CORRELATION OF LABORATORY, RADIOLOGICAL AND CLINICAL PARAMETERS IN PATIENTS WITH POPLITEAL ARTERY ANEURYSM THROMBOSIS – A CASE-CONTROL STUDY

KORELACIJA LABORATORIJSKIH, RADIOLOŠKIH I KLINIČKIH PARAMETARA KOD PACIJENATA SA TROMBOZOM ANEURIZME ZATKOLENE ARTERIJE – STUDIJA SLUČAJEVA

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Abstract

Introduction. Popliteal artery aneurysm may present with acute thrombosis, a complication associated with limb-threatening ischaemia. This study aimed to identify clinical, radiological, and laboratory features associated with acute popliteal artery aneurysm thrombosis at presentation, rather than to establish causal risk factors for future thrombosis. **Material and Methods.** A case-control study included 142 patients with popliteal artery aneurysm treated at a tertiary vascular surgery centre between January 2017 and December 2024. Cases (n=71) presented with acute thrombosis (Rutherford categories I through IIb); controls (n=71) had asymptomatic popliteal artery aneurysm without thrombosis confirmed by imaging. Demographic, clinical, radiological and laboratory parameters were compared. Multivariable logistic regression identified features independently associated with thrombosis at presentation. **Results.** Patients with thrombosis were older (67.4 versus 61.2 years; p=0.002), more often smokers (73.2% versus 47.9%; p<0.001), and diabetic (43.7% versus 25.4%; p=0.009). Aneurysm diameter was larger (29.3 versus 23.7 millimetres; p<0.001), mural thrombus was more frequent (67.6% versus 29.6%; p<0.001), and D-dimer was higher (1,842 versus 724 nanograms per millilitre; p<0.001). The strongest independent associations were observed for mural thrombus (odds ratio 3.82), aneurysm diameter above 25 millimetres (odds ratio 3.21) and D-dimer above 1,000 nanograms per millilitre (odds ratio 3.14). **Conclusion.** In this single-centre case-control sample, acute popliteal artery aneurysm thrombosis was associated with larger aneurysm diameter, mural thrombus, and elevated D-dimer levels, alongside older age, smoking, and diabetes. Laboratory biomarkers should be interpreted as contemporaneous markers of the acute thrombotic state rather than antecedent predictors. Prospective validation is needed before diagnostic application.

Key words: Popliteal Artery Aneurysm; Thrombosis; Diagnostic Imaging; Signs and Symptoms; Fibrinogen; Biomarkers; Fibrin Fibrinogen Degradation Products

Sažetak

Uvod. Aneurizma poplitealne arterije može se manifestovati akutnom trombozom, komplikacijom povezanom sa ishemijom koja ugrožava ekstremitet. Cilj studije bio je identifikacija kliničkih, radioloških i laboratorijskih karakteristika povezanih sa akutnom trombozom aneurizme poplitealne arterije pri prezentaciji, a ne utvrđivanje kauzalnih faktora rizika za buduću trombozu. **Materijal i metode.** Sprovedena je studija slučajeva i kontrola koja je obuhvatila 142 pacijenta sa aneurizmom poplitealne arterije lečenih u tercijarnom centru za vaskularnu hirurgiju (januar 2017–decembar 2024). Pacijenti su bili slučajevi (n = 71) sa akutnom trombozom (*Rutherford* kategorije I do IIb), kontrole (n = 71) pacijenti sa asimptomatskom aneurizmom bez tromboze potvrđenom imidžingom. Poređeni su demografski, klinički, radiološki i laboratorijski parametri. Multivarijantnom logističkom regresijom identifikovane su karakteristike nezavisno povezane sa trombozom pri prezentaciji. **Rezultati.** Pacijenti sa trombozom bili su stariji (67,4 naspram 61,2 godine; p = 0,002), češće pušači (73,2% naspram 47,9%; p < 0,001) i imali su dijabetes (43,7% naspram 25,4%; p = 0,009). Dijametar aneurizme bio je veći (29,3 naspram 23,7 milimetara; p < 0,001), muralni tromb češći (67,6% naspram 29,6%; p < 0,001), D-dimer viši (1.842 naspram 724 nanograma po mililitru; p < 0,001). Najsnažnije nezavisne asocijacije uočene su za muralni tromb (odnos šansi 3,82), dijametar iznad 25 milimetara (odnos šansi 3,21) i D-dimer iznad 1.000 nanograma po mililitru (odnos šansi 3,14). **Zaključak.** U ovom jednocentričnom uzorku, akutna tromboza aneurizme poplitealne arterije bila je povezana sa većim dijametrom aneurizme, prisustvom muralnog tromba i povišenim D-dimerom, uz stariju životnu dob, pušenje i dijabetes.

