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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Cerebral embolism in the perioperative period in patients post interventional treatment of carotid artery stenosis: a preliminary report

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Abstract

Background: During carotid revascularisation, embolic material originating from unstable atherosclerotic plaques and thrombi forming within the stent is generated. The significance of a cerebral embolism in the first days after restoring the patency of the arteries has not been established.

Aim: To evaluate the occurrence of microembolic signals in the middle cerebral artery on the day preceding, and on the second day after, endarterectomy and angioplasty with stenting of the ipsilateral internal carotid artery.

Methods: The study included 44 patients (mean age: 68.46 ± 6.2 years; 28 men and 16 women) in whom endarterectomy (31 patients) or stenting (13 patients) were performed due to internal carotid artery stenosis ≥ 70%. All of the patients had their history taken and underwent physical as well as neurological examinations; they also had complete blood count tests, transcranial ultrasound, duplex Doppler ultrasound of carotid and vertebral arteries including evaluation of the degree of stenosis and the morphology of atherosclerotic plaques. In addition, cerebral embolism in the middle cerebral artery was detected in them in the period preceding the intervention and on the second day after the intervention.

Results: The incidence of microembolic signals before and after the procedure was not significantly different in patients who underwent endarterectomy and stenting (38% and 53.5%, respectively, $p > 0.05$). There was no occurrence of stroke, and the periprocedural frequency of other neurological events did not differ significantly between patients depending on the type of revascularisation. In patients with symptomatic carotid artery stenosis, compared to individuals with asymptomatic stenosis, microembolic signals were detected significantly more frequently on the second day (63.2% vs. 28%, $p = 0.0197$) after the intervention. Furthermore, in these patients, microembolic signals of a frequency > 10 dB were found significantly more often (63.2% vs. 8%, $p = 0.0001$). In patients with microembolic signals of intensity > 10 dB, elevated thrombocyte counts were observed significantly more frequently compared to patients with lower intensity signals (80% vs. 25.6%, respectively, $p = 0.01$). Patients with symptomatic carotid stenosis significantly more frequently used statins prior to hospitalisation (84.3% vs. 52%, $p = 0.0256$). Symptomatic internal carotid artery stenosis (55.2% and 20.0%, respectively, $p = 0.02$) and microembolic signals after intervention (55.2% and 20.0%, respectively, $p = 0.02$) were observed significantly more frequently in patients using statins in the preoperative period than in patients not taking these medications.

Conclusions: 1. Microembolic signals in the middle cerebral artery detected with the use of transcranial ultrasound examination in the early period after carotid revascularisation of the internal carotid artery are usually asymptomatic. 2. Cerebral embolism in the early period after carotid revascularisation is more frequently found in patients with symptomatic carotid artery stenosis subjected to a repair procedure. 3. The clinical significance of cerebral emboli found after carotid revascularisation requires further research.

Key words: carotid revascularisation, cerebral embolism

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INTRODUCTION

Cerebral microembolic signals (MES), examined using transcranial Doppler ultrasonography, are often recorded during carotid endarterectomy (CEA) and carotid artery stenting (CAS). Available publications indicate a possible link between emboli and adverse effects of the above-mentioned procedures [1–4].

The sources of microemboli generated during the procedures to restore patency in the carotid artery may include unstable atherosclerotic plaques [1, 5–7] and thrombi potentially formed on the surface of unstable atherosclerotic plaques or within the stent [8]. Embolisation is encouraged by artery dissection during endarterectomy as well as by the procedures related to the introduction of neuroprotection and stent implantation [1, 9]. Previous studies have suggested that both the morphology of atherosclerotic lesions and the method of revascularisation have a significant impact on the generation of emboli. The results of the CREST study indicate that the incidence of clinically overt cerebral strokes is comparable among patients subjected to CEA and CAS [10]. However, stenting is associated with more frequent incidents revealing symptoms of transient ischaemic attack (TIA) or reversible ischaemic neurological deficit, probably partly resulting from a drop in arterial pressure and hyperperfusion syndrome [11]. The observations concerning increased risk of embolic complications in the group of patients undergoing CAS have also been confirmed by other authors [12–15]. The use of neuroprotection in the case of intravascular procedures and antithrombotic treatment (including anti-aggregation treatment) is to reduce neurological complications. On the basis of high-sensitivity neuroimaging examination (diffusion-weighted imaging — DWI), it has been shown that these methods do not protect completely against embolisation of cerebral vessels [13, 16]. In the available literature are numerous reports related to the results of early and late interventional treatment for carotid artery stenosis and cerebral embolism during CEA or CAS. On the other hand, very few studies relate to the monitoring of embolism in the first days after revascularisation. It is important to discover whether carotid artery revascularisation has an impact on the process of emboli generation (recorded using transcranial ultrasound imaging) in the middle cerebral artery in the immediate period after the procedure.

