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Age-Dependent Cognitive Sequelae of Advanced Carotid Disease after Carotid Endarterectomy

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Abstract

Aim: To analyze age-associated cognitive impact of carotid endarterectomy (CEA) in asymptomatic patients diagnosed with severe carotid disease. Our previous research showed that such patients often have subtle cognitive abnormalities detectable by Montreal Cognitive Assessment (MoCA).

Methods: Baseline cognitive status 1-2 months before CEA and cognitive follow-up using MoCA was done in 47 patients 6-12 months after CEA and associations between total cognitive change presented as differences in total MoCA score were examined. Patients were classified as cognitively impaired at MoCA score ≤ 26. Z test was used to test differences in proportions of cognitively impaired and normal participants in four age groups. Cognitive results for four age groups were examined and adjusted for baseline cognitive scores, age, gender and vascular risk factors.

Results: Differences in total MoCA scores were significant in patients 60-69 years of age ($p<0.05$). Following CEA, proportions of cognitively impaired participants were significantly decreased in younger participants (from 45-59, 60-69 and 70-79 years). Variables associated with increased cognitive decline after CEA were older age (OR 0.71, CI 0.612-0.902), hypertension (OR 3.87, CI 0.734-27.332) and ever smoking (OR 3,94, CI 0.759-29.164).

Conclusion: Positive cognitive impact of CEA is not present in older patients. Besides older age, arterial hypertension and ever smoking seem to be additional factors negatively influencing the cognitive benefit of CEA.

Keywords: Cognitive impairment; Carotid stenosis; Carotid endarterectomy; Aging

Introduction

Advanced carotid disease has been recognized as an independent risk factor for cognitive decline, besides being associated with either symptomatic cerebrovascular disease (ipsilateral stroke or TIA) or with silent cerebral infarctions and brain hypoperfusion [1-5]. Although not consistent, results of previous research mostly showed negative impact of advanced carotid disease on cognitive functions, caused by either brain hypoperfusion and/or by microembolizations from unstable carotid plaque [6-9]. Carotid disease exerts further negative repercussions on impact of normal ageing on cerebral autoregulation and neurovascular coupling which are physiological processes essential for vascular brain functions [10]. It is well known that number of patients diagnosed with advanced carotid disese progresses with age. It is diagnosed in 0.7% men and 0.4% women aged 35-39 years, but its incidence increases to 7% in men and 4.3% in women between 75 and 79 years of age [11].

Due to the effect of advances in both prevention and available healthcare worldwide which leads to increased life expectancy and progressive aging of the global population, the number of people with carotid stenosis increased by almost 60% during 2000-2020, with significant risk factors being male sex, ever smoking, diabetes, hypertension and a lower level of HDL [11]. The extent of cognitive impairment that was previously described in patients with advanced carotid disease can differ regarding severity and number of particular cognitive domains affected [2,6-9,12]. This can largely be attributed to differences in cognitive testing methodology used and the differences in sample sizes [2]. In our previous research we have shown that patients with advanced carotid stenosis or occlusion that are considered to be asymptomatic due to the absence of prior ipsilateral stroke or TIA symptoms, often do exhibit subtle cognitive abnormalities which are easily detectable by Montreal Cognitive Assessment (MoCA) [12,13]. We have therefore proposed that the use of MoCA as a brief cognitive screening tool can be recommended as a part of routine clinical assessment in such patients [12]. Although those cognitive abnormalities are mostly mild and might not meet clinical criteria for dementia, if not detected in timely manner, they are subject to steady progression which leads to a pronounced negative impact on both patients quality of life and on public health, followed by detrimental socio-economic consequences [14]. Our initial study also showed positive relationship of particular vascular risk factors and declined cognition in patients with advanced carotid disease, with advanced age, concomitant diabetes and hypertension having the most negative cognitive impact [12]. Carotid revascularization procedures, both carotid endarterectomy (CEA) and carotid stenting (CAS) in patients with severe carotid stenosis are primarily aimed at reducing stroke risk [15,16]. A substantial number of previous studies focusing on cognitive outcomes after both carotid endarterectomy (CEA) or carotid stenting (CAS) produced mixed and sometimes conflicting results [2,17-35]. This can partly be explained by methodological issues, especially the lack of correct assessment of preoperative cognitive status for each patient that should be compared to cognitive status of healthy subjects matched for demographic variables and for vascular risk [2,17-35]. So

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far no standard or specific battery of cognitive tests specifically aiming at the issues related to carotid disease and revascularization has been described [2]. This aids to vast heterogeneity in type of cognitive tests used in research, as well as differences in study designs, methods and timing of testing [2,17, 35].

