

The Role of Large Artery Atherosclerosis and Predictive Risk Factors in Symptomatic and Asymptomatic Ischemic Stroke

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ABSTRACT

Introduction: Large-artery atherosclerosis (LAA) is common in acute ischemic stroke, before determinants of whether LAA presents symptomatically or remains silent are unclear. We aimed to identify risk factors distinguishing symptomatic from asymptomatic LAA.

Method: We retrospectively analyzed 411 consecutive patients (January 2020–January 2025) with acute ischemic stroke and imaging-confirmed $\geq 50\%$ stenosis/occlusion. Demographics, comorbidities; Hypertension (HT), Diabetes mellitus (DM), Hyperlipidemia (HL), Atrial fibrillation (AF), and fasting laboratories were recorded; triglyceride-to-high-density lipoprotein (TG/HDL) and triglyceride-to-glucose (TG/GI) indices were calculated. Vascular territory was classified (carotid, vertebrobasilar (VBS), carotid-vertebrobasilar system) via Doppler, brain computerised tomography (CT), and/or magnetic resonance imaging (MRI) angiography. Patients with $\geq 50\%$ stenosis or occlusion in extra or intracranial arteries were classified as having LAA. Those with neurological deficits attributable to LAA were considered symptomatic, while patients with unrelated stroke etiologies were classified as asymptomatic. Group comparisons used chi-square, Mann-Whitney U, and Kruskal–Wallis tests.

Results: The mean age was 70.4 ± 10.6 years; 38.2% were women. Territory involvement was 49.6% carotid, 21.2% vertebrobasilar, and 29.2% carotid-

vertebrobasilar system. In younger patients (40–60 years), AF was associated with VBS involvement ($p = 0.035$). In the oldest group (81–99 years), HL was associated with VBS/carotid-VBS atherosclerosis ($p = 0.003$). Overall, 285 patients were symptomatic and 126 were asymptomatic. Hyperlipidemia was associated with symptomatic LAA (OR = 1.64; $p = 0.023$), remaining significant in men. AF was more frequent among asymptomatic LAA in those aged 61–99, suggesting cardioembolism predominated the index event while LAA remained silent. TG/HDL was higher in symptomatic women and elevated across symptomatic patients aged 40–80 ($p = 0.010$; borderline at 61–80 ages, $p = 0.050$). TG/GI did not discriminate symptom status in any subgroup.

Conclusion: In elderly patients, hypertension; in men, hyperlipidemia; and in women and middle-to-older age groups, elevated TG/HDL are linked to symptomatic conversion of LAA. In the presence of AF, concomitant LAA often remains clinically silent. Tight control of modifiable risks, may delay or prevent symptomatic transition of LAA and is an alternative for patients ineligible for intervention.

Keywords: Asymptomatic, atherosclerosis, stroke, symptomatic

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INTRODUCTION

In the classification of ischemic cerebrovascular disease, large artery atherosclerosis (LAA) is defined as $\geq 50\%$ stenosis or occlusion of extracranial or intracranial arteries and accounts for approximately 16.6% of all ischemic strokes (1).

Symptomatic LAA is a consequence of systemic and progressive atherosclerosis, with traditional vascular risk factors playing a central etiological role. The term symptomatic denotes LAA that produces ipsilateral neurological deficits, whereas asymptomatic refers to the absence of neurological findings attributable to the atherosclerotic lesion within the past six months. While the prevalence of symptomatic LAA is well-documented in the general population, the true prevalence of asymptomatic LAA remains uncertain, largely because large-vessel imaging is not routinely performed (2).