The aim of this study was to evaluate the occurrence of embolic signals in the middle cerebral artery on the preceding day and on the second day after CEA and CAS of ipsilateral internal carotid artery (ICA).

METHODS

The study included 44 patients (mean age 68.46 ± 6.2 years; 28 men and 16 women) who underwent endarterectomy (Group I: 31 patients) or stenting (Group II: 13 patients) due to ≥ 70% stenosis of the ICA in the Department of General and Vascular Surgery, Medical University of Silesia in Katowice. 19 patients had symptomatic stenosis (previous cerebral stroke, reversible ischaemic neurological deficit [RIND] or TIA within six months before revascularisation); the remaining patients reported no clinical signs of cerebral ischaemia before being qualified for revascularisation. All revascularisation procedures in symptomatic patients were performed in the period over 14 days from the ischaemic episode (from 21 days to six months).

The following criteria for exclusion from the study were adopted for the study protocol: (1) carotid artery stenosis after radiation or surgical treatment; (2) potential causes of cerebral embolism other than atherosclerotic lesions in the carotid artery: atrial fibrillation, mitral valve stenosis, aortic valve stenosis, mechanical heart valve, acute myocardial infarction, patent foramen ovale or another leak within the heart chambers, interatrial septal aneurysm, hypo/akinesia of the cardiac walls, cardiac cavity myxoma, or the presence of thrombus in the left ventricle or atrium; (3) occlusion of the contralateral carotid artery.

All patients were rated for the presence of comorbidities, including arterial hypertension, ischaemic heart disease (IHD), diabetes, chronic limb ischaemia, chronic renal failure and lipid metabolism disorders. During the period before surgery, patients were consulted cardiologically and neurologically. In the process of qualification to the surgery, all patients underwent duplex Doppler ultrasound of carotid and vertebral arteries using the 7.5 MHz linear probe and Elegra, SIEMENS US machine, along with the assessment of the degree of stenosis and atherosclerotic plaque morphology. The degree of stenosis was rated by the NASCET criteria [17], and plaque morphology was based on the Gray-Weale classification and evaluation of the Greyscale Median (GSM) after normalisation of ultrasound images [18]. Intracranial arteries were examined by the Doppler method to evaluate haemodynamic parameters of arterial flow and register MES (on the day preceding the surgery and on the second day after CEA or CAS). Registration of cerebral embolism was conducted in the middle cerebral artery (for 30 min at a depth of 55–60 mm on the side of the artery which was subjected to the corrective procedure). The incidence of all embolic signals was evaluated, then the signals were divided into MES ≤ and > 10 dB. The signals of higher frequencies were found in order to check whether they carried a higher risk of cerebral ischaemia in the course of carotid artery stenosis. A Pioneer 2002 TC device by EME, equipped with a 2 MHz head and a two-way head to register cerebral embolism, was used in the test. The registered embolic signals met the Spencer criteria [19].

CEA qualification included patients with highly calcified atherosclerotic plaques at the site of stenosis, lesions of very low echogenicity (GSM < 25), lesions with present thrombus or suspected thrombus within the lumen of the vessel or on plaque surface, with a difficult vascular access into the

carotid artery (e.g. kinking), and no vascular access within the peripheral vessels.

The indications for CAS as a treatment of choice included: high location of the ICA stenosis, high division of the common carotid artery, previous surgical procedures within the neck, and a short neck.

Endarterectomy was performed by eversion under loco-regional or local anaesthesia after previous administration of 5,000 U of heparin. Intraoperatively, none of the patients required the use of temporary cerebral flow (shunt). Heparin was administered by continuous infusion in all patients within the first 24 h after surgery; starting on the second postoperative day, acetylsalicylic acid (ASA) 150 mg + low molecular weight heparin in a prophylactic dose were routinely used. The CAS procedure was performed after prior saturation of the patient for a few days with ASA preparations (1 × 150 mg) and clopidogrel (1 × 75 mg) or by direct administration of 300 mg of clopidogrel and 300 mg of ASA. All patients hospitalised for carotid artery stenosis were admitted to the Department of General and Vascular Surgery in the course of treatment with ASA preparations (75–150 mg) and received such treatment until the day before surgery inclusive. 2,500 U of heparin were administered during the procedure; a distal neuroprotection system was used in each case. The procedure was performed with a typical femoral approach using selective predilatation for tight stenosis. After the procedure, each patient was observed in the post-operative intensive care department and re-evaluated by a neurologist. The pressure dressing was removed after 12–24 h.

