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LETTER TO THE EDITOR

Central retinal artery occlusion and internal carotid artery dissection after COVID-19 nasopharyngeal swab testing

Occlusion de l'artère centrale de la rétine et dissection de l'artère carotide interne après un prélèvement nasopharyngé pour COVID-19

Isolated central retinal artery occlusion (CRAO) is a rare manifestation of internal carotid artery dissection (ICAD) [1]. ICAD may occur spontaneously or following minor trauma. We report an unusual case of CRAO secondary to ICAD in which the only visual symptom onset coincided with a nasopharyngeal swab for SARS-CoV-2 on reverse-transcriptase-polymerase-chain-reaction (PCR) assay.

A 56-year-old man with a dense posterior subcapsular cataract in his right eye (RE), was subjected to a PCR test to exclude SARS-CoV-2 infection two days prior to the cataract surgery. His medical history included asthma, hypertension, depression and cigarette use of two packs per day. Right at the end of the PCR procedure, he experienced a sudden, painless vision loss in his right eye that was not recovered. He denied any other accompanying symptom or previous transient visual loss. On examination, visual acuity (VA) was hand motion in his RE and 20/25 in his left eye. There was a right relative afferent pupillary defect without anisocoria. There was no ptosis and extraocular motility was full. The anterior segment examination was unremarkable apart from the posterior subcapsular cataract in his RE. Intraocular pressure was 16 mm Hg in both eyes. Fundus examination showed right retinal edema and cherry-red spot without visible emboli. With the diagnosis of acute right CRAO, the patient was referred to neurology emergently. The examination revealed no focal neurological deficit. There was a normal sinus rhythm on electrocardiogram. Acute phase reactants were within normal limits. Computed tomography (CT) angiography of the neck and head revealed right internal carotid artery dissection from its origin, with complete occlusion and re permeabilization distal to the level of the origin of the ophthalmic artery, that presented an endoluminal thrombus with occlusion (Fig. 1). Diffusion magnetic resonance imaging (MRI) demonstrated four high intensity small focus of acute ischemia located in the border between the middle cerebral artery and posterior cerebral artery territories of the right brain hemisphere.

On repeated questioning, the patient recalled sneezing once while his neck was in hyperextension during the PCR procedure, just before the visual loss. He denied any recent head or neck trauma prior to the PCR performance; the result of which was negative. Medical treatment was started with clopidogrel and heparin. One week later, VA in his RE remained unchanged and retinal oedema with cherry-spot could still be observed (Fig. 2). No focal neurological deficit was added.

Internal carotid artery dissection is a well-known cause of stroke in populations below the age of 70 years, accounting for up to 2.5% of all first strokes and up to 20% in younger patients [2]. The most common clinical manifestations of ICAD include a sudden onset ipsilateral cervical pain or headache, Horner syndrome and transient or persistent focal neurological deficits, which may appear specially during the first 2 weeks after the dissection occurs [1,3]. Two-thirds of patients with ICAD have ophthalmological manifestations, which are often the initial presentation of this condition. The most common ocular sign, ipsilateral Horner syndrome, has been reported in 44% to 58% in large series. Transient monocular visual loss has been reported in 6% to 38%. Less frequent ocular manifestations are oculomotor nerve palsy and permanent visual loss secondary to ischemic optic neuropathy, CRAO or ocular ischemic syndrome [4,5]. CRAO is a rare complication of ICAD and its pathophysiologic mechanism is believed to be from haemodynamic compromise with ocular hypoperfusion or, less frequently, secondary to thromboembolic phenomena originated from the dissected area [6]. CRAO due to ICAD is usually associated with other ophthalmologic or neurologic manifestations and cervical or facial pain [5]. There are few cases with an isolated CRAO secondary to ICAD reported to date as is the case of our patient [3,6,7]. Recognising cases that do not present with typical signs and symptoms of ICAD is a challenge because of the risk of hemispheric stroke within the first two weeks after the onset of symptoms.

