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Changes in Cognitive Functions after Carotid Endarterectomy and Carotid Stenting: A Decade-Apart Comparison

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Abstract: Background: This study investigates cognitive function changes in severe carotid stenosis patients undergoing carotid endarterectomy (CEA) and carotid stenting (CAS), across two decades. Methods: We compared cognitive function within 30 days post-procedure in 267 patients (first 100 each for CEA and CAS in two periods: 2008–2012 and 2018–2022) at a single institution. Assessments used Adenbrooke's Cognitive Examination–Revised (ACE-R), Mini-Mental State Examination (MMSE), Speech Fluency Test (SFT), and Clock Drawing Test (CDT), conducted before and 30 ± 2 days after surgery. Results: Patients (mean age 67.2 years, 70%+ carotid stenosis) exhibited different cognitive changes over the periods. In 2008–2012, significant declines in MMSE (CEA, $p = 0.049$) and CDT (CAS, $p = 0.015$) were noted among asymptomatic patients. Conversely, in 2018–2022, improvements were observed in ACE-R and MMSE for both symptomatic and asymptomatic patients undergoing CEA and CAS. Conclusion: Over a decade, advancements in interventional techniques and patient management have reduced cognitive decline risks in asymptomatic carotid stenosis patients, also improving cognitive functions in both symptomatic and asymptomatic individuals.

Keywords: carotid stenosis; carotid endarterectomy; carotid artery stenting; cognitive functions

1. Introduction

Internal carotid artery (ICA) stenosis is among the most common causes of ischemic stroke,[1] and the risk of ischemic stroke increases in proportion to the severity of ICA stenosis.[2] In line with current recommendations, interventional treatment, such as surgical carotid endarterectomy (CEA) or intraluminal carotid angioplasty with stenting (CAS), are indicated for specific patients with ICA stenosis greater than 50%.[3-5] Both interventions are considered to be relatively safe and effective, with estimated risks of perioperative morbidity and mortality being less than 6%.[6,7] Moreover, peri-operative stroke and death rates are less than 2% for asymptomatic stenosis cases and less than 4% for symptomatic stenosis cases.[8] However, it is important to note that CAS is still associated with a higher risk of silent cerebral infarctions.[9] According to a recent meta-analysis, diffusion-weighted magnetic resonance imaging (DW-MRI) detected new ischemic cerebral lesions significantly more often after CAS than after CEA, with rates of 40.3% and 12.2% respectively.[10] Furthermore, these brain infarctions, although often asymptomatic at the time of clinical onset, may contribute to an increased risk of future cognitive impairment and dementia.[11]

Still, the effect of carotid endarterectomy and stenting on cognitive function remains uncertain. The pathophysiological basis for cognitive dysfunction is believed to be microembolization originating from an atherosclerotic plaque (parts of the plaque or attached thrombus) or from hypoperfusion caused by arterial stenosis.[12-19]

As a result, following the removal of arterial stenosis, an improvement in cognitive functions can be anticipated. Conversely, cognitive deterioration may also occur during the operation due to the exacerbation of hypoperfusion when clamping the artery or as a result of microembolizations during the procedure. [20-24]

The attempt at a meta-analysis of studies on CAS (Carotid Artery Stenting) and CEA (Carotid Endarterectomy) and their effects on cognition included 13 studies, involving 403 patients that underwent CEA and 368 patients that underwent CAS. In most studies, no changes were observed in cognitive tests. However, a meta-analysis could not be performed due to the limited number of patients, with the sample sizes ranging from 26 to 129 patients. Additionally, there was notable heterogeneity in the neuropsychological tests conducted, the timing of these tests, and the duration of patient follow-up. [19]

The study by Maggio et al. also evaluated the impact of new DWI (Diffusion-Weighted Imaging) lesions, which were found to have a detrimental effect on cognition. Once again, a limitation was the small number of patients, with only 37 patients involved.[25]

The Sonobuster study revealed a statistically significant reduction in DWI lesions following CAS and CEA with intraoperative sonolysis. However, the study did not indicate a significant impact of DWI lesions on cognitive functions in either the group with sonolysis or the group without it. It is worth noting that the study did not have an adequate sample size to reach statistical significance for this particular outcome.[26]

Advances in surgical techniques and stenting devices for CEA over the past several decades have improved the prognosis of patients with carotid stenosis. In the current study, we examined the progress made over a decade through a retrospective analysis of carotid interventions recorded in a single hospital registry.

