Type IIIb endoleak were not consistently available. These variables are likely important for Type III prediction and should be prospectively collected in future EVAR registries. Similarly, formal analysis of adherence to device-specific IFU was not available, limiting direct conclusions about off-label anatomy or device-specific outcomes. Nonetheless, the available morphometric data identify a clinically coherent Type III risk phenotype centered on aneurysm burden and iliac mechanical complexity.

From a surveillance perspective, the findings support risk-adapted imaging. Standard follow-up remains appropriate for uncomplicated EVAR, but intensified surveillance should be considered when proximal hostile-neck variables cluster or when large elongated sacs coexist with iliac tortuosity and iliac aneurysm involvement. In Type Ia-risk patients, early postoperative imaging should focus on the proximal seal and neck remodeling. In Type III-risk patients, follow-up should extend beyond simple endoleak detection to assess component overlap, limb geometry, modular integrity, and subtle signs of device deformation.

Limitations

This study has several important limitations. First, it is a retrospective single-center observational study derived from an institutional EVAR database. The same institutional experience has also supported previous publications with different objectives, and this relationship is disclosed and cited. The present manuscript should therefore be interpreted as an original mechanism-specific Type Ia/Type III endoleak investigation from an existing institutional database, rather than as a duplicate of the previous global endoleak or volumetric-surveillance report.

Second, the retrospective database was constructed from patients who met final imaging and follow-up eligibility criteria. The initial number of screened EVAR patients, exclusion counts for each criterion, the exact number of imaging examinations at every scheduled follow-up interval, and outcomes of patients followed exclusively at other centers could not be reliably reconstructed. Patients who died early, were lost to follow-up, or could not undergo CTA may have been excluded, introducing selection bias and potentially affecting endoleak rates and time-to-event estimates.

Third, event numbers were small: Type Ia endoleak occurred in 16 patients and Type III endoleak in 21 patients. The Type Ia multivariable model therefore has a low events-per-variable ratio and is vulnerable to instability and overfitting. The Type III analysis was limited to univariate comparisons and ROC analysis because of collinearity among sac-size variables and the absence of key device-specific variables. No internal validation, calibration assessment, or formal goodness-of-fit testing was performed.

Fourth, device-related and detailed procedural variables were not consistently available, including endograft manufacturer and generation, modular versus unibody configuration, oversizing, component overlap length, proprietary versus non-proprietary extensions, IFU adherence, migration, limb kinking, and Type IIIa versus Type IIIb classification. These omissions are especially important for Type III endoleak, and the present findings cannot establish that anatomy alone independently predicts Type III failure.

Fifth, post-intervention technical success, recurrent or persistent endoleak, rupture, and endoleak-specific mortality after secondary treatment were not consistently recorded. Formal interobserver and intraobserver reproducibility testing for morphometric measurements was not performed. Finally, the proposed mechanical explanation linking thrombus burden, sac length, iliac tortuosity, and Type III endoleak is biologically plausible but remains hypothetical because no biomechanical modeling or device-strain analysis was performed. The 6-cm aneurysm concept should therefore be interpreted as a descriptive large-aneurysm marker rather than a definitive clinical cutoff.

CONCLUSION

Type Ia and Type III endoleaks after EVAR share high-pressure clinical relevance but arise from different anatomical and mechanical risk domains. Type Ia endoleak was primarily associated with hostile proximal neck anatomy, including conical neck morphology, severe neck–sac angulation, neck thrombus, and neck calcification. Type III endoleak was associated with larger aneurysm burden, thrombus burden, iliac tortuosity, and iliac aneurysm involvement, supporting a possible anatomy-device interaction phenotype rather than a purely device-related event.

In practical terms, the strongest Type III signals in this cohort were TR/AO ratio > 52%, aneurysm length > 139 mm, and thrombus volume > 118 cm³. Maximum aneurysm diameter around or above 6 cm may be a useful marker of large aneurysm morphology, but it should not be interpreted as an independent or definitive Type III risk threshold because its discriminatory performance was lower and no multivariable Type III model was performed. Similarly, the presence of an intraoperative Type Ia endoleak should not be regarded as a trivial angiographic finding; even when corrected during the index procedure, it may indicate a vulnerable proximal seal zone and should prompt careful seal optimization and closer postoperative surveillance.

A mechanism-specific preoperative assessment may improve EVAR planning and risk-adapted surveillance. Proximal neck complexity should inform Type Ia prevention, whereas aneurysm length, thrombus burden, iliac complexity, graft configuration, and available procedural information should inform Type III surveillance and repair planning. Because Type III device-specific data were incomplete, these findings should guide future prospective data collection rather than be used as definitive independent prediction rules.

Ethics Committee Approval: Health Sciences University Ankara Bilkent City Hospital Medical Research Scientific and Ethical Review Board, TABED E1-21-1721, approval date 14/04/2021.

Patient Consent for Publication: The requirement for additional written informed consent was waived by the ethics committee because of the retrospective design and use of de-identified institutional data.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: All authors contributed equally to the article.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

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