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Silent cerebral embolism after carotid endarterectomy: a two-center experience

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ABSTRACT

Objectives. Carotid endarterectomy (CEA) is considered the most effective treatment for stroke prevention in patients with critical carotid stenosis. The incidence of new ischemic lesions ranges from 0% to 33% at diffusion-weighted magnetic resonance imaging (DW-MRI) after CEA in previous studies. We determined the rate of silent cerebral embolism in CEA patients by DW-MRI in this study. ***Methods.*** This study was conducted between January 2016 and April 2016 in two centers. Thirty-five consecutive patients (three with bilateral) with 38 CEAs were included in the study. There were no new postoperative symptoms in all patients. Preoperative and postoperative brain DW-MRIs were performed within one day preoperatively and second day postoperatively. Two DW-MRIs were screened and compared in terms of newly occurring lesions. Thus, we attempted to find the rate of silent cerebral embolism. ***Results.*** New brain lesions were detected in six (6/38 CEAs; 15.8%) cases with unilateral CEAs. All of these lesions were ischemic. In five cases, new lesions were located within the operated carotid artery territory (ipsilateral parietal lobe). However, in one case, a new lesion was located outside of the operated carotid artery territory (ipsilateral occipital lobe). Thirty-day morbidity and mortality rates were 0% and 2.85% (1/35), respectively. ***Conclusion.*** Silent cerebral embolisms may frequently occur during postoperative period in CEA patients. Even if these lesions are asymptomatic, we have to be rigorous to avoid microembolism during all stages during surgery.

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Keywords: Cerebral embolism, carotid endarterectomy, magnetic resonance imaging

Introduction

More than 150 known causes play role in the etiopathogenesis of stroke. The most common causes of them are cardiac and small vessel diseases. The other causes are exta- or intra-cranial atherosclerosis, dissection, coagulopathy, vasculitis, metabolic disease with arteriopathy and unknown.

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Almost 20% of all strokes are related to carotid stenosis [1]. Carotid endarterectomy (CEA) is the most effective treatment for stroke prevention in patients with critical carotid stenosis [2-4]. Recent studies have demonstrated that surgical morbidity is approximately 6% for symptomatic stenosis and 3% for asymptomatic stenosis in CEA patients [5, 6].

Most of perioperative ischemic neurological complications are commonly caused by an embolism released from the fragile plaque in the course of arterial dissection, shunt insertion and cross-clamping, or uncommonly by hemodynamic hypoperfusion [7-9]. Intraoperative hypoperfusion should rarely be an issue because brain perfusion can be maintained by collateral circulation or selective shunting. Conversely, a small embolism arising from a fragile plaque during arterial dissection, shunting and cross-clamping constitutes a risk of perioperative ischemic complications. In cardiovascular diseases, plaque size, luminal narrowing, and plaque structures are also considered causally related to the development of cardiovascular cases [10-12].

Diffusion-weighted magnetic resonance imaging (DW-MRI) is a highly sensitive tool for detecting cerebral ischemia [13]. Ischemic areas smaller than 3 mm in diameter can be indicated only on DW-MRI sequences. It is a suitable new method for improving quality control in cerebrovascular interventions.

The main objective was to evaluate the incidence of silent ischemic brain lesions in CEA patients by using DW-MRI.

Methods

Patients

This study consisted of 40 consecutive patients with high grade carotid artery stenosis who were treated between January 2016 and April 2016 in two cardiovascular surgery centers. Two of 40 patients with new postoperative ischemic symptoms were excluded from the study. Color doppler-assisted duplex investigations and computerized tomography (CT) with contrast was applied for the diagnoses of carotid stenosis. There were bilateral critical carotid stenoses in three patients. Therefore, CAE was separately performed bilaterally in three of 38 patients. The contralateral internal carotid artery (ICA) was occluded in two patients. A critical carotid stenosis was defined as a stenosis of ≥70% for symptomatic patients and a stenosis of ≥80% for asymptomatic patients. Preoperative and postoperative brain DW-MRIs were performed within 1 day before the operation and again 2 days after the operation in patients. Three patients whose unilateral carotid stenosis had contraindications (one with a cardiac pacemaker, one with a mechanical mitral valve prosthesis and one with claustrophobia) for DW-MRI and were excluded from the study. Thus, 38 CEA (35 patients) outcomes were examined by DW-MRI.

