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CLINICAL COMMUNICATION

Combined central retinal vein and cilioretinal artery occlusion with anterior ischaemic optic neuropathy in a haemodialysis patient

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Introduction

Most cases of anterior ischaemic optic neuropathy (AION) are non-arteritic (90–95%) and usually unilateral.[1] Non-arteritic AION may have several causes such as atherosclerosis, diabetes, hypertension, obstructive sleep apnoea syndrome, hypercoagulable conditions and immune vasculitides. Sudden loss of blood volume, hypotension, chronic haemodialysis and some medications may also lead to optic nerve ischaemia.[2]

Vascular supply of the optic nerve head may vary among individuals. Branches from short posterior ciliary arteries go to the relevant parts of the optic nerve head, peripapillary choroid, the circle of Zinn (when present) and also branches to the retrolaminar pial vascular plexus. All these constitute the main source of blood supply to the optic nerve head.[3] Cilioretinal artery may arise either from the vessels entering the choroid or from the circle of Zinn. When present, the corresponding sector of the surface layer of the optic nerve head usually gets its blood supply from this vessel.[4]

While this is the case, coexistence of AION and cilioretinal artery occlusion is rare in clinical practice. Cilioretinal artery occlusion often occurs in the presence of a central retinal vein occlusion (CRVO).[1,2] The mechanism of this association still remains unclear. There are several hypotheses proposed to explain the simultaneous development of cilioretinal artery occlusion and CRVO, such as haemodynamic blockage, nocturnal hypotension, choroidal steal, optic disc oedema and embolism.[5–7]

Here, a case of coexisting CRVO, cilioretinal artery occlusion and non-arteritic AION, is described in a patient with chronic renal failure receiving haemodialysis treatment. Such a combination does not appear to have been reported before.

Case report

A 68-year-old male patient was referred to the ophthalmology department with a 7-day history of suddenly decreased vision in the right eye. He was hospitalised to receive haemodialysis due to hypertension-related chronic kidney disease. He lost vision in the left eye months ago after a cardiac catheterisation. The patient did not report any history of headaches, scalp tenderness, jaw claudication or polymyalgia rheumatica. At presentation, visual acuity was light perception on the right and counting fingers at 2 m on the left side. There was mild relative afferent pupillary defect in the right eye. Colour vision test and visual field testing could not be performed due to severe visual impairment.

Examination of the right eye showed pale optic disc swelling, dilatation and tortuosity of the retinal veins and diffuse retinal haemorrhages with retinal whitening in the distribution of the cilioretinal artery (Figure 1). Examination of the fellow eye showed optic atrophy with tortuous veins, more prominently in the lower quadrants and few dot-blot haemorhages in the peripheral fundus.

Fundus fluorescein angiography revealed normal choroidal perfusion, delayed venous filling and delayed emptying without any non-perfused retinal areas, impaired filling of the cilioretinal artery, and hyperfluorescence of the optic disc (Figure 2). Examination of the fellow eye showed optic atrophy with tortuous vessels in the lower quadrants.

Systemic evaluation including complete blood cell count, haemoglobin, C-reactive protein, erythrocyte sedimentation rate, coagulation profile, collagen vascular diseases, carotid and temporal artery Doppler ultrasound and echocardiogram revealed no obvious abnormalities. Neurological examination and diffusion magnetic resonance imaging were also normal.

According to these findings, a diagnosis of non-arteritic AION with non-ischaemic CRVO and cilioretinal artery occlusion is secondary to haemodialysis. Despite prompt systemic corticosteroid and intravitreal bevacizumab treatments, there was no improvement in visual acuity during the 6-month follow-up.

Discussion

Central retinal vein occlusion may occur with central or branch retinal artery occlusion or cilioretinal artery occlusion, the latter accounting for the majority of cases.[5] Though the pathogenesis of combined CRVO and cilioretinal artery occlusion still needs further evaluations, several theories have been suggested in different studies.

McLeod and Ring proposed that the main occluded vessel was cilioretinal artery and that the co-occurrence of CRVO may be due to occlusion of the posterior ciliary arteries with optic neuropathy.[6] The authors also suggested the theory of 'choroidal steal', which refers to blood diverted from cilioretinal artery to the choroidal bed after CRVO.