The primary aim of this study was to compare the differences in cognitive test outcome following CEA and CAS within individual decades. The secondary objectives were to determine whether CEA and CAS result in alterations in cognitive functions and to make a comparison of changes in cognitive tests between patients who underwent CEA and those who underwent CAS.

2. Methods

This retrospective analysis adhered to the principles outlined in the Declaration of Helsinki (1964) and its subsequent amendments, including the most recent one in 2013. All procedures were performed in accordance with both national and institutional guidelines. The study was approved by the Ethics Committee of the University Hospital in Ostrava (approval nos. MZ10-FNO and 497/2017). Data from patients who signed an informed consent for the anonymous use of their hospital registry records for scientific purposes were exclusively included in this analysis.

Out of a total of 400 patients who underwent carotid endarterectomy, the initial 50 consecutive patients in each group who had cognitive tests performed both before and after the intervention were selected. The records of the initial 32 consecutive patients with symptomatic stenosis in P1 and 39 in P2 were included. Furthermore, the records of the first 35 consecutive patients with asymptomatic carotid stenosis in P1 and 35 in P2, who were treated by CEA, were also incorporated. Additionally, the records of the first 28 consecutive symptomatic patients in P1 and 29 in P2, as well as the records of the first 33 consecutive asymptomatic patients in P1 and 36 in P2, who were treated by CAS, were included. In total, 267 patients were recruited for this study from the University Hospital in Ostrava over the two periods (January 2008 to December 2012 [P1] and January 2018 to December 2022 [P2]). These patients met the following criteria: 1) carotid stenosis ≥ 70% and indications for CEA or CAS

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according to current guidelines[7,27-29] 2) underwent preoperative and postoperative cognitive tests, and 3) signed informed consent.

2.1. Carotid endarterectomy

During the first study observation period (P1), carotid endarterectomy was performed under general anesthesia by an experienced neurosurgeon with a minimum of 5 years of experience and a record of at least 25 CEA procedures annually. A longitudinal skin incision was made along the anterior edge of the sternocleidomastoid muscle to expose the common carotid artery and its two major branches, namely the internal and external carotid arteries. Subsequently, 100 IU per kg body weight of unfractionated heparin was administered and after 3 minutes, blood flow was arrested by clamping. The ipsilateral middle cerebral artery was continuously monitored using transcranial Doppler (DWL MultiDop T1, DWL, Sipplingen, Germany) to determine the need for shunt insertion. Longitudinal arteriotomy was then performed to conduct endarterectomy of the common and internal carotid arteries and plaque removal. Following plaque extraction, the artery was sutured and the clamp released. Protamine sulfate (1 mL/1000 IU of heparin) was administered 5 minutes after flow was restored. A thorough homeostasis check was performed, a Redon drain was inserted, and the wound was anatomically closed. Patients were transferred to the Intensive Care Unit, and after 24 hours, brain MRI and duplex ultrasound of the ICA were performed.

During the second period (P2), several changes were made to the surgical procedure. Specifically, (i) a transverse skin incision replaced the longitudinal incision, (ii)systemic blood pressure target values were individually predefined based on mean blood flow velocity in the ipsilateral middle cerebral artery, as monitored by transcranial Doppler, (iii) the anticoagulation strategy was adjusted to 7,000 IU heparin preoperatively for patients weighing ≥ 80 kg and 5,000 IU for patients weighing < 80 kg, and (iv) protamine sulfate was not administered. The surgical team remained unchanged in both periods.

