

Segmental left ventricular hypokinesia is associated with embolic signals in transcranial Doppler

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KEY WORDS

cardiogenic stroke,
cerebral
microembolism,
ultrasonography

ABSTRACT

INTRODUCTION Global left ventricular (LV) hypokinesia is considered to be the cause of stroke, while the significance of segmental wall motion abnormalities is still unknown.

OBJECTIVES The aim of the study was to determine the frequency of embolic signals in the middle cerebral artery in patients with segmental LV wall hypokinesia in the course of coronary artery disease (CAD) with and without stroke.

PATIENTS AND METHODS The study included 68 patients (aged 21–87 years) with segmental LV wall hypokinesia (33 patients without and 35 with stroke). The reference group comprised 30 patients (aged 43–76 years) with atherosclerotic risk factors; the control group comprised 37 healthy volunteers (aged 41–71 years). All subjects underwent echocardiography and carotid and transcranial Doppler ultrasound with the detection of microembolic signals.

RESULTS Embolic signals were observed significantly more often in patients with segmental LV wall hypokinesia than in the control and reference groups. In patients with CAD and stroke, an increased number of embolic signals (≥ 3) was observed significantly more often compared with patients without stroke. Compared with other locations, interventricular septum and apex hypokinesia was associated with a higher frequency of embolic signals in the middle cerebral arteries.

CONCLUSIONS Segmental LV motion abnormalities in the form of hypokinesia in patients with CAD are associated with the presence of embolic signals in the middle cerebral arteries, which may have clinical significance as a risk factor for stroke.

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INTRODUCTION Cerebral artery embolisms of cardiac origin constitute a clinically significant risk factor for stroke.¹ The well-established causes of cardiogenic cerebral embolism are atrial fibrillation, mitral stenosis, mechanical heart valves, recent myocardial infarction, atrial myxoma, or the presence of thrombus in the left ventricle (LV). The relative causes include mitral valve prolapse, mitral ring calcification, patent foramen ovale, interatrial septal aneurysm, aortic valve calcification, atherosclerotic plaques in the aortic arch, and segmental LV wall motion abnormalities.² The risk of cardiogenic cerebral infarction is still controversial in these cases, so the diagnosis of any of the above conditions is

not considered to be an indication for treatment with anticoagulants.

The available data suggest a relationship between advanced impairment of LV systolic function and the occurrence of a cardiogenic embolism.³ It is assumed that the presence of LV hypokinetic segments may also be conducive to the occurrence of thrombus.⁴ There is still little data on the subject and it is difficult to explain this issue due to the co-occurrence of other probable causes of an embolism.⁵

The assessment of embolic signals (high-intensity transient signals – HITS) by transcranial Doppler ultrasonography may be helpful in establishing a causative relationship between

cardiogenic stroke and segmental LV wall motion abnormalities. The relationship between the presence of HITS and the occurrence of a clinically apparent stroke is well-recognized.⁶⁻¹⁰ HITS can be identified in most patients with stroke of embolic etiology within 48 hours from the onset of the disease.¹¹ Their occurrence has been recognized as a risk factor for another cerebral ischemic event.¹⁰

The presence of HITS was first confirmed in patients with a cerebral embolism and the absolute risk factors for the disease,^{12,13} namely, in 55% of the subjects with artificial valves and in 21% of those with atrial fibrillation.¹³

Of note, only few publications are related to the detection of HITS in conditions that are considered to be relative causes of a cardiogenic cerebral embolism.¹⁴ These conditions include segmental LV wall motion abnormalities in the form of hypokinesis. In addition, strokes often occur in patients with hypokinesis, hence our interest in this topic.

The aim of the study was to assess whether the presence and location of LV wall hypokinesis in the course of coronary artery disease (CAD) are related to the incidence of embolic signals in patients with and without stroke.

