Vision loss due to ophthalmic artery occlusion secondary to spontaneous internal carotid artery dissection

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Case

A 47-year-old male smoker with hypertension, diabetes mellitus, and hyperlipidaemia complained of right eye vision loss on waking. His best-corrected visual acuity was reduced to light perception only. Physical examination revealed anisocoria without ptosis and no extraocular movement deficit. The right eye pupil was larger than the left, and the difference was more obvious under a light environment. Direct and consensual pupil reflexes were both present but there was a marked right relative afferent pupillary defect. Slit lamp examination revealed normal anterior segments of the eyes, but a right pale optic disc without rim thinning, and a typical cherry-red spot over the right fovea surrounded by generalised whitened retina. Vessels were not tortuous and no emboli were seen nor retinal haemorrhage over different layers (Figs 1 and 2). Left eye posterior segment was normal. The patient also reported right-sided headache and left-sided numbness. Thorough physical examination revealed no other cranial nerve deficit or systemic focal neurological deficit. Temporal pulse was easily palpable and non-tender, and no carotid bruit was heard. The patient worked on a construction site and denied any trauma. He commenced hyperbaric oxygen therapy but no improvement was observed. Blood tests and brain imaging were all normal, but carotid Doppler showed significant obstruction >90% of the internal carotid artery, accounting for the absence of bruit. Computed tomography angiography confirmed dissection of a long segment of the right internal carotid artery (Figs 3 and 4), not amendable to stenting or bypass surgery. A diagnosis of ophthalmic artery occlusion was made and explained the ineffectiveness of hyperbaric oxygen therapy due to choroidal ischaemia. Symptomatic internal carotid artery dissection is a rare but major cause of young-onset stroke, itself uncommon.[1] Neck trauma is a major aetiology, and there is a slight male predominance with mean age of onset in the 40s.[2] Apart from the neurological signs and symptoms of stroke, the ophthalmological presentation of internal carotid artery dissection is more similar to that of painful Horner's syndrome due to compression of the adjacent third-order sympathetic chain fibres; followed by cranial nerve palsy, caused by direct local compression or compromise of feeder vessels.[3] Ophthalmic artery occlusion is rare. Computed tomography angiography has 80% sensitivity for diagnosis and patients need to be closely monitored for massive stroke that may occur weeks to months after first presentation.[4] Hyperbaric oxygen therapy is indicated for central retinal artery occlusion, but

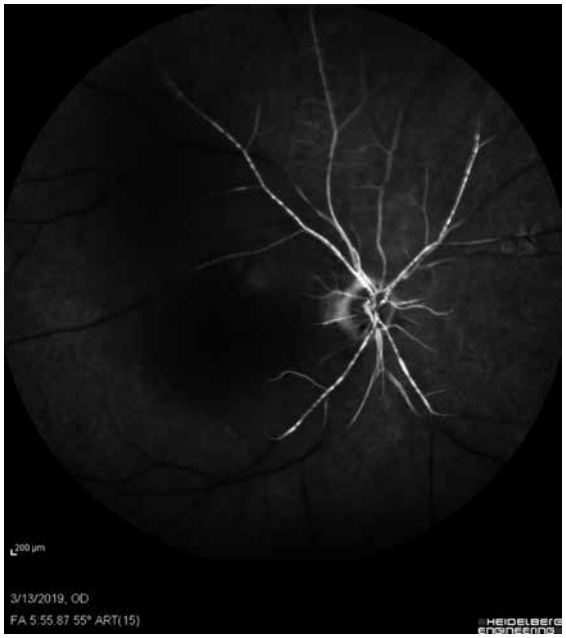

FIG 1. Fundus fluorescein angiography of the right eye showing delayed and incomplete perfusion of the retina evidenced by the incomplete filling of arteries by fluorescein at around 6 minutes (normal is 10-13 s)

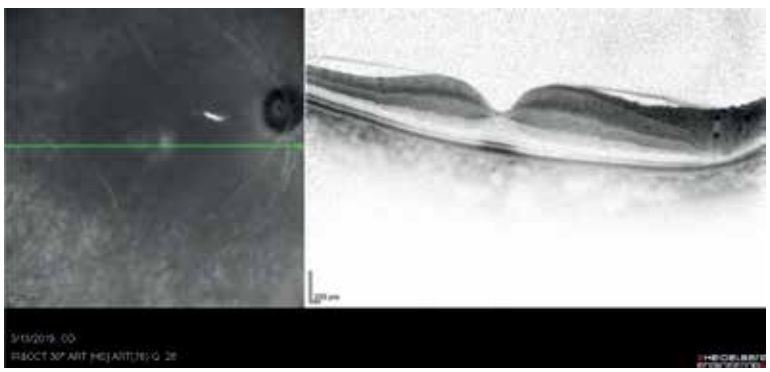

FIG 2. Optical coherence tomography of the right eye showing generalised retinal oedema secondary to retinal ischaemia that marks the typical clinical sign of cherry red spot on fundus examination

