BNP and Particular Left Ventricle Segment Asynergy Associate with Cardioembolic Stroke from Old Myocardial Infarction

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Grant support

This study was supported in part by research grants from JSPS KAKENHI (Grant Number 23590598, 15K08615), the Japan Science and Technology Agency (AS242Z02592P), Bristol-Myers Squibb, the Smoking Research Foundation, and the Tsuchiya Foundation.

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Cover title: **Cardioembolism from Old Myocardial Infarction**

Key word: ischaemic stroke; stroke subtypes; cardioembolism; myocardial infarction, brain natriuretic peptide; sonography

Abstract

Background and Purpose

It is important to determine the usage of anti-coagulant by defining the actual risk of cardioembolic stroke patients with old myocardial infarction. In the present study, we aimed to more precisely evaluate the risks of each segment associated with cardioembolic stroke using a 16-segment model. The usage of the plasma brain natriuretic peptide (BNP) associated with cardioembolic stroke was also evaluated in comparison with a left ventricle ejection fraction <40%.

Methods

There were a total of 190 ischemic stroke patients who had premorbid myocardial infarction. Excluding patients with atrial fibrillation or acute myocardial infarction, 143 ischemic stroke patients with old myocardial infarction were available for evaluation. Their left ventricle wall motion abnormality and the level of plasma BNP at their admission were analyzed.

Results

Hypertension and plasma BNP ≥ 206.9 pg/mL, determined from the receiver operating characteristic curve, were independently associated with cardioembolic stroke ($\chi^2=35.6$, $R^2=0.30$, $p<0.001$). Adjusting for these factors, statistically independent high risk was observed at the basal-inferior, basal-inferolateral, mid-anterior, mid-anteroseptal, apical-anterior, and apical-septal left ventricles.

Conclusion

High plasma BNP levels and left ventricular wall motion abnormalities in the segments perfused with left anterior descending coronary artery or right coronary artery show a high risk for cardioembolic stroke in patients with old myocardial infarction. Considering these factors, it could be possible to more precisely define the risk of cardioembolic stroke and to perform appropriate anti-thrombotic treatments in old myocardial infarction patients.

Introduction

Myocardial infarction is a disease under the spectrum of atherothrombosis, but it is also a possible cause of cardioembolism resulting from left ventricular wall motion abnormality. Visible left ventricular thrombus is frequently detected in acute myocardial infarction in the anterior (33.8-54.1%) compared with the inferior region (0-4.7%) based on observational studies prior to the advent of thrombolysis and percutaneous coronary intervention.¹⁻⁴ According to the American College of Chest Physicians Evidence-Based Clinical Practice Guidelines, an anticoagulant is recommended for preventing left ventricular thrombus in patients with anterior ST-segment elevation myocardial infarction and left ventricle ejection fraction <40%.⁵ These guidelines also recommend warfarin treatment (prothrombin time-international normalized ratio [PT-INR], 2.0 to 3.0) for several months in those patients followed by the discontinuation of warfarin treatment thereafter. The incidences of myocardial infarction and stroke were 1.44 and 1.38%/year in patients with established cardiovascular disease.⁶ Stroke occurs more frequently than myocardial infarction in patients with established coronary artery disease, especially in Asia and Europe⁶. There could be a potent risk of cardioembolic stroke even in patients with old myocardial infarction, especially in patients with inferior myocardial infarction. It is important to determine the usage of anti-coagulant by defining the actual risk of cardioembolic stroke in each patient.

In the present study, we aimed to more precisely evaluate the risks of each segment that is associated with cardioembolic stroke using a 16-segment model.⁷ Usage of the plasma brain natriuretic peptide (BNP) level in association with cardioembolic stroke is also evaluated in comparison with left ventricle ejection fraction.

Methods

In the Brain Attack Center Ota Memorial Hospital, 2,590 patients with acute ischemic stroke were admitted from January 2012 to December 2014. The study protocol was governed by the guidelines of the national government based on the Helsinki Declaration revised in 1983, and it was approved by the Institutional Research and Ethics Committee of our hospital.