PATIENTS AND METHODS A total of 68 patients with CAD and LV hypokinesis were included into the study. Patients were divided into 2 groups: group 1 including 33 patients without the clinical symptoms of stroke (9 women and 24 men aged 21–71 years; mean, 53.7; standard deviation [SD], 10.85 years), and group 2 including 35 patients in the acute phase of an embolic stroke (9 women and 26 men aged 37–87 years; mean, 62.7; SD, 12.6 years). The reference group comprised 30 patients (aged 43–76 years) with atherosclerotic risk factors. The control group consisted of 37 healthy volunteers matched for age and sex (23 women and 14 men aged 41–71 years; mean, 54.1 years; SD, 8.98 years).

The inclusion criteria for groups 1 and 2 were as follows: stable CAD previously confirmed by noninvasive and invasive tests; segmental LV motion abnormalities in the form of hypokinesis diagnosed using transthoracic echocardiography (TTE). In addition, for group 2: cardioembolic stroke on the basis of the criteria by Adams et al.,¹⁵ including heart disease associated with an absolute or relative risk of embolism, exclusion of carotid and cerebral artery stenosis based on ultrasonography, hypodense focus in head computed tomography (CT), confirmation of a stroke by head CT. The accepted age range was from 35 to 85 years.

The exclusion criteria were as follows: LV motion abnormalities in the form of akinesia / dyskinesia, LV systolic dysfunction (LV ejection fraction <50%), severe LV dilatation, history of stroke, significant valve disease (grade II/III), implanted prosthetic valves, cardiac arrhythmia, atherosclerotic lesions in the aortic arch observed on plain

chest X-ray, thrombus in the left atrium or ventricle, and stroke in mechanism associated with the Valsalva maneuver. The study was approved by the Bioethical Committee of the Medical University of Silesia. All study participants were informed about the objective of the study and provided written consent.

Medical history of all subjects was recorded and physical examination was performed to collect data on CAD duration, comorbidities, etc. The presence and location of hypokinesis (groups 1 and 2) of the LV were analyzed by TTE. The 17-segmental model of LV division was used. According to the location of hypokinesis, the following regions were considered in the analysis: interventricular septum (IVS; segments: 1, 6, 7, 12, 13), anterior wall (segments: 4, 10, 16), lateral wall (segments: 2, 8, 14), and posteroinferior wall (segments: 3, 5, 9, 11, 15) as well as the apex (segment 17). Transesophageal echocardiography (TEE) was performed in patients with bad acoustic window, in whom TTE did not allow for an objective analysis. Also, 12-lead electrocardiography and Doppler examination of the intra- and extracranial arteries were performed.

Cerebral embolism in the middle cerebral artery (at a depth of 60 mm for 30 minutes on each side) was detected in the first 24–48 hours of the disease in patients with stroke, during hospital stay in patients without symptoms of stroke, and on an outpatient basis in the control group.

The total number of embolic signals in an hour and their intensity were determined in each patient. The detected signals met the accepted Spencer criteria.¹⁶

The statistical analysis was conducted using the Statsoft Statistica 7.1 software. During the first stage of the analysis, the basic descriptive statistics (arithmetic mean, median, quartiles, variance, SD, etc.) were determined. The χ^2 test for independence or the Fisher's exact test was used to compare the distribution of nonparametric variables. Parametric variables were compared using either the *t* test (age – normal distribution) or the Mann-Whitney *U* test (duration of CAD that did not meet the condition of normal distribution). The null hypothesis related to the conformity of a given parameter to normal distribution was assessed using the Kolmogorov-Smirnov or Shapiro-Wilk tests. The accepted level of statistical significance (type I error) was set at a *P*-value less than 0.05.

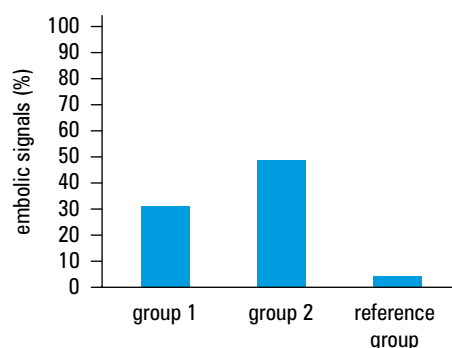
RESULTS Clinical characteristics of the participants Patients in group 1 were significantly younger (*P* = 0.0024) compared with those in group 2. The mean age of patients in groups 1 and 2 did not differ significantly compared with the control group.

