ORIGINAL ARTICLE

DOI: 10.4274/nkmj.galenos.2026.79446
Nam Kem Med J 2026;14(2):203-214

Morphological and Biomechanical Determinants of Persistent Sac Expansion Following Endovascular Aortic Repair: A Longitudinal Analysis Focused on Endotension and Non-shrinking Sacs

Endovasküler Aort Tamiri Sonrası Kalıcı Kese Genişlemesinin Morfolojik ve Biyomekanik Belirleyicileri: Endotension ve Küçülmeyen Keselere Odaklanan Boylamsal Bir Analiz

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ABSTRACT

Aim: The fundamental objective of endovascular aortic repair (EVAR) is the effective exclusion and subsequent shrinkage of the aneurysm sac. Nevertheless, a clinical challenge persists in a subset of patients who exhibit progressive sac enlargement despite the total absence of detectable endoleaks, a phenomenon known as endotension or type V endoleak. This study investigates the morphological, biomechanical, and systemic determinants of persistent sac expansion following EVAR.

Materials and Methods: A longitudinal analysis was performed on 41 consecutive patients with a minimum 18-month follow-up. We evaluated anatomical metrics via thin-slice computed tomography angiography, biochemical markers including high-sensitivity C-reactive protein (hs-CRP), and systemic comorbidities. Multivariable logistic regression was employed to identify independent predictors of adverse sac dynamics.

Results: Sac expansion (≥5 mm) was observed in 14.6% of the cohort during the follow-up period. Multivariable analysis demonstrated that a preoperative sac diameter exceeding 60 mm [odds ratio (OR): 3.24] and an increased mural thrombus burden (OR: 2.86) were the strongest independent biomechanical predictors of persistent enlargement. Patients with adverse sac behavior frequently exhibited elevated hs-CRP levels and a higher prevalence of systemic conditions such as obesity, chronic obstructive pulmonary disease, and hypothyroidism

Conclusion: Post-EVAR sac expansion is a multifactorial systemic phenotype influenced by landing zone quality, mural thrombus biomechanics, and host inflammatory status. These findings suggest that in endotension cases, the transmission of pulsatile energy through the mural thrombus may maintain sac pressurization even without a visible flow channel. Recognizing persistent expansion as a distinct phenotype necessitates a shift in post-EVAR surveillance from a binary endoleak-focused approach toward a more comprehensive assessment of longitudinal sac dynamics and biomechanical risk factors.

Keywords: EVAR, aneurysm sac expansion, endotension, mural thrombus biomechanics, , hs-CRP, longitudinal analysis

ÖZ

Amaç: Endovasküler aort tamiri (EVAR) sonrası başarının temel ölçütü anevrizma kesesinin küçülmesidir. Ancak bazı olgularda, herhangi bir endoleak saptanmamasına rağmen kesenin büyümeye devam ettiği "endotension" tip V endoleak fenotipi gözlenmektedir. Bu çalışma, EVAR sonrası kalıcı kese genişlemesine yol açan morfolojik, biyomekanik ve sistemik faktörleri boylamsal bir perspektifle incelemeyi amaçlamaktadır

Gereç ve Yöntem: Çalışma kapsamında, EVAR uygulanan ve en az 18 ay takip edilen 41 ardışık hastanın verileri retrospektif olarak analiz edilmiştir. Bilgisayarlı tomografi anjiyografi üzerinden alınan anatomik ölçümler; yüksek duyarlılıklı C-reaktif protein (hs-CRP) düzeyi gibi biyokimyasal parametreler ve komorbidite profilleri ile karşılaştırılarak, kese genişlemesini öngördüren faktörler multivaryant lojistik regresyon modeli ile belirlenmiştir.

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Received: 08.02.2026 **Accepted:** 30.03.2026 **Publication Date:** 16.06.2026

Cite this article as: Onar LÇ. Morphological and biomechanical determinants of persistent sac expansion following endovascular aortic repair: a longitudinal analysis focused on endotension and non-shrinking sacs. Nam Kem Med J. 2026;14(2):203-214

Bulgular: Takip süresince hastaların %14,6'sında ≥5 mm kese genişlemesi saptanmıştır. Yapılan analizlerde, 60 mm üzerindeki preoperatif kese çapı [olasılık oranı (OR): 3.24] ve yüksek mural trombüs yükü (OR: 2.86), kalıcı kese büyümesinin en güçlü bağımsız biyomekanik belirleyicileri olarak öne çıkmıştır. Genişleme gösteren hastalarda sistemik inflamasyonun bir göstergesi olan hs-CRP düzeylerinin belirgin şekilde yüksek olduğu ve bu hastalarda kronik obstrüktif akciğer hastalığı, obezite ve hipotiroidi gibi sistemik durumların daha sık görüldüğü tespit edilmiştir.

Sonuç: Post-EVAR kese genişlemesi, sadece teknik bir yetersizlik değil; landing zone kalitesi, mural trombüs biyomekaniği ve hastanın enflamatuvar profilinin etkileşimiyle ortaya çıkan sistemik bir fenotiptir. Görünür bir sızıntı olmasa dahi, mural trombüsün basınç iletimini kolaylaştırması kesenin gerilmesine neden olabilmektedir. Bu durum, takip süreçlerinde endoleak varlığından ziyade kese hacmi ve biyomekanik risk faktörlerine odaklanan kişiselleştirilmiş stratejilerin önemini vurgulamaktadır.

Anahtar Kelimeler: EVAR, anevrizma kese genişlemesi, endotension, mural trombüs biyomekaniği, hs-CRP, landing zone kalitesi

INTRODUCTION

Endovascular aortic repair (EVAR) has fundamentally transformed the therapeutic landscape for abdominal aortic aneurysms and selected dissections, offering a minimally invasive alternative with superior perioperative morbidity and recovery profiles compared to open surgical repair[1-3]. The traditional paradigm of EVAR success relies on the effective exclusion of the aneurysmal sac from systemic pressure, typically manifesting as sac shrinkage or stabilization over time[3].

Despite technical advancements, long-term durability is frequently jeopardized by persistent sac expansion[3,4]. While endoleaks-categorized into well-defined subtypes based on their source are considered the primary drivers of expansion, clinical practice has revealed a paradoxical subgroup of patients[4]. These individuals demonstrate progressive sac enlargement despite the total absence of detectable endoleaks on high-resolution serial imaging, a phenomenon known as "endotension" or type V endoleak[5,6]. This phenomenon suggests that sac pressurization may persist through occult mechanisms, including graft porosity, transmission of pulsatile energy through the mural thrombus, or micro-collateral flow[5,6].

