Retinal neovascularization in a patient with chronic central serous chorioretinopathy

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A 45-year-old male presented with diminution of vision in both eyes since the last 2 years. The best-corrected visual acuity was 20/200 in his right eye and 20/600 in left eye. BE fundi had changes of chronic CSCR with PED and NSD in the RE and subretinal fibrosis in the left eye. Both eyes had peripheral pigmentary changes. Multimodal imaging showed peripheral avascular retina in both eyes with neovascularization at disc in the right eye which promptly resolved with a single injection of anti-VEGF. Retinal neovascularization is an unusual finding in the setting of CSCR and has not been reported in the literature.

Key words: CSCR, NVD, pigmentary changes

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Central serous chorioretinopathy (CSCR) is characterized by serous macular detachment (SMD) and/or serous retinal pigment epithelium (RPE) detachment.[1] The exact etiopathogenesis of CSCR still remains elusive. Broadly, it has two varieties, acute and chronic. While acute CSCR is simple to diagnose and self-limiting, chronic CSCR can be a great masquerader with presentations mimicking other overlapping pathologies. Diffuse retinal pigment epitheliopathy, exudative retinal detachment, choroidal neovascularization, and polypoidal choroidal vasculopathy are few of its varied presentations.[2]

We hereby report the occurrence of retinal neovascularization in a patient with chronic CSCR. To the best of our knowledge, this is the first report describing such a complication.

Case Report

A 45-year-old male presented with diminution of vision in both eyes (BE) since the last 2 years. He was a night shift worker, nonsmoker and nonalcoholic, with no history of steroid intake. There were no systemic complaints. The best-corrected visual acuity (BCVA) was 20/200 in his right eye (RE) and 20/600 in left eye (LE). Anterior segment examination was nonremarkable in BE. Dilated fundus examination of the RE revealed pigmentary changes at fovea and a large serous RPE detachment (RPED) along superotemporal arcade. Right eye optic disc showed vascular proliferation at the inferotemporal margin [arrow, Fig. 1a]. Left eye on similar lines showed pigmentary changes

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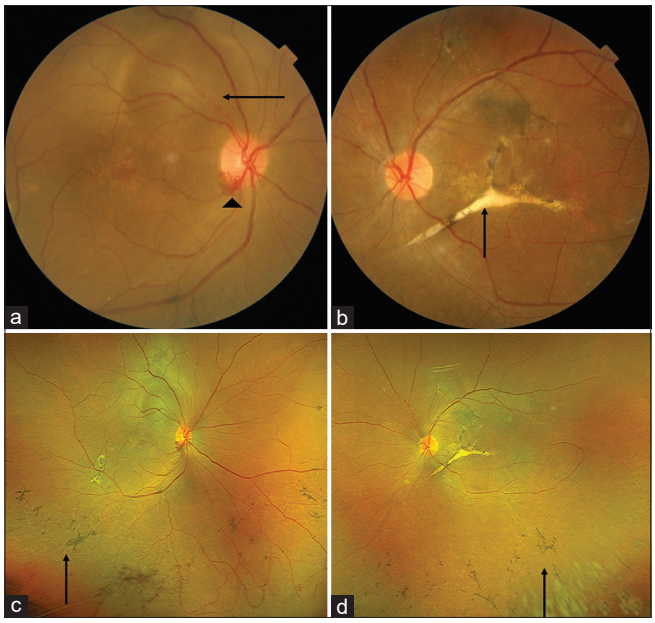

Figure 1: (a) Fundus image of right eye showing PED (arrow) and neovascularization at disc (arrow head) with pigmentary changes at fovea. (b) Fundus image of LE showing large area of chronic subretinal fibrosis. (c) Ultra widefield image of OD showing inferior old vitreous hemorrhage with peripheral pigmentary changes in the shape of bony spicules. (d) Ultra widefield image of OS showing inferior old vitreous hemorrhage with peripheral pigmentary changes in the shape of bony spicules

