

Original

Prevalence of Atrial Cardiopathy Biomarkers Across Ischemic Stroke Subtypes

Yuji Kato¹, Shintaro Nakano², Takeshi Hayashi¹, Shunsuke Kido¹,
Kiichiro Oryu¹, Noriko Arai¹, Toru Nakagami¹, Ichiro Deguchi¹,
Shinichi Takahashi¹, Shinya Kohyama³, Satoshi Suda^{1,4} and Junya Aoki¹

¹Department of Neurology and Cerebrovascular Medicine, Saitama Medical University International Medical Center, Saitama, Japan

²Department of Cardiology, Saitama Medical University International Medical Center, Saitama, Japan

³Department of Endovascular Neurosurgery, Saitama Medical University International Medical Center, Saitama, Japan

⁴Department of Neurology, Nippon Medical School, Tokyo, Japan

Background: Atrial cardiopathy (AC) has been proposed as a potential embolic mechanism in embolic stroke of undetermined source (ESUS); however, the distribution of AC biomarkers across ischemic stroke subtypes remains unclear.

Methods: We retrospectively analyzed 533 consecutive patients with acute ischemic stroke. Stroke subtypes were classified as small-vessel occlusion (SVO), branch atheromatous disease (BAD), large-artery atherosclerosis (LAA), ESUS, or cardioembolism (CE). AC was defined according to ARCADIA trial criteria as the presence of at least one of the following: P-wave terminal force in lead V1 (PTFV1) $>5,000 \mu\text{V} \cdot \text{ms}$, N-terminal pro-brain natriuretic peptide (NT-proBNP) $>250 \text{ pg/mL}$, or left atrial diameter index (LADI) $\geq 3 \text{ cm/m}^2$. We evaluated the prevalence and overlap of these biomarkers across subtypes.

Results: AC biomarkers were significantly more frequent in CE than in other subtypes. Using ≥ 1 biomarker, AC prevalence was 43% in SVO, 49% in BAD, 53% in LAA, 54% in ESUS, and 97% in CE. With ≥ 2 biomarkers, prevalence decreased to 8%, 7%, 13%, 19%, and 78%, respectively. NT-proBNP elevation was the most common biomarker across all groups. Left atrial enlargement was rare in non-CE subtypes ($\sim 5\%$) and showed no overlap with PTFV1 in SVO, BAD, or LAA.

Conclusions: While AC defined by ARCADIA criteria is highly prevalent in CE, it is also widely observed across other stroke subtypes. Refinement of AC definitions, including stricter thresholds and/or biomarker combinations, may better identify atrial dysfunction with causal relevance to stroke.

(J Nippon Med Sch 2026; 93 (5): 394–401. https://doi.org/10.1272/jnms.JNMS.2026_93-513)

Keywords: P-wave terminal force in lead V1, N-terminal pro-brain natriuretic peptide, left atrial diameter index, embolic stroke of undetermined source

Introduction

Ischemic stroke is classified into several etiological subtypes, including small-vessel occlusion (SVO), large-artery atherosclerosis (LAA), and cardioembolic stroke (CE)¹. Among these, CE is typically associated with atrial fibrillation (AF) and other cardiac disorders that serve as embolic sources.

In recent years, the concept of atrial cardiopathy (AC)

has emerged as a potential mechanism of thromboembolism independent of atrial fibrillation². Structural, electrical, and functional abnormalities of the atrium may promote thrombus formation and increase the risk of ischemic stroke². A number of biomarkers have been proposed to identify AC, including elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP), abnormal P-wave terminal force in lead V1 (PTFV1), and enlargement

Correspondence to Yuji Kato, yujik@saitama-med.ac.jp

https://doi.org/10.1272/jnms.JNMS.2026_93-513

Received: April 13, 2026; Accepted: July 22, 2026

Copyright © 2026 The Medical Association of Nippon Medical School. This is an open access article under the CC BY-NC-ND 4.0 license (<https://creativecommons.org/licenses/by-nc-nd/4.0/>).

of the left atrium³⁻⁶. These markers are thought to reflect atrial remodeling and hemodynamic stress that may contribute to thromboembolic events³⁻⁶. Previous studies have suggested that AC may be involved in the pathogenesis of embolic stroke, particularly in patients with cryptogenic stroke or embolic stroke of undetermined source (ESUS)^{2,6,7}.

However, the clinical significance of AC in terms of treatment strategies remains uncertain. The Atrial Cardiopathy and Antithrombotic Drugs in Prevention After Cryptogenic Stroke (ARCADIA) trial failed to demonstrate the superiority of apixaban over aspirin in patients with ESUS and AC, raising questions regarding the specificity and clinical relevance of currently used AC criteria⁸.

