

Utility of the CHA₂DS₂-VASc score for predicting cerebral embolic events in patients with infective endocarditis

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ABSTRACT

Aims: Cerebral embolic events (CEEs) are among the most serious complications of infective endocarditis (IE), yet risk stratification remains challenging in routine clinical practice. This study aimed to evaluate the utility of the CHA₂DS₂-VASc score in predicting CEEs during the active phase of IE, using a simple and accessible clinical tool.

Methods: This retrospective single-center study included 158 patients with definite IE. CEEs were defined as ischemic stroke or transient ischemic attack confirmed by neuroimaging. The association between the CHA₂DS₂-VASc score and CEEs was assessed using logistic regression. Two models were constructed; model 1 (baseline clinical and echocardiographic variables) and model 2 (model 1+CHA₂DS₂-VASc). Model performance was evaluated using AUC, AIC, Nagelkerke R², Brier Score, calibration curve, and decision curve analysis.

Results: CEEs occurred in 32 patients (20%). The CHA₂DS₂-VASc score was significantly higher in patients with CEEs (p=0.011) and remained an independent predictor in multivariate analysis (OR 3.00, 95% CI: 1.26-7.18, p=0.013). Incorporating the CHA₂DS₂-VASc score into the predictive model led to a substantial improvement in discrimination, increasing the AUC from 0.686 to 0.732. A threshold score of 2 provided optimal classification, demonstrating favorable sensitivity and specificity for identifying patients at risk of CEEs. Notably, the predictive value of the score remained robust across key subgroups, including those with left-sided IE, prosthetic material, atrial fibrillation, and preserved ejection fraction.

Conclusion: The CHA₂DS₂-VASc score is a strong and independent predictor of CEEs in patients with IE. Its integration into clinical assessment may enhance embolic risk stratification, particularly during the active phase of the disease, and support timely decision-making in this high-risk population.

Keywords: Infective endocarditis, CHA₂DS₂-VASc, intracranial embolism, risk assessment

INTRODUCTION

Infective endocarditis (IE) remains a severe and life-threatening condition with high morbidity and mortality despite advances in antimicrobial therapy and surgical interventions.¹ Cerebral embolic events (CEEs) represent one of its most devastating complications, occurring in up to 35% of patients, while silent cerebrovascular lesions, including ischemic infarctions and microhemorrhages, have been reported in as many as 80%.² Given their substantial impact on clinical outcomes, early risk stratification for CEEs is crucial in guiding therapeutic decisions, including the timing of surgery and the use of antithrombotic strategies.³ Although certain factors-such as *Staphylococcus aureus* infection and the presence of large vegetations (>10 mm)-have been linked to an increased risk of systemic embolism,⁴ a widely accepted and standardized predictive model for embolic risk in IE is still lacking.

The CHADS₂-VASc score, originally developed for thromboembolic risk assessment in atrial fibrillation (AF), has been extensively investigated across a broad range of other cardiovascular conditions.⁵ Several of its components-including heart failure, hypertension, and vascular disease-are established risk factors for thromboembolism beyond AF and have been associated with an increased predisposition to systemic embolization.⁶ Since these factors contribute to a prothrombotic state, endothelial dysfunction, and vascular inflammation, it is plausible that they may also play a role in the occurrence of CEEs in IE.⁷ Given the high embolic burden in IE, the potential applicability of CHADS₂-VASc in predicting stroke in this setting warrants further investigation.

In this study, we aimed to evaluate the predictive value of CHADS₂-VASc for CEEs in patients with IE, with the goal of determining its utility in identifying patients at higher risk for

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embolic complications and improving clinical management strategies in this population.

METHODS

Ethics

Data were obtained from comprehensive medical records, including clinical, laboratory, and echocardiographic evaluations. Patients with an indeterminate diagnosis of IE, a history of prior stroke or known cerebrovascular disease before the diagnosis of IE, incomplete medical records preventing CHA₂DS₂-VASc score assessment were excluded. The study was approved by the Başakşehir Çam and Sakura City Hospital Ethics Committee (Date: 21.04.2025, Decision No: KAEK/16.04.2025-117), and all procedures were conducted in accordance with the Declaration of Helsinki. As this was a retrospective study, informed consent was waived.