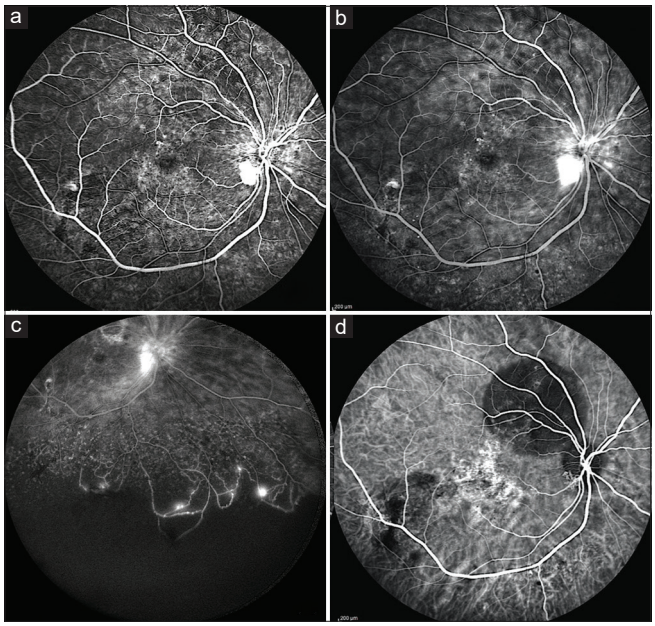

Figure 3: (a) FFA of right eye showing window defects at posterior pole with hyperfluoresent lesion at disc, suggesting NVD. (b) Late phase of FFA confirming NVD with extensive leakage. (c) Peripheral fundus shows avascular retina (CNP areas) on FFA with adjacent areas of fine NVDs. (d) ICG angiography of right eye showing hyper permeable choroid with an area of hypo fluorescence superotemporal to the disc suggesting PED

at the macula along with a linear area of subretinal fibrosis extending inferonasally from the center of fovea [Fig. 1b]. Bony

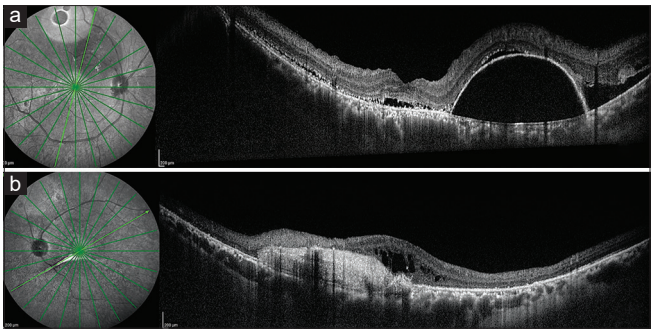

Figure 2: (a) SD-OCT of Right eye showing subfoveal NSD with fibrin deposition at outer retinal layers and large serous PED along superotemporal arcade. Pachychoroid was evident. (b) SD-OCT of Left eye showing fusiform subretinal thickening with intraretinal fluid. Pachychoroid was evident

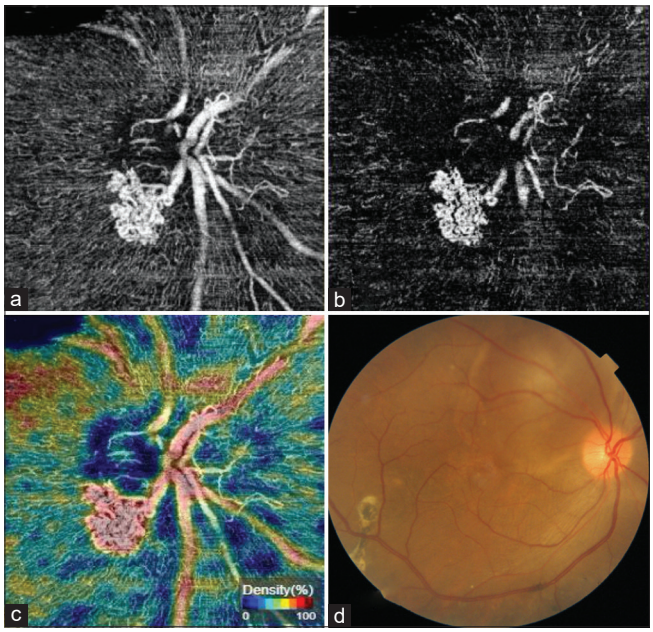

Figure 4: (a-c) OCT angiography confirms the presence of NVD. (d) Postoperative fundus photo showing immediate regression of NVD post anti-VEGF injection

spicules (pigmentary changes) were seen in the inferior retina in BE [Fig. 1c and d].