Therefore, the present study aimed to investigate the prevalence of AC biomarkers across ischemic stroke subtypes, including SVO, branch atheromatous disease (BAD), LAA, ESUS, and CE in order to better clarify the distribution and potential clinical implications of AC biomarkers in patients with acute ischemic stroke.

Materials and Methods

Study Population and Setting

In this hospital-based cross-sectional analytic study, we retrospectively reviewed clinical data of patients admitted to Saitama Medical University International Medical Center with acute ischemic stroke (AIS) between June 2023 and May 2024. AIS was defined as presentation within 7 days of the onset of symptoms. Data were obtained from electronic medical charts and summaries. We collected demographic and clinical patient characteristics including age, sex, body weight, body mass index, underlying risk factors for stroke (history of hypertension, diabetes mellitus, dyslipidemia, AF, current smoking, prior stroke, coronary artery disease, and congestive heart failure), electrocardiogram and echocardiographic findings, laboratory data on admission (NT-proBNP levels), and use of antithrombotic (antiplatelet and/or anticoagulant) agents before the index stroke. AF included paroxysmal, persistent, and permanent AF, which was either previously known or diagnosed during index hospitalization.

Etiologic subtypes of ischemic stroke were classified according to the TOAST (Trial of Org 10,172 in Acute Stroke Treatment) criteria¹. From the patients with other determined etiology, we selected those with BAD based on the following criteria: (1) a single, isolated deep infarct; (2) infarctions visible on ≥ 3 axial magnetic resonance imaging (MRI) slices (slice thickness, 5 mm) within

the deep perforator territories (e.g. the lenticulostriate artery) or extending from the basal surface of the pons within the paramedian pontine artery territory; (3) the absence of LAA and no evidence of CE^{9,10}. Unlike SVO resulting from lipohyalinotic degeneration, BAD was attributed to atherosclerosis¹¹.

From the patients with undetermined etiology, we selected those who met the diagnostic criteria for ESUS: (1) detection of a non-lacunar infarct on brain computed tomography/MRI; (2) exclusion of $\geq 50\%$ atherosclerotic stenosis proximal to the infarct with any imaging modality; (3) exclusion of a major-risk cardioembolic source with echocardiography and cardiac monitoring for ≥ 24 h; and (4) no other specific causes¹².

Atrial Cardiopathy Assessment

The biomarkers of AC, including a 12-lead electrocardiogram and NT-proBNP levels, were routinely assessed within 24h of admission. Transthoracic echocardiography was performed within 1 week of admission, and left atrial diameter index (LADI) and left ventricular ejection fraction (LVEF) were also assessed. We manually calculated PTFV1 on electrocardiogram traces by multiplying the duration (ms) of the terminal negative component of P-wave by its amplitude (μV) in lead V1. For patients with AF, the PTFV1 could not be calculated and was considered to be $>5,000 \mu\text{V} \cdot \text{ms}$. Left atrium size was calculated as the maximal antero-posterior diameter of the left atrium in systole on B-mode transthoracic echocardiogram. AC was defined according to criteria used in the ARCADIA trial as the presence of one or more of the following: $\text{PTFV1} > 5,000 \mu\text{V} \cdot \text{ms}$, serum NT-proBNP $> 250 \text{ pg/mL}$, or $\text{LADI} \geq 3 \text{ cm/m}^2$ ⁷. The prevalence and overlap of AC biomarkers were compared across stroke subtypes.

Statistical Analysis

Continuous variables are presented as means (standard deviation). Categorical variables are expressed as frequencies with proportions. One-way analysis of variance for continuous variables and the Pearson χ^2 test or Fisher's exact test, as appropriate, for categorical variables were used to compare differences among the five patient groups (patients with SVO, BAD, LAA, ESUS, and CE). For each variable, post-hoc Bonferroni correction was applied only when the global comparison yielded a $p\text{-value} < 0.05$. All statistical analyses were performed with EZR (Saitama Medical Center, Jichi Medical University, Saitama, Japan), a graphical user interface for

