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Prospective observation of neurological symptoms attributable to cerebral hyperfusion syndrome after CEA and CAS

Prospektywna analiza objawów neurologicznych towarzyszących zespołowi hiperperfuzji mózgowej po CEA i CAS

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Abstract

Introduction: Neurological symptoms are considered as most clinically significant symptoms with various pathogenesis, including cerebral hyperperfusion syndrome (CHS) and microembolism, in patients shortly after endarterectomy (CEA) and stenting (CAS) for internal carotid artery stenosis (ICA).
Aim: This study aimed to compare the structure of neurological symptoms attributable to CHS after carotid artery revascularization collected retrospectively and prospectively in large patient cohorts.
Material and methods: Prospective analysis included 1047 consecutive patients treated with CEA (n = 477) or CSA (n = 570) in a single centre from 2011 to 2015. In 2012 was introduced strict monitoring of pain in patients with headache and blood pressure (BP) and more intensive antihypertensive treatment in patients with an increase in BP post-procedure. The occurrence of neurological symptoms attributable to CHS was compared with a historical, retrospectively analysed less strictly monitored cohort (n = 1386).
Results: Neurological symptoms attributed to CHS were observed less frequently in prospectively than retrospectively analysed cohort: 8.3% vs 10.6% (p = 0.03) of CEA and 5.7% vs 8.0% (p = 0.10) of CAS group, respectively. The profile of neurological symptoms was similar in both cohorts. The prospective analysis revealed more episodes of transient bradycardia and/or hypotension in the CAS group (10.4 vs 8.8% and 11.2 vs 9.2%, respectively).
Conclusion: The incidence of neurological symptoms attributable to cerebral hyperperfusion syndrome after carotid artery revascularization in short-term observation is similar regardless of the method used. Strict monitoring of BP slightly decreased the prevalence of neurological symptoms after carotid artery revascularization.

Key words: cerebral hyperperfusion syndrome (CHS); carotid artery revascularization; carotid endarterectomy (CEA); carotid artery stenting (CAS)

Streszczenie

Wstęp: Objawy neurologiczne spostrzegane jako znaczące o różnej patogenezie włącznie z zespołem przekrwienia mózgu (CHS) i mikrozatorowością u pacjentów w okołooperacyjnym okresie po endarteriektomii tętnic szyjnych (CEA) i ich stentowaniu (CAS) leczonych z powodu zwężenia tętnic szyjnych wewnętrznych (ICA).

Cel pracy: Celem pracy było porównanie struktury objawów neurologicznych towarzyszących CHS po rewaskularyzacji tętnic szyjnych wewnętrznych w obserwacji retrospektywnej i prospektywnej w dużej kohorcie pacjentów.

Materiał i metody: Prospektywna analiza obejmowała 1047 kolejno leczonych pacjentów przyjmujących CEA (n = 447) oraz CAS (n = 570) w jednej klinice w latach 2011–2015. Wprowadzono restrykcyjny monitoring bólu u pacjentów z bólem głowy oraz wysokim nadciśnieniem (AH), stosując intensywne jego leczenie po wykonanej procedurze. Występowanie neurologicznych objawów sugerujących CHS u 1386 pacjentów leczonych przed 2011 rokiem oceniano retrospektywnie na podstawie historii chorób.

Wyniki: Objawy CHS spostrzegano rzadziej w badaniu retrospektywnym w porównaniu z grupą ocenianą prospektywnie: 8,3% *vs.* 10,6 (p = 0.03) po CEA i 5,7% *vs.* 8,0% (p = 0,10) po CAS ocenianym prospektywnie. W badaniu prospektywnym wychwycono więcej epizodów przemijającej bradykardii i nadciśnienia w grupie leczonych CAS (10.4% i 11,2% *vs.* 9,2%) odpowiednio.

Wnioski: Incydenty występowania CHS towarzyszące zespołowi hiperperfuzji po rewaskularyzacji tętnic szyjnych w krótkim okresie pooperacyjnym są metodologicznie jednakowo leczone, dostosowując leczenie nasilenia objawów neurologicznych po rewaskularyzacji tętnic szyjnych.