During the preoperative and postoperative period (14 days after surgery, the first ambulatory check-up), the patients were assessed for potential neurological complications (cerebral stroke, RIND, TIA) and regional complications (wound haematoma or haematoma in the area of vascular access).

The following were analysed: the age and sex of the patient, the occurrence of selected conditions (IHD, chronic lower limb ischaemia, diabetes, arterial hypertension, past neurological incident caused by carotid artery stenosis), treatment with statins, the presence of embolic signals before and after the intervention (also with a frequency > 10 dB) and potential postoperative complications (of neurological nature: cerebral stroke, RIND, TIA, others), and local complications related to the intervention. The characteristics described above were compared between the groups according to the type of revascularisation, neurological symptoms associated with past stenoses, the presence of embolic signals in the period before and after the intervention, and the use of statins in therapy. The patients came from various sites, which is the explanation for the non-heterogeneous therapies, and were likewise various in terms of statin use.

Leukocyte and thrombocyte count was also evaluated; the incidence of elevated leukocyte and thrombocyte count was also compared among the patients with present and absent MES, and in patients with MES of a frequency of ≤ 10 dB and > 10 dB. The following reference ranges were adopted: for leukocytes: 4.00–10.00 × 103/μL, and for platelets: 135–350 × 10^3/μL.

Table 1. Characteristics of patients undergoing endarterectomy and stenting

Parameter	CEA (n = 31)	CAS (n = 13)	P (test χ^2)
Age*	68.77 ± 6.2	67.0 ± 7.0	0.4088*
Female	12 (38.7%)	4 (30.8%)	0.6174
Symptomatic stenosis	16 (51.6%)	3 (23.1%)	0.0813
Past MI	15 (48.4%)	6 (46.2%)	0.8924
CAD	5 (16.1%)	2 (15.4%)	0.9509
PAOD	9 (29.0%)	7 (53.8%)	0.1185
Diabetes mellitus	10 (32.3%)	2 (15.4%)	0.2515
Arterial hypertension	30 (96.8%)	7 (53.8%)	0.0004
Statin therapy	21 (67.7%)	8 (61.5%)	0.6921

*Student's t-test; CEA — carotid endarterectomy; CAS — carotid stenting; CAD — coronary artery disease; MI — myocardial infarction; PAOD — peripheral atherosclerotic obstructive disease

Chi-squared test and Student's t test were used for statistical analysis; the level of statistical significance for statistically significant differences was adopted for the value of $p < 0.05$.

RESULTS

When comparing Groups I and II, there was no statistically significant difference in the occurrence of neurological symptoms within the period of six months preceding the intervention (51.6% vs. 23.1%, respectively; Table 1), which could also result from such a small number of study patients. There were no significant differences in the prevalence of concomitant diseases, except arterial hypertension (in the group of patients undergoing endarterectomy: 96.8% and stenting: 53.8%, $p = 0.0004$; Table 1).

The incidence of embolic signals before and after surgery did not differ significantly between Groups I and II (CEA: 38% vs. CAS: 53.5%; $p > 0.05$; Table 2).

Clinically overt neurological symptoms (TIA, transient worsening of the existing post-stroke paresis) within 14 days after the coronary artery revascularisation procedures were observed in five patients after CEA (16.1%) and in one patient (7.7%) after CAS ($p = 0.45$) (Table 2). Cerebral strokes were not found in the short-term observation period in both groups. There was no difference in the incidence of complications other than neurological complications (Table 2).

