Technical Complications and Cognitive Outcome in Carotid Stenting with–Versus without Protection Device

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Abstract

Background: Carotid stenting is a widely employed procedure for carotid artery stenosis therapy, a condition that can lead to stroke. Embolic protection devices (EPDs) usage during carotid stenting has been shown to decrease periprocedural stroke and neurocognitive deficits risk. However, the use of EPDs can also lead to technical complications, such as device-related issues, access site complications, and vascular injury. Furthermore, the cognitive outcomes following carotid stenting with and without EPDs are still not well understood. The purpose of this work was to analyze protection device usage impact on procedural related complication rate and cognitive function.

Results: Cognitive assessment was done on 13 patients only before and after an intervention. (2 patients did not come for follow up). A substantial post-intervention development was observed in PALT score (p value=0.01), Trail A (p value=0.01), Trail B (p value<0.001) and BVRT (p value=0.03). Cognitive assessment was done on 12 patients only before and after the intervention. (3 patients did not come for follow up). A substantial post-intervention development in scores of PALT (p value=0.01), Trail A (p value=0.01), Trail B (p value=0.01) and BVRT (p value=0.02). No statistically substantial change was seen among both groups regarding the post-intervention improvement in PALT scores (p value=0.37), Trail A (p value=0.94), Trail B (p value=0.11) and BVRT (p value= 0.95).

Conclusions: Using protection device in carotid stenting doesn't have a statistically significant impact in improving cognitive function or reducing the possibility of development of new ischemic lesions.

Keywords: Technical Complications, Cognitive Outcome, Carotid Stenting, Protection Device.

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Background:

Atherosclerosis of carotid-artery is a crucial reason for ischemic stroke. Thrombotic material could embolize from the carotid artery to the intracranial vessels [1].

In addition to being a standalone risk factor for ischemic stroke, carotid artery stenosis is linked to diminished cognitive activity. Cerebral hypoperfusion and embolic stroke are two pathophysiological factors that contribute to the carotid artery stenosis that causes cognitive impairment [2].

Carotid stenting has been established as an effective procedure for stroke prevention in carotid artery stenosis patients [3].

The risk of cerebral embolism after carotid artery stent insertion is a significant worry. In the last few years, cerebral protection devices (CPDs) have been developed. However, concerns about these protection devices have been raised because they make the process more complicated, risky, and expensive [4].

This work aimed to analyze protection device usage impact on procedural related complication rate and cognitive function.

Methods:

This is a randomized prospective study that aimed to evaluate the cognitive activity of symptomatic carotid stenosis individuals who underwent carotid artery stenting (CAS) versus cerebral protection device (CPD) filter and those who underwent CAS without the filter. The study included 30 patients, 15 of whom received CAS with CPD filter and 15 received CAS without the filter.

The patients were recruited from the neurology clinic at XXXX University Hospital in collaboration with XXXX Institute Hospital, and the research ethics committee for the faculty of medicine at XXXX University gave its approval to the study (FMBSU-REC). All participants in the study gave their informed permission.

The inclusion criteria were age between 30 and 80 years, symptomatic carotid stenosis (≥50-99%) and (≥60-99%), as assessed using the NASCET (North American Symptomatic Carotid Endarterectomy Trial) criteria, and ability to read, write, and do simple calculations. The exclusion criteria included acute cerebral infarction necessitating immediate thrombolysis and/or immediate stent insertion prior to the operation, acute angulation of ICA origin

warranting treatment by micro guidewire without protection device, fresh plaque as evidenced by duplex ultrasound study or CTA to be considered as direct indication for protection device, nonvascular factors that cause cognitive disorders, psychiatric disorders like anxiety, psychosis, or depression, severe comorbidities like liver, kidney and heart diseases, hemorrhagic blood diseases, or malignancy, intracranial hemorrhage, intracranial tumors, hemorrhagic stroke, intracranial aneurysm, or arteriovenous malformation, planned intervention to a contralateral carotid artery within two months, and any planned major surgery within two months post-procedure.

All patients underwent a thorough history taking, including information on age, gender, education, systemic arterial hypertension, diabetes mellitus, tobacco smoking, and drug abuse. Neurological assessment was performed for all patients using the National Institutes of Health Stroke Scale (NIHSS) preoperatively and one month postoperatively. Cognitive assessment was conducted before CAS and one month after CAS by a consultant neuropsychologist who was not aware of the patient's radiological findings.