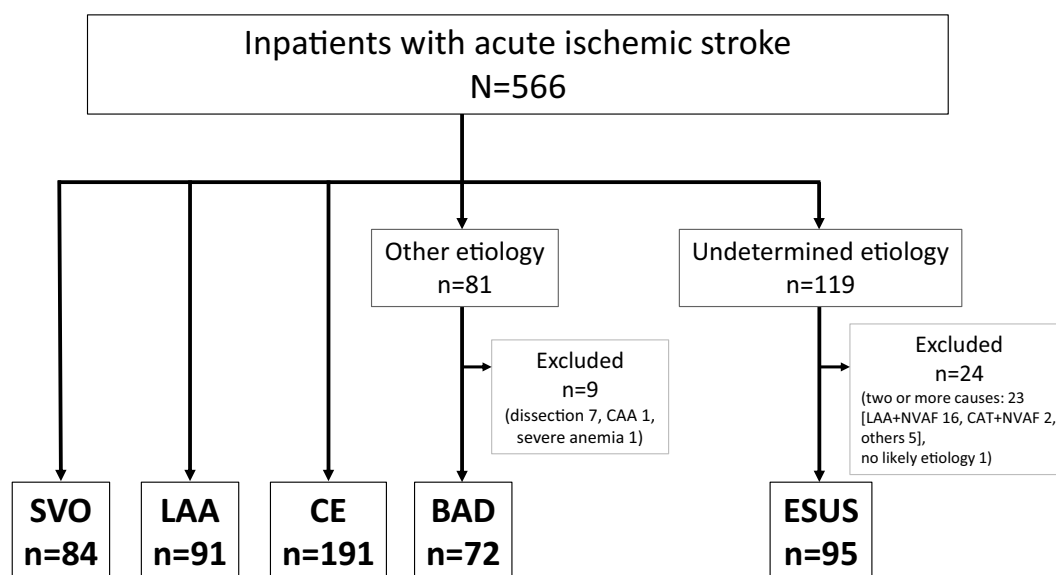


Figure 1 Flow chart of study population

SVO: small-vessel occlusion, BAD: branch atheromatous disease, LAA: large artery atherosclerosis, ESUS: embolic stroke of undetermined source, CE: cardioembolism, CAA: cerebral amyloid angiopathy, CAT: cancer-associated thrombosis, NVAF: non-valvular atrial fibrillation.

R (The R Foundation for Statistical Computing, Vienna, Austria)¹³. Statistical significance was set at $p < 0.05$ in the global comparison and at $p < 0.005$ in the post-hoc analysis.

Ethical Approval

This retrospective study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Institutional Review Board of Saitama Medical University International Medical Center (approval number: 2024-043). Patients were not required to give informed consent to participate in the study because the analysis used anonymized clinical data that were obtained after each patient had provided written consent for treatment. Furthermore, we applied an opt-out method to obtain consent for this study through an announcement on our hospital website. The announcement was approved by the institutional review board.

Results

Of the 566 inpatients diagnosed with AIS during the study period, 33 patients with undetermined etiology who did not meet the ESUS criteria, multiple potential causes, or etiology other than SVO, BAD, LAA, ESUS, or CE were excluded. Thus, 533 patients were included in the study (mean age 75.3 years, 41% women). The patient population included 84 (16%) patients with SVO, 91 (17%) patients with LAA, and 191 (36%) patients with CE, based on TOAST classification. The remaining 201

patients had other or undetermined etiology, including 72 (13%) patients with BAD and 95 (18%) patients with ESUS (Figure 1).

The background characteristics of the five patient groups are shown in Table 1. The patient age and prevalence of hypertension, diabetes, dyslipidemia, atrial fibrillation, current smoking, coronary artery disease, congestive heart failure, and oral anticoagulant administration were different across groups, while those of prior stroke and antiplatelet drug administration were similar among groups. Underlying conditions associated with CE included AF ($n=177$), akinetic/hypokinetic left ventricular segment ($n=9$), dilated cardiomyopathy ($n=2$), mechanical prosthetic valve ($n=2$), and recent myocardial infarction ($n=1$).

All three AC biomarkers were significantly higher in the CE group than in the other stroke subtypes (Figure 2). Although 177 of the 191 patients in the CE group had a history of AF, PTFV1 could only be measured directly in the 52 patients who were in sinus rhythm at the time of electrocardiography. Although not statistically significant, the mean NT-proBNP levels tended to be higher in the ESUS group than in the SVO, BAD, and LAA groups. LVEF also differed among stroke subtypes and was significantly lower in the CE group than in the other subtypes (Figure 2).