In patients with symptomatic carotid artery stenosis, compared to those with asymptomatic stenosis, embolic signals recorded on the second day after the intervention were

Table 2. Characteristics of patients undergoing endarterectomy and stenting in the periprocedural period

Parameter	CEA (n = 31)	CAS (n = 13)	P (test χ^2)
MES^{-1}	12 (38.%)	7 (53.8%)	0.3551
MES^{+2}	16 (51.6%)	3 (23.1%)	0.0813
PNS	5 (16.1%)	1 (7.7%)	0.4569
C	4 (12.9%)	0	0.1744
MES > 10 dB	10 (32.3%)	4 (30.8%)	0.9229

CEA — carotid endarterectomy; CAS — carotid stenting; MES^{-1} — microembolic signals one day before intervention; MES^{+2} — microembolic signals on 2nd day after intervention; PNS — postoperative neurological symptoms; C — non neurological postoperative complications

Table 3. Characteristics of patients with symptomatic stenosis (SS) and asymptomatic stenosis (AS)

Parameter	SS (n = 19)	AS (n = 25)	P (test χ^2)
Age*	70.05 ± 5.7	66.88 ± 6.7	0.1047*
Female	7 (36.8%)	9 (36%)	0.9541
Coronary artery disease	9 (47.4%)	12 (48%)	0.9669
Chronic kidney disease	4 (21.1%)	3 (12%)	0.4161
PAOD	6 (31.6%)	10 (40%)	0.5652
Diabetes mellitus	1	7 (28%)	0.9011
Arterial hypertension	17 (89.5%)	20 (80%)	0.3948
Statin therapy	16 (84.2%)	13 (52%)	0.0256
MES^{-1}	8 (42.1%)	11 (44%)	0.9000
MES^{+2}	12 (63.2%)	7 (28%)	0.0197
PNS	2 (10.%)	4 (16%)	0.6002
C	0	4 (16%)	0.0675
MES > 10 dB	12 (63.2%)	2 (8%)	0.0001

*Student's t-test; PAOD — peripheral atherosclerotic obstructive disease; MES^{-1} — microembolic signals one day before intervention; MES^{+2} — microembolic signals on 2nd day after intervention; PNS — postoperative neurological symptoms; C — non neurological postoperative complications

significantly more frequent (63.2% vs. 28%, p = 0.0197), and embolic signals of a frequency of > 10 dB were found significantly more often in those patients (63.2% vs. 8%, p = 0.0001) (Table 3).

These differences were observed despite the fact that patients with symptomatic stenosis of the ICA significantly more often used statins before hospitalisation (84.3% vs. 52%, p = 0.0256; Table 3). There were no statistically significant differences between the group of symptomatic patients and asymptomatic patients in terms of age, sex, the prevalence of comorbid conditions (IHD, chronic lower limb ischaemia, diabetes, arterial hypertension) and intervention complications (Table 3).

There were no statistically significant differences between the occurrence of abnormal thrombocyte and leukocyte count in patients with present embolic signals and the ones without the signals. However, an increased thrombocyte count was observed significantly more often in patients with MES > 10 dB vs. patients with MES of lower intensity (80% vs. 25.6%, respectively; p = 0.01). No significant differences were shown in the incidence of abnormal leukocyte count between the patients with MES of ≤ 10 dB and the patients with MES > 10 dB (45.5% vs. 75.8%, respectively; p = 0.06).

Among the patients taking statins during the preoperative period, compared to patients not taking them, symptomatic stenosis of the ICA (55.2% and 20.0%, respectively; p = 0.02) and embolic signals after the intervention (55.2% and 20.0%, respectively; p = 0.02) were observed significantly more often despite no differences in the frequency of embolic signals before the surgery in the group of patients taking or not taking statins). The patients using statins were significantly older then the patients in the comparison group (69.72 ± 5.1 vs. 65.4 ±7.8, respectively; p = 0.03). Comparing the groups of patients using statins preoperatively and the patients who did not, there were no significant differences in the following parameters provided in the study protocol: the patient's sex, the incidence of selected diseases (IHD, chronic lower limb ischaemia, diabetes, arterial hypertension), the type of procedure, the presence of embolic signals on the preceding day and the occurrence of post-surgical complications.

DISCUSSION

Microembolic signals in patients with carotid artery stenosis are considered to be one of the markers of atherosclerotic plaque instability [20]. The aim of restorative procedures on carotid arteries is to improve the haemodynamic conditions of intracerebral circulation and eliminate the potential source of cerebral embolisation, which is atherosclerotic plaque. However, patency-restoring procedures are associated with the risk of embolic complications; the available evidence suggests the presence of MES reported during the procedure [21]. The clinical significance of MES in the perioperative period after carotid endarterectomy is undetermined [13, 14, 22–24]. However, there are suggestions that cognitive disorders may be a remote consequence of MES [25].