Furthermore, emerging evidence indicates that the host's systemic environment encompassing chronic inflammation, collagen metabolism disorders (e.g., fibrillin gene defects), and metabolic dysregulation may critically influence the failure of sac regression[6,7]. This study aims to move beyond binary endoleak classifications to explore the integrative morphological, biochemical, and biomechanical determinants of persistent sac expansion in a well-defined longitudinal cohort.

MATERIALS AND METHODS

Study Design and Population

This retrospective observational study was conducted at the Department of Cardiovascular Surgery, a tertiary referral center. We screened 41 consecutive patients who underwent primary endovascular stent-graft implantation for aortic aneurysm between January 2009 and February 2013. The study protocol received Maltepe University Faculty of Medicine Clinical Research Ethics Committee (approval number: AN023, date: 27.11.2013) and all participants provided written informed consent.

Patients were eligible for inclusion if they were treated with endovascular stent-graft implantation for primary aortic pathology and possessed comprehensive preoperative, perioperative, and postoperative clinical data. A strict minimum follow-up duration of 18 months with standardized imaging assessments was mandatory, alongside voluntary consent for medical record analysis. Conversely, the study excluded patients presenting with congenital aortic malformations, penetrating aortic ulcers, or those requiring emergency EVAR under hemodynamically unstable conditions that precluded standardized preoperative imaging. Furthermore, patients were excluded if they possessed severe concomitant valvular stenosis or regurgitation that could significantly alter systemic aortic hemodynamics, or if they had incomplete follow-up records.

Imaging Protocol and Definitions

Preoperative and postoperative assessments were performed using contrast-enhanced computed tomography angiography (CTA) with thin-slice acquisitions (≤1.5 mm). In the postoperative period, serial imaging was conducted according to a standardized surveillance protocol, with contrast-enhanced CTA performed at 3, 6, 12, and 18 months, provided that clinical conditions permitted intravenous contrast administration. In addition, Doppler ultrasonography was routinely used for interim evaluation at 9 months. In patients in whom intravenous contrast administration was contraindicated, follow-up assessments were performed using Doppler ultrasonography and/or non-contrast-enhanced computed tomography to ensure continuity of sac diameter measurements. Aneurysm sac diameter was defined as the maximal transverse diameter measured perpendicular to the centerline of flow. Persistent sac expansion was strictly defined as a ≥5 mm increase in the maximal sac diameter relative to the first postoperative baseline measurement. Endotension

was classified as persistent sac expansion in the absence of any detectable type I-IV endoleak on serial arterial- and delayed-phase CTA.

Clinical and Biochemical Assessment

In addition to standard anatomical metrics (neck angulation, thrombus distribution), we evaluated systemic comorbidities including obesity (body mass index), spirometry results, presence of systemic hypertension, complete blood count and thyroid function tests. Inflammatory status was quantified using high-sensitivity C-reactive protein (hs-CRP) and the neutrophil-to-lymphocyte ratio.

Statistical Analysis

Statistical analyses were performed using specialized software (SPSS version 25.0). Continuous variables were assessed for normality using the Shapiro-Wilk test and expressed as mean ± standard deviation or median with interquartile range. Categorical variables were reported as frequencies.

To identify predictors of sac expansion, we employed a rigorous two-stage modeling approach. Initially, univariable analysis (Student's t-test, Mann-Whitney U, or chi-square/Fisher's exact test as appropriate) was used to screen potential risk factors. Variables demonstrating clinical relevance or statistical significance ($p<0.10$) were then entered into a multivariable logistic regression model using a backward stepwise elimination method. This was done to determine independent associations and calculate odds ratios (ORs) with 95% confidence intervals. To account for the dynamic nature of sac changes, longitudinal data were further analyzed using generalized estimating equations to evaluate the impact of time-dependent variables on sac diameter. A p-value<0.05 was considered statistically significant.

RESULTS

Patient Characteristics

Between January 2009 and February 2013, a total of 41 patients who underwent EVAR for aortic aneurysm or aortic dissection met the inclusion criteria and were included in the final analysis. All patients completed a minimum follow-up duration of 18 months, with serial imaging available for longitudinal assessment of aneurysm sac behavior.

The study cohort consisted predominantly of male patients and represented a typical EVAR population with advanced age and a high prevalence of cardiovascular risk factors. Baseline demographic characteristics and comorbid conditions were comparable across the cohort and are summarized in Table 1.

Sac Dynamics and Procedural Outcomes

Technically successful stent-graft deployment was achieved in all 41 cases, with no immediate type I or III endoleaks detected on completion angiography. However, the 18-month longitudinal follow-up revealed that aneurysm sac behavior is

Table 1. Demographic and clinical characteristics of the study cohort

Variable	Patients (n=41) / value
Age (years), mean ± SD	68.59±6.68
Gender, n (%)	
Female	11 (26.8%)
Male	30 (73.2%)
Body mass index (kg/m²), median (range)	27.13 (22.91-39.54)
Comorbidities, n (%)	
Hypertension	19 (46.3%)
Diabetes mellitus	12 (29.3%)
Hyperlipidemia	13 (31.7%)
Coronary artery disease	9 (22.0%)
Carotid artery disease	10 (24.2%)
Chronic obstructive pulmonary disease	13 (31.7%)
Smoking status, n (%)	
Never smoked	6 (14.6%)
Former smoker	21 (51.2%)
Current smoker	14 (34.1%)
Laboratory and vital signs	
Pulse (bpm), median (range)	81 (65-121)
Systolic blood pressure (mmHg), median (range)	130 (100-185)
Diastolic blood pressure (mmHg), median (range)	85 (60-110)
Creatinine (mg/dL), median (range)	1.1 (0.8-1.5)
Fasting blood glucose (mg/dL), median (range)	100 (90-305)
HbA1c (%), median (range)	5.5 (4.0-8.2)
LDL cholesterol (mg/dL), mean ± SD	131.12±29.24
HDL cholesterol (mg/dL), median (range)	40 (28-90)
Triglycerides (mg/dL), mean ± SD	403.1±122.96
hsCRP (mg/L), median (range)	2.17 (0.8-4.10)
Hematocrit (%), mean ± SD	39.90±5.11
Neutrophil-to-lymphocyte ratio, median (range)	1.60 (1.10-2.90)

BMI: Body mass index, COPD: Chronic obstructive pulmonary disease, bpm: beats per minute, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, hsCRP: high-sensitivity C-reactive protein, SD: Standard deviation, EVAR: Endovascular aortic repair, systolic and diastolic blood pressure as well as pulse rate values represent mean measurements obtained from repeated outpatient assessments and ambulatory blood pressure monitoring during the 18-month follow-up period. All laboratory parameters reflect averaged results from serial analyses performed preoperatively and at 3, 9, 12, and 18 months after EVAR

a complex, multifactorial process that cannot be adequately explained by endoleak classification alone. Persistent sac expansion, defined as an increase of >5 mm, was recorded in 6 patients (14.6%), highlighting that expansion can occur despite apparently complete aneurysm exclusion. This observation underscores the inherent limitations of a purely binary success–failure paradigm, as the absence of traditional endoleaks did not guarantee sac stability or shrinkage in our cohort. Anatomical and procedural characteristics of the study population are summarized in Table 2.

