

Results of ocular hemodynamics in chronic ocular ischemic syndrome after bilateral carotid endarterectomy

Kronik oküler iskemik sendromunda iki taraflı karotis endarterektomi sonrasında oküler hemodinami sonuçları

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Background: The aim of this study was to investigate the effect of carotid endarterectomy on chronic ocular ischemic syndrome due to internal carotid artery stenosis progressed with transient ischemic attacks (TIAs).

Methods: We examined 14 patients (12 males 2 females; mean age 62.3±5.4 years; range 47 to 71 years) with ocular ischemic syndrome due to bilateral internal carotid artery stenosis (>80% stenosis) who were treated by carotid endarterectomy. All patients had TIAs and complicated chronic ocular ischemic syndrome due to the ipsilateral internal carotid artery lesion. Preoperatively, dominant ocular sign was amaurosis fugax in 11 patients, quarantanopia in two, and blindness in one. Ophthalmic artery color Doppler flow imaging indicated ophthalmic artery flow direction and peak systolic flow velocity and was performed before and at 24 hours, one week, one month, and three months after surgery.

Results: The ophthalmic artery flow directions were reversed in nine patients and antegrade in five patients preoperatively. In the six patients who experienced antegrade ophthalmic artery flow before carotid endarterectomy, the average peak systolic flow velocity was -0.029 ± 0.05 m/s. Preoperative reversed flow resolved in each patient one week after surgery. All patients showed antegrade ophthalmic artery flow after surgery. The average peak systolic flow velocity in the patients, measured 24 hours after operation, when compared with preoperative antegrade flow values, rose significantly to 0.32 ± 0.14 m/s ($p < 0.05$). There was no significant difference when the first 24-hour findings were compared with those observed one week, one month and three months after endarterectomy. During the follow-up period (mean, 18.5 months), no recurrent visual symptoms were observed.

Conclusion: Carotid endarterectomy was effective for improving or preventing the progress of chronic ocular ischemia caused by internal carotid artery stenosis.

Key words: Carotid endarterectomy; ocular ischemic syndrome; ophthalmic artery; transient ischemic attack.

Amaç: Bu çalışmada transiyen iskemik atakla (TİA) seyreden internal karotis arter stenozunda, karotis endarterektomi girişiminin kronik oküler iskemik sendrom üzerindeki etkileri araştırıldı.

Çalışma planı: İki taraflı internal karotis arter stenozuna bağlı (>80 stenoz) kronik oküler iskemik sendromu olan ve karotis endarterektomi ile tedavi edilen 14 hasta (12 erkek 2 kadın; ort. yaş 62.3±5.4 yıl; dağılım 47-71 yıl) incelendi. Hastaların tümünde TİA ve aynı taraf internal karotis arter lezyonuna bağlı gelişen kronik oküler iskemik sendrom komplikasyonu vardı. Ameliyat öncesi dominant oküler bulgu; 11 olguda amaurosis fugax, iki olguda quarantanopia, bir olguda körlük idi. Oftalmik arter renkli Doppler akım ölçümleri oftalmik arter akım yönünü ve sistolik akım hızındaki en üst seviyeyi gösterdi ve ameliyattan önce ve ameliyattan 24 saat, bir hafta, bir ay ve üç ay sonralarında yapıldı.

Bulgular: Ameliyat öncesi oftalmik arter akımı dokuz olguda antegrad, beş olguda ters akım olarak saptandı. Karotis endarterektomi öncesinde antegrad akım saptanan altı hastada ortalama sistolik tepe akım hızı -0.029 ± 0.05 m/s bulundu. Ameliyat öncesi ters akım saptanan hastalarda ameliyat sonrası birinci haftada akım düzeldi. Ameliyat sonrasında bütün hastalarda antegrad oftalmik arter akımı gözlemlendi. Ameliyat öncesi antegrad akım değerleri ile kıyaslandığında ilk 24 saatlik ortalama sistolik tepe akım hızı 0.32 ± 0.14 m/s bulundu ($p < 0.05$) ve anlamlı artış gözlemlendi. Endarterektomi sonrasında ilk 24 saatlik ölçüm bulguları ile bir hafta, bir ay ve üç ay sonrasında yapılan ölçüm bulguları arasında fark saptanmadı. Ortalama 18.5 aylık izlem süresinde vizüel semptomlarda tekrarlama görülmedi.