The cognitive assessment involved the use of several psychometric tests, including the Trail A and B test, the Benton Visual Retention Test, and the Paired Associate Learning Test (PALT). The PALT was used to measure verbal memory using the theory of semantic cueing, while the visual perception, visual memory, visual motor, and visuoconstructive abilities were assessed using the BVRT. To evaluate executive function, attention, and psychomotor speed, the Trail A and B test was utilized.

In the filter group, post-CAS cognitive function had been done only for 13 patients. The other two patients had a stroke during the intervention and managed immediately by mechanical thrombectomy, and the other one did not come in the follow-up. In the nonfilter group, the post-CAS cognitive tests had been done in 12 patients. They did not perform in the other 3 patients as there was one case complicated by hyperperfusion syndrome, and the other two patients did not come in the follow-up. The cognitive tests were scored according to established methods and the results were analyzed to determine any differences in cognitive function between the two groups.

Statistical analysis: Utilizing version 15 of the statistical software for social science, the data were coded and inputted (SPSS v 15). Descriptive data were described for instance mean ± SD, number (%) for resounding variables. For comparing the means of two unpaired clusters of quantitative variables, the student t-test was helpful. matched example For comparison of the means of two paired clusters of quantitative variables, the t-test was helpful. Chi-square test was helpful for comparing two sets of certain data. The probability/ implication value (P-value) <0.05 is considered statistically significant.

Results:

Regarding the Demographic Data in the studied groups: Patients ages were between 30-80 years with a mean value of 63.33 (SD=6.51) years in group I and 60.6 (SD=11.84) years in group II. 66.7% (n=10) of patients in group I was males, and 33.3 % (n=5) were females. As regards patients in group II, males were 73.33 % (n=11), and females were 26.4% (n=4). **Table 1**

Regarding the Clinical cerebrovascular risk Factors: In group I, 53.3% (n=8) of patients were smokers, while in group II, 46.7% (n = 7) of patients were smokers. No substantial considerable change was observed among both groups (P-value = 0.72). In group I, 46.7 % (n=7) of patients were diabetic, while in group II, 60% (n=9) of patients were diabetic. No substantial critical change was seen among both groups (P-value = 0.46). In group I, 80 % (n=12) of patients were hypertensive, while in group II, 60 % (n=9) of patients were hypertensive. No substantially critical change was present among both groups (P-value = 0.23). In group I, 46.7 % (n=7) of patients have IHD, while in group II, 20% (n=3) of patients have IHD. No critically substantial change was observed among both groups (P-value = 0.12). **Table 2**

Comparison between both groups regarding post-intervention ischemic lesions: In group I, 86.7 % (n=13) of patients did not have any new ischemic lesions following carotid stenting, 6.7 % (n= 1) had large vessel occlusion and 6.7 % (n= 1) had small ischemic lesions. In group II, 73.3 % (n=11) of patients did not have any new ischemic lesions following carotid stenting, 26.7 % (n= 4) had small ischemic lesions and no patients had large vessel occlusion. No substantially critical change was seen among both groups (p value=0.23). **Table 3**

Cognitive assessment was done on 13 patients only before and after an intervention. (2 patients did not come for follow up). A considerable post-intervention enhancement was present in the PALT scores (p value=0.01), Trail A (p value=0.01), Trail B (p value<0.001) and BVRT (p value=0.03). **Table 4**

Cognitive assessment was done on 12 patients only before and after the intervention. (3 patients did not come for follow up). A substantial post-intervention enhancement was seen in the scores of PALT (p value=0.01), Trail A (p value=0.01), Trail B (p value=0.01) and BVRT (p value=0.02). **Table 5**

No statistically substantial change was seen among both groups regarding the post-intervention improvement in the PALT scores (p value=0.37), Trail A (p value=0.94), Trail B (p value=0.11) and BVRT (p value= 0.95). **Table 6**

Table 1: Demographic data in the study population

Demographics		Group I (n=15)	Group II (n=15)	P-value
Age [Mean (SD)]		63.33 (6.51)	60.6 (11.84)	0.44
Sex	Males [n (%)]	10 (66.7%)	11 (73.3%)	0.69
	Females [n (%)]	5 (33.3%)	4(26.7%)	

Data were expressed as mean ± SD (standard deviation), frequency (&).

Table 2: Clinical cerebrovascular risk Factors.