Słowa kluczowe: zespół przekrwienia mózgu; rewaskularyzacja tętnicy szyjnej; endarteriektomia tętnic szyjnych; stentowanie tętnicy szyjnej

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Introduction

In general endarterectomy (CEA, carotid endarterectomy) [1, 2] and stenting (CAS, carotid stenting) [3, 4] are two equally effective methods of carotid artery stenosis treatment. Carotid artery revascularization is followed by an increase in blood flow by 20–40% over baseline, lasting for several hours, up to 2 weeks [5, 6, 8, 9]. Intracranial vascular bed hyperperfusion (> 100% increase over baseline) related to insufficiency of autoregulatory mechanisms in the chronically hypoperfused vascular bed, with hyperaemia especially in vertebrobasilar circulation, occurs in a subset of patients and a few of them become symptomatic [6–8]. The incidence of CHS is estimated at up to 3% [10–12] with greater prevalence in patients with diabetes, arterial hypertension, coronary artery disease, and decreased left ventricular ejection fraction.

Cerebral hyperfusion syndrome (CHS) is usually manifested by a new onset headache ipsilateral to the carotid revascularization with or without focal neurological deficits and seizures [10]. Neurological symptoms attributable to CHS include eye and face pain, vomiting, confusion, visual disturbances, focal neurological deficits, focal motor seizures and intracerebral or subarachnoid haemorrhage [5]. The low specificity of CHS symptoms may cause their attribution to other post-operative complications such as microembolism, stroke, and hypotension [5].

Recently, the prevalence of neurological symptoms attributed to CHS in 10.6% of patients after CEA and 8.0 % after CAS [13] was reported. The analysis, however, was based on data retrospectively retrieved from medical records, therefore potentially biased by lower detection of less manifested symptoms.

Therefore, this study aimed to compare the structure of neurological symptoms attributable to CHS after carotid artery revascularization collected retrospectively and prospectively in large patient cohorts.

Material and methods

The analysis of the neurological symptoms' occurrence was performed in two cohorts treated for stenosis within an extracranial segment of the internal carotid artery in the Department of General, Vascular Surgery, Angiology and Phlebology in Katowice. The first cohort consisted of 1386 consecutive patients treated between 2005 and 2011, with 625 of them subjected to carotid endarterectomy (CEA) and 761 to percutaneous carotid angioplasty and stenting (CAS); data were extracted from medical records (collected retrospectively) [13]. In the second, 1047 patients cohort, treated from 2011 to 2015, with 477 of them subjected to CEA and 570 to CAS, data were prospectively collected.

In both cohorts, the grade of the carotid artery stenosis was verified by duplex-ultrasound according to NASCET (The North American Symptomatic Carotid Endarterectomy Trial) criteria. Qualification of the patients for the reconstructive procedure was the degree of stenosis > 60% for patients before CABG and > 70% in the remaining patients. The choice of the procedure was based on the analysis of plaque intra-luminal surface (Rutherford classification) [14] and grey-scale median score (GSM) echogenicity score [15]. Those with a score below 25, typical for unstable plaque, were referred to the eversion method of CEA, while the others were to CAS.

Reconstructive procedure

Both eversion CEA and CAS procedures were not changed in the entire period covered by the analysis.

As described previously [13], the eversion CEA procedure was performed in local anaesthesia with maintained verbal contact during the procedure in patients who received acetylsalicylic acid (150 mg daily) and clopidogrel (75 mg daily) for at least two days before it. Before probationary clamping of the common carotid artery, 5,000 U of unfractionated heparin (UFH) was given intravenously. Only in 3 patients, the necessity of shunt during the procedure has occurred.

CAS procedures were performed with distal neuroprotection preferentially by the right femoral artery in the radiology operating theatre. Each patient received intravenously 2,500 U of UFH after cannulation of the femoral artery and 0.5–1.0 mg of atropine directly before stent implantation.

Restrictive post-procedure surveillance was carried out in the intensive care unit (ICU) during the first 24–48 hours post-procedure, initially by an anaesthesiologist (first 2 hours), then by a physician, neurologist and experienced nurse team. During the subsequent days, patients remained under the supervision of a cardiologist and a neurologist, and a qualified team of nurses.