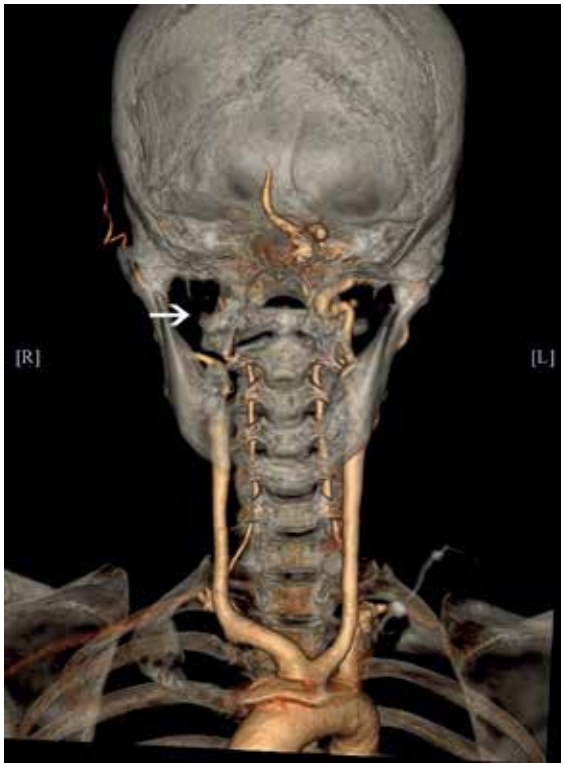

FIG 3. Three-dimensional computed tomography angiography showing the presence of left, but the absence of right internal carotid artery (indicated by white arrow) perfusion by contrast

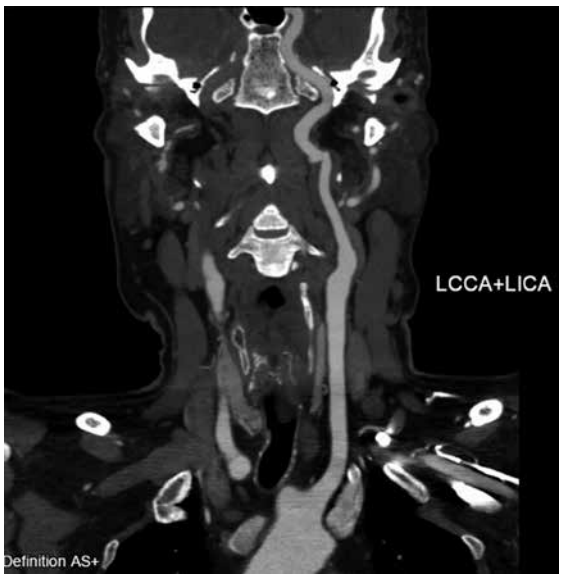

FIG 4. Coronal computed tomography angiography showing the presence of left, but the absence of right internal carotid artery perfusion by contrast

not ophthalmic artery occlusion. It aims to reperfuse the ischaemic retina with oxygen by diffusion from the choroidal circulation, bypassing the obstructed retinal vasculature. The choroidal arteries are supplied by the posterior ciliary arteries that branch from the ophthalmic artery. If the ophthalmic artery is occluded, hyperbaric oxygen therapy has no means to tackle the compromised posterior ciliary vessels.[5]

In conclusion, vision loss due to internal carotid artery dissection is uncommon. Multidisciplinary care is essential.

Author contributions

Concept or design: SCL Au.
Acquisition of data: SCL Au.
Analysis or interpretation of data: SCL Au.
Drafting of the manuscript: SCL Au.
Critical revision of the manuscript for important intellectual content: All authors.

All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of interest

All authors have disclosed no conflicts of interest.

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Ethics approval

This study was conducted in accordance with the principles outlined in the Declaration of Helsinki. Relevant patient consent was obtained for the purpose of this case study.

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