Endoleak Patterns and the Endotension Phenotype

A distinct subgroup of patients (n=2, 4.9%) developed progressive sac enlargement in the total absence of any detectable endoleak on serial CTA, consistent with the phenomenon of endotension (type V endoleak). In our study, endotension emerged as a clinically meaningful postoperative phenotype with potential implications for long-term outcomes, rather than a rare incidental finding. Conversely, persistent collateral flow (Arc of Riolan) was identified as a driver in two other expansion cases, specifically linked to patent lumbar arteries where retrograde filling was confirmed in delayed-phase imaging.

Our findings demonstrate a significant dissociation between endoleak status and sac stability; while some patients with documented low-flow endoleaks showed stable or regressing diameters, others without any detectable leak exhibited continued expansion. This reinforces the concept that endoleak presence alone is an insufficient surrogate marker for aneurysm stability, requiring clinicians to interpret imaging findings within the broader context of longitudinal sac dynamics and systemic forces.

Shared Features and Independent Predictors

A comparative analysis revealed that adverse sac behavior was associated with specific systemic and anatomical factors. Patients with expanding sacs shared common features such as elevated hs-CRP levels (mean 3.8 mg/L), uncorrected hypothyroidism, chronic anemia, and persistent diastolic hypertension. Furthermore, clinical features suggestive of subclinical connective tissue vulnerability, potentially related to fibrillin associated matrix fragility, were more frequently observed in the expansion group; however, no molecular confirmation was available, and this observation should be interpreted as hypothesis-generating.

In multivariable analysis (Figure 1), a larger preoperative sac diameter (>60 mm; OR: 3.24, p = 0.031) and a high mural thrombus burden (OR: 2.86, p = 0.042) emerged as the strongest independent predictors of persistent post-EVAR sac expansion, whereas procedural factors and the brand of the graft were not significantly associated with adverse sac behavior. Independent predictors identified in multivariable logistic regression analysis are summarized in Table 3. These results reinforce the concept that post-EVAR sac dynamics are predominantly driven by pre-existing anatomical and biological factors rather than isolated procedural or imaging-defined findings.

Table 2. Anatomical and procedural characteristics of the study population

Variable	Value (n=41)	p-value*
Aortic pathology, n (%)		
Infrarenal abdominal aortic aneurysm	39 (95.1%)	0.64
Thoracic aortic aneurysm	2 (4.9%)	0.64
Preoperative anatomical metrics		
Maximum aneurysm sac diameter (mm), mean ± SD	58.4±8.2	0.018
Proximal neck length (mm), mean ± SD	22.1±5.4	0.011
Proximal neck angulation (degrees), mean ± SD	32.5±12.1	0.032
Presence of mural thrombus, n (%)	28 (68.3%)	0.041
Procedural parameters		
Stent-graft main body diameter (mm), mean ± SD	28.2±3.1	0.27
Stent-graft oversizing (%), mean ± SD	15.4±4.2	0.19
Total procedure time (min), mean ± SD	115±25	0.48
Fluoroscopy time (min), mean ± SD	18.4±6.5	0.56
Contrast medium volume (mL), mean ± SD	95±30	0.61

*p-values refer to the association between each variable and the presence of persistent sac expansion and/or any type of endoleak during follow-up, SD: Standard deviation, EVAR: Endovascular aortic repair, the majority of patients were treated for infrarenal abdominal aortic aneurysms, with thoracic aortic aneurysms representing a small minority of cases. Preoperative imaging demonstrated moderate aneurysm size and generally acceptable proximal neck anatomy, although mural thrombus was frequently observed. Procedural parameters reflected standard EVAR practice with appropriate stent-graft sizing and oversizing, and no excessive procedural or fluoroscopic durations were recorded

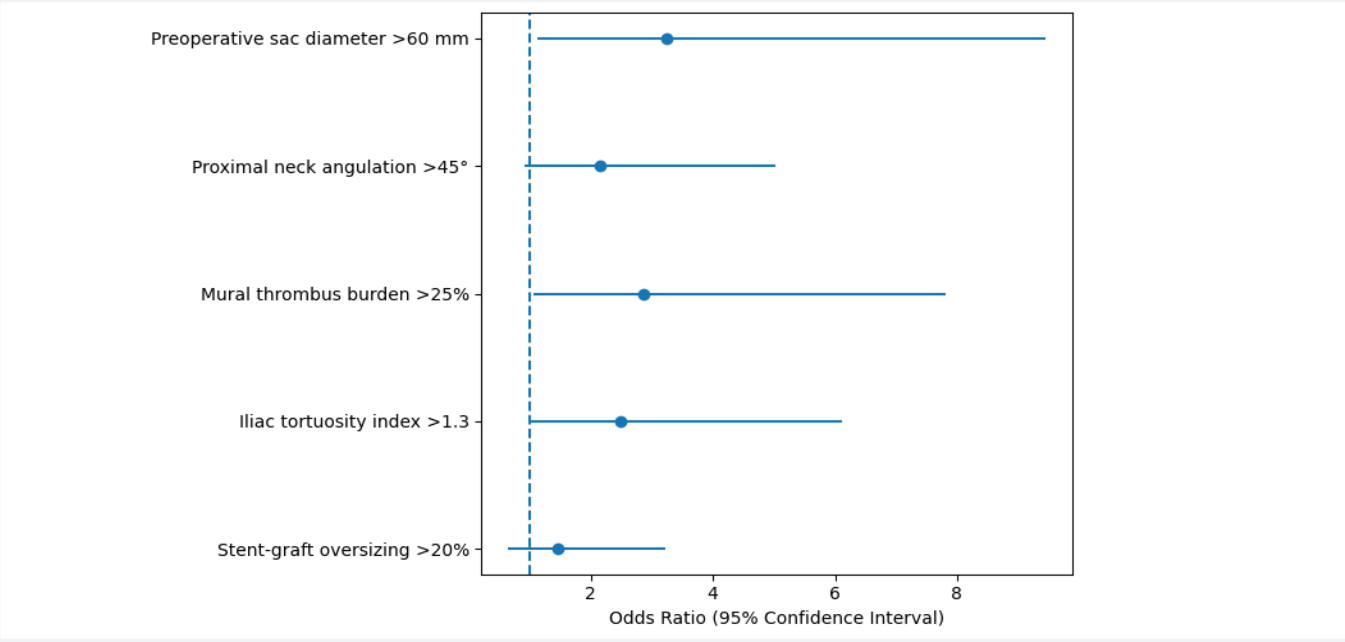

Figure 1. Multivariable predictors of persistent sac expansion after EVAR Forest plot illustrating odds ratios and 95% confidence intervals for variables included in the multivariable model. Increased preoperative sac diameter, mural thrombus burden, and iliac tortuosity index were associated with adverse sac behavior, whereas procedural oversizing was not a significant determinant. The vertical dashed line represents the null effect (odds ratio=1)