All patients were administered with acetylsalicylic acid (100 mg) and statins (40 mg) before the operation. Hypertension was defined as a blood pressure of 140/90 mmHg or higher. Diabetes mellitus was defined as a fasting blood glucose of >126 mg/dl on two measurements, or if the patient was being treated with insulin or oral antidiabetic medication. Chronic obstructive pulmonary disease (COPD) was defined as an FEV1/FVC less than 70%, or if they were undergoing bronchodilator medication.

This study was approved by the ethics committee of the university hospital. We also obtained the patients' written informed consents to be included in the study.

Carotid Endarterectomy

The CEA operations were performed under general anesthesia. Near-infrared spectroscopy were implement to all patients for assessment of neurological status during surgery. The ICA, ECA (external carotid artery), and CCA (common carotid artery) were adequately exposed. Intravenous heparin (100 U/kg) was administered; then the ECA, CCA, ICA, and superior thyroid artery were cross clamped. An arteriotomy was performed from the CCA to ICA and fully extended high above the upper extent of the lesion to be removed. The lumen was rinsed with heparinized saline for removing crumbs. A shunt was first placed into the CCA and the balloon of the proximal tube was inflated adequately with saline. Then, the distal tube of the shunt was inserted into the ICA and the balloon was inflated very gently to avoid intimal damage and debris embolization. The distal and proximal tubes of the shunt were fixed by using silicon vessel tape. Arterial flow in the shunt was tested. Thus, cerebral perfusion was restored through the shunt. All of the atherosclerotic plaques were rigorously removed in the subintimal plane and the lumen was washed with saline. The endarterectomized area was thoroughly inspected. If there was any intimal flep, it was skinned or fixed with 7-0 prolene suture. The arteriotomy was closed with a running 6-

0 prolene suture with a patch. A few loops before completion, the shunt device was removed and the ICA and CCA were clamped. Before tying the suture, the lumen was washed with heparinized saline. After tying, the ICA was unclamped for 5 seconds, then clamped again. The ECA and CCA were then declamped and then the ICA was finally declamped. Thus, any possible particulates were prompted into the ECA. Aspirin (100 mg/day) and statins (40 mg/day) were routinely administered after surgery. The excised plaques were examined macroscopically for classification after surgery. Near infrared spectroscopy (NIRS) were used for hemodynamic changes of brain function in all patients during the surgery.

Diffusion-Weighted Magnetic Resonance Imaging

DW-MRIs were performed on the brain within the 1 day before and again 2 days after the operation with a MAGNETOM Avanto 1.5-T scanner (Siemens AG, Erlangen, Germany). Diffusion gradients were applied in each of the x, y, and z directions with three b values. The imaging protocol was the same in all patients. Conventional T1- and T2-weighted spin-echo imaging with a fluid-attenuated inversion recovery (FLAIR) sequence was also performed at each examination. On the DW-MRI, any signal-intensity abnormalities were recorded. For all diffusion-weighted abnormalities, we identified the size, vascular distribution, lobe, and area of the brain in which the lesion was situated. All diffusion-weighted abnormalities were correlated with the findings of the T2-weighted and FLAIR images. The DW-MRI images were then evaluated by two neuroradiologists blinded to the clinical status of the patients. The presence of any new hyperintensities in the brain was interpreted as a sign of new ischemic lesions after CEA.

Statistical Analysis

Statistical analyses were performed by using NCSS (Number Cruncher Statistical System) 2007 Statistical Software (Utah, USA) package. In the evaluation of the data, descriptive statistical methods (mean, standard deviation, frequency and percentage distributions) as well as repeated measures of variance were used in repeated measures of the groups, Newman Keuls multiple comparison test in subgroup comparisons and Mc Nemar's test in repeated measures of qualitative data. Values of $p<0.05$ were significantly evaluated.