Sonuç: Karotis endarterektomi girişiminin, internal karotis arter stenozuna bağlı kronik oküler iskemisinin düzelmesinde ve ilerlemesinin önlenmesinde etkin olduğu görüldü.

Anahtar sözcükler: Karotis endarterektomi; oküler iskemik sendrom; oftalmik arter, transiyen iskemik atak.

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Severe extracranial carotid artery stenosis causes ocular ischemia, including the acute manifestations after a retinal embolism (amaurosis fugax, central retinal artery occlusion, and branch retinal artery occlusion) and chronic progressive ocular ischemia (ocular ischemic syndrome).^[1-3] These deficits may be caused by structural brain damage or by the chronic compromise of the cerebral blood flow.^[1,4,5] The effect of a carotid endarterectomy on embolisms in the retinal circulation is well known.^[6,7] However, no clear evidence has been found regarding the efficacy of a carotid endarterectomy on chronic ocular ischemic syndrome caused by severe internal carotid artery stenosis. It is important to understand the flow pattern and condition of the ophthalmic artery in patients with internal carotid artery stenosis before and after carotid endarterectomy.

We analyze the effect of a carotid endarterectomy on chronic ocular ischemic syndrome due to internal carotid artery stenosis at its origin on the basis of data obtained from ophthalmic artery color Doppler flow imaging (CDFI) scans.

PATIENTS AND METHODS

We examined 14 patients (12 males 2 females; mean age 62.3 ± 5.4 years; range 47 to 71 years) with ocular ischemic syndrome due to internal carotid artery stenosis (>80% stenosis), who were to be treated by a carotid endarterectomy. The risk factors were identified as smoking in 12 patients (86%), diabetes mellitus in four patients (29%) and hyperlipidemia in six patients (43%).

All patients had had transient ischemic attacks (TIAs) before. The symptoms of the patients enrolled had to have occurred within the six months prior to the inclusion in this study. Patients who had a stroke within the last six months were excluded from the study. All patients had the complaint of chronic ocular ischemic syndrome on the side ipsilateral to the affected internal carotid artery. Preoperatively, the dominant ocular signs were diagnosed as amaurosis fugax in 11 patients, quadrantanopia in two, and blindness in one.

The ophthalmic artery CDFI findings from the eye ipsilateral to the carotid endarterectomy were analyzed. The ophthalmic artery CDFI indicates the ophthalmic artery flow direction and the peak systolic flow velocity. The CDFI evaluation was performed using a computed sonography of 128XP/10 with a 5 MHz probe. The power chosen was less than 50 mW/cm^2 and the examination was completed within five minutes. All patients received this CDFI with the aim of confirming the carotid artery occlusion. This ophthalmic artery CDFI performed before the carotid endarterectomy, was also repeated at time intervals of 24 hours, one week, one month and three months after of the operation.

According to the results of the CDFI before surgery, eight patients were found to have a reversed flow in the ophthalmic artery. It was recognized that these negative peak systolic flow velocity values would create erroneous data points in the calculation of the mean values for the entire group, so these measurements were excluded from the statistical analysis. The postoperative comparison in patients with a reversed ophthalmic artery flow direction was done on the basis of the restoration of normal, antegrade flow in the ophthalmic artery and the restoration of normal flow velocities.

Statistical analysis

The statistical analysis was performed using the statistical software SPSS 11.0 for windows (SPSS Inc., Chicago, Illinois, USA). The data are expressed as the mean value \pm standard deviation for the continuous variables and as percentages for the categorical variables. Differences between the categorical variables were tested using the χ^2 test; differences between continuous variables were tested using the unpaired t-test.