The duration of CAD varied from 6.2 to 21.4 years (mean, 13.8 years). In group 1, it ranged from 6.2 to 10.4 years (mean, 7.9 years; SD, 2.6); in group 2, it ranged from 1 to 21.4 years

TABLE 1 Location of left ventricular hypokinesia in groups 1 and 2

Location of LV hypokinesia	Group 1, n = 33	Group 2, n = 35	P
IVS, n (%)	18 (58.1)	10 (28.6)	0.03
anterior wall, n (%)	0	2 (5.7)	NS
lateral wall, n (%)	5 (16.1)	12 (34.3)	NS
posteroinferior wall, n (%)	1 (3.2)	2 (5.7)	NS
apex, n (%)	7 (22.6)	9 (25.7)	NS

Abbreviations: IVS – interventricular septum, LV – left ventricular, NS – nonsignificant

FIGURE Presence of embolic signals in groups 1 and 2 and in the reference group**TABLE 2** Number of embolic signals in groups 1 and 2

		Group 1, n = 33	Group 2, n = 35
HITS, n (%)		10 (30.3)	17 (48.6)
patients with, n (%)	1 signal	4 (12.1)	2 (5.7)
	2 signals	6 (18.2)	9 (25.7)
	3 signals	0 (0)	5 (14.3)
	7 signals	0 (0)	1 (2.9)
	1–2 signals	10 (30.3)	11 (31.4)
	3–7 signals (Fisher's test, $P = 0.047$)	0 (0)	6 (17.1)

Abbreviations: HITS – high-intensity transient signals

(mean, 9.2; SD, 11.2) years. The differences were not significant.

Myocardial infarction was reported in 53 patients: 23 patients (74.2%) in group 1 and 27 patients (77.1%) in group 2.

The following comorbidities were identified: arterial hypertension in 39 patients (16 in group 1 [51.6%]; 23 in group 2 [65.75%]; nonsignificant); type 2 diabetes in 22 subjects (9 in group 1 [29%]; 13 in group 2 [37.1%]; nonsignificant); lipid metabolism disorders in 23 patients (1 in group 1 [3.2%]; 22 in group 2 [62.8%]; $P < 0.001$).

In group 2, 11 patients had a partial anterior circulation infarct, 6 had a total anterior circulation infarct, 8 had a lacunar infarct, and 10 had a posterior circulation infarct.

In the acute phase of stroke, patients were treated with aspirin or intravenous heparin (for progressive stroke) according to the recommendations of the Polish Society of Neurology. Patients in group 2 took acetylsalicylic acid in a daily dose of 75 mg.

Location of left ventricular hypokinesia Hypokinesia of the IVS was significantly more common in group 1 than in group 2 ($P = 0.030$). The incidence of other locations of LV hypokinesia did not differ between the groups (TABLE 1).

Incidence of embolic signals Embolic signals were detected in 26 patients (39.4%): 9 patients (29.0%) in group 1, 17 patients (48.6%) in group 2, and 1 patient (3.4%) in the reference group. No embolic signals were detected in the control group (FIGURE).

Embolic signals occurred in patients with segmental LV wall motion abnormalities (groups 1 and 2) significantly more often than in the control group. The difference between groups 1 and 2 was not statistically significant.

Number of embolic signals In group 1, only 1 or 2 HITS per patient were recorded. In the other group, 1, 2, 3, or 7 signals were identified (TABLE 2). An increased number of embolic signals (3–7) was observed significantly more often in group 2 than in group 1 ($P < 0.05$).

Embolic signals and the location of wall motion abnormalities In group 1, embolic signals were detected in patients with IVS and apex hypokinesia. No HITS were observed in patients with hypokinesia of the lateral and posteroinferior walls (TABLE 3).

In group 2, the frequency of embolic signals was similar in patients with septal abnormalities and those with the abnormalities of the LV posteroinferior wall.