As shown in Figure 3, the prevalence of AC varied among the groups: SVO 43%, BAD 49%, LAA 53%, ESUS 54%, and CE 97%. Among the 186 CE patients with AC,

Table 1 Background characteristics of patients

Characteristics	SVO n = 84	BAD n = 72	LAA n = 91	ESUS n = 95	CE n = 191	Global p-value	*Significant Pairwise p-value
Age, years	73.4 ± 9.4	74.2 ± 11.2	74.4 ± 9.9	71.1 ± 15.4	80.7 ± 9.3	<0.001	SVO vs. CE: <0.001 BAD vs. CE: <0.001 LAA vs. CE: <0.001 ESUS vs. CE: <0.001
Female sex, n (%)	32 (38)	31 (43)	33 (36)	44 (46)	78 (41)	0.667	NA
Body weight (kg)	57.8 ± 10.5	60.2 ± 12.3	61.9 ± 13.9	58.2 ± 11.6	59.0 ± 12.0	0.140	NA
BMI (kg/m ²)	22.5 ± 3.0	23.4 ± 4.0	23.6 ± 4.0	22.6 ± 3.6	23.0 ± 3.6	0.218	NA
Risk factors							
Hypertension, n (%)	65 (77)	52 (72)	69 (76)	61 (64)	157 (82)	0.019	ESUS vs. CE: 0.001
Diabetes mellitus, n (%)	25 (30)	16 (22)	43 (47)	15 (16)	44 (23)	<0.001	LAA vs. ESUS: <0.001 LAA vs. CE: <0.001 BAD vs. LAA: 0.001
Dyslipidemia, n (%)	44 (52)	39 (54)	53 (58)	48 (51)	73 (38)	0.011	LAA vs. CE: 0.002
Atrial fibrillation, n (%)	0 (0)	0 (0)	0 (0)	0 (0)	177 (93)	<0.001	SVO vs. CE: <0.001 BAD vs. CE: <0.001 LAA vs. CE: <0.001 ESUS vs. CE: <0.001
Current smoking, n (%)	13 (15)	11 (15)	18 (20)	10 (11)	4 (2)	<0.001	SVO vs. CE: <0.001 BAD vs. CE: <0.001 LAA vs. CE: <0.001 ESUS vs. CE: 0.003
Prior stroke, n (%)	23 (27)	14 (19)	17 (19)	23 (24)	46 (24)	0.630	NA
Coronary artery disease, n (%)	5 (6)	2 (3)	6 (7)	3 (3)	23 (12)	0.024	NS
Congestive heart failure, n (%)	0 (0)	1 (1)	6 (7)	4 (4)	48 (25)	<0.001	SVO vs. CE: <0.001 BAD vs. CE: <0.001 LAA vs. CE: 0.004 ESUS vs. CE: <0.001
Antithrombotic drug							
Antiplatelet drugs, n (%)	22 (26)	14 (19)	21 (23)	22 (23)	43 (23)	0.906	NA
Anticoagulant drugs, n (%)	1 (1)	1 (1)	1 (1)	3 (3)	58 (30)	<0.001	SVO vs. CE: <0.001
DOAC, n (%)	1 (1)	1 (1)	1 (1)	3 (3)	46 (24)	<0.001	BAD vs. CE: <0.001 LAA vs. CE: <0.001 ESUS vs. CE: <0.001
Warfarin, n (%)	0 (0)	0 (0)	0 (0)	0 (0)	12 (6)	<0.001	NS

* For each variable, post-hoc Bonferroni correction was applied only when the global comparison yielded a p-value <0.05. A p-value <0.005 was considered statistically significant in the post-hoc analysis.

SVO: small-vessel occlusion, BAD: branch atheromatous disease, LAA: large artery atherosclerosis, CE: cardioembolism, BMI: body mass index, DOAC: direct oral anticoagulant, NA: not analyzed.

only eight fulfilled the AC criteria solely because PTFV1 was considered positive in the presence of AF without meeting either of the other biomarker criteria. Using a diagnostic threshold of at least two positive criteria, the prevalence of AC in the different patient groups was SVO 8%, BAD 7%, LAA 13%, ESUS 19%, and CE 78%. In both analyses, the prevalence of AC was significantly higher in CE than in the other stroke subtypes, with no statistically significant differences among the non-CE subtypes.

Finally, the overlap in AC biomarker positivity across stroke subtypes is shown in **Figure 4**. NT-proBNP eleva-

tion was the most frequently positive biomarker across all subtypes, followed by PTFV1 and LADI. There was more overlap of positive criteria in the ESUS and CE groups than in the SVO and BAD groups. LADI was observed in only 5% of non-CE subtypes (6% in SVO, 3% in BAD, 2% in LAA, and 9% in ESUS) compared with CE (27%; $p < 0.001$), and did not overlap with PTFV1 in SVO, BAD, or LAA subtypes.