Clinical cerebrovascular risk factors		Group I (n=15)	Group II (n=15)	P-value
Smoking	With [n (%)]	8(53.3%)	7 (46.7 %)	0.72
	Without [n (%)]	7 (46.7%)	8 (53.3 %)	
DM	With [n (%)]	7 (46.7%)	9 (60 %)	0.46
	Without [n (%)]	8 (53.3%)	6 (40 %)	
HTN	With [n (%)]	12 (80 %)	9 (60%)	0.23
	Without [n (%)]	3 (20%)	6 (40%)	
IHD	With [n (%)]	7 (46.7%)	3 (20%)	0.12
	Without [n (%)]	8 (53.3%)	12 (80%)	

DM: Diabetes mellitus, HTN: hypertension, IHD: ischemic heart disease.

Table 3: Comparison between both groups regarding post-intervention ischemic lesions:

Ischemic lesions	Group I (n=15)	Group II (n=15)	P-value
No ischemic lesions [n (%)]	13 (86.7%)	11 (73.3%)	0.23
Small vessel occlusion [n (%)]	1 (6.7%)	4 (26.7%)	
Large vessel occlusion [n (%)]	1 (6.7%)	0 (0%)	

Table 4: Cognitive tests in group I before and after intervention:

Cognitive tests	Before intervention [Mean (SD)]	After intervention [Mean (SD)]	P-value
PALT	10.73(2.79)	12.04 (3.16)	0.01*
Trail A	125.77 (31.08)	106.54 (27.26)	0.01*
Trail B	221.92 (46.97)	183.08(33.26)	<0.001*
BVRT	12.54 (2.47)	14.38 (3.52)	0.03*

PALT: Paired Associate Learning Test, BVRT: Benton Visual Retention Test, SD: standard deviation, *: significant as P-value < 0.05.

Table 5: Cognitive tests in group II before and after intervention:

Cognitive tests	Before intervention [Mean (SD)]	After intervention [Mean (SD)]	P-value
PALT	12.33 (2.16)	14.33 (2.39)	0.01*
Trail A	146.67 (92.39)	128.17 (60.11)	0.01*
Trail B	226.25 (89.11)	205.75 (82.77)	0.01*
BVRT	13.67 (2.27)	15.58 (2.23)	0.02*

PALT: Paired Associate Learning Test, BVRT: Benton Visual Retention Test, SD: standard deviation, *: significant as P-value < 0.05.

Table 6: Comparison between the two groups regarding the difference in the cognitive tests:

Cognitive tests	Group I (n=13)	Group II (n=12)	P-value
Difference in PALT	1.31(1.56)	2.0 (2.18)	0.37
Difference in Trail A	-19.23 (22.72)	-18.5 (22.13)	0.94
Difference in Trail B	-38.85 (29.31)	-20.5 (24.72)	0.11
Difference in BVRT	1.85 (2.7)	1.92 (2.54)	0.95

PALT: Paired Associate Learning Test, BVRT: Benton Visual Retention Test.

Discussion

In addition to being a standalone risk factor for ischemic stroke, carotid artery stenosis is linked to diminished cognitive activity. Carotid artery stenosis has several pathophysiological reasons, including cerebral hypoperfusion and embolic stroke, that contribute to cognitive impairment [2].

Regarding the filter effect, we have two cases in our study. The used filter was catching clots, and the post-intervention diffusion MRI of both cases was free from any new ischemic lesions compared with the distribution done before the intervention. **According to Maleux and colleagues** the existence of debris captured by EPDs does not mean that future cerebral ischaemic cases are stopped [5].

The hypothesis is to explain the inconsistent lesions due to emboli from the preserved stenotic ICA and continue through intracranial recompense supply success the contralateral hemisphere. In contrast to this hypothesis, **Poppert and colleagues** and **Schluter and colleagues** reported that inconsistent DWI lesions later CAS but not later CEA are due to manipulations on the aortic arch and natural vessels, which were considered a major role in incidence of varying silent cerebral ischemia and this is more logic and agree with the two cases as both of them are old age [6, 7].

Cho and colleagues in their Meta-analysis included 20670 CAS procedures from 25 studies. There were 142 strokes (3.4%) in unprotected CAS and 326 (2.0%) in protected CAS. After CAS, the usage of the cerebral protection device greatly reduced stroke (p=0.001) [8]. Our study targeted any new ischemic lesion, whether symptomatic or not, but this Meta-analysis targeted the occurrence of stroke and not the asymptomatic signal change on brain MRI.