The aim of our follow-up study was to investigate cognitive outcome of CEA using MoCA and focusing especially on age-associated cognitive effects of carotid endarterectomy (CEA) in asymptomatic patients diagnosed with severe carotid stenosis, which were all previously included in our initial research [12]. Although it is anticipated that patients will show cognitive benefit following CEA, it is not clear from the existing literature what the cognitive outcomes may be for specific subgroups of older patients. Another aim was to further examine the possible impact of vascular risk factors on cognitive outcome after CEA.

Materials and Methods

Initial study

Initial study group included 70 consecutive right-handed patients (26 women; median age 67.5; range 43-85 years) admitted to the University Department of Neurology, Sestre milosrdnice University Hospital in Zagreb from May 2009 until June 2010 and diagnosed with severe ICA stenosis (more than 70%; ICAs or occlusion (ICAo) [12]. Patients were recruited and enrolled after the study was approved by the institutional Ethic Committee and informed consent was obtained. All patients included were stroke/TIA free and reported no subjective cognitive problems. Exclusion criteria for the study were: a history or clinical signs of cerebral/retinal ischemic disorders, malignant diseases, dementia diagnosed according to criteria stated according to DSM-IV, depression disorder diagnosed using DSM-IV criteria and inability to complete cognitive testing due to major sensorial inabilities [12,36]. Our study was restricted to right-handed participants as previously described (12). The degree of carotid stenosis was assessed using Color Doppler Flow Imaging (CDFI) assessment of carotid arteries, performed on commercially available equipment (Prosound SSD-550 and Alfa 10, Aloka, USA) with linear 10 MHz transducer according to previously defined procedure[37,38]. Baseline cognitive testing in all participants was done using Montreal Cognitive Assessment (MoCA) and MMSE test according to the well-defined protocols (12,13,39]. The detailed protocol of our initial study was previously described elsewhere [12]. Advanced ICAs was diagnosed in a total of 61 patients included in the initial study. Advanced stenosis of the left ICA was found in 27 and advanced stenosis of the right ICA in 28 patients, 3 patients had bilateral advanced ICA stenosis, 2 patients were diagnosed with left ICAo and advanced stenosis of the right ICA and one patient with right ICAo and advanced left ICAs. All patients diagnosed with advanced ICAs were subsequently referred to vascular surgeon and in 54 patients CEA was performed while 7 patients underwent carotid stenting (CAS). Due to a small number of CAS patients, only patients after CEA were finally included in the present study. All CEA procedures were done by board-certified and well-experienced vascular surgeons using established surgical techniques (regular or eversion CEA technique and shunt inserting during carotid clamping). Surgical procedures were performed using deep and superficial cervical plexus block and intraoperative neurological monitoring was done. Five out of 54 patients developed transient mild soft tissue swelling, otherwise, no neurological or other significant complications during or immediate post CEA were recorded. During the follow-up period of 12 months after the surgical procedure, non of the patients had cerebrovascular ischaemic symptoms or suffered stroke/TIA.

Follow-up study procedures

Out of 54 patients who underwent CEA a total number of 47 (12 females, median age 68.7, range 45-85 years) were included in the present study. Twenty-two patients had CEA performed on the left and 25 patients on the right ICA. In all patients, cognitive follow-up was performed 6-12 months after CEA using MoCA. MMSE was not used for cognitive follow-up because our initial study showed that MoCA is superior to MMSE in recognizing cognitive abnormalities in stoke/TIA-free patients with severe carotid disease, which corroborated with published results of the original MoCA study [12,13]. For each patient, control cognitive testing was done during the regular individually appointed check-up visits which included routine neurological examination. During check-up visits control neurolosonological exam (CDFI assessment of carotid arteries) was performed in all patients and no significant postoperative restenosis of carotid arteries was found.

Statistical analysis

The demographic and clinical data of patients were analyzed using descriptive statistics. All data were also analyzed for normality using Kolmogorov-Smirnov test. As variables were mostly not normally distributed, data were presented as median (Q1-Q3), age was presented as median (min-max), and non-parametric statistics were used for evaluation. Associations between total cognitive change presented as differences in total MoCA score (initial pre-operative MoCA score and control MoCA score; Δ MoCA) were examined. Patients were classified as cognitively impaired at MoCA score ≤ 26 [13]. A cut-off negative change of pre-operative and post CEA MoCA score of -1.73 points (Δ MoCA ≥ -1.73) was considered clinically meaningful difference pointing to cognitive decline [39,40].