Among hospitalized ischemic stroke patients, some present with symptomatic atherosclerosis, whereas in others, despite the presence of LAA, stroke etiology is unrelated, rendering the atherosclerosis

Highlights

- The incidence of asymptomatic atherosclerosis is uncertain.
- Atherosclerotic lesions do not invariably progress to a symptomatic stage.
- Hyperlipidemia and TG/HDL ratio are influential for symptomatic atherosclerosis.
- Strict control of lipids, TG/HDL ratio maybe alternative to vascular intervention.

asymptomatic. In a study, examining patients having recurrent thrombectomy in the last 10 years, demonstrated that 86.5% of them had cardioembolic stroke subtype (3). In another study, majority of patients underwent recurrent thrombectomy had cardioembolic stroke (66%), while 19% had large vessel atherosclerosis (4).

Identifying the factors that determine whether LAA manifests symptomatically or asymptotically is of clinical and prognostic importance. This study was therefore designed to investigate risk factors distinguishing symptomatic from asymptomatic LAA in acute ischemic stroke patients.

METHODS

Study Design and Sample

We retrospectively analyzed patients admitted to our neurology clinic between January 2020 and January 2025 with a diagnosis of acute ischemic stroke. Patients with LAA documented on brain imaging were included.

Demographic characteristics, comorbidities, and vascular risk factors [hypertension (HT), diabetes mellitus (DM), hyperlipidemia (HL), and atrial fibrillation (AF)] were recorded. Fasting blood glucose and lipid profiles obtained within the first three days of admission were reviewed, and the triglyceride/high-density lipoprotein (TG/HDL) and triglyceride/glucose (TG/GI) ratios were calculated.

Acute infarct features were documented on brain computerized tomography (CT) and magnetic resonance imaging (MRI) diffusion sequences, and arterial stenosis was assessed with carotid and vertebral Doppler ultrasonography, CT angiography, and/or MR angiography. Patients with $\geq 50\%$ stenosis or occlusion in extra or intracranial arteries were classified as having LAA. Those with neurological deficits attributable to LAA were considered symptomatic, while patients with unrelated stroke etiologies were classified as asymptomatic. Electrocardiography and transthoracic echocardiography were performed to further investigate the etiology of ischemic stroke.

Patients with intracerebral hemorrhage or rare stroke causes (e.g., venous thrombosis, arterial dissection) were excluded.

Statistical Analyses

All statistical analyses were performed using the the Statistical Package for the Social Sciences for Windows, Version 22.0. Kolmogorov-Smirnov measured the distribution of variables. Normal distribution variables are expressed as mean \pm standard deviation-the p-values obtained from the Mann-Whitney U-test and Kruskal Wallis tests. By displaying the percentages of the categorical variables and accounting for the expected value, the chi-square test p value is obtained. The statistical significance level was assumed to be $p < 0.05$.

This study was approved by our hospital's ethics committee, with the decision number 2025/06/10/062. The study was conducted in accordance with the Declaration of Helsinki.

RESULTS

A total of 411 patients diagnosed with acute ischemic cerebrovascular disease and imaging confirmed large artery atherosclerosis (LAA) were included in the study. Of these, 38.2% were female and 61.8% were male, with a mean age of 70.43 ± 10.57 years (range: 40–99).

Carotid artery atherosclerosis was identified in 204 patients (49.6%), vertebrobasilar system (VBS) atherosclerosis in 87 patients, and combined anterior-posterior circulation atherosclerosis in 120 patients. The distribution of comorbidities across carotid, vertebrobasilar, and combined anterior-posterior circulation involvement is presented in Figure 1. No statistically significant associations were found between atrial fibrillation, hypertension, diabetes mellitus, hyperlipidemia, and the vascular territory affected across these groups.