Ključne reči: aneurizma poplitealne arterije; tromboza; dijagnostički imidžing; znaci i simptomi: fibrinogen; biomarkeri; produkti razgradnje fibrina i fibrinogena

Introduction

Popliteal artery aneurysm (PAA) is the most common peripheral arterial aneurysm and may present with acute thrombosis, a complication that can lead to limb-threatening ischaemia [1,2]. The prevalence of PAA in the general population is approximately 1%, with a strong male predominance and higher in-

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Abbreviations

PAA – popliteal artery aneurysm
CTA – computed tomography angiography
IQR – interquartile range
OR – odds ratio
CI – confidence interval
AUC – area under the receiver operating characteristic curve
VIF – variance inflation factor
SD – standard deviation
FEU – fibrinogen equivalent units
EPV – events per variable

cidence among older patients with cardiovascular comorbidities [3]. Bilateral PAAs are present in 50–70% of cases, and associated abdominal aortic aneurysms are found in 40–50% of patients [4].

Acute thrombosis of PAA is a limb-threatening emergency; in a systematic review of 895 cases, the early amputation rate reached 14% [5]. Unlike abdominal aortic aneurysms, which primarily present with rupture, PAAs more commonly thrombose due to altered haemodynamics and intraluminal thrombus accumulation [6]. Current Society for Vascular Surgery guidelines recommend elective repair for symptomatic aneurysms of any size and for asymptomatic aneurysms exceeding 20 mm in diameter or containing mural thrombus [7].

Risk stratification for PAA thrombosis has traditionally focused on aneurysm size and morphological features. However, morphology-only assessment may miss systemic prothrombotic signals [8]. Several biomarkers, particularly D-dimer and fibrinogen, have been linked to adverse cardiovascular outcomes in peripheral arterial disease [9,10], and hypoalbuminaemia has been associated with poor results after vascular surgery [11,12]. A recent multicentre study also suggested that per cent thrombus burden may be more informative than diameter alone in identifying high-risk PAAs [13].

Previous reports on PAA thrombosis have focused on morphological correlates and surgical outcomes [8,9], with limited attention to laboratory biomarkers measured at the time of acute presentation. The study aimed to identify clinical, radiological, and laboratory features associated with acute PAA thrombosis at presentation, rather than to establish causal risk factors for future thrombosis.

Material and Methods

Study design and setting

This case-control study was conducted at a tertiary referral centre for vascular emergencies serving a population of approximately 2 million. Data were collected from patients treated between January 2017 and December 2024. The study was approved by the Institutional Ethics Committee (approval number: 00-20/522) and conducted in accordance with the Declaration of Helsinki. The need for individual informed consent was waived due to the study's retrospective design. This report follows STROBE recommendations for case-control studies [14]. A completed STROBE checklist is provided as Supplementary Table S1.

Participants

Of 180 patients diagnosed with PAA during the study period, 142 were included in the final analysis **(Figure 1).** Thirty-eight patients were excluded: incomplete clinical or laboratory data (n=22; predominantly missing D-dimer in 14 and albumin in 11, with some patients missing both), previous ipsilateral revascularisation (n=8), active malignancy (n=5), and current anticoagulation therapy (n=3). Exclusion reasons were mutually exclusive (primary reason assigned). Characteristics of included and excluded patients were compared; no significant differences in age, sex, or aneurysm diameter were observed (all $p>0.10$).