Differences in proportions of cognitively impaired and normal participants in four age groups (from 45 to 59, 60-69, 70-79 and from 80 to 85 years) were tested using z-test and variables were analyzed using non-parametric Mann-Whitney Rank Sum Test. Cognitive results for four age groups were examined and adjusted for baseline cognitive scores, age, gender and vascular risk factors. MoCA subtests scores covering different cognitive domains before and after the CEA procedure were compared.

Unadjusted odds ratios (ORs) with corresponding 95% confidence intervals (CIs) were further calculated in order to examine the associations between total cognitive change (presented as Δ MoCA) and vascular risk factors. Univariate logistic regression was performed using scores as a dependent variable while as independent variables demographic and vascular risk parameters were used. Those variables that were found to be significantly associated with Δ MoCA scores in univariate analysis were then used in multivariate modeling in order to determine further associations between cognitive status change and those variables. For all tests, differences were considered to be statistically significant at $p<0.05$. All statistical procedures were done using the statistical software SigmaStat 3.0, SPSS Inc.

Results

Out of 61 patient that were included in the initial study and diagnosed with advanced ICAs that were subsequently referred to vascular surgeon, in 54 CEA was performed. Two patients refused to continue to participate in the study and 5 patients were lost during follow up for mainly social reasons, so finally a total number of 47 patients (12 females, median age 68.7, range 45-85 years) were included in the present study. Demographic data and clinical characteristics of those patients are presented in Table 1.

Variable		Patients (n=47)
Age/years (median, min-max)		68.7 (45-85)
Age groups/years (proportion, n)	45-59	0.19 (9)
	60-69	0.32 (15)
	70-79	0.21 (10)
	79-85	0.28 (13)
Females (proportion, n)		0.25 (12)
Education/years (mean, SD)		10.25 (2.03)
Hypertension (proportion, n)		0.91 (43)
Diabetes (proportion, n)		0.40 (18)
Hyperlipidaemia (proportion, n)		0.61 (29)
Coronary disease (proportion, n)		0.38(18)
Current smoking (proportion, n)		0.32 (15)
Ex-smoking (proportion, n)		0.40 (19)
Obesity (proportion, n)		0.29 (14)
CEA left (proportion, n)		0.47 (22)
CEA right (proportion, n)		0.53 (25)

Table 1: Demographic and clinical characteristics of patients.

Following CEA, differences in total MoCA scores were significant in all patients ($p<0.05$). Proportions of abnormal total MoCA scores (≤ 26) before and after CEA did not differ significantly in the oldest age group (80-85 years) but were significantly decreased in younger participants (from 45 to 59, 60-69 and from 70 to 79 years; $p<0.05$) (Table 2).

Comparing median cognitive outcomes of CEA, presented as total MoCA score and regarding age groups, we observed that differences in total MoCA scores were significant only in patients from 60 to 69 years of age ($p<0.05$). Patients aged from 45 to 59, 70-79 and from 80 to 85 years did not differ significantly in median MoCA scores when results before and after the procedure were compared (Table 3).

Post CEA, patients performed significantly better at total MoCA score as well as on MoCA subtests of visuospatial and executive functions, attention and delayed recall ($p<0.05$). (Table 4) Overall cognitive results of patients did not differ significantly regarding the side of CEA performed. In multiple logistic regression analysis, variables associated with lower cognitive performance after CEA (presented as Δ MoCA ≥ -1.73) were older age (OR 0.71, CI 0.612-0.902), hypertension (OR 3.87, CI 0.734-27.332) and ever smoking (OR 3,94, CI 0.759-29.164). Those variables remained significant after adjusting for the side of stenosis, gender and vascular risk parameters (Table/data not shown).

	Total MoCA score ≤ 26		p
	Before CEA proportion (n)	After CEA proportion (n)	
Patients (n=47)	0.81 (38)	0.51 (23)	<0.05 *
Age groups /years (n)			
45-59 (9)	0.55 (5)	0.22 (2)	<0.05*
60-69 (15)	0.86 (13)	0.46 (7)	<0.05*
70-79 (10)	0.8 (8)	0.5 (5)	<0.05*
79-85 (13)	1.0 (13)	1.0 (13)	0.671

Table 2: Proportions of abnormal total MoCA scores (defined as MoCA cut-off score ≤ 26; indicating cognitive impairment) in patients before and after CEA.

