Combined central retinal artery and vein occlusion with optic perineuritis following herpes zoster dermatitis in an immunocompetent child

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A 15-year-old healthy boy developed acute, rapidly progressing visual loss in left eye following herpes zoster dermatitis, with a combined central retinal artery occlusion (CRAO) and central retinal vein occlusion (CRVO), along with optic perineuritis. Laboratory tests were negative. Despite an empirical, intensive antiviral treatment with systemic corticosteroids, and vision could not be restored in the affected eye. Herpes zoster dermatitis, in an immunocompetent individual, may be associated with a combined CRAO and CRVO along with optic perineuritis, leading to profound visual loss.

Key words: Central retinal artery occlusion, central retinal vein occlusion, herpes zoster dermatitis, optic perineuritis

Herpes zoster dermatitis has been reported to be followed by acute visual loss due to acute retinal necrosis (ARN),[1,2] optic neuropathy,[3,4] and central retinal vein occlusion (CRVO).[4] Browning *et al*. have reported a period of 5 days to 3 months between skin infection and ARN.[2] Biswas *et al*. reported a patient with no light perception 4 days after a VZV dermatitis presenting with an acute optic neuropathy with CRVO.[5] Here, we describe an immunocompetent child developing an acute visual loss secondary to a combined central retinal artery occlusion (CRAO) with CRVO following an episode of herpes zoster dermatitis.

Case Report

A 15-year-old healthy boy presented with loss of vision in left eye since about 15 h. He had vesicular lesions on the left side of his forehead and upper eyelid (suggestive of herpes zoster infection), which had erupted 10 days earlier [Fig. 1].

Access this article online

Quick Response Code:

Website: www.ijo.in

DOI: 10.4103/ijo.IJO_480_17

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Manuscript received: 09.06.17; Revision accepted: 19.08.17

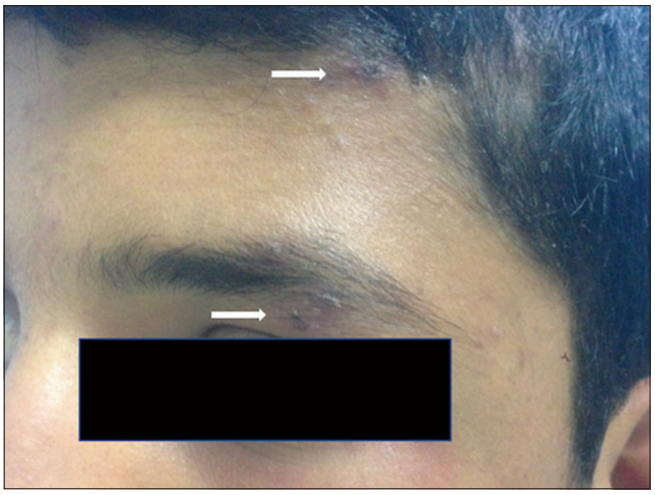

Figure 1: Skin lesions on left side of forehead and upper eyelid

The right eye was normal. Left eye had hand motion vision, intraocular pressure 10 mmHg, a relative afferent pupillary defect, optic disc edema, dilated and tortuous veins, and multiple retinal hemorrhages [Fig. 2a]. Fluorescein angiography revealed extremely sluggish blood flow in the arteries, delayed venous filling suggesting a combined CRAO with CRVO, and frosted branch angiitis in the posterior pole [Fig. 2b]. Optical coherence tomography revealed massive retinal thickening, intraretinal, and subretinal fluid that precluded identification and differentiation of retinal layers [Fig. 2c]. Given his history of VZV dermatitis and rapidly progressing visual loss, intravenous acyclovir was begun. The next day, he spoke of no light perception in the left eye. A VZV optic perineuritis was suspected on MRI [Fig. 2d]. Intravenous methylprednisolone (for 3 days) was started, followed by oral prednisolone. Complete blood count, erythrocyte sedimentation rate, lipid profile, serum homocysteine, protein C, protein S, HIV, tuberculin skin test, Computed tomography (CT) chest, *Treponema pallidum* hemagglutination test, herpes/toxoplasmosis serology, antinuclear antibody, p-ANCA, and c-ANCA were normal. Cerebrospinal fluid analysis was negative for tuberculosis, HSV, VZV, toxoplasmosis, and Cryptococcus. Intravitreal bevacizumab was injected during follow-up as a mild vitreous hemorrhage was observed, followed by panretinal photocoagulation. Viral Polymerase chain reaction (PCR) (HSV, VZV, and CMV) of aqueous tap was negative. Oral corticosteroids were tapered and stopped after 3 months. At 6 months, oral acyclovir was continued in the maintenance dose, the right eye remained uninvolved, and the left eye vision remained dark with no light perception.

Cite this article as: Bansal R, Singh R, Takkar A, Lal V. Combined central retinal artery and vein occlusion with optic perineuritis following herpes zoster dermatitis in an immunocompetent child. Indian J Ophthalmol 2017;65:1233-5.