2.2. Carotid percutaneous transluminal angioplasty with stenting

Patients indicated for CAS underwent diagnostic and interventional procedures using digital subtraction angiography. The procedure was performed through the femoral approach by an experienced interventional radiologist with a minimum of 5 years of experience and a record of at least 25 CAS procedures annually. All patients were prescribed long-term aspirin (100 mg/day) and received a 525-mg loading dose of clopidogrel. A dose of 10,000 IU of unfractionated heparin was administered at the beginning of the intervention. Whenever technically feasible, a distal cerebral protection device (FilterWire EZ™; Boston Scientific, Natick, Massachusetts, USA) was used during the procedure. Following the insertion of the filter/wire through the area of the stenosis, pre-dilatation of the stenosis was performed as required using a balloon, followed by the insertion of the stent. The selection of the carotid stent and other specific interventional strategies were determined by the judgment and the expertise of the interventional radiologist. After pre-dilatation, if deemed necessary, an appropriate stent (such as the Carotid Wall Stent Boston Scientific Corp., Natick, MA, USA) was implanted for each stenosis. Based on the angiographic findings, any remaining stenosis within the stent was addressed by further dilation with a balloon, after which the protection device was subsequently removed. Following the procedure, a follow-up angiography was performed, and the results were assessed.

In the second period, all patients were tested for acetylsalicylic acid and/or clopidogrel resistance. Additionally, the dose of unfractionated heparin at the beginning of the intervention was reduced to 5,000 IU for patients weighing < 80 kg and 7,500 IU in patients weighting ≥ 80 kg. Blood pressure was more precisely controlled through continuous saline solution infusion. Moreover, advanced instrumentation, including a new type of double-layered stent (Roadsaver™ Carotid Artery Double-Layer Stent, Terumo Corporation, Tokyo, Japan) was used. The interventional radiology team remained unchanged in both periods.

2.3. Magnetic resonance imaging

Magnetic resonance imaging was performed both prior to surgery and at 24 ± 4 hours post-surgery, using a 1.5-T Avanto system (Siemens, Erlangen, Germany). The MRI protocol remained consistent with that of a previously published study.[12] Concisely, the protocol included four sequences: (i) T2-weighted spin echo [echo time = 100 ms; repetition time = 4310 ms; section thickness = 5.0 mm; matrix size = 192 × 256; gap = 0.5 mm; field of view (FOV) = 250 mm; FOV ph = 75%; echo train length = 9; number of excitations = 1]; (ii) diffusion-weighted imaging (echo time = 130 ms; repetition time = 4500 ms; b, representing a factor of diffusion-weighted sequences b ¼ 0 and b ¼ 1000 s/mm^2; section thickness = 5.0 mm; gap = 1 mm; matrix size = 192 × 192; FOV = 255 mm; FOV ph = 100%; number of excitations = 4; echo spacing = 0.93 ms; bandwidth = 1240 Hz/Px); apparent diffusion coefficient maps were obtained in all cases; (iii) T2 star-weighted gradient-recalled echo sequence; (iv) fluid-attenuated inversion recovery (FLAIR). The sequences were applied at the same slice thickness and cut number, with a slice thickness consisting of the cut thickness (5 mm) plus a 10% gap. The standard number of slices was 25, positioned at a modified skull base level to minimize distant-artefacts in the echo planar imaging sequence.

The results were evaluated by a radiologist, and in cases of uncertainty, a neurologist was consulted for a review of each scan separately. In instances of divergent findings, a consensus was reached through a discussion regarding the number and volume of lesions. New cerebral ischemic lesions were defined as hyperintensities on post-intervention DW-MRI that were absent on pretreatment DW-MRI.