	Before CEA			After CEA			p
	Median	Q1	Q3	Median	Q1	Q3	
Patients (n=47)	21	18	25	25	18	27	<0.05*
Age groups /years (n)	79-85 (13)	79-85 (13)	79-85 (13)	79-85 (13)	79-85 (13)	79-85 (13)	79-85 (13)
45-59 (9)	25	23	27	26	23	27	0.534
60-69 (15)	22	19	26	25	23	27	<0.05*
70-79 (10)	22	18	23	23	18	24	0.613
79-85 (13)	18	17	21	19	17	22	0.272
*sign. difference (Mann-Whitney Rank Sum test)							

Table 3: Differences in median total MoCA scores in different age groups before and after CEA.

	Before CEA (n=47)			After CEA (n=47)			p
	Median	Q1	Q3	Median	Q1	Q3	
MoCA(total score)	21	18	25	25	18	27	<0.05**
MoCA subtests							
VSE*	3	1	4	4	3	5	<0.05**
Naming	3	2	3	3	3	3	0.792
Attention	4	2	5	5	4	6	0.019**
Language	2	2	3	2	2	3	0.219
Abstraction	1	1	2	1	1	2	0.692
Delayed recall	2	0	3	3	2	5	<0.05**
Orientation	6	6	6	6	6	6	0.431
*VSE-Visospatial/executive functions; **sign. difference (Mann-Whitney Rank Sum test)							

Table 4: MoCA scores (total and subtests scores) for patients before and after CEA.

Discussion

The main result of our follow-up study is that there is overall cognitive benefit in patients with advanced carotid stenosis after CEA is performed. We observed significant improvement in cognitive functioning, presented as total MoCA score in all patients. Although previous studies of cognition after CEA have been published with mixed results, our findings are in line with those that described improvement in certain cognitive domains as well as in overall cognitive abilities following surgical revascularization procedure. [17-28]. However, some studies concluded that there is no change in cognitive abilities in patients following CEA [29-34]. One study described both cognitive impairment and cognitive decline [35]. In others, impairment or contradictory results were described [17,33]. It should be noted that most of the previously published studies do not easily allow for comparison, due to differences in study designs and a vast heterogeneity of cognitive testing methods used. Our results are best compared to those of Pucite et al. who showed that patients after CEA performed significantly better on the total MoCA scores after 6 and 12 months [40,41]. Another possible rationale that can influence the direct comparison of some older studies results is the recent improvement in surgical skills and modern operating techniques pertaining to CEA as well as advanced medical treatment of modifiable vascular risk factors [17,41,42]. Issues that further aid to challenging comparison of study results are heterogeneity in patient populations studied, presence of vascular risk factors, previous cerebrovascular incidences and the side od stenosis [17,35]. It is possible that differences in the genetic profile including the presence or absence of the APOE-ε4 allele can influence the cognitive effects of CEA [43]. In our earlier study which included 70 stroke/TIA-free patients diagnosed with severe carotid stenosis or occlusion we demonstrated the usefulness of MoCA as a simple tool for detection of cognitive impairment. Our observation was that the MMSE test, although widely used in clinical practice, did not prove to be sensitive enough for recognition of subtle cognitive changes in those patients [12]. Previously published studies recommended MoCA and some specific MoCA subtests (5-word recall, word list generation, trail-making, abstract reasoning and cube copy) for use as an optimal brief cognitive tool in vascular cognitive impairment [44,45]. As our present longitudinal research represents the cognitive follow-up of the same patients recruited from the previous study after the operative revascularization procedure was performed, we opted for the use of the same testing tool (MoCA) to further explore possible changes in cognition after CEA. Some authors concluded that postoperative cognitive assessment should preferably be performed after at least 3 months in order to detect lasting cognitive effects [46,47]. It was shown that previous practice or repeating cognitive testing can also cause a learning effect and thus influence the results as patients can become "test-wise" [48,49]. Therefore, in order to minimize the possible learning effect of MoCA, we performed a control cognitive assessment during the later period from 6 to 12 months after the procedure. As cognitive results can further be biased by individuals administering the tests, all cognitive testing was performed by the same neurologist experienced in cognitive testing. To minimize the possible cognitive impact of previous cerebrovascular incidents all subjects included in our study were asymptomatic (stroke/TIA free) and were referred to vascular surgeon for primary prevention of cerebrovascular events. Although we did not aim to determine the etiological relationship of cognitive changes following CEA, we hypothesize that cognitive changes following CEA can most probably be attributed to effects of revascularisation and consequent changes in cerebral hemodynamics, as was postulated by previously published studies [17,18,20,50-52].