Enhanced depth imaging on spectral domain optical coherence tomography (SD-OCT) noted chronic SMD and large serous RPED in the RE [Fig. 2a]. The LE OCT revealed hyper-reflective fusiform thickening corresponding to subretinal fibrosis above RPE along with few intraretinal cystoid spaces [Fig. 2b]. Pachychoroid was evident in BE.

Fundus fluorescein angiography (FFA) showed window defects at the macula and intense hyperfluorescence from retinal neovascularization in RE [Fig. 3a and b]. In addition, inferior retina of RE showed large area of capillary nonperfusion and adjoining retinal neovascularization [Fig. 3c].

Indocyanine green (ICG) angiography showed late choroidal hyperpermeability along with dilated choroidal vessels and an area of hypofluorescence corresponding to

RPED in the RE [Fig. 3d]. Choroidal hyperpermeability was evident in LE as well. Optical coherence tomography angiography confirmed neovascularization at the edge of the optic disc in RE [Fig. 4]. A diagnosis of chronic CSC with resolved inferior exudative retinal detachment in BE and resolved submacular fibrin in LE was made. The patient underwent intravitreal injection of Bevacizumab in the RE which resulted in prompt resolution of neovascularization [Fig. 4d].

Discussion

Chronic CSCR or Bullous CSCR can be misdiagnosed as rhegmatogenous retinal detachment, choroidal inflammation, or malignancies. Recurrent neurosensory detachments are seen in nearly 1/3 cases of CSCR. These detachments persist for more than 6 months and are mainly inferior in location owing to gravitational spread of subretinal fluid. Pathogenesis of such detachment is attributed to diffuse retinal pigment epitheliopathy, pachychoroid, and scleral thickening leading to decreased transcleral outflow.[3]

Both hypermeability of choroid and dysfunctional retinal pigment epithelium together result in longstanding neurosensory detachments. It has been shown earlier that chronic retinal detachments may lead to ischemia of inner retinal layers and degeneration of neurons due to thickening of basal lamina of retinal vessels and their subsequent occlusion.[4] This ischemia, in turn, upregulates VEGF causing neovascularization of retina or optic nerve. It is our contention that longstanding neurosensory detachment and nonperfusion of the peripheral retina led to similar retinal neovascularization in our case. In a recent report, it has been reported that long-term neurosensory detachment may lead to cellular death which may, in turn, lead to release of inflammatory mediators and peripheral vasculitis like picture and thereby leading to peripheral vascular occlusion and avascular areas.[5] Management of such neovascularization poses a challenge, as treatment of choice for their regression is laser photocoagulation. However, in such detachments, eliminating these ischemic areas is not always possible, thus, anti-VEGF may be a viable option as was seen in our case.

Few differentials that we may need to keep in mind while evaluating a case with neovascularization are vessel occlusions, diabetic retinopathy, ocular ischemic syndrome, and vasculitis. In our case, these differentials were effectively ruled out by relevant clinical and imaging features all suggestive of chronic CSCR.

Although retinal neovascularization has been reported earlier in chronic rhegmatogenous retinal detachments, no reports of the same were found in a setting of chronic CSC. Thus, our case highlights another varied presentation of chronic CSC making it further diagnostic conundrum. In addition, such neovascularizations can be managed successfully with anti-VEGF injection, although closer follow-up and eventual laser photocoagulation may be required later.

Conclusion

To conclude, this is the first case reporting the occurrence of retinal neovascularization in chronic CSC.

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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Conflicts of interest

There are no conflicts of interest.

References

1. Wang M, Munch IC, Hasler PW, Prünte C, Larsen M. Central serous chorioretinopathy. Acta Ophthalmol (Copenh) 2008;86:126-45.
2. Hanumunthadu D, Tan AC, Singh SR, Sahu NK, Chhablani J. Management of chronic central serous chorioretinopathy. Indian J Ophthalmol 2018;66:1704-14.
3. Yannuzzi LA, Slakter JS, Kaufman SR, Gupta K. Laser treatment of diffuse retinal pigment epitheliopathy. Eur J Ophthalmol 1992;2:103-14.
4. Piccolino FC. Vascular changes in rhegmatogenous retinal detachment. Ophthalmologica 1983;186:17-24.
5. Madanagopalan VG, Shah K, Nagesha CK, Baskaran P. Peripheral retinal avascularity and capillary leakage in central serous chorioretinopathy. J Curr Ophthalmol 2019;31:220-4.