Bilateral arterial occlusions masking retinitis in a HIV-positive male

Kalpana Babu, Bhagya Sudheer, Krishna R Murthy

We report an interesting case of 36-year-old HIV-positive male with uveitis, cilioretinal artery occlusion in OD, and superotemporal branch retinal artery occlusion in OS. Hypercoagulability, cardiovascular, and rheumatologic workups were unremarkable. Aqueous taps were negative for toxoplasma, viruses, and *MTb* by multiplex polymerase chain reaction. Patches of retinitis were seen on clearing of retinal edema. Serology was positive for toxoplasma and rickettsia. Management included doxycycline, azithromycin, bactrim DS, and oral steroids. Vision improvement to 6/60 and 6/24 in OD and OS refer to the right eye and left eye, respectively, were noted at 4-month follow-up. Infections should be considered in arterial occlusions associated with inflammation in HIV-positive individuals.

Key words: Arterial occlusions, bilateral, HIV, retinitis, rickettsia, toxoplasma

Branch retinal artery occlusions in young patients are rare. They can occur as a result of conditions that promote formation of emboli, hypercoagulable states, or infections. Causes of inflammatory branch retinal artery occlusions include toxoplasmosis, rickettsiosis, West Nile virus, syphilis, tuberculosis, idiopathic retinal vasculitis, aneurysms and neuroretinitis, Behcet disease, and Crohn's disease.[1]

We report an interesting case of bilateral arterial occlusions masking bilateral retinitis in a HIV-positive male on antiretroviral therapy (ART).

Case Report

A 36-year-old retrovirus-positive male presented with sudden diminution of vision of 2 days duration. He had a history of fever 2 weeks ago. He was on ART for 3 years, and his CD4 counts were 166 cells/mm^3 at the time of examination.

His best-corrected visual acuity was count fingers at 2 m (OU [both eyes]). Slit lamp examination showed an anterior chamber (AC) reaction of 1+ (SUN)[1] in both eyes. Fundus examination showed a cilioretinal artery occlusion in the right eye and superotemporal branch retinal arterial occlusion in the left eye. Peripheral retina showed scattered areas of arteriolar occlusion and vasculitis [Fig. 1a and b]. There was minimal vitritis in both eyes. Aqueous was negative for toxoplasma, HSV, VZV, CMV, and *MTb* by multiplex polymerase chain reaction (PCR) in both eyes. Coagulation testing including prothrombin time, activated partial thromboplastin time, D Dimer, protein S, C, factor 5, factor 8, and serum homocysteine was normal. Rheumatologic workup including antinuclear antibodies, anticardiolipin antibodies, and lupus anticoagulant was negative. Erythrocyte sedimentation rate and C-reactive protein were normal. Complete blood counts and basic metabolic panel including liver enzymes, lipid profile, renal function, blood sugars, and urine routine were normal. Electrocardiography and echocardiography revealed no cardiac abnormalities. Carotid Doppler was normal. As the retinal edema cleared, patch of retinitis distinct from the arterial occlusion was noted [Fig. 1c and d]. Fluorescein angiography confirmed areas of vascular occlusion (OU) and hypofluorescent areas near foveal avascular zone with a marginal hyperfluorescence in late phases [Fig. 2]. ELISA for toxoplasma showed increased titers of IgG antibodies (400 IU/ml) and negative IgM antibodies. Venereal disease research laboratory test and Treponema pallidum haemagglutination test (VDRL and TPHA) for syphilis were negative. Weil–Felix

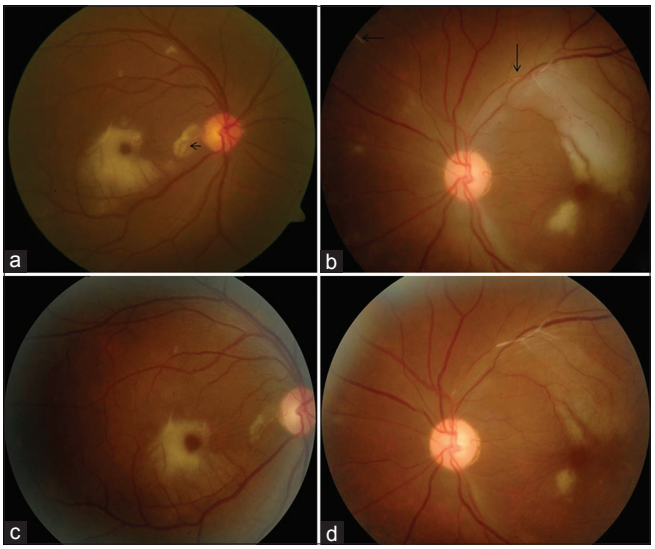

Figure 1: Fundus photograph showing cilioretinal artery occlusion with cherry red spot in OD (a) and superotemporal branch retinal artery occlusion with multiple areas of arteriolar occlusion (black arrow) and cotton-wool spots in OS (b). After 1 week with resolving retinal edema, a patch of retinitis near the foveal avascular zone and temporal to the optic disc (black arrows) in OD (c) and a distinct patch of retinitis near the fovea (black arrow) in OS (d)

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Prabha Eye Clinic and Research Center, Vittala International Institute of Ophthalmology, Bengaluru, Karnataka, India

Correspondence to: Dr. Kalpana Babu, Prabha Eye Clinic and Research Centre, 504, 40th Cross Road, 8th Block, Jayanagar, Bengaluru - 560 070, Karnataka, India. E-mail: kalpanbabumurthy@gmail.com