Study Population

This retrospective study included 158 patients diagnosed with definite IE who experienced a CEE either at the time of admission or during hospitalization at a tertiary center. All events occurred during the active phase of endocarditis. The diagnosis of IE was based on the modified Duke criteria, while CEEs were identified through neurological evaluation supported by neuroimaging, in accordance with standard clinical definitions.

Definitions

Infective endocarditis was defined based on the modified Duke criteria.¹ A definite diagnosis required the presence of two major criteria, one major and three minor criteria, or five minor criteria. The major criteria included positive blood cultures with typical IE pathogens *Staphylococcus aureus*, *Streptococcus viridans*, *Enterococcus* spp., or HACEK group [*Haemophilus* spp., *Aggregatibacter* spp., *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* spp.) and evidence of endocardial involvement on echocardiography (vegetation, abscess, new dehiscence of a prosthetic valve, or new valvular regurgitation). The minor criteria consisted of predisposing heart conditions or intravenous drug use, fever ($\geq 38^{\circ}\text{C}$), vascular phenomena (e.g., embolic events, Janeway lesions), immunologic phenomena (e.g., glomerulonephritis, Osler's nodes), and positive blood cultures not fulfilling the major criteria.

Cerebral embolic events were defined as the occurrence of either ischemic stroke or transient ischemic attack (TIA) during the active phase of infective endocarditis. Events were included if they occurred either at the time of admission or during hospitalization. Ischemic stroke was defined as a focal neurological deficit lasting more than 24 hours and confirmed by neuroimaging [either magnetic resonance imaging (MRI) or computed tomography (CT)] demonstrating acute cerebral infarction.⁸ TIA was defined as a transient neurological deficit resolving within 24 hours without evidence of acute ischemia on neuroimaging, consistent with tissue-based definitions recommended in recent guidelines.⁹

The CHADS₂-VASc score was calculated for each patient based on the presence of congestive heart failure (1 point), hypertension (1 point), age ≥ 75 years (2 points), diabetes mellitus (1 point), prior stroke or transient ischemic attack (2 points), vascular disease (1 point), age 65-74 years (1 point), and female sex (1 point).¹⁰

Echocardiographic Evaluation

All patients underwent transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) as part of their diagnostic workup. TTE was performed using a Philips Epic CVx ultrasound system with an X5-1 phased-array transducer, and TEE was conducted using the same system with a Philips X8-2t matrix-array transesophageal transducer. The procedure was performed under oropharyngeal anesthesia and intravenous sedation. Vegetations were defined as oscillating intracardiac masses attached to a valve or endocardial surface, with the longest dimension recorded for analysis.¹¹ An abscess was identified as a non-vascularized perivalvular echo lucent area. Pseudoaneurysms were defined as pulsatile perivalvular cavities communicating with the cardiac chamber.¹² Fistulas were considered as abnormal communications between cardiac chambers or major vessels. Dehiscence of a prosthetic valve was defined as partial or complete detachment, leading to abnormal motion on TEE.¹²

Statistical Analysis

Continuous variables were tested for normality using the Kolmogorov-Smirnov test and were presented as mean $\pm$ standard deviation (SD) for normally distributed data or median [interquartile range (IQR)] for non-normally distributed data. Categorical variables were expressed as counts and percentages (n, %) and were compared using the Chi-square test or Fisher's exact test, as appropriate.

To evaluate the association between clinical parameters and CEEs in IE patients, univariate and multivariate logistic regression analyses were performed. Variables with $p < 0.25$ in the univariate analysis were included in the multivariate models. Two separate models were constructed, where the first model included baseline clinical and echocardiographic parameters, and the second model incorporated all variables from the first model with the addition of the CHADS₂-VASc score as an independent predictor.