2.4. Clinical evaluation

Physical and neurological examinations were performed by a board-certified neurologist 24 hours and 30 days after the intervention. Neurological deficits were assessed with the National Institutes of Health Stroke Scale (NIHSS), self-sufficiency with the modified Rankin Scale (mRS), and cognitive functions with the Adenbrooke's Cognitive Examination–Revised (ACE-R), Mini-Mental State Examination (MMSE), Speech Fluency Test (SFT), and Clock Drawing Test (CDT). All tests were performed as per established protocols 12–48 hours before surgery and 30 ± 2 days after surgery.

2.5. Statistical analysis

The sample size for the study was determined by considering an anticipated difference of 4 points in follow-up cognitive test results between intervention in period 1 (estimated 86±5 points) and period 2 (90 points). Calculations indicated that a minimum of 25 patients in each group would be required to detect a significant difference with an α value of 0.05 (two-tailed) and a β value of 0.8.

The normality of data distributions was tested using the Shapiro–Wilk test. Categorical variables are presented as relative frequencies, while continuous variables are reported as mean ± standard deviation. Continuous variables were compared between groups using the Mann–Whitney U-test, while categorical variables were analyzed using the chi-square test for independence in contingency tables. Changes in cognitive test performance scores were compared using the Wilcoxon matched-pairs signed rank test. The significance level was defined as $p < 0.05$ for all tests.

3. Results

Out of 400 patients who underwent carotid intervention in both study periods, the medical records were examined for 267 patients (181 males, mean age 67.2 ± 7.4 years) with carotid stenosis ≥70 % (mean 80.3 % ± 8.6 %) receiving CEA (93 males and 48 females, mean age 66.6 ± 7.7 years) or CAS (88 males and 38 females, mean age 67.8 ± 7.1 years) during the two study periods (2008–2012 [P1] and 2018–2022 [P2]) and completed cognitive tests both prior to and 30 days after intervention were included in the statistical analysis. All demographic data are summarized in Table 1.

Table 1. Demographic data.

	CEA period 1		CEA period 2		CAS period 1		CAS period 2	
	AS	SS	AS	SS	AS	SS	AS	SS

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Number of participants; n	35	32	35	39	33	28	36	29
Age (years); mean±SD; years	65.7±8.5	65.9±6.9	67.6±6.8	67.1±8.7	66.4±7.6	67.1±7.3	68.9±6.7	68.6±6.9
Male sex; n (%)	23 (66)	21 (66)	24 (69)	25 (64)	22 (67)	20 (71)	25 (70)	21 (72)
Right-sided stenosis; n (%)	20 (57)	17 (53)	19 (54)	22 (56)	17 (52)	14 (50)	19 (53)	16 (55)
Severity of stenosis; mean±SD; %	80.1±8.1	80.9±8.7	79.2±.7.9	79.8±9.2	81.1±9.6	80.3±8.9	80.1±7.7	80.6±8.7
Contralateral stenosis >50%; n (%)	12 (34)	10 (31)	11 (31)	13 (33)	15 (45)	11 (39)	14 (39)	11 (38)
Arterial hypertension; n (%)	29 (83)	28 (88)	31 (89)	33 (85)	30 (91)	25 (89)	34 (94)	26 (90)
Diabetes mellitus; n (%)	11 (31)	11 (34)	11 (31)	13 (38)	13 (39)	12 (43)	13 (36)	10 (34)
Hyperlipidemia; n (%)	25 (71)	23 (72)	26 (74)	30 (77)	22 (67)	21 (75)	26 (72)	22 (76)
Coronary heart disease; n (%)	10 (29)	11 (34)	12 (34)	11 (28)	13 (39)	11 (39)	13 (36)	9 (31)
Atrial fibrillation; n (%)	2 (6)	2 (6)	1 (3)	2 (5)	2 (6)	1 (4)	1 (3)	2 (7)
Smoking; n (%)	11 (31)	10 (31)	12 (34)	12 (31)	9 (27)	8 (29)	13 (36)	10 (34)
Alcohol abuse; n (%)	1 (3)	0 (0)	2 (6)	2 (5)	2 (6)	1 (4)	0 (0)	2 (6)
Statin use; n (%)	24 (69)	21 (66)	26 (74)	30 (77)	21 (64)	21 (75)	26 (72)	22 (76)
Antiplatelets; n (%)	32 (91)	30 (94)	34 (97)	38 (97)	33 (100)	28 (100)	36 (100)	29 (100)