Discussion

This study comprehensively evaluated the prevalence of three biomarkers of AC across different ischemic stroke

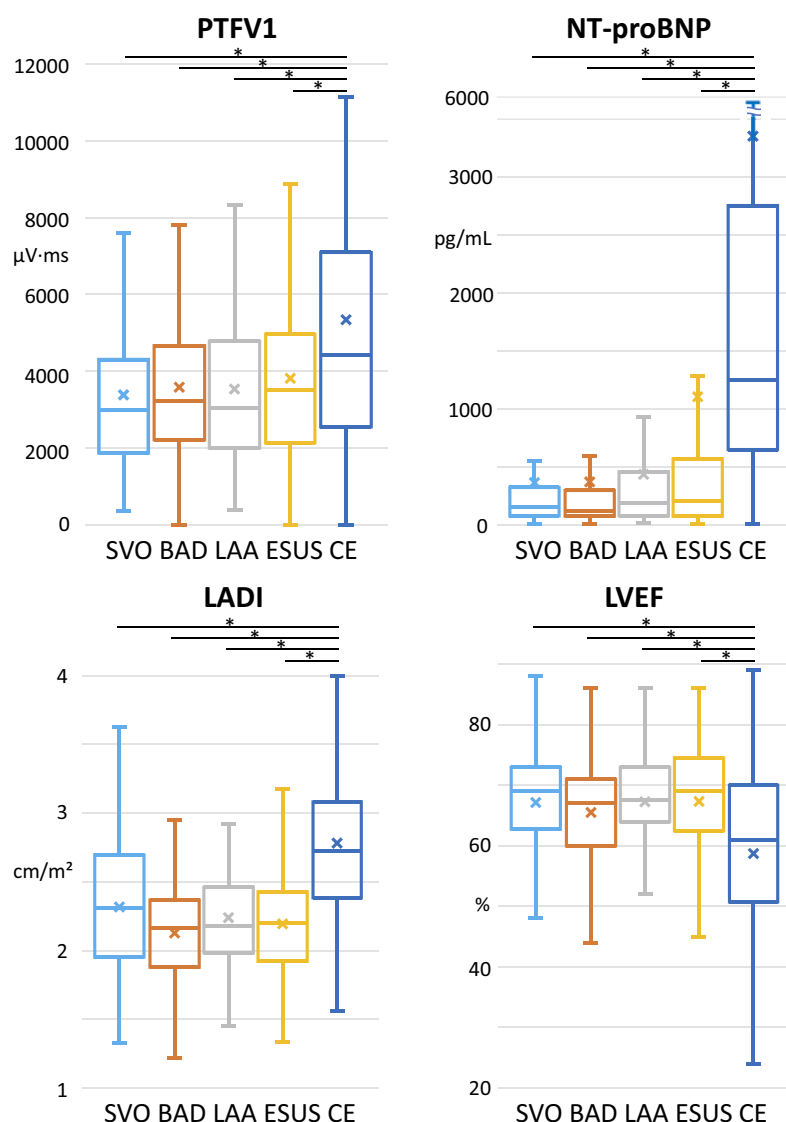


Figure 2 Comparison of atrial cardiopathy biomarkers and left ventricular ejection fraction across stroke subtypes

*Bonferroni correction showed that atrial cardiopathy biomarkers were significantly higher, whereas left ventricular ejection fraction was significantly lower, in the CE group than in the other groups.

×mean value

PTFV1: P-wave terminal force in lead V1, NT-proBNP: N-terminal pro-brain natriuretic peptide, LADI: left atrial diameter index, LVEF: left ventricular ejection fraction, SVO: small-vessel occlusion, BAD: branch atheromatous disease, LAA: large artery atherosclerosis, ESUS: embolic stroke of undetermined source, CE: cardioembolism.

subtypes. The principal finding was that AC biomarkers were markedly more prevalent in patients with CE, whereas no significant differences were observed among the four non-CE stroke subtypes (SVO, BAD, LAA, and ESUS). These results suggest that AC, as defined by the current ARCADIA criteria, is not specific to ESUS but is widely distributed across ischemic stroke subtypes other than CE.

AC has been proposed as an important mechanism underlying ESUS, and previous studies have suggested that AC is more frequent in ESUS than in non-cardioembolic stroke. Jalini et al.¹⁴ reported that AC was significantly more common in ESUS patients than in those with SVO or LAA. The prevalence estimates vary substantially across studies depending on the diagnostic thresholds and the number of biomarkers used. According to the