Cases (n=71) were patients presenting with acute PAA thrombosis confirmed by computed tomography angiography (CTA) or duplex ultrasonography, defined as complete occlusion of the popliteal artery

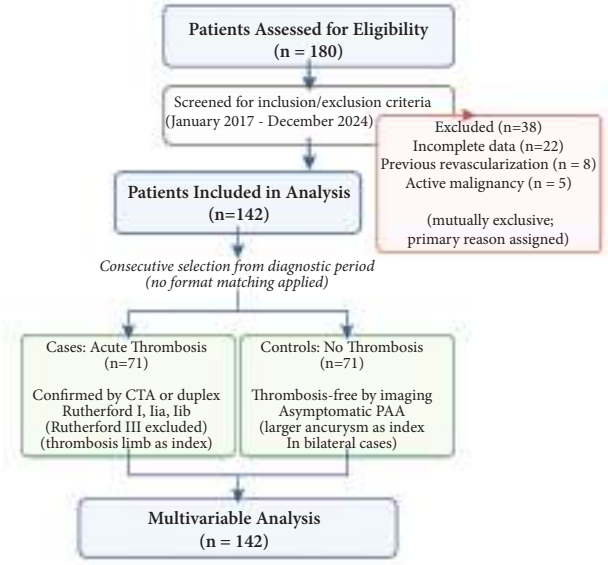

Figure 1. Patient selection flow diagram. Of 180 patients with popliteal artery aneurysm assessed for eligibility, 142 were included (71 cases with acute thrombosis, 71 asymptomatic controls confirmed to be thrombosis-free by imaging). Exclusion reasons are mutually exclusive (primary reason assigned). In bilateral cases, the thrombosed limb was analysed, and the larger aneurysm in controls. Cases included Rutherford categories I, IIa and IIb [19]; Rutherford III was excluded.

within the aneurysmal segment with clinical signs of acute limb ischaemia (Rutherford categories I, IIa and IIb) [19]. Patients with irreversible ischaemia (Rutherford III) were excluded. Controls (n=71) were patients with asymptomatic PAA diagnosed during the same period and confirmed to be thrombosis-free by imaging. Controls were selected consecutively from the institutional database at a 1:1 ratio without formal matching. In patients with bilateral PAAs, the thrombosed limb was analysed in cases, whereas the larger aneurysm was used as the index lesion in controls.

Variables analysed

Demographic data included age and sex. Clinical variables included smoking status (current or former smoker versus never smoker; categories combined owing to similar vascular risk profiles), diabetes mellitus, hypertension and dyslipidaemia. Radiological parameters were obtained from CTA (primary modality, n=118; 83%) or duplex ultrasonography (n=24; 17%). The distribution of imaging modalities was similar between groups (CTA: cases 82%, controls 85%). Parameters assessed included maximum outer-to-outer aneurysm diameter, presence of bilateral PAA, presence of mural thrombus in the aneurysm sac, and infrapopliteal arterial stenosis (defined as >50% stenosis in any tibial artery, assessed by diameter reduction on CTA or peak systolic velocity ratio >2.5 on duplex). Radiologists were blinded to laboratory results and the study hypothesis. Inter-observer agreement for mural thrombus detection was substantial ($\kappa = 0.78$).

Laboratory biomarkers included D-dimer (ng/mL, fibrinogen equivalent units, immunoturbidimetric assay), fibrinogen (g/L, Clauss method) and serum albumin (g/dL). Laboratory samples were obtained within 6 hours of emergency department presentation (cases) or at outpatient diagnostic workup (controls), prior to therapeutic anticoagulation or thrombolysis. This difference in sampling context is an inherent limitation of the case-control design and is addressed in the Discussion.