All of those patients underwent magnetic resonance imaging, magnetic resonance angiography of the cranial and carotid arteries, electrocardiogram, and carotid ultrasonography evaluation at their admission, except if there were contraindications to performing these tests. When an abnormal electrocardiogram was detected at admission, transthoracic echocardiography was also performed. The final diagnosis of the stroke subtype was made before discharge, based on the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification, and the etiology of the qualifying ischemic event was rated as cardioembolism (high-risk cardiac source for cerebral emboli, e.g., akinetic left ventricular segment), large vessel disease (occlusion or >50% stenosis of an appropriate large extracranial or intracranial artery, or occlusion of appropriate stem, division, or branch artery), small vessel disease (lacunar infarction on brain CT or MRI), or other.⁸

Detailed data were collected from all patients by stroke physicians, including baseline characteristics (age and sex) and vascular risk factors (hypertension, diabetes mellitus, and dyslipidemia). Hypertension, diabetes mellitus, and dyslipidemia were diagnosed based on the following criteria. Patients were designated as hypertensive if they were taking anti-hypertensive agents or had a systolic blood pressure ≥ 140 mmHg and/or a diastolic blood pressure ≥ 90 mmHg. Patients were diagnosed with diabetes mellitus if they were being treated with oral hypoglycemic agents or insulin and/or their serum fasting blood glucose level was ≥ 7 mmol/L. Patients were diagnosed with dyslipidemia if they were

taking anti-dyslipidemia medication or presented levels of serum low-density lipoprotein cholesterol ≥ 3.62 mmol/L, triglycerides ≥ 1.69 mmol/L and/or high-density lipoprotein cholesterol < 1.03 mmol/L.

Echocardiography

Two-dimensional echocardiography was performed with an ultrasound system (iE33 xMATRIX; Royal Philips, Amsterdam) and a medium- and short-focused 2.5 or 3.5 MHz transducer or both. Each study included all standard echocardiographic views. Myocardial infarction was diagnosed by a typical medical history or the identification of diagnostic Q waves with responsible left ventricular wall motion abnormality using echocardiography by cardiologists blinded to the characteristics of ischemic stroke. Left ventricular wall motion was qualitatively defined in each segment individually as normokinesis, hypokinesis, akinesis, or dyskinesis with a 16-segment model.⁷ The left ventricular ejection fraction was determined with a modified Simpson's rule.⁹ The frequency of patients with left ventricular ejection fraction $< 40\%$ was determined.¹⁰

Brain Natriuretic Peptide (BNP)

Blood was immediately separated into chilled tubes containing potassium ethylenediaminetetraacetic acid and centrifuged for 30 minutes. Plasma BNP concentrations were determined with a specific immunoradiometric assay (Shionoria BNP Kit, Shionogi Co., Ltd., Tokyo, Japan).¹¹ Blood samples were taken at admission.

Statistical analysis

The statistical analyses were performed using JMP 12.0.1 statistical software (SAS Institute Inc., USA). Summary statistics were expressed as mean $\pm$ standard deviation for continuous variables and frequencies and percentages for discrete variables. Comparisons of the clinical characteristics were made with Student's t-test (for continuous variables) or Fisher's exact tests (for discrete variables). The logistic regression analysis model is

reported with all variables forced to be in the model. Statistical significance was established at two-tailed $p < 0.05$.

Results

Among the acute ischemic stroke patients, 190 patients had premorbid myocardial infarction. There were 33 patients with atrial fibrillation and 6 patients with an onset of myocardial infarction within 6 months prior to ischemic stroke onset. Additionally, 8 patients had no transthoracic echocardiography evaluation. Excluding these patients, 143 subjects were available for evaluation. Left ventricular wall motion abnormality ($\geq$ akinesis), which was described as a high-risk cardiac source for cerebral emboli, was frequently observed in the basal-inferior, mid-anterior, mid-anteroseptal and apex segments (Figure 1). To evaluate the features of cardioembolic stroke due to old myocardial infarction, the subjects were classified into either cardioembolic or non-cardioembolic infarction (e.g., atherothrombotic brain infarction, lacunar infarction, and other ischemic stroke) according to the subject's ischemic stroke subtype. Twenty-two subjects (15.4%) were classified as having cardioembolic stroke.