Another result of our follow-up study is that older patients did not show cognitive benefit after CEA when tested using MoCA. Although differences in total MoCA scores showing cognitive recovery were significant in all patients after surgical procedure, the proportion of abnormal cognitive results in patients in the oldest age group (from 80 to 85 years) did not change significantly. It is possible that older patients still improved cognitively, but that the degree of cognitive change in this age group was mild and not detectable by MoCA. Another possible explanation is that mechanisms proposed to have an impact on cognitive benefit after surgical revascularisation, such as improved blood flow, cannot contribute enough to provide restoration of cerebral perfusion and associated cognitive functioning due to critically impaired vascular functioning in older individuals. It has been shown that age-related neuroanatomical changes as well as impairment of vasculature that inevitably occur with aging reduce or even prevent positive effects of revascularisation on cognition [52-55]. We hypothesize that the presence of multiple vascular risk factors, which share similar mechanisms of impact on vascular dysfunction, probably additionally contributes to lower cognitive benefits of CEA in older patients. This is in line with our previously published results showing lower total MoCA scores in older patients with advanced carotid disease [12]. Our present results are comparable to those of Lattanazi et al. who found age to be an independent predictor of cognitive changes after CEA, albeit the latter study was performed on a larger number of patients and used more complex cognitive testing methods [56]. Although using additional cognitive battery besides MoCA (Cantab), the study by Turowicz et al. also showed positive cognitive effects of carotid revascularisation but more pronounced in younger patients with worst cognitive performance before surgery [57]. In a sample of older patients Bo et al. found that patients with symptomatic left ICA disease undergoing CEA have a greater risk of cognitive decline [58]. Our results also coincide with conclusions by Zuniga et al. who found age was an independent predictor for a long-term significant decline in cognitive status after carotid interventions [59]. In a study by Wasser et al, examining the cognitive effects of both CEA and CAS in symptomatic and asymptomatic patients, transient cognitive decline was observed in patients ≥ 68 years old who underwent CAS, while patients that underwent CEA had a persistent cognitive decline at 3 months postoperatively [60]. Mocco et al. also concluded that increased age and diabetes increase the risk of cognitive decline following CEA in both symptomatic and asymptomatic patients [61]. Younger patients showed better global cognitive improvement and information processing in a study by Ortega et al., although the research was done on patients following CAS [62]. In a study by Migliara et al. cognitive status improved after CEA, especially in patients over 75 years of age [22]. As opposed to our results, the study by Baracchini et al. implied that only elderly symptomatic patients with severe carotid stenosis showed significant cognitive improvement after CEA, suggesting that CEA might provide some protection against cognitive decline in the elderly [63]. However, the latter study included both symptomatic and asymptomatic patients with advanced carotid stenosis, included a control group and used both MoCA and MMSE for cognitive assessment. Assessment of cerebrovascular reserve was not performed in our patients, however, we believe that functional impairment of cerebral blood flow causing reduced cerebral vascular reactivity probably contributes to the poorer cognitive outcome of CEA that is most often found in older individuals [17,20-23,64].

One interesting result of our study became evident when median cognitive outcomes of CEA among age groups were compared. Only patients aged from 60-69 years showed significant improvement in median total MoCA scores. This result should, however, be interpreted with caution as it could be biased because the particular age group involved the largest number of patients, despite the overall smaller sample size of patients included in the study. The influence of other factors that could probably affect cognitive test results, such as patient's

motivation and possibly better control of modifiable vascular risk factors can also not be ruled out as well.

MoCA subtest analysis showed that significant improvement was present after CEA in multiple cognitive domains, including visuospatial/executive functions, attention and delayed recall. Our results are in line with those of Turowicz et al. who evaluated patients 6 months after CEA and found improvement in five cognitive domains when tested by MoCA: visuospatial/executive, naming, language, abstraction and delayed recall [57] Wooley et al showed better executive functions and Sinforiani el al. described improvement in cognitive domains of attention and verbal memory in patients following CEA [18,19]. Despite the use of a more complex battery of neurocognitive tests, Kougias et al. similarly found that carotid revascularization, either CEA or CAS, improves memory and attention within the first 6 postoperative months [65]. Using MoCA, Pucite et al. also found significant improvement in cognitive domains of attention, language, abstraction and delayed recall after CEA [41]. In a study by Watanabe et al. comparing cognitive changes after CEA and CAS, the CEA group showed improvement in executive and memory MoCA subtest scores, which is partially congruent with our findings [28]. Earlier study by Fukunaga et al, using the Wisconsin Card Sorting Test (WCST) for frontal lobe function assessment in patients who underwent CEA, found significant improvements in all indexes of WCST, suggesting that CEA improves frontal lobe function, which certainly involves executive functions. Moreover, the authors of the latter study concluded that CEA improves frontal lobe function in patients with severe carotid stenosis or reduced cerebral perfusion reserve [23]. Results of the study by Relander et al. aiming to examine the associations between postoperative cognitive changes after CEA and long-term survival showed that the most often improved cognitive domain was executive functioning [66].