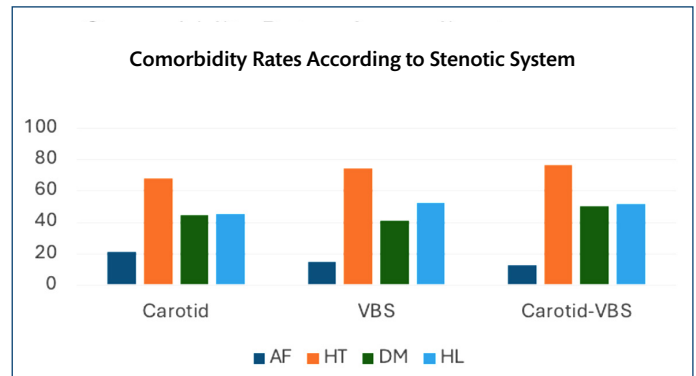


Figure 1. Comorbidity Rates According to Stenotic Region.

The chart shows the prevalence of atrial fibrillation (AF), hypertension (HT), diabetes mellitus (DM), and hyperlipidemia (HL) across different stenotic systems (carotid, vertebrobasilar system (VBS) and carotid-VBS).

When stratified by age, a significant association emerged between AF and vascular involvement in the younger age group (40–60 years). Specifically, VBS atherosclerosis was significantly more frequent among younger patients with AF ($p = 0.035$). A relationship between HL and vascular territory was only observed in the oldest age group (81–99 years), where patients with HL had significantly higher rates of VBS and combined carotid-VBS atherosclerosis ($p = 0.003$). No significant associations were observed between HT or DM and vascular involvement in any age group (Table 1).

Of the total cohort, 285 patients were symptomatic and 126 were asymptomatic. Within the asymptomatic group, 62 patients had small vessel disease, 25 had left ventricular systolic dysfunction on echocardiography, 35 had AF, 2 had cancer-related etiologies (bladder and colon adenocarcinoma), 1 had vasculitis, and 1 had patent foramen ovale (PFO) with an intracardiac mass. There was no significant difference in gender between the symptomatic and asymptomatic groups.

The distribution of comorbidities and their relationship to symptomatic versus asymptomatic LAA is shown in Table 2. The presence of AF and HL was significantly associated with symptomatic LAA. Hyperlipidemia was present in 48.9% of patients overall and was significantly more frequent in symptomatic cases ($p = 0.023$), with a stronger association observed in older patients ($p = 0.017$)(Table 2).

Gender-stratified analysis revealed no significant association in women, whereas HL was significantly more prevalent among men with symptomatic LAA (Table 3).

AF was detected in 71 patients. It was present in 12.6% of symptomatic LAA cases. While AF prevalence did not differ between symptomatic and asymptomatic patients in the younger age group, AF was significantly more common in asymptomatic LAA patients in the middle-aged and elderly groups. By contrast, HT prevalence was significantly higher only among symptomatic elderly patients (Table 4).

In female patients, the TG/HDL ratio was found to be significantly higher in the symptomatic BAA group compared to the asymptomatic group (Table 5).

Regarding metabolic indices, in the 40–60 age group, the TG/HDL ratio was significantly elevated in symptomatic patients, irrespective of sex ($p = 0.01$). In the 61–80 age group, this association reached borderline significance ($p = 0.050$). The TG/GI ratio showed no significant differences across age or gender categories (Table 6).

Table 1. Distribution of Risk Factors by Age and Stenotic System

Risk Factor	40-60 years			61-80 years			81-99 years			p-value
	Carotid	VBS	Carotid-VBS	Carotid	VBS	Carotid-VBS	Carotid	VBS	Carotid-VBS	
AF	0/28	4/26	0/13	25/134	6/44	9/79	18/42	3/17	6/28	0.035
HT	17/28	17/26	8/13	88/134	33/44	59/79	34/42	15/17	25/28	>0.05
DM	15/28	12/26	7/13	63/134	21/44	44/79	14/42	3/17	11/28	>0.05
HL	16/28	16/26	7/13	66/134	19/44	38/79	11/42	11/17	17/28	0.003

AF:Atrial fibrillation; DM: Diabetes mellitus; HL: Hyperlipidemia; HT: Hypertension; VBS: Vertebrobasilar system.