EVAR: Endovascular aortic repair

Table 3. Multivariable logistic regression analysis of factors associated with persistent sac expansion after

Variable	Odds ratio	95% Confidence interval	p-value**
Anatomical factors			
Preoperative sac diameter >60 mm	3.24	1.12-9.45	0.031*
Proximal neck angulation >45°	2.15	0.92-5.02	0.078
Mural thrombus burden >25%	2.86	1.05-7.82	0.042*
Iliac tortuosity index >1.3	2.48	1.01-6.12	0.047*
Procedural factors			
Stent-graft oversizing >20%	1.45	0.65-3.22	0.365
Follow-up findings			
Presence of type II endoleak	1.22	0.48-3.10	0.672
Presence of type I/III endoleak†	N/A	N/A	N/A

**Variables with p < 0.10 in univariable analysis were entered into the multivariable logistic regression model using backward stepwise elimination, *Statistically significant (p<0.05), †Type I and type III endoleaks were excluded from the regression model because no such events were observed in this cohort

Illustrative Case Scenarios of Adverse Sac Behavior Guiding Post-EVAR Clinical Decision-making

Case 1: Type II Endoleak with Progressive Sac Enlargement Leading to Open Conversion

A 67-year-old female patient with diabetes mellitus and collagen tissue disease underwent EVAR for an infrarenal abdominal aortic aneurysm with a preoperative sac diameter of 69.7x107.1mm (Figure 2A). After the EVAR procedure follow-up imaging demonstrated limited sac regression, measuring 69.6x106.4 mm at 6 months and 69.5x107.0 mm at 12 months. At the 18-month CTA, the sac diameter increased to 69.6x110.9 mm, corresponding to a cumulative 3-mm expansion (Figure 2B). The Doppler ultrasonography demonstrated persistent pulsatile flow within the sac, and contrast-enhanced CTA confirmed a type II endoleak originating from lumbar arteries (Figure 2C).

Given the documented sac enlargement exceeding guideline-recommended thresholds and ongoing sac pressurization, an endovascular attempt at lumbar artery embolization using occluder devices was performed but proved unsuccessful due to extensive and complex collateral flow. As sac pressurization persisted despite endovascular intervention, open surgical conversion was undertaken, resulting in definitive resolution.

Case 2: Early Type II Endoleak with Moderate Sac Expansion Managed with Intensified Surveillance

A 69-year-old male patient underwent EVAR for an infrarenal abdominal aortic aneurysm with a preoperative sac diameter of 60 mm. At the 6-month follow-up, contrast-enhanced CTA demonstrated a type II endoleak, with the aneurysm sac measuring 63 mm, corresponding to a 3-mm increase in diameter (Figure 3A). Detailed imaging analysis identified retrograde sac perfusion through lumbar arteries supplied via collateral flow from the arc of Riolan, indicating a mesenteric–lumbar collateral pathway as the source of the endoleak (Figure 3B, C).

Given the moderate degree of sac enlargement, the early timing after EVAR, and the absence of rapid expansion or high-risk features, a strategy of repeated imaging surveillance was adopted rather than immediate reintervention, with a low threshold for secondary intervention should further sac expansion be observed.

The moderate degree of sac enlargement, early post-EVAR timing, and identification of a collateral-driven type II endoleak without high-risk features supported a management strategy of intensified imaging surveillance rather than immediate reintervention, with a low threshold for secondary intervention should further sac expansion occur.

Case 3: Endotension Phenotype with Minimal Sac Growth Managed Conservatively

A 71-year-old male patient with hypertension and chronic obstructive pulmonary disease underwent EVAR for an infrarenal abdominal aortic aneurysm with a baseline sac diameter of 58mm. Serial CTA demonstrated 58 mm at 6 months, 59 mm at 12 months, and 60 mm at 18 months, reflecting a total sac enlargement of 2 mm over 18 months (Figure 4A, B). No demonstrable endoleak was identified on repeated imaging, and no graft-related complications were observed. Given the low expansion rate, absence of endoleak (Figure 4C), and stability over time, the patient was classified as having an endotension phenotype and managed with close radiological surveillance rather than reintervention, in accordance with guideline-based recommendations.

Case 4: Minimal Sac Enlargement with Type II Endoleak Managed Conservatively

A 73-year-old female patient with hypertension and obesity underwent EVAR for an aneurysm with a baseline sac diameter of 62 mm. Follow-up imaging demonstrated 62 mm at 6 months and 63 mm at 12 months, corresponding to a 1-mm increase over one year. CTA revealed

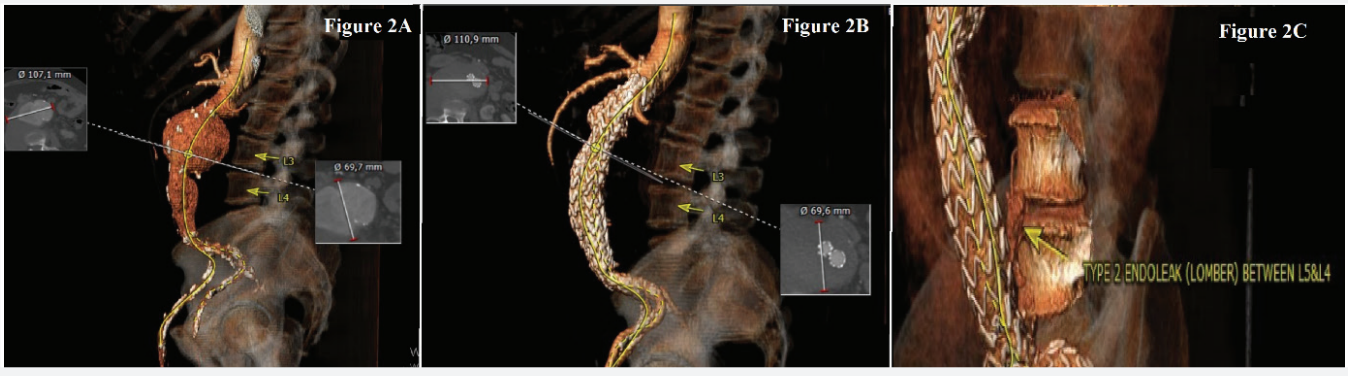

Figure 2. Serial computed tomography angiography demonstrating adverse sac behavior associated with type II endoleak