The baseline characteristics of the subjects with cardioembolic or non-cardioembolic infarction are presented in Table 1. The subjects with cardioembolic stroke were significantly younger than the subjects with non-cardioembolic infarction ($p = 0.02$). Old myocardial infarction was highly distributed at the anteroseptal, anterior, lateral, inferior, and septal left ventricle in the subjects with cardioembolic stroke compared with non-cardioembolic infarction ($p < 0.01$, respectively). There were high cross-over rates at the location of the old myocardial infarction, which was associated with a wider infarction area in the subjects with cardioembolic stroke. The left ventricular ejection fraction was lower and plasma BNP was higher in the subjects with cardioembolic stroke ($p < 0.001$,

respectively). The frequency of patients with left ventricular ejection fraction <40% was higher in the subjects with cardioembolic stroke ($p < 0.001$). The cutoff point of plasma BNP for predicting cardioembolic stroke was set as 206.9 pg/mL based on ROC curve analysis (sensitivity 0.524, specificity 0.851, $\chi^2 = 9.28$, $p = 0.002$, area under curve = 0.635; Figure 2). Premorbid warfarin was used frequently in the subjects with cardioembolic stroke, although its use was not significant (Table 2). The subjects who had PT-INR ≥ 1.6 were only those with non-cardioembolic infarction (Table 1).

To define the predictive factors of cardioembolic stroke, multivariate logistic regression analysis was conducted ($\chi^2 = 35.6$, $R^2 = 0.30$, $p < 0.001$, Table 3). Hypertension and plasma BNP ≥ 206.9 pg/mL were independently associated with cardioembolic stroke.

Adjusting for all factors listed in Table 3, each left ventricular segment with $\geq$ akinesis was evaluated for its association with cardioembolic stroke (Figure 3). Based on these results, the basal-inferior, basal-inferolateral, mid-anterior, mid-anteroseptal, apical-anterior, and apical-septal left ventricles had a high odds ratio.

Discussion

In the present study, the clinical features of cardioembolic stroke due to old myocardial infarction were evaluated. The patients with cardioembolic stroke had high plasma BNP levels at their admission. Additionally, a high plasma BNP level was an independent factor for differentiating cardioembolic stroke from non-cardioembolic infarction compared with the left ventricle ejection fraction in the patients with old myocardial infarction. Additionally, the prevalence of left ventricular wall motion abnormality and its association with cardioembolic stroke were different among the segments.

In the present study, the patients who had cardioembolic stroke were younger than the patients who had non-cardioembolic infarction. The duration until their stroke onset

from myocardial infarction events was slightly shorter in the patients with cardioembolic stroke (8.3 ± 3.3 years) compared with the patients with non-cardioembolic infarction (11.3 ± 1.2 years, $p=0.40$; data not shown because the medical records of 63.6% of the patients with myocardial infarction onset were not in our hospital). This shorter onset of stroke from myocardial infarction event could explain the younger onset in the patients with cardioembolic stroke.

Premorbid anti-thrombotic treatments included an anti-coagulant in 7% of patients in this study and an anti-platelet in 46%. In the 5 patients who had $PT-INR\geq 1.6$ with warfarin, none was diagnosed as having cardioembolic stroke under the TOAST criteria. Anti-coagulation can decrease ischemic stroke more than 50%, but it increases all bleeds 2.32-fold controlling PT-INR at 2 to 3 in patients with acute coronary syndrome who are followed up for 3 months to 5 years.¹² Approximately 2.5% of patients suffer an ischemic stroke within 6 months of discharge from acute myocardial infarction.¹³ The incidences of myocardial infarction and stroke were 1.44 and 1.38%/year, respectively, in patients with established cardiovascular disease.⁶ Constructing selection strategies for patients at high risk for cardioembolic stroke and preventive strategies for cardioembolic stroke in selected high-risk patients with a history of myocardial infarction is a necessity.