Results

Clinical outcomes

There were ischemic symptoms in two (5%) patients of 40 CEA procedures postoperatively (hemiplegia and transient ischemic attacks [TIA]). These patients were excluded from the study. The mean age of the 35 participants was 66 years (range; 42-80 years). There were 11 (31.4%) females and 24 (68.6%) males. Fifteen (42.9%) patients had no symptoms preoperatively. Additionally, there were 6 (17.1%) patients with stroke and 14 (40%) patients with TIAs preoperatively. The contralateral ICA was totally occluded in two patients and three patients had critical stenoses in the contralateral internal carotid artery. So, CAE was separately performed bilaterally in these patients. The mean percent of stenosis in the operated carotid artery was 86.85% (range: 70%-98%). Saphenous vein patch were used in 15 (15/38; 39.5%) cases, synthetic patch were used in 14 (14/38; 36.8%) cases and external jugular vein patch were used in 9 (9/38; 23.7%) cases to close the arteriotomy. None of the arteriotomies were closed primarily. The mean cross clamp time was 1.86 minutes before shunting and 2.6 minutes after removing the shunt. Mean arterial pressure was between 80 mmHg and 115 mmHg in all patients and hypotensive period was not observed in all patients during the surgery. When the excised plaques were identified macroscopically, 15 (15/38; 39.5%) plaques had ulcerations, 25 (25/38; 65.8%) plaques had calcifications, 12 (12/38; 31.6%) plaques had thromboses, and 20 (20/38; 52.6%) plaques had signs of degeneration. During the postoperative period, there was no case revealed postoperative hyperperfusion syndrome. Control carotid doppler ultrasound were performed in all patients postoperatively and results were satisfactory. The 30-day morbidity and mortality rates were 0% and 2.85% (1/35), respectively. Cause of mortality was myocardial infarction. The other clinical and laboratory parameters are demonstrated in Tables 1 and 2.

DW-MRI outcomes

There were relatively bigger ischemic lesion (9 mm and 13 mm) in ipsilateral parietal lobe in two patients that excluded from the study due to postoperative ischemic symptoms. Therefore, rate of postoperative ischemic symptoms were 5% in the study. Six (6/38; 15.7%) cases whose no new symptoms postoperatively had new brain lesions in

Table 1. The clinical and laboratory parameters of patients (n=35)

Parameters	Data
Age (year)	66.02±10.37 (42-80)
Gender	
Male	24 (68.6)
Female	11 (31.4)
PAD	7 (20)
DM	11 (31.4)
HT	24 (68.6)
COPD	8 (22.9)
Smoking	18 (51.4)
Symptom	
Asymptomatic	15 (42.9)
Stroke	6 (17.1)
TIA	14 (40)
LDL (mg/dL)	128.68±35.9 (48-189)
HDL (mg/dL)	23±10.66 (12-68)
Cholesterol (mg/dL)	200±50.01 (108-302)
Trigliserid (mg/dL)	134.4±40.96 (56-229)
Urea (mg/dL)	20.54±8.38 (8-46)
Creatinin (mg/dL)	1± 0,95 (0.5-6.3)
Hemoglobin (g/dL)	12.76±1.62 (9.1-16)
Hematocrit (%)	38.76±4.42 (27.9-48)
Platelet ($x10^9$/L)	231.4± 59.76 (151-388)
Ipsilateral Stenosis (%)	86.57±6.96 (70-98)
Contralateral Stenosis (%)	32.82±24.12 (0-90)

Data are shown as mean±standart deviation (range; mininumum-maximum) or number (percent). COPD=chronic obstructive pulmonary disease, DM=diabetes mellitus, HDL=high-density lipoprotein, HT=hypertension, LDL=low-density lipoprotein, PAD=peripheric arterial disease

postoperative DW-MRIs. All lesions were ischemic. In five cases, new ischemic lesions were located within the operated carotid artery territory (ipsilateral parietal lobe). In one case, new lesion was located outside of the operated carotid artery territory (ipsilateral occipital lobe). The range of these lesion sizes were from 3 mm to 6 mm (Table 3, Figure 1). The other 29 patients (32 CEA cases) were normal according to the DW-MRI results.

Discussion

In this consecutive series of 35 patients undergoing elective CEA, the stroke rate was 0% and the mortality rate (from any cause) was 2.85% in first

Table 2. The operative parameters (n=38)

Parameters	Data
Patch	
Saphenous	15 (39.5)
Synthetic	14 (36.8)
EJV	9 (23.7)
First Clamp Time (min)	1.86±0.77 (1-4)
Second Clamp Time (min)	5±1.12 (1-2.6)
Shunt Time (min)	32.63±9.84 (15-62)
Ulcer	15 (39.5)
Calcification	25 (65.8)
Thrombosis	12 (31.6)
Degeneration	20 (52.6)

Data are shown as mean±standart deviation (range; mininumum-maximum) or number (percent). EJV=external jugular vein

30 postoperative days. The incidence of postoperative silent brain ischemia that demonstrated by DW-MRI was six (6/38 CAEs; 15.8%) cases.