If case of neurological symptoms occurrence, each patient was examined by a neurologist, and Doppler sonography (to exclude thrombosis) and CT were performed twice (the second after 24 h) to exclude stroke.

Data collection

In the first (historical), retrospectively analysed cohort, neurological signs and symptoms were extracted from medical records and nursing documentation as was previously described [13]. Those not related to ischaemic stroke were attributed to hyperperfusion syndrome (CHS) when occurred in a patient with new onset of headache ipsilateral to the carotid revascularization.

In 2012 a program 'patient without pain' with careful supervision of all patients with headache and blood pressure (BP) monitoring and more intensive antihypertensive treatment in patients with an increase in BP post-procedure was introduced to the hospital. In addition, in 2011 prospective monitoring of all neurological symptoms in patients with carotid revascularization was started. That decreased the chance to overlook neurological signs and symptoms in these patients.

Statistical analysis

Statistical analysis was performed using the software package Statistica 11.0 PL (StatSoft Inc., USA). Results are presented as mean values or percentages for CEA and CAS groups, and both cohorts, separately. Chi^2 and t-tests were applied. The value of $p < 0.05$ was considered statistically significant.

Results

Study group characteristics

In both cohorts patients who qualified for CAS were slightly older and had significantly tighter carotid artery stenosis on average. The prevalence of past stroke episodes, peripheral artery disease (symptomatic claudication) and diabetes was greater, and coronary revascularization procedures were smaller in both CEA and CAS groups. While the prevalence of hypertension and chronic kidney disease was significantly higher in the retrospectively analysed CEA group, only (Table I).

The prospectively analysed CEA group had a significantly greater prevalence of coronary revascularisation procedures than the retrospectively analysed group. While prospectively analysed CAS group greater percentage of past myocardial infarcts (Table I).

Table I. Characteristics of retrospectively (n = 1386) and prospectively (n = 1047) analysed study groups

	CEA			CAS		
	Retrospective (n = 625)	**Prospective (n = 477)**	**p**	**Retrospective (n = 761)**	**Prospective (n = 570)**	**P**
Age [years]	70 (57–82)	71 (58–84)	0.82	73 (62–83)	74 (64–84)	0.89
Carotid artery stenosis (%)	80 (76–84)	81 (77–85)	0.76	86 (81–91) ^	87 (82–93) ^	0.69
Concomitant diseases (n/%)						
Prior stroke/TIA	176/28.1	135/28.3	0.99	145/19.0 ^	108/18.9 ^	0.98
Coronary artery disease (CAD)	375/60.0	290/60.8	0.84	449/59.0	336 / 58.9	0.97
Past MIC	124/19.8	94/19.7	0.98	165/12.3 ^	90/15.8	0.009
Past PCI/CABG	93/14.8	95/19.9	0.03	158/20.4**	97/17.0*	0.10
Peripheral artery disease (PAD)	137/21.9	104/21.8	0.98	59/7.7 ^	43/7.5 ^	0.96
Hypertension	489/78.2	372/78.0	0.98	395/51.9 ^	463/81.2	< 0.001
Diabetes	70/11.2	50/10.5	0.78	123/16.1**	91/16.0*	0.98
Chronic kidney disease (CKD)	68/11.0	53/11.1	0.98	57/7.4*	41/7.2	0.92

*Defined as serum creatinine concentration > 1.5 mh/dl. Statistical significance between corresponding CEA vs CAS — *$p < 0.05$; **$p < 0.01$; ^ $p < 0.001$

Table II. Neurological and cardiological symptoms noticed in the first hours after the procedure in prospectively and retrospectively analysed groups