In the present study, we did not observe any correlation between the occurrence of embolic signals and clinically overt cerebral stroke in the early postoperative period. As found by other authors, embolic signals were mostly clinically silent. In studies by Pinero et al. [16], patients in the early period after CEA and CAS revealed mostly asymptomatic fresh ischaemic foci in DWI in the area supplied by the artery subjected to corrective surgery. However, there was no close correlation between the number of embolic signals recorded in transcranial Doppler ultrasonography and the amount of fresh lesions in the brain in DWI [13, 16]. According to various authors, in

17–43% of patients after CAS these were reported in the region supplied by the carotid artery subjected to revascularisation, but they have also been observed in brain regions not directly related to the repaired carotid artery [13, 16, 26–28]. Some authors obtained results indicating a link between the emboli generated during revascularisation, the foci of acute ischaemia in DWI, and fresh cerebral stroke [16, 29].

In the present study, embolic signals were observed on the second day after CEA and CAS. There was no relationship between the type of revascularisation and the frequency or intensity (≤ 10 dB and > 10 dB) of embolic signals. However, MES were reported significantly more often in the postoperative period in patients with symptomatic stenosis than in asymptomatic patients. All procedures were performed with a temporary deferral after acute cerebral ischaemia and the repair mechanisms possibly allowed stabilisation of the plaque causing ICA stenosis. Reassessment of destabilisation during surgery could be the cause of MES occurring significantly more frequently in this group of patients. The instability of atherosclerotic plaques in the ICA wall, expressed clinically as symptoms of cerebral ischaemia, seems to favour the presence of MES also after corrective procedures. The mechanism of emboli generation during the postoperative period is unclear. Its elements may include damage to the arterial wall after CEA and the presence of a stent. The underlying cause of emboli is believed to be platelet adhesion and aggregation, which is why antiplatelet therapy is applied. Clopidogrel reduces the number of emboli as early as during the first day of inclusion; however, it is impossible to tell which mechanism of drug action is more significant: the anti-inflammatory or the anti-aggregation mechanism [30]. An association between an inflammatory condition and atherogenesis, atherosclerotic plaque rupture and thrombosis in the vessels of the heart and brain has been demonstrated [31]. The effect of an inflammatory condition on embolisation is unclear. Embolic material generated during the procedure comprises plates, cholesterol, lipid-filled macrophages, fibrin and endothelial cells [32]. Among others, the composition may suggest that the inflammatory process plays a role in embolisation. According to Aronow et al. [31], a high leukocyte count found before revascularisation is a predictor of emboli generation during the procedure, which emphasises the important role of inflammatory condition. Nasr et al. [33] observed MES in patients with symptomatic stenosis of the ICA and a high neutrophil count. In the present study, there was no significant correlation between an abnormal number of leukocytes and thrombocytes and a more frequent generation of embolic signals. However, there was a relationship between an increase in leukocyte count and the presence of MES intensity > 10 dB. The mechanisms responsible for destabilisation of atherosclerotic plaques causing ICA narrowing and the occurrence of clinically overt emboli can probably be responsible for the incidence of MES in the early period after revascularisation.

Cerebral ischaemia depends on the size of embolism and hypoperfusion distally from the site of vessel stenosis [29]. Perfusion disorders play a special role during CEA. The passage of emboli along the vessels, especially with impaired cerebral perfusion, may damage the endothelium and consequently activate leukocytes initiating the inflammatory process. Impaired perfusion and cerebral autoregulation observed in patients with significant stenosis of the ICA makes it difficult to 'wash out' the emboli, which can also promote their presence in the first days after the procedure [34].

In the present study, the use of statins did not significantly affect the reduction of embolic signals. Statins were taken especially by people with symptomatic stenosis who significantly more often reported embolic signals in the first days after revascularisation.

Identification of risk factors for embolism during carotid revascularisation may significantly optimise the methods of treatment, including pharmacological therapy, during the preparatory period and after the procedure.

Limitations of the study

The authors are aware of the limitations of the present study, the results of which should be verified in a larger group of patients. An important element likely to affect a better understanding of the formation of microembolisms in the perioperative period may also be simultaneous monitoring of MES signals in the middle cerebral artery on the opposite side and in other cerebral arteries. Modification of hypolipemic treatment and the study-adopted regimen of antiplatelet therapy differing among the groups may also influence the results.

CONCLUSIONS

1. Embolic signals in the middle cerebral artery recorded by transcranial ultrasound test in the early period after restoring patency in the ICA are usually asymptomatic.
2. Cerebral embolism in the early period after carotid revascularisation is often found in patients with symptomatic carotid artery stenosis subjected to surgical treatment.
3. The clinical significance of cerebral emboli found after carotid artery revascularisation requires further research.