Based on our results, cardioembolic stroke is associated with a high plasma BNP level, independently from a left ventricular ejection fraction $<40\%$.¹⁰ The plasma BNP level could be a useful biomarker for differentiating cardioembolic stroke from non-cardioembolic infarction, due not only to atrial fibrillation but also myocardial infarction.^{14, 15} Additionally, the plasma BNP level is associated with the cardiovascular disease risk including stroke.¹⁶ Therefore, a larger-population study could clarify the utility of the plasma BNP level to predict cardioembolic stroke.

A visible left ventricular thrombus was frequently detected in patients with anterior

or apex infarction with a low ejection fraction within 6 months from their myocardial infarction onset.⁴ Most mural thrombi are detected within 2 weeks after acute myocardial infarction.¹⁷ Such a mural thrombus after myocardial infarction poses an increased risk of embolization.¹⁸ Based on our results, a strong association of a left ventricular wall motion abnormality with cardioembolic stroke was observed at the basal-posterior to inferior and the mid- and apical-anterior to anteroseptal segments. It has been suggested that old myocardial infarction patients with left ventricular wall motion abnormality on those segments could have a higher risk of cardioembolic stroke onset. Conversely, the location of acute myocardial infarction has been reported as anterior in 41.2%, lateral in 4.2%, inferior in 52.9%, and posterior in 1.6%.¹⁹ In our study, the location of old myocardial infarction was distributed almost similarly among these regions. Therefore, a higher distribution of old myocardial infarction in the lateral and posterior areas was observed in our study compared with previous epidemiological studies. This distribution difference could be a result of the selection bias of stroke patients with old myocardial infarction.

This study contains several limitations. First, this study is a cross-sectional study of the patients who were admitted with ischemic stroke conducted in one local high-volume stroke center, which could cause a selection bias. However, with this setting, consistencies of echocardiography evaluation and stroke subtype diagnosis are retained. Second, based on our results, no suggestive treatment can be recommended for preventing cardioembolic stroke in patients with old myocardial infarction because it is a cross-sectional study. To construct preventive strategies of cardioembolic stroke in old myocardial infarction patients, it is necessary to perform prospective randomized studies in selective patients to minimize the risk of cerebral hemorrhage. Third, in the present study, any atrial fibrillation was excluded from analysis to evaluate actual association of particular left ventricle segment asynergy from old myocardial infarction on cardioembolic stroke. However, paroxysmal atrial fibrillation may

not be excluded, because of its diagnosis difficulty.

High plasma BNP levels and left ventricular wall motion abnormalities in the segments perfused with left anterior descending coronary artery or right coronary artery show a high risk for cardioembolic stroke in patients with old myocardial infarction. Considering these factors, it could be possible to more precisely define the risk of cardioembolic stroke and to perform appropriate anti-thrombotic treatments in old myocardial infarction patients. To more accurately associate the risk factors with cardioembolic stroke, performing a prospective study following up patients with old myocardial infarction is necessary.

Acknowledgments

We would like to show our sincere appreciation to Ms. Tomoko Fukushima in the Brain Attack Center Ota Memorial Hospital for her data management support. We also thank Dr. Shiro Nozaki, MD, PhD, FACC (Nozaki Clinic of Cardiology, Takamatsu, Japan) for his meaningful discussions.

This study was supported in part by research grants from JSPS KAKENHI (Grant Number 23590598, 15K08615), the Japan Science and Technology Agency (AS242Z02592P), Bristol-Myers Squibb, the Smoking Research Foundation, and the Tsuchiya Foundation.

Contributors' statement

Naohisa Hosomi, Masaru Kuriyama, and Masayasu Matsumoto were contributed in study design, data analysis, data interpretation, and manuscript construction.

Takeshi Yoshimoto, Yuhei Kanaya, Shuichiro Neshige, Naoyuki Hara, Takahiro Himeno, Ryuhei Kono, Shinichi Takeshima, Yutaka Shimoe, Kazuhiro Takamatsu, and Taisei Ota were contributed in data collection.

Tomoko Ota, Yoshinori Miyamoto, and Kotaro Yasuda were contributed for diagnosing myocardial infarction.

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Figure Legends

Figure 1. Distribution of left ventricular wall motion abnormalities ($\geq$ akinesis) with a 16-segment model.

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Figure 2. ROC curve of plasma BNP for predicting cardioembolic stroke.