Figure 2A. Baseline contrast-enhanced computed tomography angiography of a 67-year-old female patient with diabetes mellitus and collagen tissue disease demonstrating an infrarenal abdominal aortic aneurysm prior to endovascular aortic repair. The preoperative aneurysm sac dimensions measured 69.7×107.1 mm; Figure 2B. Follow-up computed tomography angiography obtained at 18 months after endovascular aortic repair demonstrates adverse sac behavior characterized by interval sac enlargement. While early follow-up showed limited sac regression, with measurements of 69.6×106.4 mm at 6 months and 69.5×107.0 mm at 12 months, the sac diameter increased to 69.6×110.9 mm at 18 months, corresponding to an overall expansion of approximately 3 mm; Figure 2C. Contrast-enhanced computed tomography angiography and Doppler ultrasonography demonstrate persistent pulsatile flow within the aneurysm sac, with contrast opacification originating from lumbar arterial branches, consistent with a type II endoleak. These findings explain the failure of sac regression and ongoing sac pressurization despite technically successful endograft deployment

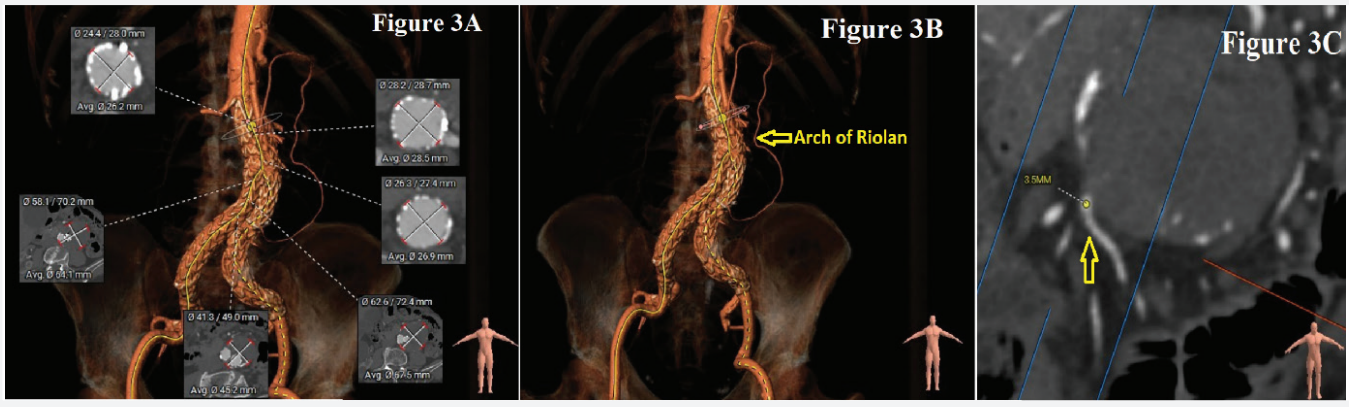

Figure 3. Type II endoleak mediated by mesenteric-lumbar collateral flow via the arc of riolan

Figure 3A. Baseline three-dimensional volume-rendered computed tomography angiography following endovascular aortic repair in a 69-year-old male patient demonstrates appropriate endograft deployment and exclusion of the infrarenal abdominal aortic aneurysm. The preoperative aneurysm sac diameter measured 60 mm. Multiplanar reconstructions and cross-sectional measurements illustrate the spatial relationship between the endograft and the aneurysm sac without evidence of proximal or distal sealing failure at this stage; Figure 3B. computed tomography angiography obtained at the 6-month follow-up reveals a type II endoleak with interval sac enlargement to 63 mm, corresponding to a 3-mm increase in diameter. Volume-rendered imaging demonstrates retrograde filling of lumbar arteries supplied via collateral flow from the arc of Riolan, highlighting a mesenteric–lumbar collateral pathway as the source of persistent sac perfusion. Proximal and distal graft fixation zones remain intact; Figure 3C. Axial contrast-enhanced computed tomography angiography confirms contrast opacification within the aneurysm sac adjacent to a lumbar arterial branch, consistent with retrograde low-pressure filling. The measured contrast column within the collateral vessel further supports the diagnosis of a lumbar artery-mediated type II endoleak without evidence of high-pressure systemic inflow

a type II endoleak originating from lumbar arteries. Given the minimal degree of sac enlargement, potential measurement variability, and absence of rapid progression, the patient was managed with conservative, very close follow-up, without immediate reintervention (Figure 5A, B).

Case 5: Accelerated Late Sac Expansion Following Aorto-uni-iliac EVAR and Distal Type I Endoleak Prompting Open Conversion

A 75-year-old male patient underwent EVAR for an infrarenal abdominal aortic aneurysm with a preoperative maximum sac diameter of 96.4 mm. Due to anatomical considerations, the procedure was performed using an aorto-uni-iliac stent-graft configuration, combined with a femoro–femoral crossover bypass to maintain bilateral lower limb perfusion. Early postoperative imaging confirmed appropriate graft positioning and satisfactory exclusion of the aneurysm sac. During follow-up, the aneurysm sac initially appeared stable; however, at the 6-month evaluation, interval sac enlargement was detected, raising concern for persistent sac pressurization.

Given imaging findings (Figure 6A, B) suggestive of retrograde pelvic inflow, and the assumption that the left internal iliac artery was chronically occluded, an endovascular occlusion of the left internal iliac artery using an occluder device was performed (Figure 6C). Serial contrast-enhanced CTA suggested a high-pressure endoleak originating from the distal sealing zone near the right common iliac artery, consistent with a distal type I endoleak (Figure 6D). As the aneurysm sac diameter exceeded 101 mm (Figure 6B), and given the progressive nature of expansion despite secondary endovascular intervention, a decision was made to proceed with open surgical conversion.

Despite this intervention, subsequent imaging demonstrated continued and progressive sac enlargement. Intraoperatively, the aneurysm sac was found to be pressurized. An attempt at mechanical banding of the distal iliac neck failed to achieve a meaningful reduction in intra-sac pressure. Upon opening the sac, a relatively fresh thrombus was encountered (Figure 7), within which the trajectory of active endoleak flow could be traced circumferentially around the right common iliac artery, confirming the distal sealing zone as the source of persistent sac filling. Consequently, the endograft material was completely explanted (Figure 7), and definitive repair was achieved by performing an aortobifemoral bypass, resulting in successful exclusion and decompression of the aneurysm sac.