The predictive performance of the models was assessed using receiver operating characteristic (ROC) curve analysis, and the area under the curve (AUC) was compared to evaluate discrimination. The optimal CHADS₂-VASc cut-off for predicting CEEs was determined using the Youden Index. Calibration of the models was assessed using the Hosmer-Lemeshow goodness-of-fit test and calibration plots.

To further evaluate the clinical utility of incorporating CHADS₂-VASc into the model, ROC analysis was used to compare their discriminative ability, while decision curve analysis (DCA) was performed to assess net clinical benefit. Additionally, model performance was evaluated using Nagelkerke's R², Brier score, Akaike information criterion (AIC), and Log-Likelihood (-2LL) values. Overall, the two models were systematically compared across these metrics.

All statistical analyses were performed using SPSS version 30 (IBM Corp., Armonk, NY, USA) and R version 4.4.2 (R Foundation for Statistical Computing, Vienna, Austria). A p-value <0.05 was considered statistically significant.

RESULTS

Among the 158 patients included in the study, 32 (20%) experienced a CEE during the active phase of IE. Of these, 24 (75%) were ischemic strokes and 8 (25%) were transient ischemic attacks. Baseline characteristics were compared between patients with and without CEEs. There were no significant differences in demographic characteristics, renal function markers, inflammatory parameters, or echocardiographic indices between the groups (Table 1-3). However, patients with CEEs were more likely to have left-sided IE and a higher CHADS₂-VASc score compared to those without CEEs (p=0.041 and p=0.011, respectively). The mean CHA₂DS₂-VASc score was 3.75±1.92 in the CEE group and 2.77±1.93 in the non-CEE group.

In the univariate analysis, CHADS₂-VASc >2, left-sided IE, and female sex were associated with a higher likelihood of CEEs (p=0.014, p=0.021, and p=0.044, respectively), while EF demonstrated a borderline association (p=0.098). In the multivariate analysis, model 1 included baseline clinical and

echocardiographic parameters. When CHADS₂-VASc was included in model 2, it remained an independent predictor of CEEs (OR 3.00, 95% CI: 1.26-7.18, p=0.013) (Table 4).

Adding CHADS₂-VASc to the model improved its predictive performance, as reflected by an increase in the area under the ROC curve (AUC) from 0.686 (95% CI: 0.588-0.785) to 0.732 (95% CI: 0.647-0.818) (Figure 1). The optimal CHADS₂-VASc cutoff for predicting CEEs was 2, with a sensitivity of 66% and specificity of 78%.

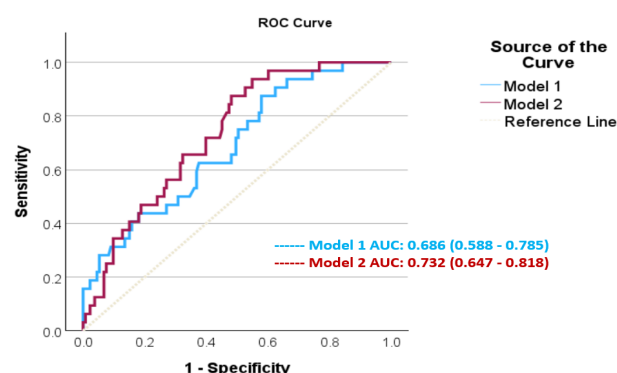


Figure 1. ROC curve comparison of two predictive models for cerebral embolic events in infective endocarditis

ROC: Receiver operating characteristic

Table 1. Baseline characteristics of patients with and without cerebral embolic events