Figure 3. Odds ratio of each segment with left ventricular wall motion abnormality ($\geq$ akinesis) associated with cardioembolic stroke.

Age, sex, hypertension, diabetes mellitus, dyslipidemia, left ventricular ejection fraction $<40\%$, brain natriuretic peptide ≥ 206.9 pg/mL, and prothrombin time-international normalized ratio ≥ 1.6 . Segments are surrounded by bold line, where statistically significant ($p < 0.05$).

This figure is constructed based on a figure published previously ²⁰.

Table 1. Baseline characteristics.

	Total (n=143)	Cardioembolism (n=22)	Non-cardioembolism (n=121)	p
Age, year	74.8±11.2	69.7±12.7	75.7±10.8	0.02
Female, n (%)	42 (29.4%)	4 (18.2%)	38 (31.4%)	0.31
Hypertension, n (%)	120 (83.9%)	15 (68.2%)	105 (86.8%)	0.052
Diabetes mellitus, n (%)	61 (42.7%)	11 (50.0%)	50 (41.3%)	0.49
Dyslipidemia, n (%)	91 (63.6%)	14 (63.6%)	77 (63.6%)	1.00
Location of OMI				
Anteroseptal	38 (26.6)	16 (72.7)	22 (18.2)	<0.001
Anterior	47 (32.9)	17 (77.3)	30 (24.8)	<0.001
Lateral	37 (26.1)	12 (54.6)	25 (20.8)	0.003
Posterior	37 (25.9)	7 (31.8)	30 (24.8)	0.60
Inferior	51 (35.7)	15 (68.2)	36 (29.8)	0.001
Septal	48 (33.6)	18 (81.8)	30 (24.8)	<0.001
LVEF, %	57.5±13.4	46.7±15.4	59.4±12.1 (n=120)	<0.001
LVEF<40, n (%)	15 (10.6)	8 (36.4)	7 (5.8)	<0.001
BNP, pg/mL	173.9±275.1 (n=137)	368.8±499.5 (n=21)	138.7±194.7 (n=116)	<0.001
PT-INR	1.02±0.23	1.01±0.12	1.03±0.25	0.82
PT-INR≥1.6, n (%)	5 (3.5)	0	5 (4.13)	1.00

OMI, old myocardial infarction; LVEF, left ventricular ejection fraction; BNP, brain natriuretic peptide; PT-INR, prothrombin time-international normalized ratio.

Table 2. Premorbid anti-thrombotic agent usage.

Anti-thrombotic agent	Total	Cardioembolic	Non-cardioembolic
	(n=143)	(n=22)	(n=121)
Warfarin, n (%)	10 (7.0)	2 (9.1)	8 (6.6)
Antiplatelet, n (%)	66 (46.2)	11 (50.0)	55 (45.5)
Aspirin, n (%)	54 (37.8)	11 (50.0)	43 (35.5)
Thienopyridine, n (%)	24 (16.8)	3 (13.6)	21 (17.4)
Cilostazol, n (%)	6 (4.2)	2 (9.1)	4 (3.3)

Table 3. Multivariate logistic analysis of cardioembolic stroke.

Factors (n=136)	Odds ratio	95% confidence interval	P
Age, per 10-year increase	0.70	0.39, 1.25	0.23
Female	0.25	0.04, 1.16	0.08
Hypertension	0.11	0.02, 0.43	0.002
Diabetes mellitus	0.98	0.29, 3.20	0.97
Dyslipidemia	3.22	0.84, 14.51	0.09
LVEF, <40%	2.13	0.32, 13.11	0.43
BNP, ≥ 206.9 pg/mL	13.88	2.48, 92.21	0.003
PT-INR, ≥ 1.6	0.00	0.00, 1.22	0.07

LVEF, left ventricular ejection fraction; BNP, brain natriuretic peptide; PT-INR, prothrombin time-international normalized ratio.

Figure 1

A. Four Chamber

B. Two Chamber

C. Long Axis

D. Base

E. Mid

F. Apex

Figure 2

Figure 3

A. Four Chamber

B. Two Chamber

C. Long Axis

D. Base

E. Mid

F. Apex