Statistical analysis

Distributional assumptions for continuous variables were assessed using visual inspection of histograms and Q-Q plots, supplemented by the Shapiro-Wilk test. Normally distributed variables were presented as mean ± standard deviation (SD) and compared using Student's t-test. Non-normally distributed variables were presented as medians with interquartile ranges (IQRs) and compared using the Mann-Whitney U test. Categorical variables were presented as absolute and relative frequencies and compared using the chi-square test; Fisher's exact test was used when expected cell counts were below 5. Univariable comparisons were considered exploratory and were interpreted primarily to describe between-group differences rather than to establish independent effects. Given this exploratory nature, no formal correction for multiple testing was applied, and univariable p-values are interpreted descriptively.

Continuous variables were dichotomised using clinically established cut-off values (age >65 years, aneurysm diameter >25 mm, D-dimer >1000 ng/mL, fibrinogen >4 g/L, albumin <3.5 g/dL) to facilitate clinical interpretability. This simplified interpretation may have reduced statistical power and obscured non-linear associations. The 25 mm diameter threshold was chosen to optimise separation in this dataset while retaining clinical relevance; guideline thresholds for elective repair (e.g. 20 mm) address a different clinical decision context.

A complete case analysis was performed. Multiple imputation was not used; the potential impact of missing data is discussed in the Limitations section. Candidate variables were entered into multivariable logistic regression based on clinical relevance. Multicollinearity was assessed using variance inflation factors (VIF); all values were <2.5. Results were reported as odds ratios (OR) with 95% confidence intervals (CI). To assess model discrimination, the area under the receiver operating characteristic curve (AUC) was calculated, with internal validation by bootstrap resampling (1,000 iterations) and 10-fold cross-validation. Model calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test, the calibration-in-the-large intercept, the calibration slope and the Brier score. Given the limited number of events relative to predictors (an events-per-variable ratio of approximately 8), the multivariable model should be considered exploratory and hypothesis-generating, despite internal resampling procedures. No formal a priori sample size calculation was performed; the number of candidate variables was constrained by the available number of cases. Statistical significance was set at $p<0.05$. Analyses were performed using R version 4.3.0 (R Foundation for Statistical Computing, Vienna, Austria) with packages 'rms' and 'pROC'.

Results

Demographic and clinical characteristics

Baseline demographic and clinical characteristics are presented in **Table 1**. Mean age in the acute thrombosis group was 67.4±9.2 years compared to 61.2±8.4 years in controls (p=0.002; mean difference 6.2 years, 95% CI 2.3–10.1). The proportion of patients older than 65 years was higher in the thrombosis

Table 1. Demographic and clinical characteristics of the study groups

Variable	Acute thrombosis (n=71)	Control (n=71)	p
Age (years, mean±SD)	67.4 ± 9.2	61.2 ± 8.4	0.002
Age >65 years, n (%)	46 (64.8)	31 (43.7)	0.012
Male sex, n (%)	63 (88.7)	58 (81.7)	0.271
Smoking*, n (%)	52 (73.2)	34 (47.9)	<0.001
Diabetes mellitus, n (%)	31 (43.7)	18 (25.4)	0.009
Hypertension, n (%)	45 (63.4)	39 (54.9)	0.291
Dyslipidaemia, n (%)	38 (53.5)	30 (42.3)	0.127

SD – standard deviation. *Smoking defined as current or former smoker versus never smoker; current and former smokers were combined due to similar vascular risk profiles

Table 2. Radiological characteristics of the study groups

Variable	Acute thrombosis (n=71)	Control (n=71)	p
Max. diameter* (mm, mean±SD)	29.3 ± 4.5	23.7 ± 3.9	<0.001
Diameter >25 mm, n (%)	54 (76.1)	28 (39.4)	<0.001
Bilateral PAA, n (%)	29 (40.8)	14 (19.7)	0.014
Mural thrombus, n (%)	48 (67.6)	21 (29.6)	<0.001
Infrapopliteal stenosis >50%†, n (%)	27 (38.0)	13 (18.3)	0.008

PAA – popliteal artery aneurysm; SD – standard deviation. *Maximum outer-to-outer diameter measured by CTA or duplex. †Defined as >50% stenosis in any tibial artery

group (64.8% vs 43.7%; p=0.012). Most patients were male (88.7% vs 81.7%; p=0.271). Smoking was more prevalent among cases (73.2% vs 47.9%; p<0.001), as was diabetes mellitus (43.7% vs 25.4%; p=0.009). Hypertension and dyslipidaemia did not differ significantly between groups.