Variable	No cerebral embolic events, n:126	Cerebral embolic events, n: 32	Total population, n: 158	p-value
Age (years)	58 (47.5-68)	62 (52.7-69)	58 (50-68)	0.157
Female sex (n, %)	46 (37%)	14 (44%)	60 (38%)	0.034
Diabetes mellitus (n, %)	54 (43%)	15 (47%)	69 (44%)	0.473
Hypertension (n, %)	80 (63%)	21 (66%)	101 (64%)	0.687
Hyperlipidemia (n, %)	53 (42%)	14 (44%)	67 (42%)	0.693
Coronary artery disease (n, %)	58 (46%)	12 (38%)	70 (44%)	0.557
Myocardial infarction history (n, %)	12 (10%)	5 (16%)	17 (11%)	0.328
Heart failure (n, %)	31 (25%)	5 (16%)	36 (23%)	0.476
Chronic kidney disease (n, %)	16 (13%)	10 (31%)	26 (16%)	0.018
Chronic obstructive pulmonary disease (n, %)	8 (6%)	3 (9%)	11 (7%)	0.518
History of endocarditis (n, %)	2 (2%)	1 (3%)	3 (2%)	0.479
Atrial fibrillation (n, %)	27 (21%)	8 (25%)	35 (22%)	0.632
Active smoker (n, %)	32 (25%)	11 (34%)	43 (27%)	0.264
Intravenous drug use (n, %)	4 (3%)	1 (3%)	5 (3%)	0.972
Malignancy (n, %)	9 (7%)	3 (9%)	12 (8%)	0.703
NYHA functional class (n, %)				0.846
Class I (n, %)	55 (44%)	13 (41%)	68 (43%)	
Class II (n, %)	31 (25%)	7 (22%)	38 (24%)	
Class III (n, %)	30 (24%)	6 (19%)	36 (23%)	
Class IV (n, %)	17 (13%)	6 (19%)	23 (15%)	
Body-mass index (kg/m ²)	25.4 (22.2-28.4)	23.38 (20.4-27.6)	24.9 (21.5-28.3)	0.091
CHA ₂ DS ₂ -VASc score	2.77±1.93	3.75±1.92	2.96±1.96	0.011
CHA ₂ DS ₂ -VASc score group				0.011
0-1	41 (32.5%)	3 (9.4%)	44 (27.8%)	
2-3	49 (38.9%)	11 (34.4%)	60 (38.0%)	
≥4	36 (28.6%)	18 (56.3%)	54 (34.2%)	

Continuous variables are presented as median (interquartile range), and categorical variables are presented as number (percentage). p-values in bold indicate statistical significance (p<0.05). NYHA: New York Heart Association

Table 2. Laboratory and echocardiographic characteristics of patients with and without cerebral embolic events

Variable	No cerebral embolic events, n:126	Cerebral embolic events, n: 32	Total population, n: 158	p-value
Hemoglobin (g/dl)	9.9 (8.4-11.4)	10.2 (8.9-12.1)	9.9 (8.4-11.5)	0.254
White blood cell count ($\times 10^3/\mu\text{l}$)	10.18 (7.13-14.68)	12.55 (9.13-15.3225)	10.43 (7.6-15.045)	0.081
Platelet count ($\times 10^3/\mu\text{l}$)	207 (155.5-284.5)	223.5 (134.7-27)	209 (150-283.5)	0.706
Creatinine (mg/dl)	1.17 (0.8-4.2)	1.16 (0.9-1.7)	1.1 (0.8-3.9)	0.551
Glomerular filtration rate (ml/min/1.73 m ²)	61.2 (12-88.5)	59.5 (40.2-83.2)	61 (14-87)	0.714
C-reactive protein (mg/L)	87 (37.8-162.3)	95.7 (56-155.7)	91.7 (39.4-156.4)	0.601
Procalcitonin (ng/ml)	0.7 (0.1-5.6)	0.4 (0.1-2.8)	0.6 (0.1-5.3)	0.443
Troponin (ng/ml)	60.4 (19.9-196.5)	51.4 (25.8-179.7)	57.7 (21.5-195)	0.683
Albumin (g/dl)	33 (29-38)	34 (29.5-40)	33 (29-38)	0.377
Total protein (g/dl)	66 (59-71)	65.5 (57.2-72.5)	66 (58.5-71)	0.822
Sodium (mmol/L)	135 (132-138)	134 (132-136)	135 (132-138)	0.214
Potassium (mmol/L)	4.4 (4.0-4.7)	4.3 (4-4.6)	4.4 (4.0-4.7)	0.705
Magnesium (mg/dl)	2 (1.8-2.22)	2.1 (1.8-2.4)	2.0 (1.8-2.2)	0.247
Calcium (mg/dl)	8.6 (8.2-9.1)	8.5 (7.9-8.9)	8.6 (8.1-9.1)	0.192
Total cholesterol (mg/dl)	150 (127.5-181)	169.5 (129.5-216.25)	152 (128.5-184)	0.067
Triglycerides (mg/dl)	142 (107-182)	155 (117-235)	150 (115-192)	0.061
LDL cholesterol (mg/dl)	89 (64-114.5)	101.5 (70-128)	91 (64.5-119)	0.103
HDL cholesterol (mg/dl)	29 (23-39)	32 (24.5-44.25)	30 (23-40)	0.536
Pro-BNP (pg/ml)	2242 (245-6763.5)	2047.5 (375.7-5872.2)	2193 (295.5-6577.5)	0.872
Echocardiographic features				
Ejection fraction (%)	61 (58 - 63)	60 (56.25-60)	60 (50-60)	0.061
Pulmonary artery pressure (mmHg)	40 (30.25-50)	36 (30.7-43.7)	40 (30.2-50)	0.563
TAPSE (mm)	21 (18-24)	16 (16-16)	21 (18-24)	0.186

