Case Report

Anomalous Retinal Neovascularisation in chronic Central Serous Chorio Retinopathy

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ABSTRACT

There are varied presentations of chronic Central Serous Chorio Retinopathy (CSCR) which include diffuse retinal pigment epitheliopathy, Exudative retinal detachment, choroidal neovascularization, and polypoidal choroidal vasculopathy. Retinal neovascularization is an exceptionally rare complication associated with it. In this report, we aim to discuss an unusual case of abnormal retinal neovascularization in a patient suffering from chronic CSCR. in both eyes. The CSCR with anomalous NVE was confirmed by Fundus Fluorescein Angiography (FFA) and Optical Coherence Tomography (OCT). The patient underwent Pan Retinal Photocoagulation (PRP) in both eyes. After 2 months, the new vessels started regressing, with a reduction in the sub retinal fluid (SRF)of both eyes. Tailored approaches and parallel treatment of NVE are required to avoid ischemia-related complications and optimal outcomes.

Keywords: Central serous chorioretinopathy, Anomalous retinal neovascularisation, Pan retinal photocoagulation.

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INTRODUCTION

Central serous chorioretinopathy (CSCR) is defined by the presence of serous detachment in the macula and the retinal pigment epithelium.[1] The precise causes of CSCR remain uncertain, but both stress and the use of corticosteroids are important risk factors. Factors such as autoimmune disorders, cardiovascular diseases, hypertension, and certain medications may also contribute to the development of CSCR. It is classified into acute and chronic forms. While acute CSCR typically resolves on its own, chronic CSCR can present in a way that mimics other overlapping conditions. Examples of these varied presentations include diffuse retinal pigment epitheliopathy, exudative retinal detachment, choroidal neovascularization, and polypoidal choroidal vasculopathy.[2] Retinal neovascularization is an exceptionally rare complication associated with CSCR. In this report, we aim to discuss an unusual case of abnormal retinal neovascularization in a patient suffering from chronic CSCR.

Case Report

We present the case of a 58-year-old woman with no significant systemic health issues who experienced a gradual decline in vision, particularly in her left eye, over the past year. On examination, her best-corrected visual acuity (BCVA) was 6/9 in the right eye and 6/60 in the left eye. The anterior segment examination was unremarkable in both eyes. Fundus examination of the right eye revealed a normal optic disc with neovascularization elsewhere (NVE) observed superior and temporal to the disc (Figure 1a, c). The left eye also had a normal optic disc, with NVE along the inferior arcade (Figure 1b). The foveal reflex was dull, with retinal pigment epithelium (RPE) alterations

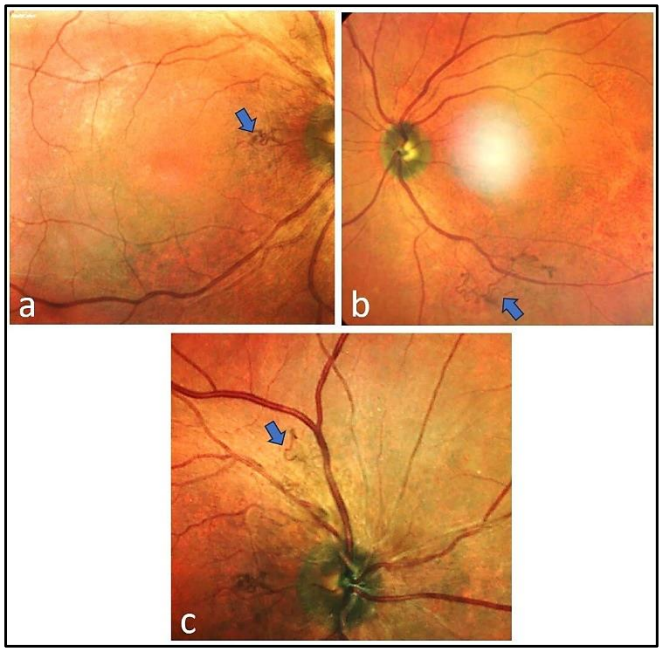

Figure 1: A and C show fundus photo of right eye with blue arrow showing retinal neo vascularisation. b shows fundus photo of left eye with blue arrow showing retinal neo vascularisation.

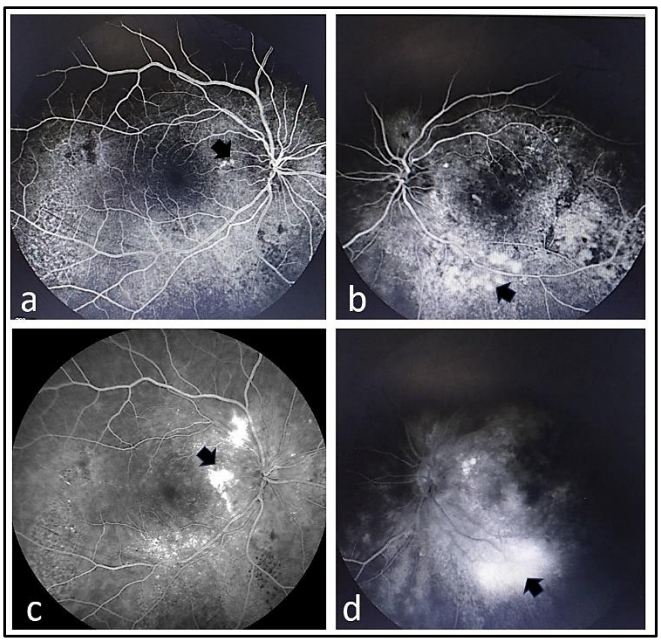

Figure 3:A and B show early phase FFA image of OD and OS with black arrow showing leakage due to retinal neo vascularisation. C and D show OD and OS late phase FFA image with increase in leakage due to retinal neo vascularisation.