Tal LaRita and colleagues found that the stroke rate was 0.8% in patients treated with distal filter devices against 3.8% in non-protected CAS group (P=0.08). No diffusion MRI was done in this study to detect any new ischemic lesion (symptomatic or asymptomatic) [9].

In the Worldwide Carotid Stenting Research (ICSS), the frequency of stroke in the community preserved with preventive equipment was greater (5.1%) than in collection in which no aid was charity (2.4 percent). In similar research, the degree of diffusion-weighted MRI aberrations after CAS was likewise greater in the safe community.

Regarding Pre- and post-procedural cognitive function assessment: The carotid artery stenting impact on cognitive performance is not apparent. Both cognitive development and regression were recorded after CAS [10-12]. The current work targeted studying CAS effect on the cognitive activity of carotid stenosis individuals. The results revealed a significant enhancement in cognitive activity after the filter protected CAS.

Several studies found that revascularization of the carotids increases cerebral blood flow, that may explain the improvement in cognitive function [13, 14].

Takaiwa and colleagues examined 26 individuals (15 treated with CAS and 11 treated by CEA). They observed that while no substantial change was seen in terms of brain performance between the two approaches, there were memory and focus enhancements. These changes have been maintained over time following carotid revascularization [15].

Stošić and colleagues assessed the cognitive activity in symptomatic and asymptomatic carotid stenosis patients before and following the CAS operation. Following CAS, patients' executive functions, memory, and attention all improved in both symptomatic and asymptomatic patients [16].

Chen and colleagues revealed that ICA stenosis patients who were "asymptomatic" showed increased cognitive performance following a successful therapy. As it is only seen in patients with baseline cerebral ischemia, this development is brought on by improved perfusion. Cognitive enhancement is anticipated three months after carotid stenting corrects the cerebral hypoperfusion. The researchers showed that changes in cognitive function correspond to changes in cerebral perfusion state [17].

The presences of silent ischemic lesions following CAS can be considered one of the causes of cognitive impairment besides hemodynamic disturbance (hypo/hyperperfusion) [18, 19]. But our study revealed that following CAS, cognitive impairment is not strongly related with the existence of new ischemic lesions on DWI.

Regarding our findings, two studies argued that original brain lesions followed by carotid revascularization have no long-term or clinically significant effects on cognitive functioning. In addition, considering the higher embolic load observed by DWI, after the CAS, CAS was not correlated with a more severe deficiency of cognitive function than CEA [20, 21]. This research examined the possible effects of new DWI lesions on intellectual functions following CEA or CAS. The absence of an association between silent ischemic lesions and cognitive deficit can be clarified by the reversibility of angioplasty DWI lesions, as shown in the **Hauth and colleagues** report [22].

Research performed by **Heyer and colleagues** reported cognitive impairment in structural evidence lack for cerebral ischemia following uncomplicated CEA. This also suggested other causes causing impairment of cognitive function not related to the new silent ischemic lesions post-CAS [23]. Additionally, hemodynamic and metabolic stressors occurring after angioplasty may be incriminated in the development of post interventional cognitive decline.

Capoccia and colleagues performed a study on two groups (CEA and CAS). In the CAS group only, they observed that cognitive function worsened. This discovery and a higher rate of lesions identified by diffusion magnetic resonance within 24 hours were found to be positively associated, according to the researchers [24].

Conclusions:

Using protection device in carotid stenting doesn't have a statistically significant impact in improving cognitive function or reducing the possibility of development of new ischemic lesions.

Declarations:

Ethics approval and consent to participate: The study was accepted by the Faculty of Medicine, Beni-Suef University research ethics committee (FMBSU-REC). Informed consent was obtained from all participants included in the study.

Consent for publication: Not applicable.

Availability for data and materials: The data is available upon reasonable request from the authors.

Competing interests: The authors declare that they have no competing interests

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List of Abbreviations

EPDs	Embolic protection devices
PALT	Paired Associate Learning Test
BVRT	Benton Visual Retention Test
CPDs	cerebral protection devices
CAS	carotid artery stenting
CTA	Computed Tomography Angiography
ICA	internal carotid artery
NIHSS	National Institutes of Health Stroke Scale
IHD	ischemic heart disease
MRI	Magnetic resonance imaging
DWI	Diffusion weighted imaging
CEA	Carotid endarterectomy

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