AS – asymptomatic stenosis; CAS – carotid angioplasty and stenting; CEA – carotid endarterectomy; SD – standard deviation; SS – symptomatic stenosis, No significant differences were observed in any demographic data between the groups.

In either P1 or P2, no patient with asymptomatic carotid stenosis suffered from a stroke, transient ischemic attack (TIA), myocardial infarction, or vascular death within the 30 days after the intervention. Among patients with symptomatic ICA stenosis undergoing CEA, one patient suffered from stroke in P1 and one patient had a TIA in P1. Similarly, in the CAS group, one patient in P1 and one patient in P2 suffered from a stroke and one patient in P1 had a TIA. Out of the 267 patients, only one patient died within 30 days following the intervention due to a stroke. This patient underwent CEA in P2 and the cause of death was a cancer.

During P1, a significant decline in MMSE score was observed among asymptomatic patients following CEA (p = 0.049), and a similar decline was noted in CDT performance among asymptomatic patients following CAS (p = 0.015) – Table 2?. In P1, no significant differences were found in other cognitive tests between the two groups. However, during P2, no significant cognitive decline was detected by any of the tests in either group.

During the P2 period, a significant improvement was detected in the ACE-R test (p < 0.01) and MMSE (p < 0.05) in both symptomatic and asymptomatic patients after CEA. In both symptomatic and asymptomatic patients after CAS, there was an improvement in the ACE-R test scores (p < 0.01).Additionally, in symptomatic patients, there was also a significant improvement in MMSE scores (p < 0.05). (Table 2).

Table 2. Results of cognitive tests prior to and 30 days after intervention.

	BS	**AS**	**Δ**	**BS**	**AS**	**Δ**	**BS**	**AS**	**Δ**	**BS**	**AS**	**Δ**
	CEA period 1 AS (n=35s)			*CEA period 1 SS (n=32)*			*CEA period 2 AS (n=35)*			*CEA period 2 SS (n=39)*		
ACER; median (IQR); mean	87 (85.5-91.5); 87.2	87 (84.5-91); 86.9	-1 (-1.5-0.5); -0.3	83.5 (70-89); 79.9	83.5 (69-89); 78.8	-0.5 (-3-0); -1.1	87 (77-91); 84.0	91 (82-95.5); 88.0	3 (1.5-7); 4.0#	83 (72.5-89); 80.6	89 (81.5-91.5); 86.5	6 (2-8); 5.9#
MMSE; median (IQR); mean	29 (28-29.5); 28.6	28 (27-29); 27.9	0 (-1.5-0); -0.7*	28 (25-29); 26.6	27.5 (25-29); 26.1	0 (-1-0); -0.5	28 (26-28.5); 27.0	29 (26-30); 27.8	1 (0-2); 0.8*	27 (24-29); 26.1	28 (27-29); 27.6	1 (0-3); 1.5*