Conflict of interest: *none declared*

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Zatorowość mózgowa w okresie okołooperacyjnym u chorych po interwencyjnym leczeniu zwężenia tętnic szyjnych: doniesienie wstępne

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Streszczenie

Wstęp: Podczas zabiegów udrażniających tętnicę szyjną generowany jest materiał zatorowy, którego źródłem są niestabilne blaszki miażdżycowe i zakrzepy formujące się w obrębie stentu. Embolizacji sprzyja preparowanie tętnicy w trakcie endarterektomii i zabiegi związane z wprowadzeniem neuroprotekcji oraz implantacją stentu. Wskazuje się na możliwy związek między występowaniem sygnałów zatorowych rejestrowanych w ultrasonograficznym (USG) badaniu transkranialnym podczas interwencji a niekorzystnymi następstwami rewaskularyzacji szyjnej. Nieustalone jest natomiast znaczenie zatorowości mózgowej w pierwszych dobach po udrożnieniu tętnic.

Cel: Celem badania była ocena występowania sygnałów zatorowych w tętnicy środkowej mózgu w dobie poprzedzającej i drugiej po endarterektomii oraz stentowaniu tożstronnej tętnicy szyjnej wewnętrznej.

Metody: Do badania włączono 44 pacjentów (średni wiek 68,46 ± 6,2 roku; 28 mężczyzn i 16 kobiet), poddanych endarterektomii (31 chorych) lub stentowaniu (13 chorych) z powodu ≥ 70% zwężenia tętnicy szyjnej wewnętrznej. U wszystkich przeprowadzono wywiad i badanie internistyczne oraz neurologiczne, badanie morfologii krwi, transkranialne badanie USG, USG *Doppler duplex* tętnic szyjnych i kręgowych, z oceną stopnia zwężenia i morfologii blaszki miażdżycowej oraz rejestrację zatorowości mózgowej w tętnicy mózgu środkowej w okresie poprzedzającym interwencję i w drugiej dobie po interwencji.

Wyniki: Częstość sygnałów zatorowych przed i po zabiegu nie różniła się istotnie u pacjentów poddanych endarterektomii oraz stentowaniu (odpowiednio: 38% i 53,5%; p > 0,05). Nie zaobserwowano udaru mózgu w okresie okołozabiegowym a częstość innych zdarzeń neurologicznych nie różniła się znamiennie między pacjentami w zależności od rodzaju rewaskularyzacji. U osób z objawowym zwężeniem tętnicy szyjnej w porównaniu z chorymi ze zwężeniem bezobjawowym istotnie częściej rejestrowano sygnały zatorowe w drugiej dobie po interwencji (63,2% vs. 28%; p = 0,0197) oraz znamiennie częściej u tych pacjentów stwierdzono sygnały zatorowe o częstotliwości > 10 dB (63,2% vs. 8%; p = 0,0001). U osób z sygnałami zatorowymi o intensywności > 10 dB istotnie częściej obserwowano podwyższoną liczbę trombocytów w porównaniu z pacjentami, u których rejestrowano sygnały o niższej intensywności (odpowiednio 80% vs. 25,6%; p = 0,01). Chorzy z objawowym zwężeniem tętnicy szyjnej wewnętrznej znamiennie częściej stosowali statynę przed hospitalizacją (84,3% vs. 52%; p = 0,0256). Wśród pacjentów stosujących w okresie przedzabiegowym statyny w porównaniu z chorymi nieprzyjmującymi leku istotnie częściej obserwowano zwężenie objawowe tętnicy szyjnej wewnętrznej (odpowiednio 55,2% i 20,0%; p = 0,02) i sygnały zatorowe po interwencji (odpowiednio 55,2% i 20,0%; p = 0,02).

Wnioski: 1. Sygnały zatorowe w tętnicy środkowej mózgu rejestrowane w transkranialnym badaniu USG we wczesnym okresie po udrożnieniu tętnicy szyjnej wewnętrznej najczęściej są bezobjawowe. 2. Zatorowość mózgową we wczesnym okresie po rewaskularyzacji szyjnej częściej stwierdza się u pacjentów z objawowym zwężeniem tętnicy szyjnej poddanej zabiegowi naprawczemu. 3. Znaczenie kliniczne zatorów mózgowych rejestrowanych po rewaskularyzacji szyjnej wymaga przeprowadzenia dalszych badań.

Słowa kluczowe: rewaskularyzacja szyjna, zatorowość mózgowa

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