Among 71 cases, Rutherford category distribution was: category I (n=17, 23.9%), category IIa (n=38, 53.5%), and category IIb (n=16, 22.5%). Median time from symptom onset to presentation was 18 hours (IQR 8–36).

Radiological characteristics

Radiological characteristics are presented in **Table 2.** Mean maximum aneurysm diameter was larger in the thrombosis group (29.3±4.5 mm vs 23.7±3.9 mm; p<0.001; mean difference 5.6 mm, 95% CI 4.2–7.0). Diameter above 25 mm was observed in 76.1% of cases versus 39.4% of controls (p<0.001). Bilateral PAA was present in 40.8% of cases compared to 19.7% of controls (p=0.014). Mural thrombus was strongly associated with acute thrombosis (67.6% vs 29.6%; p<0.001). Infrapopliteal stenosis >50% was more prevalent among cases (38.0% vs 18.3%; p=0.008).

Laboratory characteristics

Laboratory findings are presented in **Table 3.** Median D-dimer was higher in the thrombosis group (1842 ng/mL, IQR 876–3214) than in controls (724 ng/mL, IQR 412–1156; p<0.001). D-dimer above 1000 ng/mL was present in 59.1% of cases versus 26.8% of controls (p<0.001). Median fibrinogen was 4.2 g/L (IQR 3.4–5.1) in cases versus 3.5 g/L (IQR 2.9–4.2) in controls (p<0.001). Hypoalbuminaemia (albumin <3.5 g/dL) was more frequent in cases (40.8% vs 16.9%; p=0.004).

Table 3. Laboratory characteristics of the study groups

Variable	Acute thrombosis (n=71)	Control (n=71)	p
D-dimer (ng/mL FEU), median (IQR)	1842 (876–3214)	724 (412–1156)	<0.001
D-dimer >1000 ng/mL, n (%)	42 (59.1)	19 (26.8)	<0.001
Fibrinogen (g/L, Clauss), median (IQR)	4.2 (3.4–5.1)	3.5 (2.9–4.2)	<0.001
Fibrinogen >4 g/L, n (%)	33 (46.5)	17 (23.9)	0.002
Albumin (g/dL), median (IQR)	3.4 (3.1–3.8)	3.9 (3.6–4.2)	<0.001
Albumin <3.5 g/dL, n (%)	29 (40.8)	12 (16.9)	0.004

IQR – interquartile range; FEU – fibrinogen equivalent units. Laboratory samples: cases within 6 h of emergency presentation; controls at outpatient workup

Table 4. Multivariable logistic regression: features independently associated with acute PAA thrombosis at presentation

Variable	OR (95% CI)	β (SE)	VIF	p
Age >65 years	2.31 (1.24–4.32)	0.84 (0.31)	1.12	0.008
Smoking	2.58 (1.41–4.72)	0.95 (0.30)	1.18	0.002
Diabetes mellitus	2.14 (1.18–3.88)	0.76 (0.30)	1.08	0.012
Diameter >25 mm	3.21 (1.76–5.87)	1.17 (0.31)	1.24	<0.001
Mural thrombus	3.82 (2.04–7.14)	1.34 (0.32)	1.31	<0.001
Infrapopliteal stenosis >50%	2.42 (1.28–4.58)	0.88 (0.32)	1.15	0.006
D-dimer >1000 ng/mL	3.14 (1.68–5.87)	1.14 (0.32)	1.42	<0.001
Fibrinogen >4 g/L	2.48 (1.32–4.66)	0.91 (0.32)	1.38	0.005
Albumin <3.5 g/dL	2.76 (1.44–5.29)	1.02 (0.33)	1.21	0.002