Continuous variables are presented as median (interquartile range), and categorical variables are presented as number (percentage). p-values in bold indicate statistical significance ($p < 0.05$). LDL: Low density lipoprotein, HDL: High density lipoprotein, pro-BNP: pro-B-type natriuretic peptide, TAPSE: Tricuspid annular plane systolic excursion

Further evaluation of model performance metrics demonstrated that model 2 had lower AIC and -2 Log Likelihood values, indicating better fit, while Nagelkerke's R² and Brier Score also suggested improved predictive accuracy (Figure 2). DCA and calibration analysis were presented together, demonstrating both higher net clinical benefit with the inclusion of CHA₂DS₂-VAsC and improved agreement between predicted and observed probabilities (Figure 3).

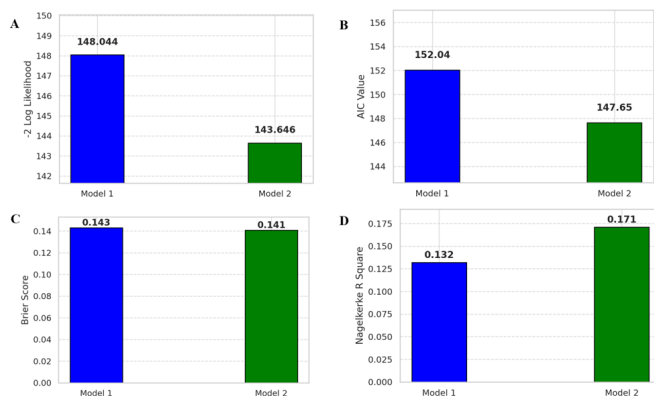


Figure 2. Comparison of model performance metrics for predicting cerebral embolic events in infective endocarditis

(A) -2 Log Likelihood values indicating overall model fit.
(B) Akaike information criterion values indicating model parsimony and fit.
(C) Brier scores reflecting overall prediction accuracy.
(D) Nagelkerke R² values representing explained variance.