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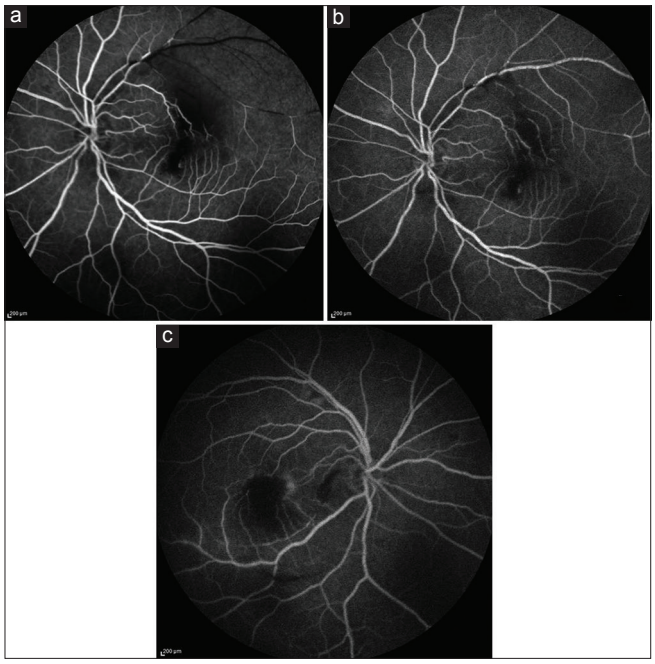

Figure 2: Fluorescein angiography shows superotemporal retinal nonperfusion in early phases and area of blocked fluorescence area adjacent to the fovea (a) and retrograde filling with mild marginal hyper fluorescence adjacent to the fovea in late phases in OD (b). Retrograde filling of cilioretinal artery and mild marginal hyperfluorescence at the area of the foveal avascular zone and area of blocked fluorescence near the optic disc in OS (c)

test (tube method) was strongly positive (1:400). Mantoux, QuantiFERON-TB gold tests, serum angiotensin-converting enzyme, and chest X-ray were negative. Magnetic resonance imaging of brain and orbits were normal. Treatment was started with doxycycline 100 mg bd followed by once a day for 6 weeks in addition to azithromycin 500 mg OD and bactrim DS twice a day. Oral steroids were initiated after 48 hours. The retinal edema cleared over 6 weeks and the retinitis resolved with a scar [Fig. 3]. At 4-month follow-up, vision improved to 6/60 (OD) and 6/24 (OS).

Discussion

We describe a rare case of cilioretinal artery occlusion (OD) and branch retinal artery occlusion (BRAO) (OS) due to infectious retinitis in a HIV-positive individual. Inspite of the AC reaction, the retinitis lesions were not initially appreciated due to the retinal edema corresponding to the vascular occlusion. PCR on the aqueous done at the initial visit was negative for toxoplasma. Rothova *et al*.[2] noted that DNA from Toxoplasma gondii was found intraocularly in 37% of cases of recently acquired disease and 4% of recurrent ocular toxoplasmosis. Diagnosis could be made once the retinal edema started to resolve and retinitis patches were distinctly seen. Vascular occlusion though rare has been reported in toxoplasmosis.[3-5] What was interesting in this case was the high titers of Weil–Felix test suggestive of a rickettsial infection. Kahloun *et al*.[1] in their series show rickettsiosis as the second most common cause of inflammatory BRAO after toxoplasmosis. The high titers of Weil–Felix test (titrated by tube method) could definitely not

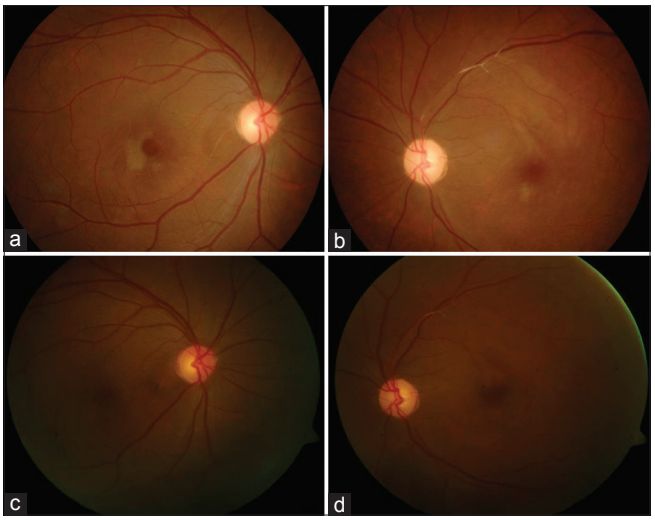

Figure 3: Fundus photograph showing resolving retinal edema and retinitis after 4 weeks in OD (a) and OS (b) and after 4 months in OD (c) and OS (d)

be ignored resulting in treatment with both antitoxoplasma and antirickettsial drugs.

Many mechanisms may be involved in the development of BRAO. It can occur as a result from direct compression of the artery by the focus of retinitis leading to interruption of the blood flow. As a response to an acute inflammatory stimulus, arteriolar contraction can occur leading to increased blood viscosity and release of heparin from the mast cells causing coagulation inhibition. Perivasculitis due to infiltration of the vessel wall by the inflammatory cells can also occur resulting in thickening of the vessel wall, disruption of blood flow, and thrombosis in the arteries.[6,7]

This case highlights the rare occurrence of bilateral retinal vascular occlusions due to infectious retinitis. The retinal edema and the vascular occlusions were overwhelming and masked our appreciation of the areas of retinitis. Earlier diagnosis in this case may have spared an otherwise exhaustive workup.

Conclusion

A rare finding of bilateral arterial occlusions with retinitis with positive serology for toxoplasma and rickettsia in a HIV-positive patient is reported. Infectious retinitis should be considered in arterial occlusions associated with inflammation in HIV-positive individuals.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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