	CEA			CAS		
	Retrospective (n = 625)	Prospective (n = 477)	p	Retrospective (n = 761)	Prospective (n = 570)	p
Paraesthesia in contralateral extremities	60 (9.6%)	39 (8.2%)	0.48	54 (7.1%)	32 (5.5%)	0.33
Ptosis of unilateral lips	31 (5.0%)	20 (4.1%)	0.65	4 (0.5%) ^	2 (0.3%) ^	0.95
Buccal trembling	8 (1.3%)	6 (1.2%)	0.81	13 (1.7%)	10 (1.7%)	0.88
Weakening muscular strength in the contralateral upper limb	39 (6.2%)	29 (5.9%)	0.99	46 (6.0%)	34 (5.9%)	0.96
Weakening muscular strength in the contralateral lower limb	29 (4.6%)	22 (4.6%)	0.90	49 (6.4%)	31 (5.4%)	0.61
Epilepsy	7 (1.1%)	3 (0.6%)	0.60	14 (1.8%)	10 (1.7%)	0.93
Bradycardia	8 (1.3%)	7 (1.4%)	0.99	62 (8.8%) ^	79 (10.4%) ^	0.001
Hypotonia	8 (1.3%)	7 (1.2%)	0.99	49 (9.2%) ^	64 (11.2%) ^	0.003

Statistical significance between the corresponding CEA vs CAS — ^ $p < 0.001$

Neurological symptoms attributable to CHS

In both cohorts the prevalence of past stroke and/or transient ischaemic attack (TIA) [n = 321 (23.1%) and n = 243 (23.2%)] and neurological deficits evaluated by the neurologist before revascularization procedure [n = 234 (16.9%) and n = 184 (17.5%)] was similar.

Neurological symptoms attributed to CHS were observed less frequently in prospectively than retrospectively analysed cohort: 8.3% (n = 39) vs 10.6% (n = 66) of CEA and 5.7% (n = 32) vs 8.0% (n = 61) of CAS group, respectively. The decline in frequency was statistically significant in CEA cohorts (p = 0.03). The were no significant differences in the frequency of neurological symptoms attributed to CHS in the combined groups of CEA and CAS (8.9 vs 6.9%, p = 0.10).

The most common neurological symptom that was observed between 2 and 12 hours after the procedure was numbness of the opposite limb paraesthesias. The symptom was reported with similar frequency in both retrospective groups (9.6% after CEA and 7.1% after CAS) and the prospective groups (8.2% after CEA and 7.1% CAS). A weakening of the muscle strength of the opposite limbs was also frequently stated with equal frequency in CEA and CAS in prospective and retrospective groups (Table II).

Epileptic attacks were reported in 34 patients with nearly similar frequency in both retrospectively and prospectively analysed study groups (Table II). The difference in the frequency of falling lips was greater in both CAS and CEA groups (Table II). Calculated related risk (RR) for falling lips in the combined cohorts was 10.27 (95CI: 4.42–23.83) in CEA compared to CAS.

Symptoms attributed to sympathetic nervous system dysregulation

Both episodes of transient bradycardia and/or hypotension were observed more frequently in prospectively than retrospectively analysed CAS cohort (Table II). The differences in the transient bradycardia between CAS and CEA groups in the prospectively analysed cohorts were even greater (10.4% vs. 1.4%) than in the retrospectively analysed cohort frequency (8.8% vs 1.3%, respectively). The calculated RR for the combined cohorts was 7.78 (95CI: 4.60–13.17) for transient bradycardia and 6.24 (3.66–10.62) for hypotension in CAS compared to CEA.

Post-procedural stroke

Neurological deficit in patients up to the 30th-day post procedure with documented stroke in CT was observed with similar frequency in retrospective (22 cases — 3.5% after CEA and 18 patients — 2.4% after CAS), and prospective cohort (18 patients — 3.7% after CEA and 11 — 1.9% after CAS).

More than half of strokes had ischaemic aetiology (33 and 21, respectively). In one-third of the patients with stroke (N = 23) symptoms of hemiparesis subsided without permanent neurological deficit.

Fatal outcome

During the first 30 days in the retrospective (n = 1386) group, 10 patients died (1.6%) in the CEA group and 6 patients (0.8%) in the CAS group. In the prospective analysis (n = 1047) similar frequency of death was observed 14 in total, including 9 in CEA (1.8%) and 5 in the CAS group (0.8%). All death episodes were in patients with very high cardiovascular risk (with a recent history of stroke, myocardial infarction, abdominal aorta aneurysm or peripheral revascularization).