CEAs reduce the stroke risk in symptomatic or asymptomatic patients with high grade carotid stenosis [2, 4, 14]. In the European Carotid Surgery Trial, among the 1,745 patients who underwent CEA, there were 122 (7.0%) major strokes or death. The death rate was 1.0%, the disabling stroke rate was 2.5%, and the non-disabling stroke rate was 3.5% in 30 days postoperatively [13]. Rapp *et al.* [15] demonstrated that new DW-MRI lesions were observed over a large range after both carotid artery stenting (9% to 70.3%) and CEA (0% to 27.27%). The incidence of new ischemic lesions after CEA was between 0% and 33% at DW-MRI in different studies [16-19].

DW-MRIs have been shown to be far more sensitive to acute cerebral infarctions than either CTs or conventional MRIs [20, 21]. Therefore, DW-MRIs are commonly used for identifying an ischemic lesion in the brain. Furthermore, this imaging method is a great for quality control in carotid interventions. Most of the lesions are ischemic at postprocedural DW-MRIs in patients with carotid interventions.

Embolization can occur during all phases of the CEA operation (e.g., dissection, shunting, clamp release, and wound closure) [22]. Intra-arterial shunting for the maintenance of cerebral perfusion is often blamed for new ischemic brain lesions in CEA patients. Aksun *et al.* [23] demonstrated that requirement of shunt usage may be clear by applying of cerebral oximetry monitoring during the CEA operation. Schnaudigel *et al.* [24] observed that the general use of intra-arterial shunts during carotid artery clamping was associated with a significantly higher incidence of new ipsilateral DW-MRI lesions. Effect of shunt to the development of cerebral ischemia is obscured in this study. Therefore, the effect of shunt to the cerebral embolism could be more clear if the comparison were performed between the patients with and those without shunt.

On the other hand CEA could performed under local anesthesia. Toktas *et al.* [25] demonstrated that CEA procedure with local anesthesia may provide better assessment of neurological status during operation. Whereas all procedure were performed under general anesthesia in this study. Therefore assessment of neurological status were performed by near-infrared spectroscopy during surgery.

Shunt were used in all study patients during the procedures. The arteriotomy area was rinsed with saline before inserting the shunt and after the endarterectomy procedure. Also, the inside of shunt was bled to the outside thorough a third tube to avoid

Table 3. Patients with silent cerebral embolism

No	Age/ Gender	Preoperative Symptom	Carotid Stenosis (%)	Contraleteral Carotid Stenosis (%)	Patch	Ulcer	Calcification	Thrombosis	Degeneration	Localization of Lesions	Size of Lesion (mm)
1	62/M	No	80	26	EJV	-	+	+	+	İpsilateral Pariatel	3
2	67/M	No	85	15	Synthetic	-	+	-	+	İpsilateral Oxipital	4
3	68/M	TİA	98	55	Synthetic	-	+	-	-	İpsilateral Pariatel	6
4	56/M	TİA	75	40	Saphenous	+	+	-	+	İpsilateral Pariatel	6
5	67/M	No	90	33	Saphenous	-	-	-	-	İpsilateral Pariatel	5
6	69/M	No	85	15	Synthetic	-	+	-	-	Ipsilateral Pariatel	4