DISCUSSION

The present study demonstrates that aneurysm sac behavior following EVAR is governed by an intricate interplay of anatomical, mechanical, and systemic factors[7]. Type II endoleaks represent the most frequently encountered endoleak subtype after EVAR, with an overall reported incidence ranging

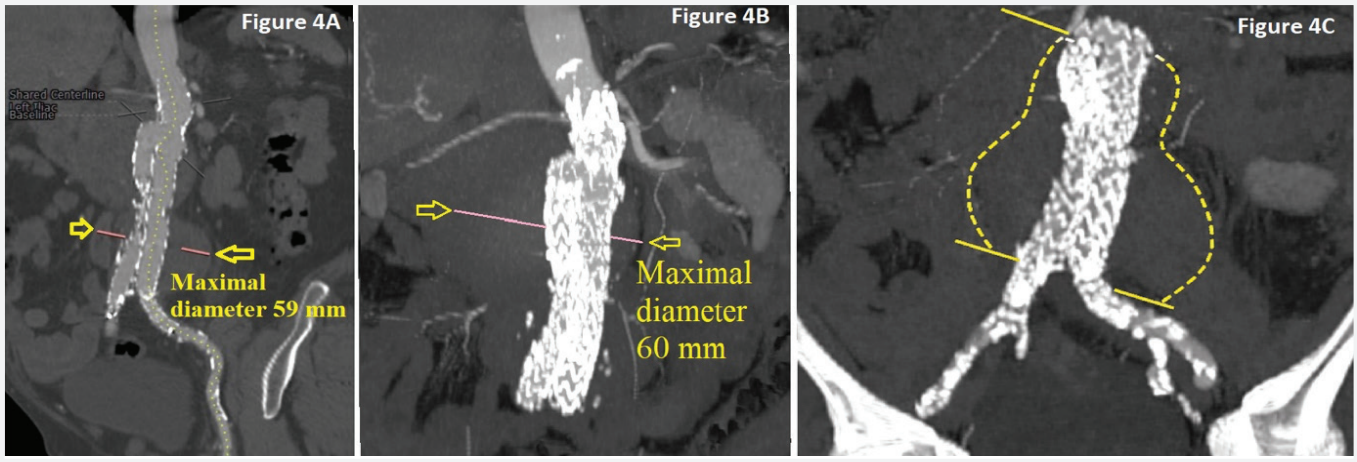

Figure 4. Type V endoleak (endotension)

A 71-year-old male with comorbidities underwent endovascular aneurysm repair for a 58 mm infrarenal abdominal aortic aneurysms. Over 18 months, the sac enlarged by 2 mm (Figure 4A, B) without detectable endoleak (Figure 4C). Due to the slow growth, stable graft, and signs of endotension, guideline-based surveillance was preferred over reintervention; Figure 4A. This coronal computed tomography angiography image shows the abdominal aortic aneurysm sac following endovascular aneurysm repair. The image is labeled with the maximal diameter of 59 mm, a 1 mm increase from baseline. Yellow dashed centerlines track the stent graft, and a double-headed arrow measures the aneurysm's maximal width. There is no visible endoleak, and the stent graft appears well-positioned. This scan has been done at the 12-month follow-up admission; Figure 4B. This follow-up computed tomography angiography, obtained 18 months after endovascular aortic repair, demonstrates a further 1 mm increase, with the aneurysm sac reaching 60 mm in maximal diameter. Again, no endoleak is identified despite the progressive but slow sac expansion over time. The stent graft remains intact with no evidence of migration or structural compromise; Figure 4C. This axial image highlights the absence of contrast extravasation, confirming no visible endoleak. Dashed yellow lines delineate the area between the endograft and the outer aneurysm sac, consistent with endotension—a condition where the aneurysm expands without a detectable leak. Yellow arrows mark the reference boundaries. The stable graft position and lack of complications support the diagnosis of an endotension phenotype

between 20% and 40% among EVAR-treated patients and accounting for nearly half of all identified endoleaks[8]. These endoleaks commonly originate from retrograde collateral flow through branches such as the inferior mesenteric artery, lumbar arteries, median sacral artery, or accessory renal arteries[8]. Importantly, type II endoleaks may remain clinically silent or radiographically occult during early follow-up and are not infrequently detected only after aneurysm sac enlargement becomes evident[8]. Consequently, in cases of continued or progressive sac expansion despite technically successful stent-graft implantation, type II endoleak is often the first and most plausible diagnosis considered in clinical practice. Consistent with these observations, type II endoleaks were not uncommon in our study cohort and constituted a substantial proportion of patients exhibiting adverse sac behavior, underscoring their continued clinical relevance in post-EVAR surveillance. One of the most important insights of the present study is the identification of a distinct subgroup of patients who developed progressive aneurysm sac enlargement in the absence of any detectable endoleak. The presence of this phenotype underscores the critical importance of prolonged follow-up and careful longitudinal assessment, as extended surveillance allows for a more accurate characterization of the factors contributing to endotension[9]. In this context, the true incidence of endotension reported in the literature may be underestimated, potentially reflecting artificially low rates driven by relatively short follow-up durations rather than a genuine rarity of the condition[10]. Accordingly, cohorts with extended imaging surveillance, such as the present study population, are particularly valuable for elucidating the determinants and natural history of endotension-related adverse sac behavior. Rather than representing a rare event, endotension in our cohort emerged as a meaningful postoperative phenotype. Several mechanisms may explain this persistent pressurization, including the transmission of pulsatile forces through the stent-graft material, microleakage below the resolution of conventional imaging, and the biomechanical effects of mural thrombus[9-11]. Our results support the concept that anatomical factors, particularly those related to landing zone quality and thrombus morphology, play a central role in determining these dynamics.

The role of persistent lumbar artery inflow in type II endoleaks deserves particular attention. While type II leaks are often considered benign, our findings in two cases suggest that

lumbar arteries can act as high-pressure conduits reinforced by systemic paraspinal circulation. This persistent inflow prevents the expected fibrotic transformation of the sac[11]. Furthermore, the association between elevated hs-CRP and sac growth suggests that a systemic inflammatory state may keep the mural thrombus in a "fluid" state, facilitating the transmission of systemic pressure even in the absence of a visible flow channel[12,13].