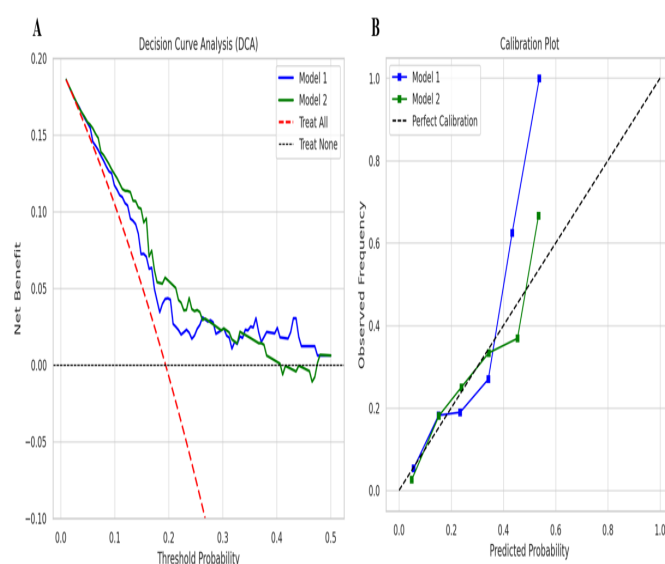


Figure 3. Decision curve analysis (A) and calibration plot (B) of model 1 and model 2 for predicting cerebral embolic events in infective endocarditis

The predictive value of the CHA₂DS₂-VAsC score for CEEs was further evaluated across specific patient subgroups (Figure 4). The score remained significantly associated with CEEs in patients with left-sided IE, prosthetic valve or pacemaker/lead involvement, preserved EF (>50%), and AF.

Variable	No cerebral embolic events, n:126	Cerebral embolic events, n: 32	Total population, n: 158	p-value
Primary infection site (n, %)				0.095
Native valve (n, %)	64 (51%)	22 (69%)	86 (54%)	
Prosthetic valve (n, %)	29 (23%)	8 (25%)	37 (23%)	
Lead and catheter infection (n, %)	33 (26%)	2 (6%)	35 (22%)	
Vegetation localization (n, %)				0.041
Left-Sided localization (n, %)	92 (73%)	29 (91%)	121 (77%)	
Right-Sided localization (n, %)	28 (22%)	2 (6%)	30 (19%)	
Bilateral localization (n, %)	6 (5%)	1 (3%)	7 (4%)	
Vegetation size (n, %)				0.537
Vegetation diameter <10 mm (n, %)	35 (28%)	8 (25%)	43 (27%)	
Vegetation diameter ≥10 mm (n, %)	91 (72%)	24 (75%)	115 (73%)	
Perivalvular complications (n, %)				0.079
No complication (n, %)	100 (79%)	21 (66%)	121 (77%)	
Perforation (n, %)	12 (10%)	3 (9%)	15 (9%)	
Paravalvular leak (n, %)	5 (4%)	0 (0%)	5 (3%)	
Pseudoaneurysm (n, %)	2 (2%)	1 (3%)	3 (2%)	
Abscess or fistula (n, %)	9 (7%)	7 (22%)	16 (10%)	
Dehiscence (n, %)	5 (4%)	0 (0%)	5 (3%)	
Causative microorganism (n, %)				0.549
No growth (n, %)	23 (18%)	4 (13%)	27 (17%)	
Coagulase-negative staphylococci (n, %)	30 (24%)	4 (13%)	34 (22%)	
<i>Staphylococcus aureus</i> (n, %)	42 (33%)	10 (31%)	52 (33%)	
<i>Streptococcus</i> spp. (n, %)	8 (6%)	5 (16%)	13 (8%)	
<i>Enterococcus</i> spp. (n, %)	9 (7%)	4 (13%)	13 (8%)	
Gram-negative bacteria (n, %)	10 (8%)	2 (6%)	12 (8%)	
<i>Brucella</i> spp. (n, %)	1 (1%)	0 (0%)	1 (1%)	
<i>Candida</i> spp. (n, %)	4 (3%)	2 (6%)	6 (4%)	
Other pathogens (n, %)	6 (5%)	1 (3%)	7 (4%)	