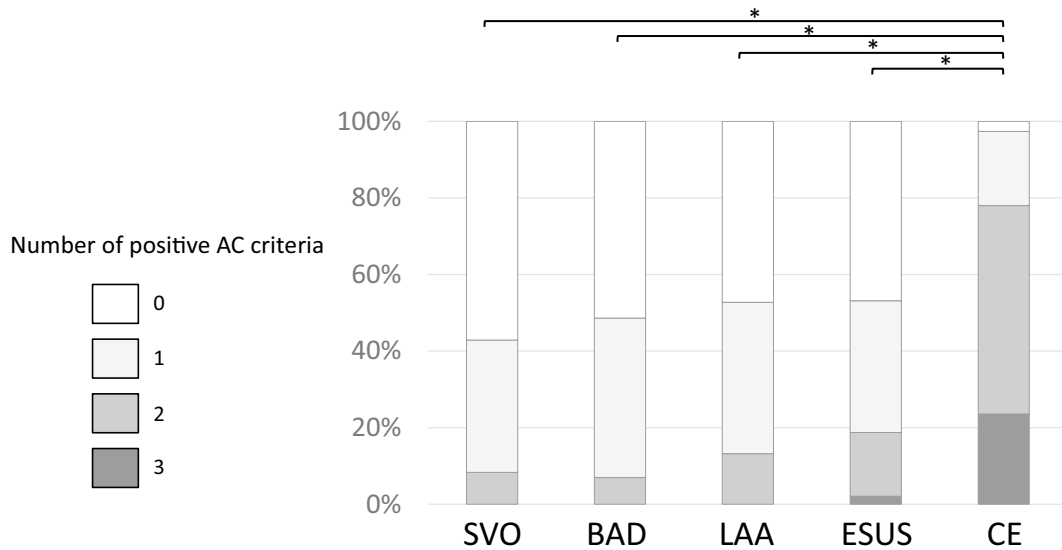


Figure 3 The prevalence of atrial cardiopathy and the number of positive diagnostic criteria across stroke subtypes

The prevalence of atrial cardiopathy varied among patient groups: SVO (43%), BAD (49%), LAA (53%), ESUS (54%), and CE (97%). Using a diagnostic threshold of at least two positive criteria, the prevalence of AC in the different patient groups was SVO 8%, BAD 7%, LAA 13%, ESUS 19%, and CE 78%. In both analyses, the prevalence of AC was significantly higher in the CE group than in the other stroke subtypes, as confirmed by Bonferroni-corrected comparisons ($p < 0.005$).

SVO: small-vessel occlusion, BAD: branch atheromatous disease, LAA: large artery atherosclerosis, ESUS: embolic stroke of undetermined source, CE: cardioembolism.

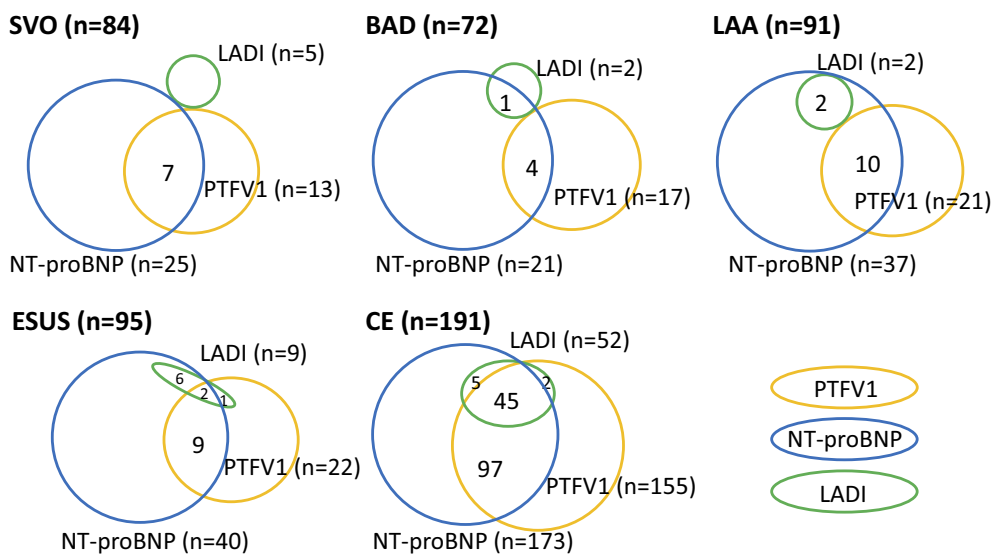


Figure 4 Overlap in atrial cardiopathy biomarker positivity across stroke subtypes

There was greater overlap of positive biomarkers in the ESUS and CE groups than in the SVO and BAD groups. The LADI criterion did not overlap with PTFV1 in SVO, BAD, or LAA subtypes.