DISCUSSION The results of our study show that the presence of a microembolism is associated with mild LV wall motion abnormalities in the form of hypokinesia. HITS were detected

TABLE 3 Location of left ventricular hypokinesia and incidence of embolism in groups 1 and 2

Location of LV hypokinesia	Groups 1 + 2, n = 68		Group 1, n = 33		Group 2, n = 35		P
	total	with embolic signals, n (%)	total	with embolic signals, n (%)	total	with embolic signals, n (%)	
IVS	28	13 (46.4)	18	7 (38.9)	10	6 (60)	NS
lateral wall	17	5 (24.9)	5	0	12	5 (41.6)	NS
anterior wall	2	0	0	0	2	0	NS
posteroinferior wall	3	1 (33.3)	1	0	2	1 (50)	NS
apex	16	7 (43.7)	7	2 (28.6)	9	5 (55.5)	NS

Abbreviations: see TABLE 1

in almost 40% of the subjects with CAD and wall motion abnormalities. HITS were also observed in patients with a recent stroke and stable CAD (48.6% and 29%, respectively); however, the difference was not statistically significant. On the other hand, HITS were not detected in the control group.

This observation is significant because the presence of HITS is a risk factor for the occurrence of ischemic stroke.¹⁷⁻¹⁹ The available data suggest that in individuals with different risk factors for stroke, embolic signals occur more often in those who have suffered from cerebral ischemia compared with asymptomatic individuals. A similar trend was observed in our study.

The relatively high incidence of HITS observed in our patients without embolic stroke is interesting. Similar results were reported by Sliwka et al.²⁰ who monitored the middle cerebral artery in patients without previous neurological events. They observed HITS in 36% of the patients with CAD, ejection fraction above 30%, and the presence of akinesia or hypokinesia of at least 3 segments in a 16-model division of the LV. A study with similar assumptions revealed the presence of HITS in 25% of the patients without stroke, who had been referred for standard echocardiography.¹⁰

The presence of HITS in patients with acute ischemic stroke depends on the time of the measurement.^{11,21-23} Grosset et al.¹¹ observed HITS in 71% of the patients within 48 hours from the onset of cerebral ischemia. We also assessed cerebral embolism in the first 24 to 48 hours after stroke and observed a similar incidence of HITS (48.6%).

The incidence of embolic signals in patients with stroke depends on the etiology and the potential co-occurrence of several risk factors for stroke.^{20,24} In a study of patients with ischemic stroke or a transient ischemic attack and cardiac diseases with a different embolic potential, the highest number of HITS was detected in subjects with mitral valve vegetations (100%).²² In the cases of atrial fibrillation and an akinetic segment of the LV, HITS were present in 33% of the patients. According to the available data on patients with cardiac diseases associated with a low risk of cardiogenic embolism, the highest incidence of HITS was reported in patients with mitral ring calcification (33%).²² Detection rate of HITS in our group with embolic stroke was similar to that reported by Kuznetsow et al.²⁴ They examined patients with cardiogenic stroke with 1 cause of a cardiogenic cerebral embolism.

A number of reports have emphasized the incidence of HITS in patients with a recent myocardial infarction and LV wall motion abnormalities, particularly if they are accompanied by thrombus or impaired ejection fraction.¹⁷ There are only a few studies reporting the relationship between segmental LV wall motion abnormalities and stroke. In a report by Deleu et al.,²⁵ a higher incidence of lacunar stroke was observed in patients with segmental LV wall motion abnormalities, but this

group was older and had a number of other potential risk factors for embolism.²⁵ It should be noted that the exclusion criteria in our study allowed us to analyze the presence of HITS in patients with segmental LV wall motion abnormalities that could be the only possible cause of cerebral embolism.

A quantitative analysis showed a significantly higher number of embolic signals in the group of patients with stroke. A difference in the number of embolic signals observed in 1 patient with HITS was also demonstrated. From a practical perspective, an increase in the number of embolic signals to 3 in a patient with segmental LV wall motion abnormalities should be regarded as a warning against embolic stroke.

