Case Report

Inflammatory choroidal neovascularization after tubercular posterior scleritis

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Abstract: A 45-year-old female presented with typical features of posterior scleritis in her left eye with visual acuity of 20/252. After treatment with oral steroids and immunosuppressive drugs, at 2 months follow-up, posterior scleritis resolved and visual acuity improved to 20/50. Five months later she presented with vision loss (20/160) associated with active choroidal neovascular membrane (CNVM) close to scar. Significant choroidal thinning (subfoveal choroidal thickness =137 microns), compared to fellow eye (subfoveal choroidal thickness =247 microns) was noted. Two doses of intravitreal bevacizumab (IVB) were given at 1 month interval. At 9 months follow-up, her visual acuity was maintained at 20/160 with scarred CNVM. In conclusion, IVB is safe and efficacious in treatment of inflammatory CNVM secondary to posterior scleritis. Choroidal changes after posterior scleritis could be contributory factor for formation of CNVM.

Keywords: Posterior scleritis; choroidal neovascular membrane (CNVM); choroidal thickness; choroid

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Introduction

Posterior scleritis is a painful and under-recognized form of scleral inflammation caused by infectious agents or non-infectious immune reactions (1). The disease may be associated with anterior scleritis and may spread to muscle and other orbital tissue or the choroid and retina (2,3). Although choroidal neovascular membrane (CNVM) in inflammatory posterior pathology is quite well-known and published widely (4,5), however, the same in posterior scleritis is under reported in literature.

We herein report a case on the occurrence of inflammatory CNVM in a patient who recovered from posterior scleritis and was treated successfully with anti-vascular endothelial growth factor (anti-VEGF) therapy.

Case presentation

A 45-year-old healthy female presented with complaints of pain and reduced vision in her right eye for last 5 months duration. On examination, best corrected visual acuity (BCVA) was 20/252 and 20/25 in the right and left eye respectively. Anterior segment examination was normal in both eyes. On fundoscopy, right eye showed disc hyperemia with blurred margins with sub retinal fluid at the posterior pole and left eye was within normal limits. Ultrasonography, fluroscein angiography (FA) and spectral domain optical coherence tomography (SD-OCT) findings confirmed the diagnosis of posterior scleritis in right eye (*Figure 1*).

Detailed systemic work-up was done which showed strongly positive Mantoux test (24×20 mm^2 after 5 IU of purified protein derivative). Her systemic examination for tuberculosis was negative. In view of presumed tuberclurosis as cause of posterior scleritis, antitubercular therapy (ATT) was initiated along with tapering doses of oral steroid with addition of azathioprine at 1 month follow-up. At 2 months follow-up, visual acuity in her right eye improved to 20/80. Fundus examination of the right eye showed RPE changes at macula with no sub retinal fluid which was confirmed

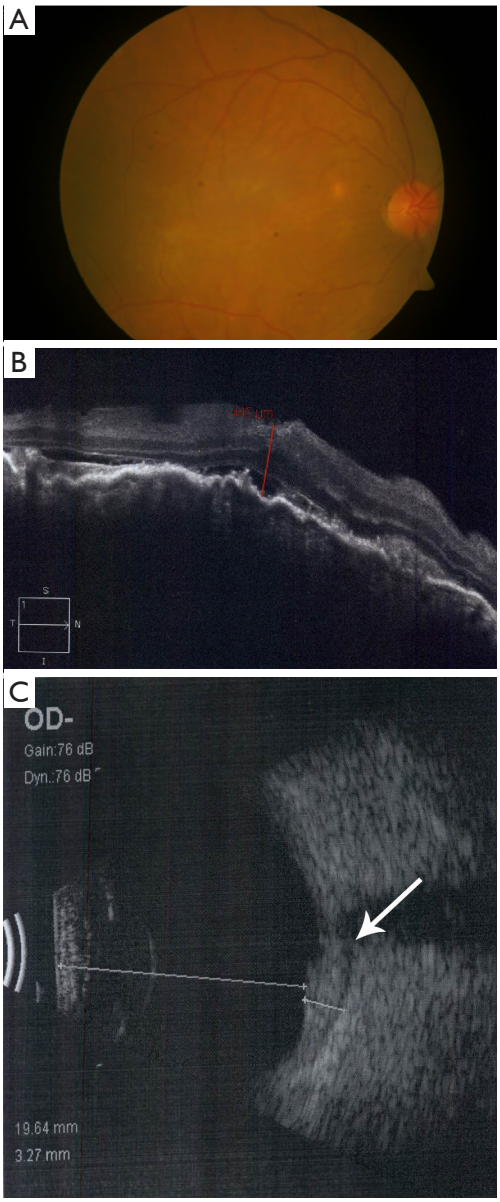

Figure 1 (A) At baseline color fundus photograph shows hyperemic disc with retinal edema; (B) spectral domain optical coherence tomography (SD-OCT) shows choroidal elevation with choroidal folds along with subretinal fluid; (C) ultrasonography shows choroidal thickening with presence of fluid (arrow) (T sign) suggestive of posterior scleritis.

with SD-OCT. She was on a regular follow-up with stable visual acuity.

Six months later she came with further vision loss in the same eye. On examination, visual acuity in her right eye was 20/160. Right eye fundus showed fresh sub retinal haemorrhages at the macula close to previously involved area, suggestive of an active CNVM (*Figure 2A*). SD-OCT, FA and indocyanine green angiography showed signs of active choroidal neovascular membrane (*Figure 2B,C,D*). After informed consent, patient underwent two injections of intravitreal bevacizumab (IVB) (1.25 mg in 0.05 mL) at 1 month interval. One month after second injection, there was no activity on SD-OCT; therefore, she was asked to continue ATT as per the physician's advice. Patient was under regular follow-up. At 9 months follow-up after last IVB, her BCVA was maintained at 20/160 and fundus examination showed scarred CNVM which was confirmed on SD-OCT (*Figure 2E*), along with RPE atrophy due to recovered posterior scleritis.

