

# Sticky situations:

On November 25, 2023, Marianne Ala-Kauhaluoma defended her thesis, "Ocular findings in patients with carotid stenosis undergoing carotid endarterectomy," at the Faculty of Medicine, University of Helsinki. The PhD project was conducted at the Dept. of Ophthalmology, University of Helsinki, and Helsinki University Hospital, with Dr. Paula Summanen, MD, PhD, and docent Lauri Soinne, MD, PhD, as supervisors.

Marianne Ala-Kauhaluoma Faculty of Medicine, University of Helsink<u>i</u>

#### Introduction

Carotid stenosis (CS) can cause circulatory disturbances in not only the brain but also the eye. The eye is only subserved by the ophthalmic artery, which is a branch of the internal carotid artery. Ocular hypoperfusion causes various findings. How carotid endarterectomy (CEA)—often needed in CS to remove the mechanical hindrance of blood flow—affects retinal findings is partly unknown.

#### **Methods**

A total of 70 CS patients and 41 controls entered our study. We objectively graded retinal microvascular abnormalities and semiautomatically evaluated central retinal arterial and venular equivalent (CRAE and CRVE) before and 6 months after CEA. A dynamic vessel analyzer assessed flicker-induced vascular reactions, and optical coherence tomography measured subfoveal choroidal thickness (SFCT).

#### **Results**

We found ocular signs of CS in 17 patients, of whom four had embolic Hollenhorst plaques and 13 had hypoperfusion-related signs. The latter included ocular ischemic syndrome (OIS), with typical mid-peripheral hemorrhages requiring ocular treatment in four, mild OIS in six, and de novo findings in three. One patient had an irreversible neovascular glaucoma with poor visual acuity, and another developed ocular hyperperfusion with macular edema 1 week after CEA. Among microvascular abnormalities, the patients showed more severe grades in arteriolar and venular tortuosity in the macula than did the controls at baseline. CRAE was similar in laterality and in patients compared to controls. At the baseline, we detected higher CRVE in the patients' ipsilateral eyes. Ipsilateral arteriolar and venular flicker-induced dilation appeared lower in patients than in controls at the baseline. After CEA, ipsilateral venular dilation increased, and a trend toward the same was evident in arteriolar dilation. SFCT was lower in patients than in controls, with no difference in laterality.

#### Conclusions

Potential novel ocular biomarkers of CS include dilated venules, higher grades of vessel tortuosity in the macula, reduced flicker-induced dilatations, and low SFCT. Ocular signs of CS should be detected before irreversible changes occur. Those patients with reduced visual acuity or ocular pain or both, before or after CEA, need prompt ophthalmological examination. CEA seems to positively influence retinal blood flow.

### Future directions:

- Precise diagnostic criteria for ocular ischemic syndrome (OIS) and guidelines for the follow-up of these patients, with or without carotid endarterectomy, are needed.
- OIS should be recognized and treated before it becomes irreversible.

## Ocular findings in patients with carotid stenosis

#### **Key points:**

- Clinicians should be aware of the ophthalmological signs and findings related to carotid stenosis (CS) to detect the underlying condition.
- Moderate and severe grades of arteriolar and venular tortuosity in the macula may be considered a sign of CS.
- The hemodynamic effect of CS induces venular dilatation, seen as a higher central retinal venular equivalent in the ipsilateral eye, which is reversible after carotid endarterectomy.
- Subfoveal choroidal thickness is lower in patients with CS.

#### References

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