EJM=external jugular vein, TIA=transient ischemic attack

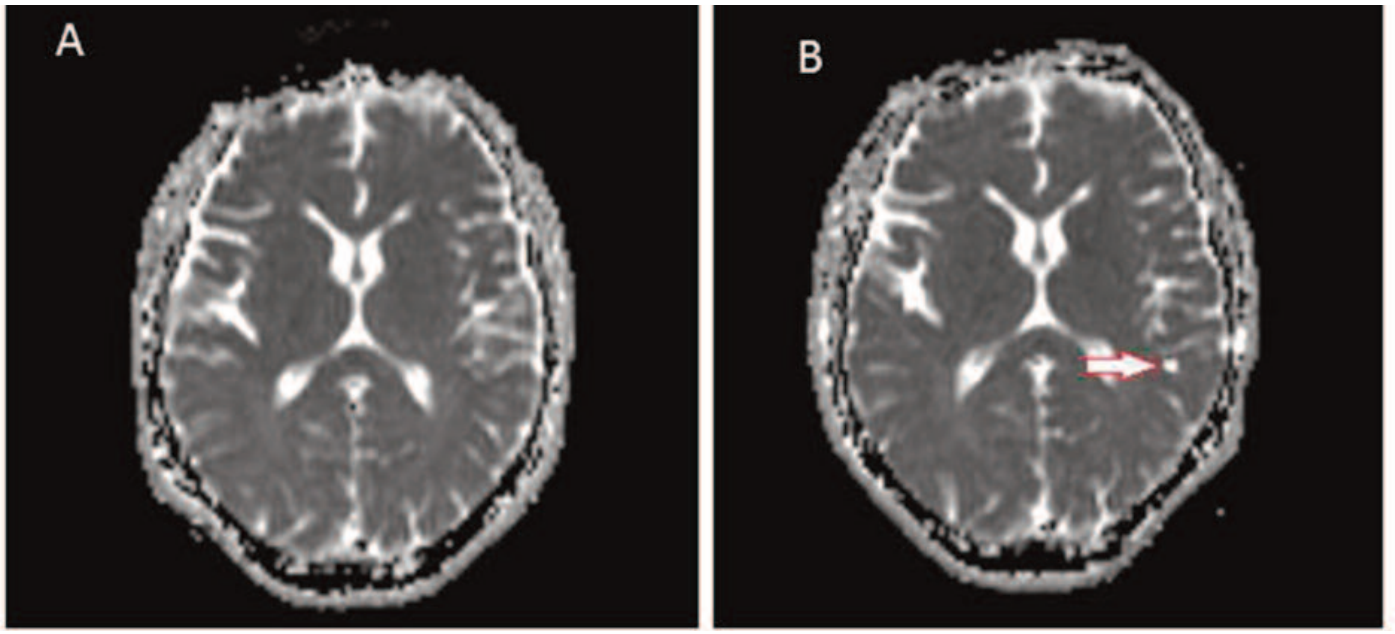

Figure 1. (A) Preoperative and (B) postoperative diffusion weight magnetic resonance imaging scans of the brain. Arrow shows the new asymptomatic microischemic lesion that was detected at the ipsilateral parietal lobe.

the formation of microemboli. Therefore, rinsing all possible particulate fragments and carefully inserting the shunt into the ICA and CCA have vital importance. The effect of plaque morphology on the CEA outcome is unclear. However, some studies have reported that microemboli occur during the dissection phase because of fragile carotid plaques [26]. Verhoeven *et al.* [22] divided carotid plaque morphology into three types (fibrous, fibroatheromatous, and atheromatous). According to this study, fibrous plaques were more related to the occurrence of microemboli than atheromatous plaques during the declamping of carotid artery and the closing of the arteriotomy. Atheromatous plaques were more related to occurrence of microemboli during the dissection phase. As a result, this study emphasized that cerebrovascular adverse events occurred more often in patients with atheromatous plaques than in patients with fibrous or fibroatheromatous plaques.

In the present study, six (6/38 CAEs; 15.8%) cases had silent cerebral emboli in the postoperative DW-MRI. In these patients, ulcerated and thrombosed plaque rates were 16.6%, the calcified plaque rate was 83.6%, and the degenerated plaque rate was 50%. This rates demonstrate that the importance of gentle handling of the carotid artery.

All ischemic lesions of brain may not be related to microembolism. Another possible mechanism might be hemodynamic compromise during the procedure. Critical reduction of the cerebral perfusion can cause cerebral ischemia in a certain group of patients [27, 28]. This mechanism may clarify

ischemic lesions outside of the treated carotid artery territory. Therefore, one (16.6%) of the new ischemic brain lesions was located outside of the operated carotid artery territory (ipsilateral occipital lobe) in the DW-MRI in our study.

Additionally, the temporary reduction of carotid blood flow can cause an ischemic event in the ipsilateral brain side in patients with inadequate cerebral collateral circulation (incomplete circle or occlusion of the circle of Willis) [29]. Therefore, systolic arterial pressure has to be at ± 20% from the preoperative baseline value during the cross clamping [30].

The Limitations of the Study

Our study has some limitations, including a relatively small sample size and there were no statistical comparisons. Long-term results of patients were also not presented. All operation were performed by using the shunt. So the comparison was not performed between patients with and those without shunt. Additionally effect of local anesthesia to the cerebral microembolism was unknown in this study. Because all operation were made under general anesthesia. Other factors that may be responsible for the silent cerebral embolism (cardiac rhythm disorders, cerebral blood flow, etc.) were not analyzed in this study. Also cerebral collateral circulation was not examined in all patients.

Conclusions

Silent cerebral ischemia may occur frequently postoperatively in CEA patients. Even if these ischemic lesions that detected with DW-MRI are asymptomatic, we have to be rigorous during all stages of the surgery. Each stage of the CEA may be responsible to cerebral embolism. Additionally, a lower than critical level of blood flow may be the cause of cerebral ischemia. Therefore, we have to maintain adequate blood pressure during the surgery to avoid cerebral hypoperfusion.

Conflict of interest

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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