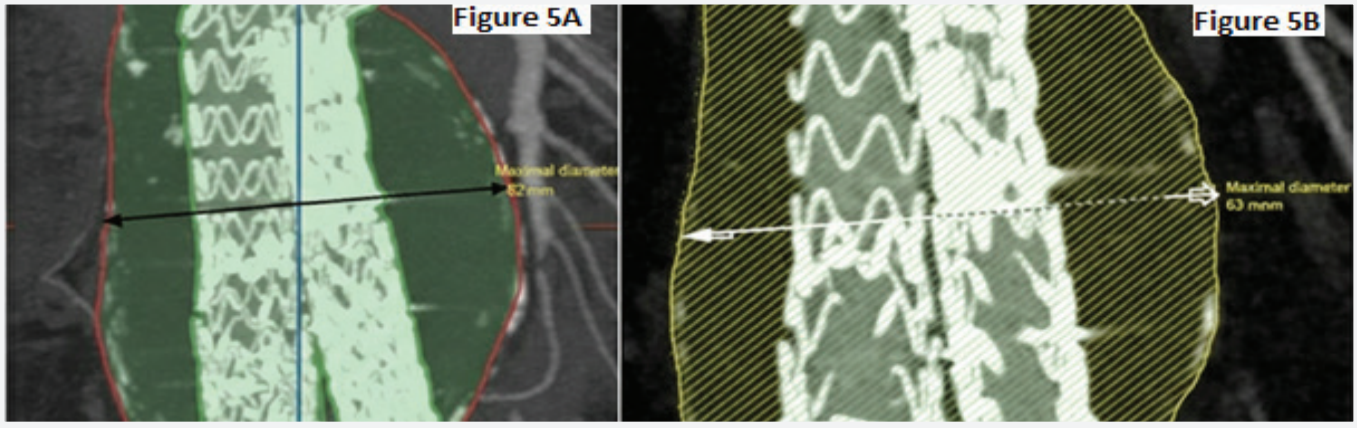

Figure 5. Minimal sac enlargement associated with lumbar artery–mediated type II endoleak managed conservatively

Figure 5A. Contrast-enhanced computed tomography angiography obtained during follow-up in a 73-year-old female patient with hypertension and obesity demonstrates an infrarenal abdominal aortic aneurysm treated with endovascular aortic repair. Three-dimensional reconstruction and cross-sectional measurement indicate a baseline aneurysm sac diameter of 62 mm, with preserved endograft position and intact proximal and distal sealing zones. No significant sac deformation or graft-related complication is evident; Figure 5B. Computed tomography angiography performed at 12 months after endovascular aortic repair demonstrates a minimal increase in aneurysm sac diameter to 63 mm, corresponding to a 1-mm enlargement over one year. Shaded segmentation highlights the aneurysm sac boundaries, and contrast opacification adjacent to lumbar arterial branches is consistent with a type II endoleak originating from lumbar arteries. Given the minimal degree of sac enlargement, potential measurement variability inherent to serial imaging, and absence of rapid progression, these findings supported a strategy of conservative management with very close imaging surveillance, without immediate reintervention

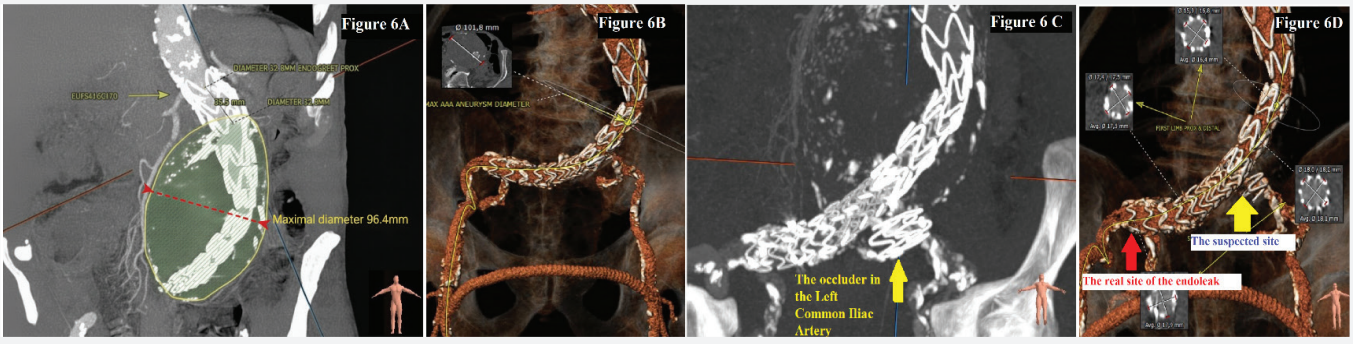

Figure 6. Imaging findings of accelerated sac expansion and distal type I endoleak after aorto-uni-ılıac endovascular aortic repai

Figure 6A. Baseline contrast-enhanced computed tomography angiography demonstrates the infrarenal abdominal aortic aneurysm treated with an aorto-uni-iliac stent-graft configuration. The aneurysm sac is clearly delineated, with an initial maximum diameter of 96.4 mm, and satisfactory early exclusion is observed; Figure 6B. Follow-up computed tomography angiography reveals progressive aneurysm sac enlargement, exceeding 101 mm, despite apparently preserved graft position. These findings indicate persistent sac pressurization and prompted further investigation for a high-pressure endoleak source; Figure 6C. Three-dimensional reconstructed imaging illustrates endovascular occlusion of the left common iliac artery using an occluder device, performed under the assumption of retrograde pelvic inflow contributing to sac pressurization; Figure 6D. Subsequent computed tomography angiography identifies a high-pressure distal Type I endoleak originating from the right iliac sealing zone, resulting in direct systemic pressurization of the aneurysm sac and explaining the continued rapid sac expansion despite secondary endovascular intervention.