ARCADIA: The Atrial Cardiopathy and Antithrombotic Drugs in Prevention After Cryptogenic Stroke, PTFV1: P-wave terminal force in lead V1, NT-proBNP: N-terminal pro-brain natriuretic peptide, LADI: left atrial diameter index, SVO: small-vessel occlusion, BAD: branch atheromatous disease, LAA: large artery atherosclerosis, ESUS: embolic stroke of undetermined source, CE: cardioembolism.

ARCADIA criteria, we observed an unexpectedly high prevalence of AC in non-CE subtypes. This may reflect shared systemic vascular and inflammatory remodeling pathways that contribute to both atherosclerosis and atrial dysfunction^{15,16}. Alternatively, it raises the possibility that some strokes classified as non-CE subtypes may have an unrecognized atrial or cardioembolic component. However, these findings also suggest that evaluating individual AC components and their combinations may help better identify patients in whom atrial pathology plays a causal role in stroke.

Although PTFV1 was assumed to be positive in patients with AF, this approach had only a limited impact on our findings. The high prevalence of AC in the CE group was primarily driven by the coexistence of multiple biomarkers rather than by the assumed positivity of PTFV1.

Our study showed that NT-proBNP >250 pg/mL was the most frequent diagnostic criterion for identifying AC. In the Warfarin Aspirin Recurrent Stroke Study, this threshold was associated with a twofold increased risk of recurrent stroke, whereas higher cutoffs (>750 pg/mL) identified patients who benefited more from anticoagulation¹⁷. Similarly, Kneihsl et al.¹⁸ reported that NT-proBNP levels below 505 pg/mL were associated with negative findings on extended cardiac monitoring. Taken together, these findings suggest that higher NT-proBNP thresholds may improve specificity by reducing overclassification related to non-atrial comorbid conditions. Because NT-proBNP may be influenced by cardiac function, we additionally evaluated LVEF, which was significantly lower in the CE group (**Figure 2**). This finding is consistent with the greater burden of underlying cardiac disease in CE patients and should be considered when interpreting NT-proBNP levels.

Left atrial enlargement is a key marker of AC and is thought to reflect more advanced atrial remodeling, often coexisting with AF¹⁹. However, its prevalence was low in our cohort, consistent with the findings of the ARCADIA trial⁸. In the ESUS and CE groups, LADI frequently overlapped with other biomarkers, whereas in the SVO, BAD, and LAA groups, it showed no overlap with PTFV1. The former pattern may reflect LADI as a late marker of AC, potentially indicating AF or a pre-AF state. In contrast, the latter may represent a dissociation between structural and electrical remodeling, suggesting limited relevance to embolic stroke mechanisms.

Our findings may help explain the neutral results of the ARCADIA trial, in which apixaban did not reduce re-

current stroke compared with aspirin in patients with cryptogenic stroke and AC⁸. One possible explanation is that current AC criteria identify a heterogeneous population in which atrial dysfunction is not the primary stroke mechanism. If AC biomarkers are widely present across stroke subtypes unrelated to CE, anticoagulation based solely on these markers may fail to identify patients who would benefit. These findings highlight the need to refine current AC definitions. More specific criteria—such as stricter biomarker thresholds, combinations of markers, or incorporation of structural and functional atrial parameters—may better distinguish causal atrial pathology from nonspecific cardiovascular abnormalities.

This study has several limitations. First, it is a single-center retrospective study and is therefore subject to selection bias. Second, most patients were Japanese, which may limit the generalizability of our findings. Third, we did not perform long-term cardiac rhythm monitoring; therefore, subclinical AF may have been missed. Finally, NT-proBNP levels may be influenced by the acute phase of ischemic stroke. However, previous studies have shown that NT-proBNP measured within the first 72 hours after stroke onset retains reasonable diagnostic accuracy for identifying CE mechanisms²⁰. Therefore, although acute-phase elevation cannot be completely excluded, the observed distribution of NT-proBNP across stroke subtypes likely reflects underlying cardiovascular conditions rather than solely an acute stress response.

In conclusion, AC defined by the ARCADIA criteria was highly prevalent in patients with CE but was similarly distributed across non-CE stroke subtypes, including SVO, BAD, LAA, and ESUS. These findings suggest that the current AC criteria may lack etiologic specificity for embolic stroke mechanisms. Refinement of AC definitions will be necessary to better identify patients in whom atrial dysfunction plays a causal role in stroke.

Author Contributions: All authors meet the authorship criteria recommended by the ICMJE. YK, SN, SKi, KO, NA, TN, and ID contributed to data acquisition; YK, SN, TH, SKo, SS, and JA contributed to data interpretation; YK drafted the manuscript; SN, TH, ST, SKo, SS, and JA critically reviewed the manuscript for important intellectual content; all authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Funding: None.