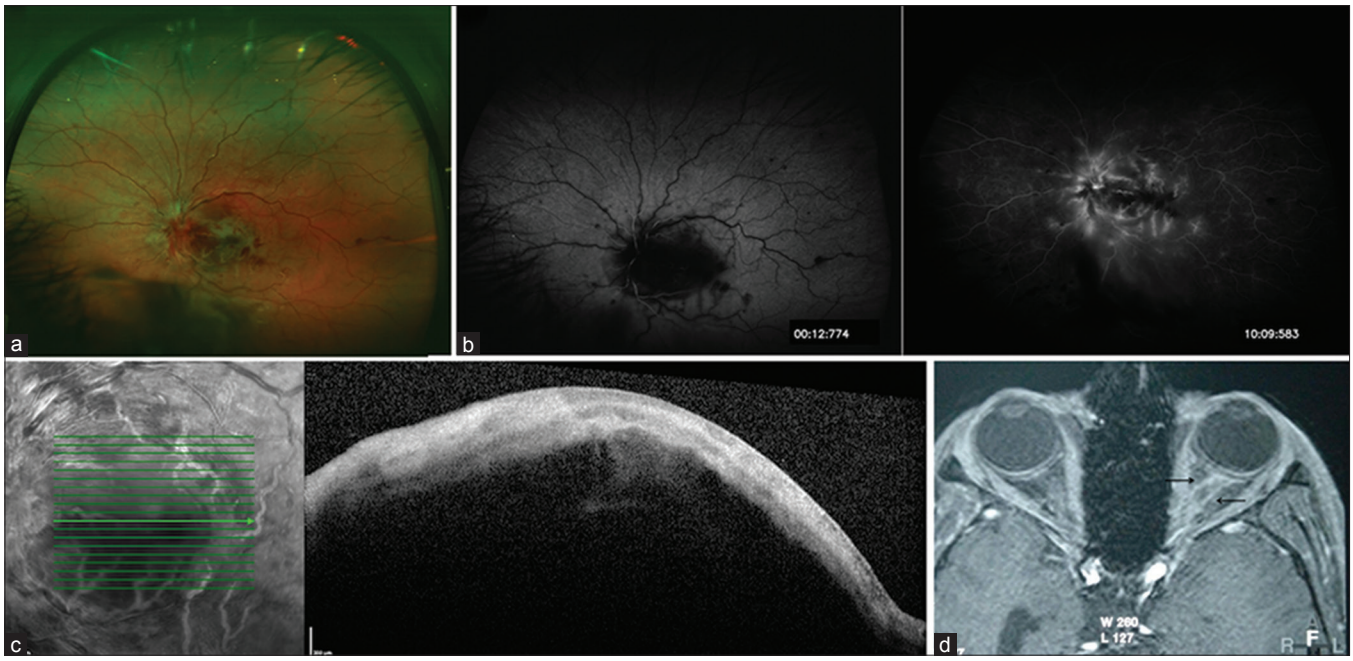

Figure 2: (a) Ultra-widefield fundus photograph of left eye illustrating extensive retinal hemorrhages over the posterior pole and optic disc, with retinal edema, and frosted branch angiitis in the posterior pole (b) Fundus fluorescein angiography showing blocked fluorescence due to dense hemorrhages in posterior pole, extremely sluggish blood flow in the arteries in early phase (left), delayed venous filling, retinal vascular leak from frosted branch angiitis in the posterior pole in the late phase (right), suggesting a combined central retinal vein occlusion and central retinal artery occlusion (c) Optical coherence tomography showing massive retinal thickening, intraretinal and subretinal fluid precluding identification and differentiation of retinal layers (d) Magnetic resonance imaging orbit showing left optic perineuritis (arrows)

Discussion

Central retinal vascular occlusion due to herpes zoster has been reported in association with ARN, either presenting along with ARN or, rarely, preceding ARN as the initial presenting sign.[4-7] A combined CRAO with CRVO has also been reported to be associated with ARN in the same eye,[6] or herald ARN in the fellow eye.[4] Certain risk factors have been identified in these patients such as ARN, elderly patient, preexisting cardiovascular disease, and AIDS.[4-7] Our patient had none of these systemic/ocular risk factors. In the absence of any intraocular signs of inflammation (such as vitritis, retinitis, or vasculitis), negative PCR from vitreous fluid, atypical presentation, and a negative laboratory workup, this case posed a significant diagnostic and therapeutic challenge. Although there was no definite evidence of CRVO, the possibility was considered due to the presence of extensive retinal hemorrhages and macular edema with dilated and tortuous veins. The clinical presentation of VZV dermatitis provided the only diagnostic clue in this case in the absence of definitive tests.

Optic neuropathy and/or central retinal vascular (arterial or venous) occlusion secondary to herpes zoster infection is often associated with profound visual loss leading to no light perception.[4-7] However, given the potential for an impending ARN in these challenging cases,[4] prompt antiviral, and anti-inflammatory therapy is warranted.

Failure of prompt antiviral therapy to restore vision, as was seen in our case, may be attributed to herpes zoster optic neuritis at the initial onset.[5,8] All tissues within the retrobulbar part of the optic nerve (meninges, pial septae, and central retinal vessels) have been shown to be affected with granulomatous inflammation (suggestive of vasculitis) in enucleated eyes with herpes zoster ophthalmicus.[9] This could explain the extensive inflammation around the optic nerve in our patient as seen on MRI. Ischemic or compressive optic neuropathy has also been implicated in causing severe visual loss in herpes zoster ophthalmicus. Association of herpes zoster optic neuropathy with CRAO corroborates ischemic mechanism, leading to severe visual loss.[10]

Conclusion

Our case was quite unusual with a severe, combined CRAO with CRVO, and sudden loss of vision following herpes zoster dermatitis in the absence of any intraocular inflammatory signs in a healthy immunocompetent individual. Although vision could not be restored in the affected eye, it is likely that a prompt and an intensive antiviral therapy could have prevented the onset of ARN in the affected/fellow eye, thereby avoiding its sequelae.

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.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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