Haemodynamic block theory by Hayreh et al.[7] brought a brought a different perspective by stating that changes in intraluminal perfusion pressure occur either when the pressure in the arteries decreases or when the pressure in the

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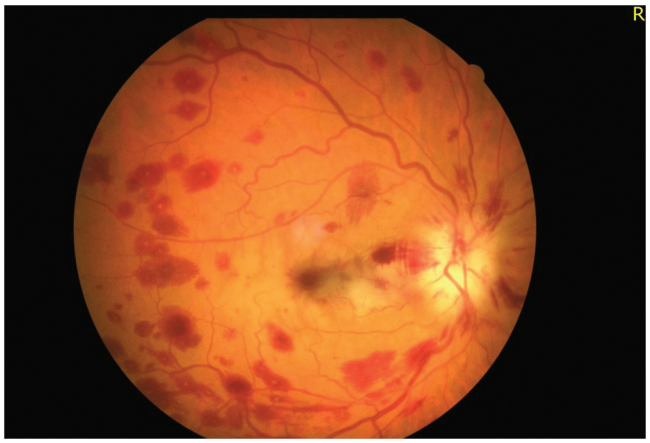

Figure 1. Diffuse haemorrhages with venous dilatation and tortuosity, retinal whitening in the distribution of the cilioretinal artery extending from the disc to the macula and marked pale optic disc swelling.

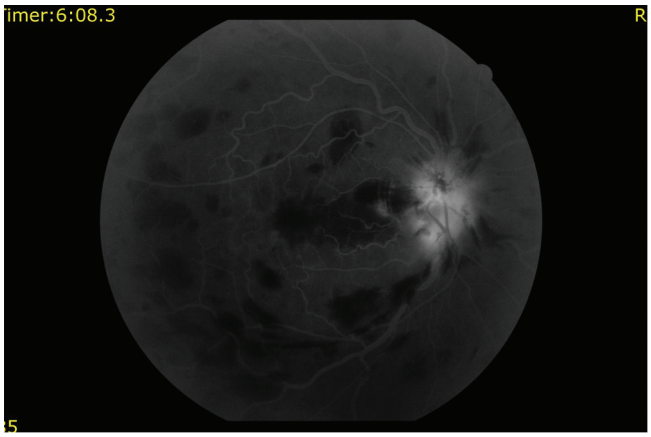

Figure 2. Impaired filling of the cilioretinal artery and hyperfluorescence of the optic disc with dilated veins in the angiography.

veins increases.[4] Therefore, due to anatomic features and lack of central retinal artery-like autoregulation in the choroidal vascular bed, the cilioretinal artery appears to be vulnerable to the haemodynamic blockade caused by CRVO. The authors also reported that nocturnal arterial hypotension may have important implications for understanding the mechanism of this combination.[7]

CLRA is found in about 20% of the population and the short posterior ciliary arteries supply the optic nerve head along with the cilioretinal artery. When a cilioretinal artery occlusion occurs in the setting of an AION, it is almost diagnostic for arteritic-AION associated with temporal arteritis. Therefore, the initial clinical presentation of the patient suggested an arteritic-AION at first. Nevertheless, the absence of common systemic symptoms (such as headache, jaw claudication, scalp tenderness and polymyalgia rheumatica) and the interval between the visual loss in both eyes being months supported the diagnosis of NAION.

Moreover, there were no signs of choroidal hypoperfusion on fundus fluorescein angiography. Temporal artery colour Doppler ultrasound, C-reactive protein and erythrocyte sedimentation rate were normal. There were not any optic disc excavations in both eyes during the follow-up. Fellow eye had tortuous retinal veins with few peripheral dot-blot haemorrhages and optic atrophy at initial examination which suggested that the patient possibly had a previous CRVO together with a non-arteritic AION in this eye.

Chronic haemodialysis patients usually have risk factors for NAION, such as anaemia, hypotension, atherosclerosis and uraemia. McIntosh et al. found that chronic kidney disease was an independent risk factor for retinal vein occlusion.[8] This patient had been undergoing haemodialysis for 5 years because of chronic kidney failure due to hypertension and potentially optic nerves may have subjected to inadequate blood flow.

Impaired blood clotting in these patients may also play a role in the development of vascular occlusions. McLeod and Ring suggested a spectrum of vascular lesions between acute CRVO and acute ischaemic optic neuropathy; thus, co-occurrence of CRVO might have been due to occlusion of the posterior ciliary arteries with ischaemic optic neuropathy.[6]

Conclusion

This report describes an unusual association of non-arteritic AION, cilioretinal artery occlusion and CRVO in a haemodialysis patient. CRVO seems to be accompanied by both cilioretinal artery occlusion and non-arteritic AION. Clinicians should be aware of this serious and irreversible complication, as close monitoring of the systemic condition of a patient may help to restore some vision and prevent possible life-threatening complications. Further studies are needed to improve current knowledge of this uncommon vascular complication.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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