Variable	Model 1				Model 2			
	p-value	OR	95% CI		p-value	OR	95% CI	
			Lower	Upper			Lower	Upper
Age (years)	0.174	1.019	0.992	1.047	0.295	1.523	0.693	3.346
Female	0.059	1.997	0.973	4.097	0.291	1.527	0.697	3.347
Native valve	0.606	1.272	0.510	3.175	0.621	1.268	0.495	3.247
Left-sided infective endocarditis	0.072	3.502	0.895	13.697	0.066	3.699	0.917	14.921
Ejection fraction (%)	0.466	1.020	0.968	1.075	0.261	1.032	0.977	1.091
CHADS ₂ -VASC score >2	.	.	.	.	0.013	3.009	1.262	7.177
OR: Odds ratio, CI: Confidence interval								

DISCUSSION

This study investigated the association between the CHA₂DS₂-VASC score and the risk of CEE in patients with IE. We found that higher CHA₂DS₂-VASC scores were significantly associated with these events, and the score remained an independent predictor after adjustment for key

clinical variables. To our knowledge, this is the first study to specifically assess the prognostic value of the CHA₂DS₂-VASC score for CEE in this patient population, highlighting its potential role as a simple and accessible tool in a complex clinical setting.

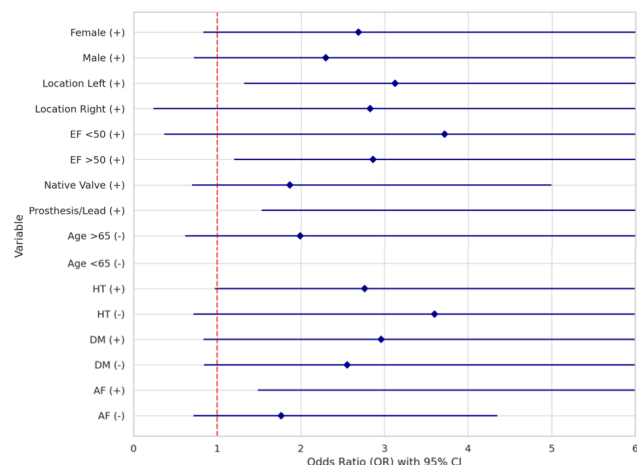


Figure 4. Forest plot of subgroup analyses for the association between the CHA₂DS₂-VAsC score and cerebral embolic events in infective endocarditis

The association between higher CHA₂DS₂-VAsC scores and CEE in IE is not unexpected, given the well-established link between the score's components and embolic risk factors. Age, hypertension, diabetes, heart failure, and prior vascular events are all independently associated with systemic vascular vulnerability and a prothrombotic state¹³ both of which contribute to the likelihood of embolization in IE. While the score was initially developed for AF, these variables are not rhythm-specific and may reflect a broader predisposition to embolic complications. A recent study reported that higher CHA₂DS₂-VAsC scores were associated with increased long-term mortality in IE, but did not find a statistically significant relationship with in-hospital stroke.¹⁴ Notably, their outcome definition did not appear to include TIA, which may have led to underestimation of embolic events. Our study used a broader definition of CEE and demonstrated a significant and independent association, highlighting the potential utility of this score in embolic risk stratification among patients with IE.

Several clinical, echocardiographic, and microbiological factors have been consistently associated with an increased risk of CEEs in patients with IE.¹⁵ These include *Staphylococcus aureus* infection,¹⁶ mitral and aortic valve involvement,¹⁷ mural vegetations,¹⁸ valvular abscess,¹⁹ and large vegetations (especially those ≥ 10 mm),²⁰ all of which reflect the underlying anatomical vulnerability to embolic events. Inflammatory and hemostatic biomarkers such as elevated D-dimer²¹ and CRP²² have also been linked with embolic complications. Moreover, both the use of antithrombotic therapy²³ (including anticoagulants and antiplatelets) and the timing of antibiotic initiation²⁴ have been shown to influence the risk of embolic events, although findings have been somewhat conflicting across studies. Although vegetation size ≥ 10 mm is considered a classical risk factor for embolic events in infective endocarditis, it did not show a statistically significant association with CEEs in our study. This finding may be explained by the high prevalence of large vegetations in our cohort, which could have reduced the discriminative power of this variable. Additionally, the relatively small sample size and the exclusive focus on clinically overt CEEs—rather than subclinical lesions—may have limited our ability to

detect this association. Differences in patient characteristics, timing of interventions, or measurement variability may also account for the discrepancy with previous studies. However, CEE were more frequent in patients with left-sided IE, female sex, and chronic kidney disease—an overall pattern consistent with previously reported embolic risk profiles.