OR – odds ratio; CI – confidence interval; β – regression coefficient; SE – standard error; VIF – variance inflation factor. Model intercept $\beta_0 = -4.21$ (verified for this dataset; identical to the logistic model fitted on the same 142-patient sample). Apparent AUC = 0.89 (95% CI 0.84–0.93); optimism-corrected AUC (1,000 bootstrap) = 0.87; optimism = 0.02. Hosmer-Lemeshow $\chi^2 = 9.4$, p = 0.21. Calibration-in-the-large intercept = −0.08; calibration slope = 0.91. Brier score = 0.14. Because of 1:1 case-control sampling, the intercept and absolute probabilities are sample-dependent and not transportable to populations with different thrombosis prevalence. The model is exploratory.

Multivariable analysis

Multivariable logistic regression results are presented in **Table 4.** Nine variables were independently associated with acute thrombosis at presentation. The strongest associations were observed for mural thrombus (OR 3.82; 95% CI 2.04–7.14; p<0.001), aneurysm diameter >25 mm (OR 3.21; 95% CI 1.76–5.87; p<0.001), and D-dimer >1000 ng/mL (OR 3.14; 95% CI 1.68–5.87; p<0.001). Smoking (OR 2.58), hypoalbuminaemia (OR 2.76), infrapopliteal stenosis >50% (OR 2.42), elevated fibrinogen (OR 2.48), diabetes (OR 2.14) and age >65 years (OR 2.31) were also independently associated. All VIF values were below 2.5.

D-dimer and fibrinogen showed moderate correlation (Spearman r = 0.41), as reflected in their VIF values of 1.42 and 1.38; both were retained, as they capture partially distinct aspects of the coagulation cascade, in which an imbalance between procoagulant and inhibitory components of haemostasis underlies the prothrombotic state [20].

Exploratory assessment of model discrimination yielded an apparent AUC of 0.89 (95% CI 0.84–0.93). Bootstrap internal validation (1,000 iterations) produced an optimism-corrected AUC of 0.87 (95% CI 0.82–0.91), and 10-fold cross-validation yielded a mean AUC of 0.86. Bootstrap correction revealed an optimism of 0.02 (apparent AUC 0.89 minus corrected AUC 0.87), which, although modest in absolute terms, should be interpreted in the context of a low events-per-variable ratio of approximately 8. The Hosmer-Lemeshow goodness-of-fit test was non-significant (χ^2=9.4; p=0.21). Calibration-in-the-large intercept was −0.08, and calibration slope was 0.91, suggesting adequate calibration within this case-control sample. The Brier score was 0.14.

Discussion

In this case-control study of 142 patients with PAA, we identified clinical, radiological and laboratory features associated with acute thrombosis at presentation. Multivariable analysis revealed nine independently associated variables, with mural thrombus, larger aneurysm diameter, and elevated D-dimer showing the strongest associations.

Our findings are consistent with previous reports linking aneurysm size and mural thrombus to PAA complications [8,9]. Martelli et al. found that superficial femoral artery occlusion and poor distal runoff were independently associated with PAA thrombosis [9]. We used infrapopliteal stenosis >50% as a simplified measure; this is not equivalent to formal runoff scores but captures clinically relevant distal disease. A recent multicentre study by Bellomo et al. suggested that per cent thrombus burden may be more informative than diameter alone for identifying high-risk PAAs [13]; this is consistent with our finding that mural thrombus carried the strongest association (OR 3.82), although whether thrombus quantity adds incremental value over its mere presence remains to be tested prospectively.

An additional aspect of this study is the inclusion of laboratory biomarkers measured at presentation, alongside clinical and imaging variables. The association between D-dimer and thrombotic events in peripheral arterial disease is well supported. Kremers et al., in a systematic review of over 21,000 patients, found that elevated D-dimer and fibrinogen were associated with increased mortality [10]. Vidula et al. reported that D-dimer levels were independently associated with near-term mortality in peripheral arterial disease [11]. However, these studies addressed general peripheral arterial disease populations rather

than PAA specifically; caution is warranted when extrapolating to acute PAA thrombosis.