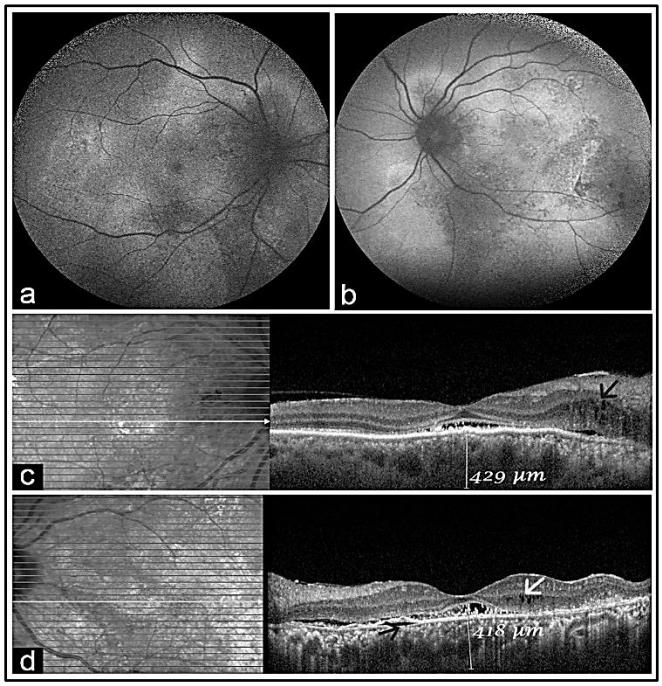

Figure 2: A and B show AF image of OD and OS with hyper auto fluorescence due to RPE alterations. C shows OD OCT macular cube 30° with horizontal line scan showing sub foveal neuro sensory detachment. OD Sub foveal choroidal thickness was 429 microns. Nasal to fovea intra retinal fluid seen near the disc (black arrow). D shows OS OCT macular cube 30° with horizontal line scan shows sub foveal neuro sensory detachment and hyper reflective dots. OS Sub foveal choroidal thickness was 418 microns. OS Temporal to fovea few intra retinal fluid spaces (white arrow) seen and nasal to fovea shallow pigment epithelial detachment seen (black arrow).

in both OCT showed shallow SRF, pigment epithelial detachment (PED) and pachychoroid in both eyes (Figure 2 C, D). Fundus FA showed hyper-fluorescence due to leakage of NVE with no capillary non perfusion (CNP) areas (Figure 3 a-d). A diagnosis of chronic CSCR with anomalous retinal neo-vascularization was made. Both eyes were treated with two sittings of Pan-retinal photocoagulation (PRP). Two months post laser, the anomalous NVE were regressing, with the reduction in the SRF of both eyes over a period of 1 year.

DISCUSSION

The suggested process for neovascularization involves chronic subretinal fluid resulting in an increase in the thickness of the basal lamina of retinal blood vessels, which in turn causes ischemia in the inner retina and the release of VEGF.[3,4] Increased levels of VEGF lead to the formation of new blood vessels in retina or around the optic disc. In our situation, as there were no areas of capillary nonperfusion detected on FFA, we cannot link retinal ischemia as the cause of NVE. Moreover, the patient was not willing for intra vitreal anti VEGF injection, as it is an invasive procedure, we performed two sittings of PRP in both eyes. The new vessels started regressing after laser treatment. Further

follow-ups will be required to assess the efficacy of treatment and determine the need for other treatment modalities in case of recurrence.

We also considered the possibility of co-existence of diabetic retinopathy and CSCR due to the presence of long-standing SRF and neovascularization.[5] However, there was no history of diabetes and no other fundus findings suggestive of diabetic retinopathy was noted. Bullous CSCR with neovascularization was also considered a differential, due to the presence of SRF at the posterior pole and minimally in the inferior periphery as well. Neovascularization due to peripheral ischaemia in long-standing bullous CSCR has been reported.[6] But in our case, there was no shifting fluid or turbidity of SRF to support this.

Long-term neurosensory detachment has been indicated to potentially result in cell death. This cellular death might trigger release of inflammatory mediators, which could contribute to the development of a condition resembling peripheral vasculitis. This inflammatory response could potentially lead to peripheral vascular occlusion and the formation of avascular areas.[7] Alternative possible differential diagnoses, such as ocular ischemic syndrome and vascular occlusive conditions, were thoroughly excluded through clinical assessment and imaging modalities.

CONCLUSION

In summary, without conclusive evidence, it is not possible to clearly attribute anomalous NVE as being either related or unrelated to CSCR. More studies would be required to establish any potential causal link or to rule out such a relationship definitively.

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Conflict of Interest: Authors declared no conflict of interest.

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