When we performed regression model analysis for prediction of cognitive worsening after CEA, older age, diabetes, hypertension and ever smoking were found to be positively associated with increased cognitive decline. Vascular risk factors are well known to have devastating long-term effects on brain health. The burden of vascular risk factors causing symptomatic or silent progression of cerebrovascular disease aids significantly to cognitive impairment probably due to further impairing cerebral vasculature both functionally and structurally [54,55]. According to the results from the ARIC study, both atrophic and ischemic changes in the brain were driven by altered glycemic and blood pressure control, beginning in midlife [67]. The study by Schröder et al. did not observe associations of hypoperfusion with cognitive performance in asymptomatic patients with advanced carotid stenosis before and after revascularization procedure, either CEA or CAS, however, the study included only a smaller number of patients [68]. Our previously published study showed positive associations for impaired cognition and presence of multiple vascular risk factors and arterial hypertension in symptomatic patients with cerebrovascular disease but also in individuals with multiple vascular risk factors who were stroke/TIA free [69]. Our present results are in line with those of Zuniga et al. who found that patients older than 80 years of age and smokers were more likely to experience long-term memory decline at 6 months [59]. Another study with comparable results is by Mocco et al. who concluded that both diabetes and older age predict cognitive decline in patients after CEA [61].

We hypothesize that, especially in older individuals, particular vascular risk factors, such as diabetes, hypertension and smoking present a certain confounding variable for poor cognitive outcome following carotid revascularisation procedure. Probable mechanism involves a contribution of vascular risk factors to impairment of already less functional small vessels in critical cortical regions, particularly in the anterior part of the brain and exhaustion of cerebrovascular reserve which leads to cerebrovascular compensatory failure. Consequently, despite the presumably restored blood flow in the carotid arteries following CEA, inadequate perfusion of already impaired cerebral vasculature in older patients does not allow for significant improvement in cognitive functioning.

The strength of the present study is its longitudinal design which involves follow-up of previously recruited patients, consideration of major vascular risk factors and a longer follow-up period of 6 to 12 months. Limitations of the present study, which should be noted when interpreting results, include a limited number of patients and non-uniform sample sizes across age groups causing obviously reduced statistical power, meaning that the results of statistical significance could change if the study is performed on a large number of patients. Another limitation is obviously lack of brain imaging procedures following CEA as possible silent cerebrovascular incidents during the follow-up period could influence the results. As stated in the previously published study, evaluation for potential confounders such as medical treatment of modifiable vascular risk factors was not done and the duration of vascular risk factors was not determined. Despite limitations noted, we believe that our results raise noteworthy questions in terms of cognitive outcomes of CEA depending on age. Impairment of cognitive functions due to advanced carotid disease has been recognized and receives increased attention due to its great clinical significance and impact on quality of life. Previously published research suggest that CEA procedures can safely be performed in older patients, in terms of restoration of blood flow [70]. Our results indicate that the expected cognitive benefit after CEA is low, if present at all, with increasing age.

Older patients with advanced carotid disease, although might present as asymptomatic, have limited life expectancy and often multiple comorbidities which makes them prone to complications and are less likely to benefit from carotid surgical intervention that is primarily aiming to stroke/TIA prevention [71]. Although mostly positive cognitive impact of carotid revascularisation has been recognized, poorer cognitive outcomes can be expected in older patients, particularly in those with more vascular risk factors present. Therefore, in older patients with advanced carotid disease, meticulous patient selection with individual assessment of not only vascular but also of cognitive status should be essential in determining the risk/benefit ratio when deciding on the treatment.

Conclusion

Positive cognitive impact of CEA is not present in older patients. Besides older age, arterial hypertension and ever smoking seem to be additional factors negatively influencing the cognitive benefit of CEA.

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