Our results are in line with similar studies in which an increase in the number of signals in cardiac diseases is associated with a higher risk of embolism.²⁶

We did not demonstrate statistically significant differences in the location of hypokinetic segments between the groups with and without stroke, but our findings may suggest such a relationship. In patients without stroke, HITS were detected only in patients with hypokinesia of the IVS or the apex. In patients with stroke, as many as 55.5% of the patients with apical hypokinesia and 60% of those with IVS hypokinesia had embolic signals and their incidence was higher than that in patients with a different location of wall motion abnormalities. Of note, wall motion abnormalities of the apex and IVS seem to be significant predisposing factors for thrombus formation. This finding concerns primarily patients with myocardial infarction.²⁷ Atrial fibrillation is also associated with thrombus formation, which may be the source of embolic material for cerebral circulation.^{28,29} Our results seem to be in agreement with the view that not only the qualitative and quantitative characteristics of HITS but also the location of embolic material formation are risk factors for cardiogenic stroke.²⁶

A relatively small number of subjects is a limitation of our study. However, the optimal selection of the groups allowed us to choose relatively similar subjects and thus to draw conclusions regarding segmental LV hypokinesia as a risk factor for the occurrence of HITS.

Previous studies that evaluated the incidence of embolic signals in the acute phase of an ischemic stroke differ in the assessment of this phenomenon. The diversity of results probably depends to a large extent on the time since the onset of stroke and its etiology. Not all patients underwent TEE but other parameters, such as sinus rhythm and correct dimensions of the left atrium, suggested that there was no thrombus in the left atrial appendage. Patients with stroke were older than patients without stroke and the control group. Considering that age is a risk factor for both atrial fibrillation and stroke, this could be an additional factor that contributes to cerebral ischemia.^{30,31}

In conclusion, segmental LV wall motion abnormalities in the form of hypokinesis in patients with CAD are associated with the presence of embolic signals in the middle cerebral artery, which may have clinical significance as a risk factor for stroke.

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Związek między odcinkowym zaburzeniem kurczliwości lewej komory pod postacią hipokinezy a sygnałami zatorowymi w przezczaszkowym badaniu dopplerowskim

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SŁOWA KLUCZOWE

kardiogeny udar
mózgu,
ultrasonografia,
zatorowość mózgowa

STRESZCZENIE

WPROWADZENIE Globalną hipokinezę lewej komory serca uznaje się za przyczynę udaru mózgu, natomiast znaczenie odcinkowych zaburzeń kurczliwości serca jest wciąż nieustalone.

CELE Celem pracy było określenie częstości sygnałów zatorowych w tętnicy środkowej mózgu u chorych z odcinkowymi zaburzeniami kurczliwości lewej komory w przebiegu choroby wieńcowej bez oraz z udarem mózgu.

PACJENCI I METODY W badaniu udział wzięło 68 chorych (w wieku 21–87 lat) z odcinkowymi zaburzeniami kurczliwości lewej komory (33 bez objawów udaru mózgu i 35 chorych z zawałem mózgu). Grupa odniesienia liczyła 30 pacjentów (43–76 lat) z czynnikami ryzyka miażdżycy, a grupa kontrolna – 37 zdrowych ochotników (41–71 lat). U każdego wykonano echokardiografię, ultrasonografię tętnic szyjnych oraz przezczaszkowe badanie dopplerowskie z rejestracją mikrozatorowości w tętnicach środkowych mózgu.

WYNIKI Sygnały zatorowe występowały znamienne częściej u chorych z odcinkowymi zaburzeniami kurczliwości lewej komory w porównaniu z grupą kontrolną i odniesienia. U pacjentów z chorobą wieńcową oraz udarem mózgu w porównaniu z pacjentami bez udaru mózgu znamienne częściej stwierdzono zwiększoną liczbę sygnałów zatorowych (≥ 3). Zaburzenia kurczliwości przegrody międzykomorowej oraz koniuszka serca w porównaniu z zaburzeniami kurczliwości o innej lokalizacji wiązały się z częstszym występowaniem sygnałów zatorowych w tętnicach środkowych mózgu.

WNIOSKI Odcinkowe zaburzenia kurczliwości lewej komory serca w postaci hipokinezy u pacjentów z chorobą wieńcową są związane z występowaniem sygnałów zatorowych w tętnicach środkowych mózgu, co może mieć znaczenie kliniczne jako czynnik ryzyka udaru mózgu.

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