DISCUSSION

Large artery atherosclerosis (LAA) accounts for approximately 15% of all ischemic strokes. According to the 2020 *Lancet Global Health* review, the prevalence of carotid atherosclerosis is 1.5%, and this prevalence increases with advancing age and male sex (5). Vertebrobasilar artery strokes comprise only about 1% of all strokes, while VBS atherosclerosis accounts for up to 27% of posterior circulation strokes (6–8).

The major modifiable risk factors for atherosclerotic stroke include hypertension, diabetes mellitus, and hyperlipidemia. Atherosclerosis begins early in life with the accumulation of foam cells in the arterial intima, progressing over time to plaque formation, with dyslipidemia acting as a key trigger (9). Hypertension and diabetes also contribute to endothelial dysfunction, accelerating atherosclerosis. Accordingly, most patients with LAA present with one or more of these risk factors. Notably,

Table 2. Association of Comorbidities Between Symptomatic and Asymptomatic Artery Atherosclerosis (BAA)

Comorbidity	Symptomatic BAA (n,%)	Asymptomatic BAA (n,%)	Pearson X ²	p-value	OR	% 95 CI
AF	36/285 (12.6%)	35/126 (27.8%)	14.026	<0.001	2.66	1.58-4.49
HT	206/285 (72.3%)	90/126 (71.4%)	0.031	0.859	0	0.60-1.55
DM	129/285 (45.3%)	61/126 (48.4%)	0.349	0.555	0	0.58-1.37
HL	150/285 (52.6%)	51/126 (40.5%)	5.166	<0.023	1.64	1.06

AF: Atrial fibrillation; CI: Confidence interval; DM: Diabetes mellitus; HL: Hyperlipidemia; HT: Hypertension; OR: Odds ratio.

Table 3. Distribution of Comorbidities in Symptomatic and Asymptomatic Artery Atherosclerosis (BAA) by Sex

Comorbidity	Female		Male		p-value Female /Male
	Symptomatic BAA (n,%)	Asymptomatic BAA (n,%)	Symptomatic BAA (n,%)	Asymptomatic BAA (n,%)	
AF (+)	17/107 (15.9%)	16/50 (32%)	19/178 (10.7%)	19/76 (25%)	0.021/0.003
AF (-)	90/107 (84.1%)	34/50 (68%)	159/178 (89.3%)	57/76 (75%)	
HT (+)	83/107 (77.6%)	40/50 (80%)	123/178 (69.1%)	50/76 (65.8%)	0.731/0.604
HT (-)	24/107 (22.4%)	10/50 (20%)	55/178 (30.9%)	26/76 (34.2%)	
DM (+)	62/107 (57.9%)	28/50 (56%)	67/178 (37.6%)	33/76 (43.4%)	0.819/0.388
DM (-)	45/107 (42.1%)	22/50 (44%)	111/178 (62.4%)	43/76 (56.6%)	
HL (+)	64/107 (59.8%)	26/50 (52%)	86/178 (48.3%)	25/76 (32.9%)	0.356/0.023
HL (-)	43/107 (40.2%)	24/50 (48%)	92/178 (51.7%)	51/76 (67.1%)	

AF: Atrial fibrillation; DM:Diabetes mellitus; HL:Hyperlipidemia; HT: Hypertension. p-values are reported separately for females and males.

Table 4. Distribution of Comorbidities in Symptomatic and Asymptomatic Artery Atherosclerosis (BAA) by Age Groups