Hypoalbuminaemia was independently associated with thrombosis in our study (OR 2.76), consistent with literature linking low albumin to adverse vascular outcomes [12,15]. Hypoalbuminaemia may contribute through reduced antithrombin-binding capacity, impaired nitric oxide transport, and heightened platelet aggregation, and may also reflect chronic nutritional depletion and systemic inflammation [16].

D-dimer, fibrinogen, and albumin should be interpreted primarily as contemporaneous markers associated with the acute thrombotic state at presentation, rather than antecedent predictors of future thrombosis in asymptomatic aneurysms. Laboratory samples in cases were obtained during the acute event, whereas controls were sampled during outpatient workup. This fundamental difference in sampling context means that the observed laboratory differences may partly reflect the acute-phase response to thrombosis itself (reverse causality) rather than pre-existing risk. Had pre-thrombotic laboratory values been available, the magnitude of the D-dimer and fibrinogen associations would likely be substantially attenuated, and the model's discrimination would decrease accordingly.

Several limitations warrant consideration. The comparison of clinically extreme phenotypes (acute thrombosis versus asymptomatic aneurysm) reflects spectrum bias inherent in the study design, inflating discrimination metrics and limiting applicability to intermediate clinical presentations. In real-world emergency triage, the diagnostic challenge often involves distinguishing acute PAA thrombosis from other causes of acute limb ischaemia or from symptomatic but non-thrombosed PAA. Controls were selected consecutively without age or sex matching; the 6.2-year age difference between groups may confound univariable comparisons, although age was included in the multivariable model. Because of the 1:1 case-control sampling scheme, the model intercept and implied absolute probabilities are sample-dependent and should not be transported directly to populations with different thrombosis prevalence.

The study is single-centre and has a relatively small sample size, yielding an events-per-variable ratio of approximately 8, which increases the risk of overfitting despite bootstrap correction. That all nine candidate variables reached conventional significance in a model with this EPV ratio warrants cautious interpretation; the possibility of unstable coefficient estimates cannot be excluded. Of 180 patients, 22 (12.2%) were excluded due to incomplete data, predominantly missing D-dimer (n=14) and albumin (n=11); multiple imputation was not performed, which may have introduced selection bias. Dichotomisation of continuous variables simplified interpretation but may have reduced statistical power and obscured non-linear associations. Radiological parameters were obtained by CTA (83%) or duplex ultrasonography (17%), introducing potential measurement heterogeneity; a sensitivity analysis restricted to the CTA subgroup would be informative but was not performed owing to sample size constraints. Finally, the 8-year study period implies potential evolution of diagnostic practices. The fibrinogen and D-dimer elevations we observed are consistent with earlier work linking these markers to arterial thrombotic events and to the severity of peripheral arterial disease [17,18]. Most cases in our cohort presented with advanced ischaemia (Rutherford categories IIa or IIb) [19], reflecting the emergent nature of acute popliteal artery aneurysm thrombosis. Because inherited and acquired thrombophilia contribute to arterial thrombotic events [20], the biomarker changes observed at presentation should be regarded as part of this acute thrombotic process rather than as isolated diagnostic signals.

Conclusion

In this single-centre case-control sample, acute popliteal artery aneurysm thrombosis was associated with older age, smoking, diabetes, larger aneurysm diameter, mural thrombus, and higher D-dimer and fibrinogen with lower serum albumin at presentation. Mural thrombus, aneurysm diameter above 25 mm, and D-dimer above 1000 ng/mL showed the strongest independent associations. The observed laboratory differences may support diagnostic recognition of acute thrombosis at presentation, but they should not be interpreted as validated prognostic markers for future thrombosis. These associations describe the presentation profile of acutely thrombosed popliteal artery aneurysm in this cohort but require prospective validation in independent cohorts before any diagnostic use.

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