RESULTS

None of the patients with preexisting cerebrovascular problems had a new stroke.

In the preoperative period, all of the patients complained of chronic ocular ischemic syndrome. The ophthalmic artery flow directions were reversed in nine patients and antegrade in five patients preoperatively. This relationship between the chronic ocular ischemic syndrome and reversed ophthalmic artery flow direction was nonsignificant.

In the other five patients who experienced antegrade ophthalmic artery flow, the average peak systolic flow velocity was $-0.029 \pm 0.05 \text{ m/s}$. The preoperative reversed flow resolved in each patient one week after the surgery and an antegrade ophthalmic artery flow was observed all patients postoperatively. The average peak systolic flow velocity measured 24 hours after operation in the patients who preoperatively had an antegrade flow rose significantly, from $-0.029 \pm 0.05 \text{ m/s}$ to $0.32 \pm 0.14 \text{ m/s}$ ($p < 0.05$). In all the patients with a preoperatively reversed ophthalmic artery flow direction, a return to the normal antegrade flow direction was observed after the operation. This redirectioning of the reversed ophthalmic artery flow direction was significant. The average peak systolic flow velocity in the patients with preoperative antegrade flow rose to $0.32 \pm 0.14 \text{ m/s}$ ($p < 0.05$), which showed a significant increase as compared with the preoperative level $-0.029 \pm 0.05 \text{ m/s}$ ($p < 0.05$). There was no significant difference in the mean peak systolic flow velocities between the patients with preoperatively reversed ophthalmic artery flow direction and the

patients with preoperative antegrade ophthalmic artery flow direction.

There was no significant change in the peak systolic flow velocity in any of the patients with preoperative antegrade flow or in the patients with preoperatively reversed flow when the values at the time point one month after the carotid endarterectomy were compared. Three months after the carotid endarterectomy, the flow direction in the ophthalmic artery was still antegrade in all patients. At the end of the study period, the visual acuity had improved in six patients and had not worsened in the other eight patients. During the follow-up period (mean, 18.5 months), no patients complained of recurrent visual symptoms.

DISCUSSION

The internal carotid artery stenosis at its origin can influence the flow dynamics of the ophthalmic artery. The hemodynamic reduction of the ocular circulation due to severe internal carotid artery stenosis causes the ocular ischemic syndrome.^[8,9] A carotid endarterectomy is the best treatment in order to directly remove the internal carotid artery stenotic lesion. Numerous prospective, randomized and multi-centered studies have been designed to evaluate the efficacy and safety of a carotid endarterectomy.^[10,11] Occlusive internal carotid artery diseases lead to ophthalmic artery flow disturbances.^[8] The disturbed ophthalmic artery flow causes ocular ischemic syndromes such as amaurosis fugax or a decline in the visual acuity.

There are two potentially favorable effects of a carotid endarterectomy:^[6] Firstly, the carotid endarterectomy removes the atheromatous plaque, which is a possible source of cerebral embolisms. Another and more hypothetical explanation of the beneficial effect is the restoration of the cerebral perfusion pressure and the improvement in the hemodynamic status of the brain. Vascular events that occur in the brain also affect the eye, giving rise to different ophthalmologic manifestations that range from amaurosis fugax to complete blindness due to central retinal artery occlusion.

A small subgroup of patients who would benefit from carotid endarterectomy may experience an ocular ischemic syndrome.^[12] Therefore, it is important to evaluate the ophthalmic artery in the patients treated by a carotid endarterectomy (for the internal carotid artery stenosis.) However, there have been few reports about the effect of the carotid endarterectomy on the ophthalmic artery.^[13,14]

In the present study, we aimed to demonstrate the correction of the abnormal flow direction and the improvement of the ophthalmic artery flow velocity as the result of a carotid endarterectomy we performed. In

order to evaluate the effect of a carotid endarterectomy on the ophthalmic artery, we examined the artery through CDFI before and after the surgery and during the 18.5-month follow-up period. There is a strong correlation between the course of the ocular ischemic syndrome and improvement of the ophthalmic artery CDFI findings during the postoperative stage. The improvement of the peak flow velocity and normalization of the reversed ophthalmic artery flow direction was observable within one week after surgery. After this period, there was no significant improvement of the ophthalmic artery peak flow velocity.