Comorbidities	40-60 years		61-80 years		81-99 years		p-value
	Symptomatic BAA (n)	Asymptomatic BAA (n)	Symptomatic BAA (n)	Asymptomatic BAA (n)	Symptomatic BAA (n)	Asymptomatic BAA (n)	
AF (+)	2/54	2/13	21/171	19/86	13/60	14/27	0.111/0.041/0.005
AF (-)	52/54	11/13	150/171	67/86	47/60	13/27	
HT (+)	33/54	9/13	118/171	62/86	55/60	19/27	0.587/0.610/0.010
HT (-)	21/54	4/13	53/171	24/86	5/60	8/27	
DM (+)	27/54	7/13	84/171	44/86	18/60	10/27	0.803/0.758/0.516
DM (-)	27/54	6/13	87/171	42/86	42/60	17/27	
HL (+)	34/54	5/13	84/171	39/86	32/60	7/27	0.108/0.568/0.017
HL (-)	20/54	8/13	87/171	47/86	28/60	20/27	

AF: Atrial fibrillation; HT: Hypertension; DM: Diabetes mellitus; HL:Hyperlipidemia p-values are presented for the comparisons within each age group (40–60, 61–80, 81–99 years, respectively).

Table 5. Comparison of TG/HDL and TG/Glucose Ratios Between Symptomatic and Asymptomatic Artery Atherosclerosis (BAA) According to Sex

Parameter	Female		Male		p-value
	Symptomatic BAA (mean rank)	Asymptomatic BAA (mean rank)	Symptomatic BAA (mean rank)	Asymptomatic BAA (mean rank)	Female/Male
TG/HDL	85.53	65.03	132.28	116.32	0.008/0.113
TG/Glucose	82.78	70.91	130.60	120.24	0.127/0.303

TG/Glucose: Triglyceride-to-glucose ratio; TG/HDL: Triglyceride-to-high-density lipoprotein cholesterol ratio, p-values are presented separately for females and males.

Table 6. TG/HDL and TG/Glucose Ratios in Symptomatic and Asymptomatic Basilar Atherosclerosis (BAA) Across Age Groups

Parameter	40-60 years		61-80 years		81-99 years		p-value
	Symptomatic BAA (mean rank)	Asymptomatic BAA (mean rank)	Symptomatic BAA (mean rank)	Asymptomatic BAA (mean rank)	Symptomatic BAA (mean rank)	Asymptomatic BAA (mean rank)	40-60/61-80/81-99 years
TG/HDL	37.00	21.54	135.43	116.21	44.15	43.67	0.010/0.050/0.934
TG/Glucose	35.94	25.92	132.81	121.42	44.83	42.15	0.096/0.247/0.646

TG/Glucose: Triglyceride-to-glucose ratio; TG/HDL: Triglyceride-to-high-density lipoprotein cholesterol ratio, p-values are given separately for each group (40-60,61-80,81-99 years)

these same risk factors also contribute to small vessel disease (SVD). However, while some patients develop LAA, others manifest only lacunar syndromes. The mechanisms underlying SVD-related lacunar infarction differ, involving impaired cerebral blood flow, inadequate autoregulation, and increased blood-brain barrier permeability, although the molecular pathways remain incompletely understood (10).

Clinical imaging of acute ischemic stroke patients frequently reveals lacunar syndromes or cardioembolic strokes associated with cardiac pathologies (e.g., AF, left ventricular dysfunction, prosthetic valves, patent foramen ovale), often coexisting with asymptomatic LAA. Understanding why some LAA lesions remain asymptomatic while others become symptomatic has important prognostic implications.

In our study, hypertension was significantly associated with symptomatic LAA, particularly in the oldest age group (81–99 years, $p = 0.01$). Thus, hypertension may contribute to the symptomatic manifestation of LAA in elderly patients.

Similarly, lipid abnormalities play a crucial role in the progression of atherosclerosis. Lipid dysfunction decreases vascular compliance and increases susceptibility to LAA. In our analysis, hyperlipidemia emerged as a significant predictor of symptomatic LAA, especially among men and older individuals.