Conflict of Interest: None.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process: Generative AI and AI-assisted technologies were not used in the preparation of this work.

References

- Adams HP Jr, Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in acute stroke treatment. *Stroke*. 1993 Jan;24(1):35–41.
- Kamel H, Okin PM, Longstreth WT Jr, Elkind MS, Soliman EZ. Atrial cardiopathy: a broadened concept of left atrial thromboembolism beyond atrial fibrillation. *Future Cardiol*. 2015 May;11(3):323–31.
- Folsom AR, Nambi V, Bell EJ, et al. Troponin T, N-terminal pro-B-type natriuretic peptide, and incidence of stroke: the atherosclerosis risk in communities study. *Stroke*. 2013 Apr;44(4):961–7.
- Kamel H, O'Neal WT, Okin PM, Loefer LR, Alonso A, Soliman EZ. Electrocardiographic left atrial abnormality and stroke subtype in the atherosclerosis risk in communities study. *Ann Neurol*. 2015 Nov;78(5):670–8.
- Yaghi S, Moon YP, Mora-McLaughlin C, et al. Left atrial enlargement and stroke recurrence: the Northern Manhattan Stroke Study. *Stroke*. 2015 Jun;46(6):1488–93.
- Kato Y, Takahashi S. Atrial cardiopathy and cryptogenic stroke. *Front Neurol*. 2022 Feb 22;13:839398.
- Kamel H, Longstreth WT Jr, Tirschwell DL, et al. The Atrial Cardiopathy and Antithrombotic Drugs In prevention After cryptogenic stroke randomized trial: rationale and methods. *Int J Stroke*. 2019 Feb;14(2):207–14.
- Kamel H, Longstreth WT Jr, Tirschwell DL, et al. Apixaban to prevent recurrence after cryptogenic stroke in patients with atrial cardiopathy: the ARCADIA randomized clinical trial. *JAMA*. 2024 Feb 20;331(7):573–81.
- Caplan LR. Intracranial branch atheromatous disease: a neglected, understudied, and underused concept. *Neurology*. 1989 Sep;39(9):1246–50.
- Nakase T, Yamamoto Y, Takagi M; Japan Branch Atheromatous Disease Registry Collaborators. The impact of diagnosing branch atheromatous disease for predicting prognosis. *J Stroke Cerebrovasc Dis*. 2015 Oct;24(10):2423–8.
- Zhou L, Yao M, Peng B, Zhu Y, Ni J, Cui L. Atherosclerosis might be responsible for branch artery disease: evidence from white matter hyperintensity burden in acute isolated pontine infarction. *Front Neurol*. 2018 Oct 9;9:840.
- Hart RG, Diener HC, Coutts SB, et al. Embolic strokes of undetermined source: the case for a new clinical construct. *Lancet Neurol*. 2014 Apr;13(4):429–38.
- Kanda Y. Investigation of the freely available easy-to-use software 'EZ' for medical statistics. *Bone Marrow Transplant*. 2013 Mar;48(3):452–8.
- Jalini S, Rajalingam R, Nisenbaum R, Javier AD, Woo A, Pikula A. Atrial cardiopathy in patients with embolic strokes of unknown source and other stroke etiologies. *Neurology*. 2019 Jan 22;92(4):e288–94.
- Almuwaqqat Z, Claxton JS, Norby FL, et al. Association of arterial stiffness with incident atrial fibrillation: a cohort study. *BMC Cardiovasc Disord*. 2021 May 20;21(1):247.
- Rachieru C, Lighezan DF, Petrescu L, et al. The significance of inflammation in atrial fibrillation. *Curr Health Sci J*. 2024 Jan-Mar;50(1):59–66.
- Longstreth WT Jr, Kronmal RA, Thompson JL, et al. Amino terminal pro-B-type natriuretic peptide, secondary stroke prevention, and choice of antithrombotic therapy. *Stroke*. 2013 Mar;44(3):714–9.
- Kneihsl M, Gattringer T, Bisping E, et al. Blood biomarkers of heart failure and hypercoagulation to identify atrial fibrillation-related stroke. *Stroke*. 2019 Aug;50(8):2223–6.
- Jordan K, Yaghi S, Poppas A, et al. Left atrial volume index is associated with cardioembolic stroke and atrial fibrillation detection after embolic stroke of undetermined source. *Stroke*. 2019 Aug;50(8):1997–2001.
- Fonseca AC, Matias JS, E Melo TP, et al. Time course of NT-proBNP levels after acute ischemic stroke. *Acta Neurol Scand*. 2013 Oct;128(4):235–40.