ICAD may be traumatic or spontaneous. A positive family history is reported in 2—18% of patients [2]. Certain genetic connective tissue diseases such as Marfan syndrome, Ehlers-Danlos syndrome, Osteogenesis Imperfecta and fibromuscular dysplasia are reported to increase the risk of ICAD. Most frequently, ICAD may be the result of genetic predisposition, risk factors as hypertension, migraine or infection and environmental triggers as minor cervical trauma or physical effort [8]. These mechanical triggers involve sudden hyperextension or rotation of the neck, or intrathoracic pressure. Coughing, vomiting, vigorous nose blowing, chiropractic manipulation, Valsalva manoeuvres, body building, practising yoga and whiplash injuries have all been implicated in ICAD [7,9,10]. Our patient had hypertension as a risk

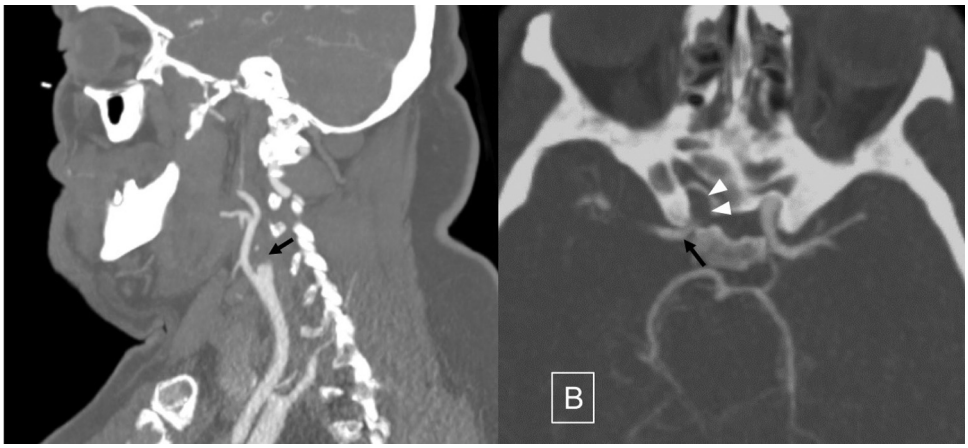

Figure 1. CT angiography with maximum intensity projection (MIP) reconstruction, suggestive of occlusive dissection of the right internal carotid artery (RICA). A. Sagittal plane: Post-bulbar RICA occlusion by dissection (black arrow). B. Axial plane: Absence of contrast in up to RICA C6 segment (white arrowheads) and re-permeabilization in RICA C7 segment (black arrow).

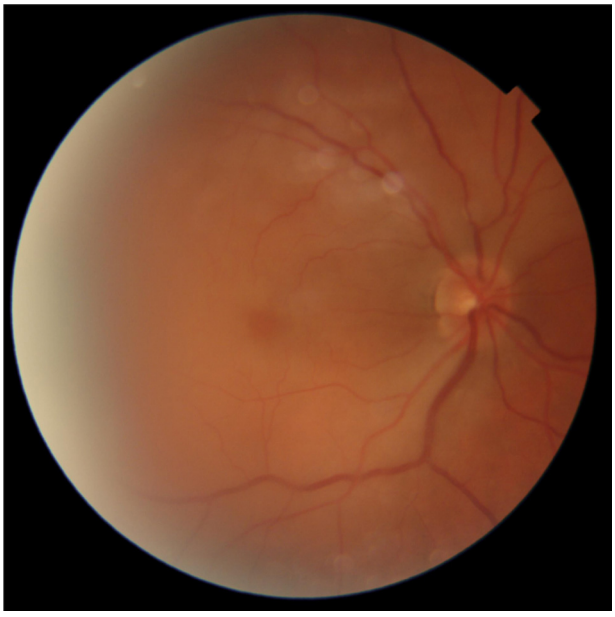

Figure 2. Right fundus photograph showing retinal oedema and cherry red spot that persisted one week after the onset of symptoms. The image quality was limited by the cataract opacity.

factor for spontaneous ICAD, but his visual loss was clearly related to PCR testing procedure. We hypothesized that the cervical hyperextension required to introduce the nasopharyngeal swab followed by the cervical flexion secondary to sneezing could have been the trigger for carotid dissection that caused CRAO and asymptomatic acute brain ischemia in this patient.

This case highlights the importance of considering carotid dissection in young patients with central retinal artery occlusion, even in the absence of other typical symptoms of ICAD, like pain, Horner syndrome or focal neurological deficit, especially if it is associated with sudden movements of the neck. Referral for urgent neurologic evaluation is important, because early diagnosis of ICAD may prevent further cerebral ischemic complications.

Disclosure of interest

The authors declare that they have no competing interest.

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