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Clock Drawing Test; median (IQR); mean	5 (4-5); 4.1	5 (4-5); 3.9	0 (0-0); -0.2	4 (1-5); 3	4 (0-5); 2.8	0 (0-0); -0.2	5 (4-5); 4.5	5 (4.5-5); 4.6	0 (0-0); 0.1	5 (4-5); 4.3	5 (4-5); 4.6	0 (0-0.5); 0.2
Speech Fluency Test; median (IQR); mean	10 (7.5-11.5); 9.4	10 (8-11); 9.5	0 (0-1); 0.1	6.5 (4-10); 7.1	7.5 (5-10); 7.1	0 (-1-1); 0	10 (8-12); 9.4	10 (9-12.5); 9.9	0 (-1-1); 0.5	10 (7-12.5); 9.3	10 (7-12); 9.5	0 (-1-1); 0.2
	CAS period 1 asymptomatic (33 patients)			*CAS period 1 symptomatic (28 patients)*			*CAS period 2 asymptomatic (36 patients)*			*CAS period 2 symptomatic (29 patients)*		
ACER; median (IQR); mean	87 (77-93); 85.0	88 (76-93); 83.9	0 (-3-1); -1.1	83 (71-90.5); 81.1	82 (68-91.5); 78.6	1 (-2-2); -2.5	87 (76-92); 83.5	89 (83-94); 86.3	3 (1-5); 2.8#	83.5 (80-87); 81.7	88 (84-94); 85.3	3 (2-6); 3.6#
MMSE; median (IQR); mean	28 (26-29); 26.9	28 (25-28); 26.8	0 (-1-1); -0.2	26.5 (25-29); 26.1	27 (25-28); 25.2	0 (0-1); -0.9	28 (25-29); 26.9	28.5 (26-29); 27.4	1 (-1-1); 0.5	28 (25-29); 26.8	28 (27-30); 27.8	1 (0-2); 1.0*
Clock Drawing Test; median (IQR); mean	4 (2-5); 3.2	3 (0-5); 2.6	0 (-1-0); -0.6*	4 (0-5); 2.9	4 (0-5); 2.8	0 (0-0); -0.1	5 (4-5); 4.5	5 (4-5); 4.5	0 (0-0); 0	5 (4-5); 4.3	5 (4-5); 4.5	0 (0-1); 0.2
Speech Fluency Test; median (IQR); mean	10 (6-12); 9.1	10 (5-12); 8.7	0 (-1-1); -0.4	10 (4-11); 8.0	8.5 (5-11); 8.4	0 (-0,5-1); -0.4	10 (8-11); 9.7	10 (8-12); 9.8	0 (-1-2); 0.1	10 (8-12); 9.6	10 (9-12); 9.6	0 (0-1); 0.0

* – $p < 0.05$; # – $p < 0.01$; ACER – Adenbrook´s Cognitive Examination – Revised; CAS – carotid angioplasty and stenting; CEA – carotid endarterectomy; IQR – interquartile range; M – month; MMSE – Mini Mental State Examination.

Both CEA and CAS resulted in an enhancement of cognitive functions as measured by the ACE-R test during the P2 period. However, concerning CAS, improvement in MMSE scores was observed exclusively in symptomatic patients.

4. Discussion

In a previously published study using the same sample of patients, new ischemic lesions on follow-up post-procedure brain diffusion-weighted MRI were significantly less frequent in the CEA group when compared to the CAS group during both P1 (23 *versus* 49 per cent; $P < 0.001$) and P2 (15 *versus* 29 per cent; $P = 0.017$), in the CAS group during P2 when compared with P1 ($P = 0.004$), and in the entire cohort during P2 when compared with P1 ($P = 0.002$). Significant reductions in the risk of new ischemic lesions on follow-up brain DW-MRI were detected after CAS, with rates decreasing from 46% in asymptomatic and 52% in symptomatic stenosis patients during P1 to 26% and to 32%, respectively, during P2. The mean ischemic lesion volume was larger in the CEA group when compared to the CAS group, although this difference did not reach statistical significance ($P > 0.050$).[30]

In line with the significant impact of new ischemic brain lesions of cognition, the reduction in risk of new ischemic lesions on the follow-up brain DW-MRI during the second period correlated with a reduced risk of cognitive decline. In fact, cognitive functions showed improvement during P2. More specifically, patients exhibited significantly higher ACE-R and MMSE scores 30 days after the procedure when compared to their score before the procedure, with the exception of MMSE among asymptomatic patients following CAS. In addition to the expected gradual restoration of cognitive

