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Nikolaos Kontopodis

Stavros Charalambous

George Vourakis

Nikolaos Galanakis

Panayiotis Mitsias
Henry Ford Health, PMITSIA1@hfhs.org

See next page for additional authors

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Authors

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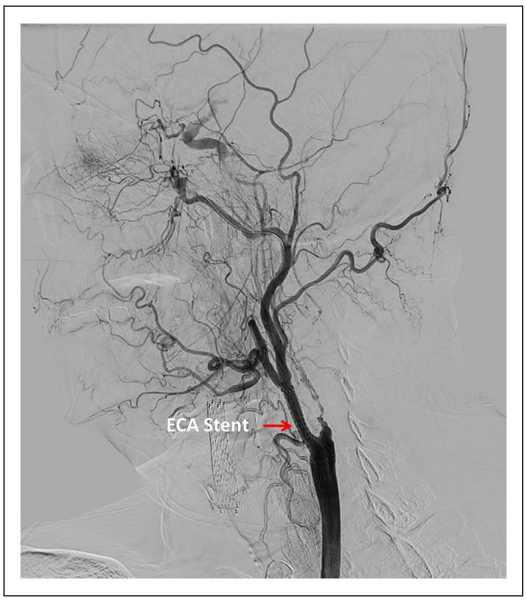
Images in Vascular Medicine: Bright light amaurosis – When external carotid artery stenosis matters

Nikolaos Kontopodis[1], Stavros Charalambous[2], George Vourakis[3], Nikolaos Galanakis[2], Panayiotis Mitsias[3,4,5], Dimitrios Tsetis[2] and Christos V Ioannou[1]

Panel A.



Panel B.

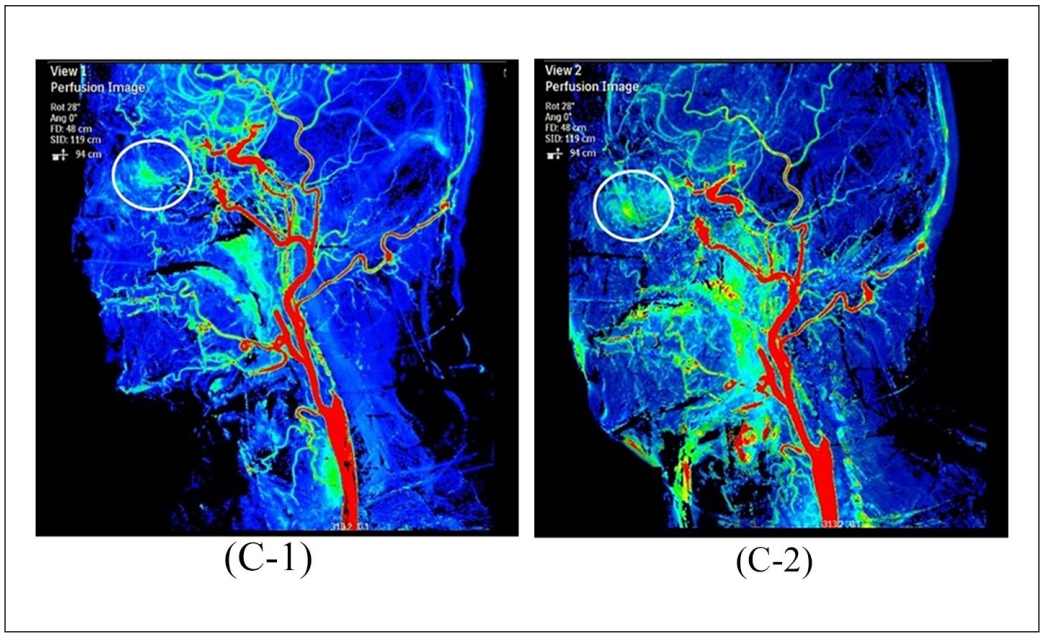
The ophthalmic artery (OA) is the first branch of the internal carotid artery (ICA) and constitutes the main arterial supply of the eye. Collateral supply from the external carotid artery (ECA) contributes to maintain adequate flow in the OA, in case of compromised inflow from a diseased ICA.[1] In the presence of ICA occlusion, hypoperfusion of the retina may result in low-flow transient ischemic attack in the form of 'retinal claudication' or bright-light amaurosis, a phenomenon originally described by Furlan et al.[2] This refers to transient blindness brought on by sudden exposure to bright light, likely caused by increased metabolic demand of the retina that cannot be matched by the necessary increase in retinal blood flow due to carotid occlusion. Retinal claudication represents a very rare clinical presentation that is frequently missed by most vascular care providers.

We encountered a 75-year-old patient who presented with retinal claudication of the left eye in the presence of a long-standing ICA occlusion, with postocclusive poor recanalization and concomitant significant stenosis (~60%) of the left ECA (Panel A). No significant stenosis was recorded from the contralateral side, but the circle of Willis was incomplete due to absent anterior communicating artery. A transcranial Doppler study revealed flow reversal in the OA with blunted monophasic waveforms, a peak systolic velocity of 30 cm/sec and pulsatility index of 1, findings consistent with a proximal occlusive process of the collateral circulation from ECA branches.

Following a multidisciplinary review of the case, the patient was deemed an appropriate candidate for ECA revascularization to assist perfusion of the OA through the collateral network[3,4] and he underwent stenting of the left ECA (Protégé RX 8/6 × 30 mm; Medtronic, Dublin, Ireland) (Panel B).

[1]Department of Cardiothoracic and Vascular Surgery, Vascular Surgery Unit, University Hospital of Heraklion, Crete, Greece
[2]Department of Medical Imaging, Interventional Radiology Unit, University Hospital of Heraklion, Crete, Greece
[3]Department of Neurology, University Hospital of Heraklion, Crete, Greece
[4]School of Medicine, University of Crete, Heraklion, Greece
[5]Department of Neurology, Henry Ford Hospital, Detroit, MI, USA

Corresponding author:
Nikolaos Kontopodis, Department of Cardiothoracic and Vascular Surgery, Vascular Surgery Unit, University Hospital of Heraklion, PO Box 1352, Heraklion, 711 10, Greece.
Emails: kontopodisn@yahoo.gr; nkontopodis@uoc.gr



Panel C.

A perfusion-based digital subtraction angiography was performed pre- and poststent deployment and a change in pixel signal intensity (SI) within a region of interest (ROI) was mapped over time, generating a SI curve. An operator-defined ROI was placed within the ocular region, as can be seen within the circle of Panel C-1 (prestenting) and C-2 (poststenting), and a detectable improvement in SI and flow parameters, such as wash-in rate (WiR), time-to-peak (TTP), width (Wid), and mean-transit-time (MTT), were observed postintervention. WiR and TTP reflect to the briskness of blood flow within a ROI, whereas Wid and MTT represent the time duration for contrast to travel through the ROI. A significant enhancement of the intraocular tissue perfusion within a ROI with a 58.3% increase in WiR after intervention was illustrated in the encircled region of Panel C-1 (prestenting) and C-2 (poststenting). The postoperative course was uneventful and symptoms resolved immediately after the intervention. Postoperative transcranial Doppler study revealed normalized waveforms in the left OA.

In conclusion, retinal claudication is a rare clinical entity that may be encountered in the presence of concomitant ICA occlusion and ECA disease. Awareness of this uncommon presentation is necessary for the definitive diagnosis, whereas restoration of blood flow in the ipsilateral ECA can increase cerebral and intraocular blood perfusion, helping to relieve symptoms.

Declaration of conflicting interests

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ORCID iD

Nikolaos Kontopodis https://orcid.org/0000-0002-6792-5003

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