Elevated plasma triglycerides and reduced HDL cholesterol are well-established risk factors for coronary artery atherosclerosis (11,12). The TG/HDL ratio has been recognized as an independent risk factor for coronary artery disease and a practical marker for atherosclerosis (13). Elevated triglycerides promote oxidative stress and inflammation (14,15), whereas HDL exerts anti-atherogenic effects and is inversely associated with both cerebrovascular and cardiovascular disease. Accordingly, elevated TG/HDL ratios may also predict the progression from asymptomatic to symptomatic LAA. In our study, higher TG/HDL ratios were significantly associated with symptomatic LAA in women and in both sexes aged 40–80 years.

Taken together, our findings suggest that hypertension in older age, hyperlipidemia in men, elevated TG/HDL ratios in women, and elevated

TG/HDL ratios in middle-aged and elderly patients (61–99 years old), irrespective of sex, are potential determinants of symptomatic conversion in LAA.

Although hyperlipidemia appears particularly relevant, diabetes mellitus also represents a critical cerebrovascular risk factor. Zhao et al. reported that elevated TG/GI ratios increase vascular stiffness (16), and subsequent studies have confirmed associations between TG/GI and atherosclerosis (17,18). Li et al. further demonstrated that TG/GI levels were positively associated with coronary lesions and carotid plaques in symptomatic coronary artery disease, though their analysis was limited to asymptomatic carotid plaques (19). In our study, elevated TG/GI did not distinguish between symptomatic and asymptomatic LAA. Prior literature has more consistently linked TG/GI to coronary artery disease, diabetic microvascular complications, and post-stroke prognosis rather than to initial stroke mechanisms (20–23). Indeed, higher TG/GI values have been associated with recurrent stroke, mortality, and poorer three-month functional outcomes following ischemic stroke. These findings suggest that the TG/GI index may serve better as a prognostic rather than a diagnostic marker in stroke patients.

In our cohort, atrial fibrillation was identified in 71 patients, 36 of whom had symptomatic LAA. AF was significantly associated with asymptomatic LAA in middle-aged and older patients (61–99 years). Thus, in the presence of AF, cardioembolism appears to be the dominant stroke mechanism, with concurrent LAA often remaining clinically silent. As expected, AF was strongly linked to cardioembolic stroke. However, clinicians should remain mindful that LAA can coexist silently in AF patients, who nonetheless remain at elevated risk of recurrent cerebrovascular events. AF prevalence increases with age (24) consistent with our finding that 27 patients in the oldest group (81–99 years) had AF.

When analyzed by vascular territories, carotid system involvement was more common among asymptomatic patients (61.9%), whereas combined carotid-VBS atherosclerosis was more frequent among symptomatic patients (33.7%). This pattern likely reflects the multi-vessel nature of progressive atherosclerosis, which often extends to multiple vascular territories through a chronic inflammatory process. Conversely, asymptomatic patients frequently developed acute infarctions through

alternative mechanisms, despite the coexistence of LAA. For instance, lacunar infarction due to SVD was observed in 62 of the 126 asymptomatic patients. Among patients with AF, only one demonstrated multi-vessel involvement, and among those with left ventricular dysfunction, multi-territory infarction was uncommon.

In summary, patients with LAA on vascular imaging may present with acute ischemic stroke due to diverse etiologies, in which case LAA remains clinically silent. Hypertension, hyperlipidemia, and elevated TG/HDL ratios emerged as significant contributors to symptomatic LAA in specific patient subgroups. Regular monitoring and rigorous control of these modifiable risk factors may help delay or prevent the transition from asymptomatic to symptomatic LAA, particularly in elderly populations where invasive therapeutic options are limited.

Ethics Committee Approval: Approval for this study was obtained from the ethics committee of Bağcılar Training and Research Hospital with decision number 2025/06/10/062.

Hakem Değerlendirmesi: Dış bağımsız.

Author Contributions: Concept - EÇ; Design - EÇ; Supervision - NKI; Resources - EÇ; Materials - EK, FK; Data Collection and/or Processing - EÇ, RK; Analysis and/or Interpretation - EÇ; Literature Search - EÇ; Writing - EÇ; Critical Review - NKI.

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