Discussion

The present prospective analysis, in line with the previous retrospective analysis [13], reports a similar incidence of neurological symptoms (focal deficits and seizures) attributed to CHS that occurred during the first 12 hours after the revascularization procedure, regardless of the method of carotid revascularization used. In this prospective analysis, the bias related to the retrospective design of the authors' previous study was eliminated [13]. It was expected that some mild neurological symptoms could not be recorded in medical and nursing records. As the procedures of revascularization remain unchanged in the period spanning the whole analysis, the slightly lower

incidence of the neurological symptoms (significant only in CAS) in the prospectively analysed cohort might be related to the growing experience of the authors' centre.

The present data demonstrate with high statistical power related to the study size, that neurological symptoms attributable to CHS develop equally frequently after CEA and CAS within the first 12 hours post-procedure. A similar incidence of CHS with neurological symptoms after CEA and CAS was previously reported by Ogasawara et al. [16]. It seems that co-morbidity and risk factors for CHS and more important and can be explained between study variability [17]. In addition, the hypothesis that prognosis and outcomes in CHS are worse after CAS than after CEA cannot be supported [18, 19].

So far reported incidences of CHS in patients after carotid endarterectomy vary in a wide range from 0.2 to 18.9%. The differences, at least partially result from the criteria CHS applied. A more restrictive definition obviously diminishes the incidence of neurological symptoms potentially attributable to CHS [20–23]. The exclusion of stroke or transient ischaemic attack after revascularization may further decrease CHS incidence. Some authors suggest that neurological symptoms after carotid artery revascularization are more common than the incidence of CHS [17, 24–27]. In the present study, the documented stroke in CT may explain neurological symptoms in less than half of patients (40 from 105 after CEA, 39 and 93 after CAS). There is, however, a grey zone of uncertainty, as strokes related to microembolism might not be diagnosed with available imaging technics [28–31].

The spectrum of neurological symptoms attributable to CHS was similar in both cohorts analysed in the present study. In both cohorts, the incidents of unilateral falling of the lips were greater after CEA [RR = 10.27 (95%CI: 4.42–23.83)], which probably result from the irritation of branches in the ramus mandibularis of the facial nerve during the procedure, and is not related to CHS. There are no literature data concerning the risk of this adverse event after carotid revascularization.

In addition to symptoms of CHS, the authors analysed the incidence of episodes of transient bradycardia and/or hypotension. Both were related to stimulation of carotid baroreceptors during catheter instrumentation and stent placement [26, 32, 33], and therefore more frequently observed after CAS than CEA group, with similar frequency in prospectively and retrospectively cohorts. The calculated RR was 10.27 (95%CI: 4.60–13.17) for transient bradycardia and 6.24 (3.66–10.62) for hypotension in CAS compared to CEA.

It is worse to be stressed that the prevention of CHS is difficult. According to some authors, the basic and most important element of CHS prevention is careful systemic blood pressure control [34–38]. In line with this recommendation, strict monitoring of blood pressure, pulse rate, blood oxygenation (finger oximetry) and severity of pain was started, tailoring analgesics and antihypertensive drugs. This strict supervision started in 2012 covering the majority of patients in the prospective cohort only slightly decrease the frequency of neurological symptoms attributable to CHS.

The authors still did not control the haemodynamic changes post-procedure routinely, and thus could not determine which neurological symptoms which occurred were accompanied by at least a doubling in cerebral blood flow in patients with new onset of headache ipsilateral to the carotid revascularization, that is the main limitation of this study. The authors would like to acknowledge that the analysis of symptoms of CHS was difficult in the group of patients with a history of stroke, neurological deficits and uncontrolled arterial hypertension. Moreover, distinguishing symptoms caused by post-procedure hyperperfusion and microembolism was not possible.

In conclusion, the incidence of neurological symptoms attributable to cerebral hyperperfusion syndrome after carotid artery revascularization in short-term observation is similar regardless of the method used. Strict monitoring of BP slightly decreases the prevalence of neurological symptoms after carotid artery revascularization.

Conflict of interest

None declared

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