Discussion

Choroidal neovascular membrane formation although uncommon is a well-known sight-threatening complication of posterior segment intraocular inflammation. The development of CNVM results either directly from an inflammatory-mediated angiogenic drive and/or secondary to a degenerative disruption in the Bruch's membrane-retinal pigment epithelium (RPE) complex. VEGF is a key inducer of neovascularization (6).

Uchihori *et al.* reported that high-penetration optical coherence tomography (HP-OCT) measurements performed in two patients with acute posterior scleritis showed thickened choroids. After treatment, the choroidal thickness decreased (7). Similarly in our patient the choroidal thickness was found to be reduced to 137 microns after the resolution of posterior scleritis compared to 247 microns in the fellow eye. Involvement of choroid in posterior scleritis is also supported by studies conducted by Auer and Herbort (8), who reported that that posterior scleritis induces major alterations in the adjacent choroid, confirmed on indocyanine green angiography. Initial enhanced depth imaging optical coherence tomography (EDI-OCT) in our case showed minimal changes in the choriocapillaries along with increased choroidal thickness, but as the posterior scleritis resolved it was seen that in addition to the reduced choroidal thickness there was loss of medium sized choroidal vessels and choriocapillaries, sparing only larger choroidal vessels (*Figure 2D,E*). From this observation, we speculate that inflammation of the posterior sclera induces alterations in the adjacent choroid, gradually leading to choroidal atrophy and changes in the integrity of Bruch's membrane finally favouring the

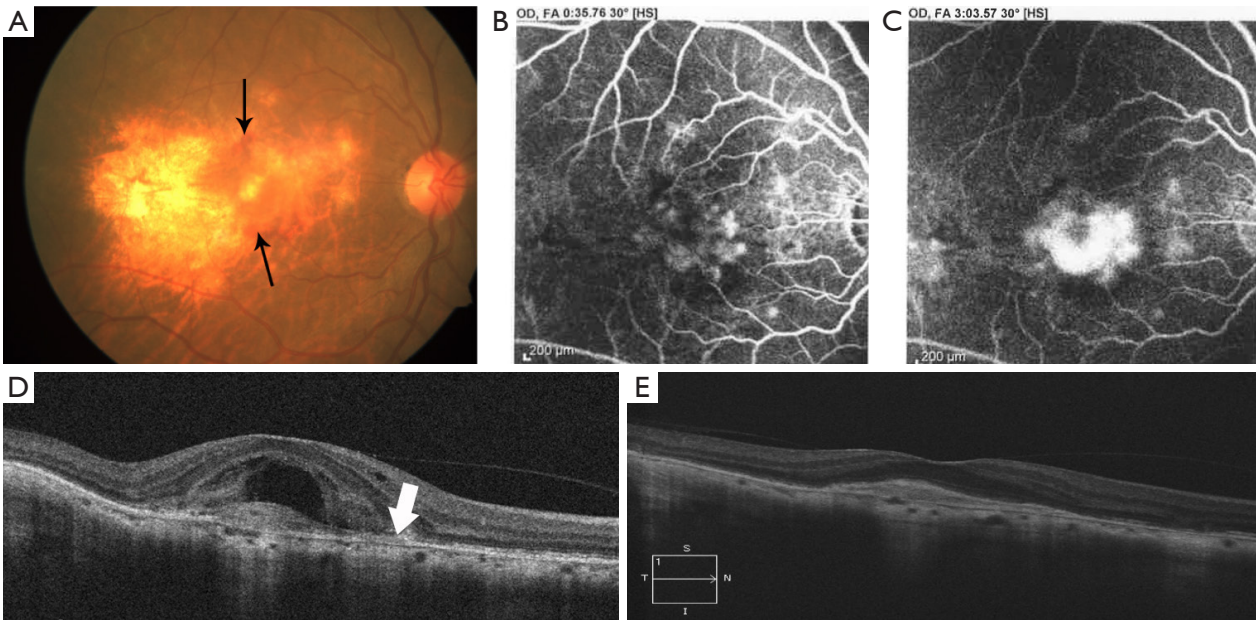

Figure 2 (A) Fundus photograph shows scarring at the macula secondary to recovered posterior scleritis along with fresh subretinal haem (black arrow); (B,C) fundus fluorescein angiography is suggestive of active choroidal neovascularization; (D) spectral domain optical coherence tomography (SD-OCT) showed presence of subretinal and intraretinal fluid along with choroidal neovascular membrane. Choroidal thinning along with loss of medium sized choroidal vessels with sparing of large vessels (white arrow) also noted; (E) at 9 months follow-up, SD-OCT scan shows no fluid with subfoveal scar.

development of a neovascular membrane.

Currently, various options are available for managing inflammatory CNVM including laser photocoagulation, local and systemic corticosteroids, and surgical removal; all with potential limitations (9,10). To date, clinical trials evaluating the treatment options for inflammatory CNVM associated with uveitis have been limited and mostly non-comparative. Successful management involves both control of intraocular inflammation and occlusion of the inflammatory neovascular membrane. Median number of anti-VEGF injections required in treatment of inflammatory CNVM has been reported to be to 1.73 (range, 1-5) (5). Similarly in our patient, two IVB injections led to CNVM regression as well as improvement and stabilization of vision with no recurence till 9 months follow-up.

Although, significant advances have recently occurred in the understanding of choroidal changes in posterior scleritis, further understanding about the vascular changes in choroid is still warranted. These significant choroidal vascular changes in various inflammatory conditions could be targeted for therapy and could be used to predict the occurrence of CNVM in future.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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