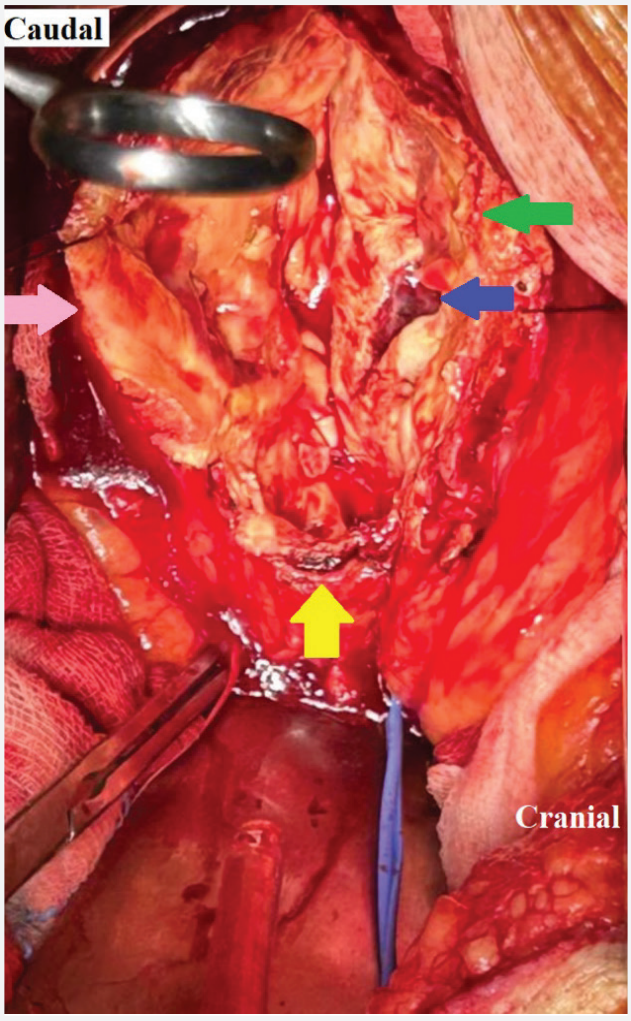

Figure 7. Intraoperative image demonstrating the site of a distal type I endoleak originating from the right common iliac artery landing zone. The blue arrow indicates relatively fresh thrombus formation within the aneurysm sac, allowing visualization of the endoleak flow trajectory, while the yellow arrow denotes the proximal landing zone. On the endoleak side, high-velocity jet flow and turbulent intra-sac hemodynamics contributed to increased intimal injury, resulting in focal irregularity and fragility of the aneurysm sac wall, as indicated by the green arrow. In contrast, the region marked by the pink arrow demonstrates a smoother, more uniform sac wall morphology with preserved structural integrity, consistent with effective sealing and subsequent thrombus organization followed by fibrotic remodeling

The failure of banding during open conversion in our series provides a critical clinical lesson. It suggests that in the setting of advanced sac expansion or endotension, the biomechanical failure is often circumferential or material-related[12]. Simple mechanical constriction of the neck may be insufficient to counteract the transmitted forces, making total graft excision a more definitive, albeit invasive, necessity[13]. This reinforces the need to view sac expansion, regardless of endoleak status, as a warning signal warranting intensified surveillance and individualized decision making[13,14]. Beyond anatomical determinants, persistent sac expansion after EVAR may also reflect a systemic vascular phenotype characterized by chronic inflammation, impaired arterial compliance, and altered extracellular matrix remodeling. Patients with adverse sac dynamics in our cohort more frequently exhibited elevated hs-CRP levels, obesity, chronic obstructive pulmonary disease, hypothyroidism, and diastolic hypertension. Although these variables did not consistently remain independent predictors in multivariable analysis, their clustering suggests that systemic inflammatory burden and vascular wall vulnerability may contribute to sustained intra-sac pressurization[14]. Obesity and chronic obstructive pulmonary disease, in particular, are well recognized to promote low-grade systemic inflammation, endothelial dysfunction, and increased arterial stiffness, mechanisms that may plausibly facilitate transmission of pulsatile forces to the excluded aneurysm sac[15-17]. Taken together, these observations support the concept that failure of sac regression should not be interpreted solely as a graft-related phenomenon but rather as the interaction between local biomechanical conditions and host-related biological susceptibility[18-20]. In this context, persistent sac expansion should not be interpreted solely as a local graft-related phenomenon but rather as the downstream manifestation of an unfavorable anatomical–biological milieu. This integrated perspective may help explain why sac regression fails to occur in certain patients despite technically successful EVAR and underscores the importance of incorporating systemic patient characteristics into postprocedural risk stratification and surveillance strategies.

Finally, the integration of metabolic and genetic factors reveals that sac behavior is a systemic phenotype. Hypothyroidism and anemia likely impair the metabolic pathways required for vascular wall remodeling, while diastolic hypertension provides a constant stressor that prevents sac contraction[18]. In patients with underlying connective tissue weakness, such as fibrillin defects, these forces are amplified[19]. Recognizing persistent sac expansion as a distinct post-EVAR phenotype may enable more refined risk stratification and contribute to improved selection of patients requiring secondary intervention by supporting surveillance strategies that integrate longitudinal sac behavior with anatomical risk markers and systemic inflammatory status, rather than relying exclusively on conventional endoleak classification[1,20-22]. In this context, persistent aneurysm sac expansion should be interpreted as a clinically meaningful indicator of adverse postprocedural dynamics reflecting the combined influence of anatomical configuration, biomechanical forces, and host-related biological susceptibility. Accordingly, longitudinal sac monitoring may represent a more robust marker of procedural

durability than endoleak status alone. This perspective is consistent with evidence from large randomized trials such as EVAR-1 (1), DREAM (21), and OVER (22), which established the central role of aneurysm sac dynamics as a surrogate marker of long-term outcomes after EVAR. Incorporation of sac behavior into postprocedural surveillance frameworks may therefore facilitate more individualized follow-up strategies and support timely decision-making regarding secondary intervention in selected patients.

Study Limitations

This study has several limitations that should be acknowledged. First, its retrospective design inherently limits causal inference and may be subject to selection bias. Second, the study was conducted at a single center with a relatively limited sample size, which may restrict the generalizability of the findings to broader populations or different EVAR platforms.

Additionally, direct measurement of intra-sac pressure was not performed, and conclusions regarding biomechanical mechanisms are therefore inferential rather than mechanistically proven. Advanced imaging modalities capable of detecting microleakage or dynamic pressure transmission were not routinely available during the study period.

Nevertheless, the study benefits from a well-defined cohort, standardized imaging follow-up, and complete longitudinal data without loss to follow-up. The homogeneity of procedural technique and surveillance protocols may, in fact, strengthen internal validity by reducing confounding variability.

CONCLUSION

Persistent aneurysm sac expansion after EVAR represents a complex and clinically relevant phenomenon that extends beyond conventional endoleak classification. Our findings demonstrate that sac behavior is influenced by a constellation of anatomical and procedural factors and should be interpreted as an independent marker of post-EVAR success or failure.

Routine follow-up strategies should place greater emphasis on longitudinal sac dynamics rather than reliance on endoleak status alone. Recognizing endotension and related sac expansion phenotypes may improve patient selection for secondary interventions and contribute to more personalized post-EVAR management strategies.

Future studies incorporating advanced imaging, biomechanical modeling, and direct pressure measurements are warranted to further elucidate the mechanisms underlying persistent sac expansion and to optimize long-term outcomes following EVAR.

Ethics

Ethical Committee Approval: The study protocol received Maltepe University Faculty of Medicine Clinical Research Ethics Committee (approval number: AN023, date: 27.11.2013).

Informed Consent: All participants provided written informed consent.

Acknowledgement

This study was originally conceived and designed by the author (Lütfi Çağatay Onar MD) as a medical specialty thesis in 2009. Ethical approval for the study was obtained in November 2013, and the work was formally completed as a thesis in 2014. During the thesis phase, the author received academic supervision from Professor Dr. Uğur Filizcan. While Professor Dr. Filizcan was not involved in the technical aspects of the manuscript preparation, and therefore is not listed among the authors of the present article, the author gratefully acknowledges his invaluable clinical guidance and contributions to patient management and postoperative follow-up, which were conducted in accordance with contemporary clinical guidelines at the time of the study.

Footnotes

Authorship Contributions

Financial Disclosure: The authors declared that this study received no financial support.

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