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function following a stroke among symptomatic carotid stenosis patients, which can be attributed to neuroplastic changes, the observed cognitive improvement may stem from better cerebral hemodynamics following CEA and CAS. Indeed, cerebral blood supply impairment may be considered as an independent and potentially reversible factor that plays a role in determining cognitive decline in patients with severe carotid stenosis.[31] Although, many studies have reported an improvement in the cognitive functions of patient after CAS[32-37] and CEA[38-40], these benefits appear to be restricted to symptomatic patients, presumably with more substantial blood flow disruption. For instance, a recent systematic review concluded that neither CAS nor CEA consistently enhance overall cognitive function in asymptomatic patients (< 2%). This is especially the case for patients who are otherwise at low risk of cognitive decline, even over the long-term with appropriate medical treatment.[41] Compared to interventional treatment, medical treatment does not show improvement in cognitive performance in patient after CAS or CEA in one year follow-up.[42]

Due to its high spatial resolution, DW-MRI is commonly used for the detection of small brain infarcts following CEA and CAS.[43,44] Moreover, DW-MRI-detected cerebral infarcts are often the primary endpoint in recent trials instead of 30-day vascular morbidity and mortality. This shift is due to considerably higher incidence of cerebral infarcts when compared to clinically symptomatic and potentially fatal vascular events, such as transient ischemic attack (TIA) or stroke.[45,46] In addition, these infarcts are associated with a higher risk of cognitive decline or dementia.[11,47-52] Our previous results[30] are consistent with a recently ublished systematic review[9] and a meta-analysis.[53] They all conclude that new ischemic lesions detected on brain DW-MRI are more common after CAS than after CEA, with the incidence ranging from 18% to 58%.[54]

In the second period, there was a significant reduction in the risk of new ischemic lesions detected on the follow up DW-MRI.[30] One possible explanation for this improvement is the greater experience of both CEA and CAS teams. However, interventional procedures were also modified, nd included more precise intraoperative blood pressure management, as well as an increased utilization of antiplatelet treatment or heparin management, and perhaps even a broader improvement in primary and secondary stroke prevention in the Czech Republic.[55] A recent study found that continuous TCD monitoring reduced the risk of new ischemic lesions detected on DW-MRI after CAS or CEA,[56] while neither the type of distal protection[57] nor stent design[58] exhibited similar effects. Furthermore, a radiological study found that the majority of microemboli (particularly the smaller and more distal ones) either showed no manifestation[59,60] or disappeared during long-term MRI follow-up.[61]

We observed a tendency for smaller ischemic lesions after CAS when compared to CEA, although this difference did not reach statistical significance.[30] This observation contrasts with two previous studies that found similar lesion volumes following both procedures.[45,62] One potential explanation for this discrepancy is that during CAS atherosclerotic debris and thrombotic material dislodged could result in multiple small emboli, each affecting only a small volume of brain tissue, whereas CEA may generate fewer emboli but they might involve a larger volume of brain tissue.

Study limitations

There are some limitations to this study. First, the retrospective single-center study design may have introduced selection bias. Thus, the results may not be universally representative of the safety comparison between CAS and CEA. Conversely, the single-center design allows for a direct and unambiguous comparison between the two methods over a decade, as both CAS and CEA intervention teams remain consistent. Secondly, cognitive tests were conducted only one month after the procedure. For a more comprehensive assessment of definitive cognitive changes, a prospective study with a one-year follow-up would be appropriate.

5. Conclusions

Our study showed that improvements in carotid endarterectomy and stenting management, along with a reduction in the frequency of microembolizations, contributed to the prevention of cognitive decline after the procedure in asymptomatic patients. Furthermore, these improvements led to enhanced cognitive functions in both symptomatic and asymptomatic patients.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study. Data from patients who signed an informed consent for the anonymous use of their hospital registry records for scientific purposes were exclusively included in this analysis.

Data Availability Statement: All source data are available from the corresponding author upon request.

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