Importantly, comorbidities such as hypertension, atrial fibrillation, hyperlipidemia, and heart failure¹³ each shown to significantly elevate embolic risk—are also core components of the CHA₂DS₂-VAsC score. The aggregation of these variables within a single scoring system likely contributes to its predictive capacity, enabling it to capture embolic risk in a more integrated manner than individual parameters alone. This may explain why the CHA₂DS₂-VAsC score remained independently associated with cerebral embolic events in our cohort, despite the lack of significance for some classical IE-specific predictors.

Beyond its overall association with CEE, the CHA₂DS₂-VAsC score also demonstrated stronger predictive performance in specific clinical subgroups. In particular, among patients with AF, the score was strongly associated with embolic risk, consistent with its original purpose and further supporting its applicability in the setting of IE. Similarly, its predictive value was more pronounced in patients with left-sided IE, preserved ejection fraction, or prosthetic material involvement—subgroups known to carry a higher risk of embolization. These findings suggest that CHA₂DS₂-VAsC may offer additive value in patients with both rhythm-related and structural risk factors, where standard imaging or laboratory data may fall short in individual risk stratification.

To date, most embolic risk models in IE have focused on vegetation characteristics, microbiological findings, or echocardiographic markers. While these variables are undeniably important, they often require advanced imaging modalities or are not available at the time of clinical decision-making. Our study proposes an alternative approach by repurposing an existing clinical score CHA₂DS₂-VAsC to stratify embolic risk using routinely collected parameters. Although the score has been previously associated with long-term mortality in IE,¹⁴ to our knowledge, this is the first study to specifically evaluate its prognostic value for CEE. Given its simplicity and widespread familiarity among clinicians, the CHA₂DS₂-VAsC score may represent a practical adjunct to existing risk assessment tools in patients with infective endocarditis.

Limitations

This study has several limitations that should be considered when interpreting the results. Its retrospective design and single-center nature may introduce selection bias and limit the external validity of the findings. Additionally, the relatively small sample size may have limited the statistical power of the analyses, particularly in subgroup evaluations, where effect estimates tend to be less precise. Only clinically apparent ischemic strokes confirmed by neuroimaging were evaluated in this study. Other neurological manifestations of IE—such as meningitis, brain abscess, encephalopathy, and infectious aneurysms—were not systematically assessed. Moreover,

the precise embolic source of stroke could not always be determined by imaging, and the presence of alternative mechanisms cannot be fully excluded. Finally, detailed data on stroke localization and antithrombotic therapy—both of which could offer valuable insights into the underlying pathophysiology—were not consistently available across all patients. These factors, along with the limited sample size, may have influenced the ability to detect some associations previously reported in the literature.

CONCLUSION

As a result, this study highlights the potential utility of the CHA₂DS₂-VASc score as a practical and readily applicable tool for predicting CEE in patients with IE, particularly during the active phase of the infection. By incorporating well-established clinical risk factors into a single composite measure, the score may help identify patients at elevated embolic risk, even in the absence of advanced imaging or laboratory markers. These findings support its role as a complementary tool in embolic risk stratification and clinical decision-making in the context of endocarditis. To the best of our knowledge, this is the first study to evaluate the CHA₂DS₂-VASc score specifically for this purpose, offering new insights into its prognostic relevance beyond AF.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of Başakşehir Çam and Sakura City Hospital Ethics Committee (Date: 21.04.2025, Decision No: KAEK/16.04.2025-117).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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