Previous reports showed the hemodynamic improvement after carotid endarterectomy according to the serial single photon emission computed tomography imaging or transcranial Doppler flow studies.^[6,13] These improvements of the ophthalmic artery CDFI findings were evident immediately after the carotid endarterectomy, thus correlating well with the clinical ischemic syndrome.

We concluded that a carotid endarterectomy was effective for improving or preventing the progress of chronic ocular ischemia caused by internal carotid artery stenosis.

REFERENCES

1. Alizai AM, Trobe JD, Thompson BG, Izer JD, Cornblath WT, Deveikis JP. Ocular ischemic syndrome after occlusion of both external carotid arteries. *J Neuroophthalmol* 2005; 25:268-72.
2. Trobe JD. Carotid endarterectomy for transient monocular visual loss and other ocular ischemic conditions. *J Neuroophthalmol* 2005;25:259-61.
3. Caplan LR, Hertzner NR. The management of transient monocular visual loss. *J Neuroophthalmol* 2005;25:304-12.
4. Boto de los Bueis A, Fernández-Prieto A, Ruiz-Martín MM, Gorospe L, Amorena Santesteban G, Fonseca Sandomingo A. Bilateral carotid occlusion in young woman. Clinical and hemodynamic ocular results. *Arch Soc Esp Oftalmol* 2003;78:227-30. [Abstract]
5. Bakker FC, Klijn CJ, Jennekens-Schinkel A, van der Tweel I, Tulleken CA, Kappelle LJ. Cognitive impairment in patients with carotid artery occlusion and ipsilateral transient ischemic attacks. *J Neurol* 2003;250:1340-7.
6. Kawaguchi S, Sakaki T, Uranishi R, Ida Y. Effect of carotid endarterectomy on the ophthalmic artery. *Acta Neurochir [Wien]* 2002;144:427-32.
7. Pribán V, Fiedler J, Chlouba V. Ocular symptoms as an indication for carotid endarterectomy. *Cesk Slov Oftalmol* 2006;62:354-9. [Abstract]
8. Lawrence PF, Oderich GS. Ophthalmologic findings as predictors of carotid artery disease. *Vasc Endovascular Surg* 2002;36:415-24.
9. Alizai AM, Trobe JD, Thompson BG, Izer JD, Cornblath WT, Deveikis JP. Ocular ischemic syndrome after occlusion of both external carotid arteries. *J Neuroophthalmol* 2005;25:268-72.

10. Lal BK, Hobson RW 2nd. Treatment of carotid artery disease: stenting or surgery. *Curr Neurol Neurosci Rep* 2007;7:49-53.
11. Eren E, Balkanay M, Toker ME, Tunçer A, Anasiz H, Güler M, et al. Simultaneous carotid endarterectomy and coronary revascularization is safe using either on-pump or off-pump technique. *Int Heart J* 2005;46:783-93.
12. Shah H, Major KM, Alexander JQ, Hood DB, Rowe VL, Weaver FA. Recanalization of a thrombosed carotid artery following endarterectomy. *Ann Vasc Surg* 2007;21:172-7.
13. Kawaguchi S, Okuno S, Sakaki T, Nishikawa N. Effect of carotid endarterectomy on chronic ocular ischemic syndrome due to internal carotid artery stenosis. *Neurosurgery* 2001;48:328-32.
14. Wolintz RJ. Carotid endarterectomy for ophthalmic manifestations: is it ever indicated? *J Neuroophthalmol* 2005;25:299-302.