



Idiopathic lenticular surface neovascularization: An unusual presentation

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ABSTRACT

Purpose: To present a case of posterior lenticular surface neovascularization in the absence of any ocular or systemic pathology.

Observations: A 29-year-old asymptomatic male was detected with the right eye (OD) posterior lenticular surface neovascularization extending for six-clock hours in the temporal portion along with the presence of mild posterior capsular haze. His best-corrected visual acuity was 20/25 in OD and 20/20 in the left eye (OS). No additional abnormality was identified in the remainder of the OD anterior segment and the fundus respectively. OS evaluation was unremarkable. OD imaging including B-scan ultrasonography and ultrasound biomicroscopy (UBM) were essentially normal. The anterior segment optical coherence tomography (AS-OCT) thickened lens capsule with a dense hyperreflective layer adhered to its posterior surface and separating from the capsule in the periphery. Few hyperreflective dots were visible posterior to the ciliary body although no CB thickening was noted. No systemic abnormality was detected. The patient is being managed conservatively and has shown no signs of progression of the neovascularization over six months.

Conclusions and importance: This is the first reported case of an isolated idiopathic posterior lenticular surface neovascularization occurring in an otherwise healthy patient. Although an unspecified breach in the posterior capsule or undetermined cyclitis can trigger such neovascularization, further histopathological studies of the capsular biopsy and ciliary body can provide better insight into its etiopathogenesis. Additionally, considering the asymptomatic nature of the condition, these patients can be observed and closely monitored.

1. Introduction

Abnormal lens capsular neovascularization has been described in eyes with proliferative diabetic retinopathy or retinal detachment and is often associated with iris/angle neovascularization.^{1–3} We hereby report a unique case that presented with lenticular surface neovascularization in the absence of any anterior segment neovascularization or posterior segment pathology.

2. Case report

A 29-year-old male patient visited our hospital for a routine ocular examination. His past ocular and systemic history were unremarkable, and he gave no history of trauma or surgical procedure. On examination, his best-corrected visual acuity (BCVA) in the right eye (OD) was 20/25 and left eye (OS) was 20/20. Slit-lamp evaluation of the OD anterior segment after full dilatation showed posterior capsular haze and surface vascularization of the temporal half of the posterior capsule extending

from 6 o'clock to 12 o'clock position (Fig. 1A and B). There was an absence of any cells or flare in the anterior chamber or the anterior vitreous. The intraocular pressure (IOP) was 12 mm Hg in both eyes by Goldmann applanation tonometry. Fundus examination of OD by slit-lamp biomicroscopy and by indirect ophthalmoscopy was normal and revealed no abnormal vessels. Anterior and posterior examination of the left eye was normal. Gonioscopic evaluation of anterior chamber angles was unremarkable in both eyes.

Ultrasound B-Scan of OD was normal except for few medium reflective dot echoes in the vitreous cavity (Fig. 2). The axial length (AL) was 22.17 mm in OD. OD Ultrasound biomicroscopy (UBM) was unremarkable (Fig. 3). Anterior segment optical coherence tomography (AS-OCT) revealed a thickened intact posterior capsule that was adhered to another thickened membrane posterior to it, which most likely represented the neovascularization layer. These two layers were coupled together centrally and separated from each other in the peripheral portion. Additionally, the AS-OCT also demonstrated few reflective signals (yellow arrow) behind the ciliary body in the quadrant having

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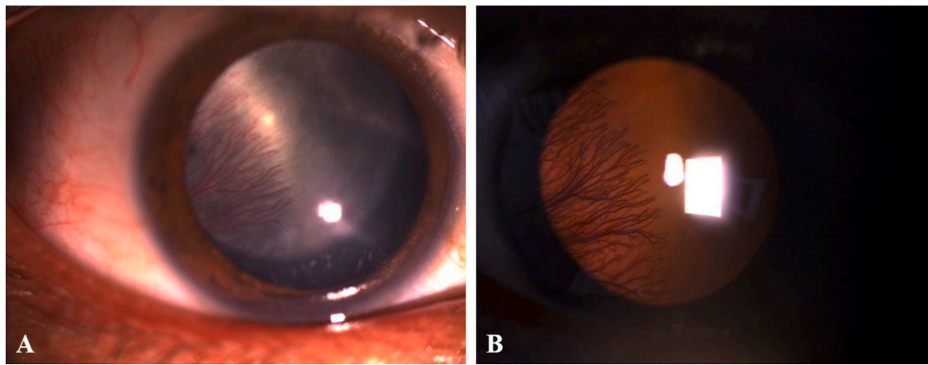


Fig. 1. Slit-lamp colour photography of the anterior segment of the right eye in diffuse illumination (A) showing the showing posterior lens capsule haze with vascularization. The vascularization was better appreciated on retroillumination (B). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

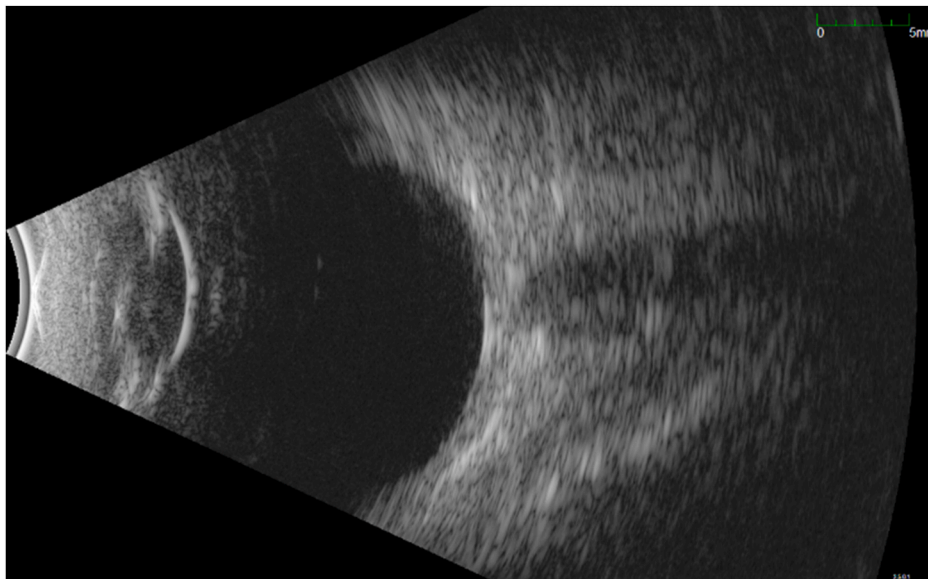


Fig. 2. Posterior Ultrasound B scan of the right eye showing medium reflective dot echoes in the vitreous cavity and a normal retinochoroidal complex and the optic nerve head.

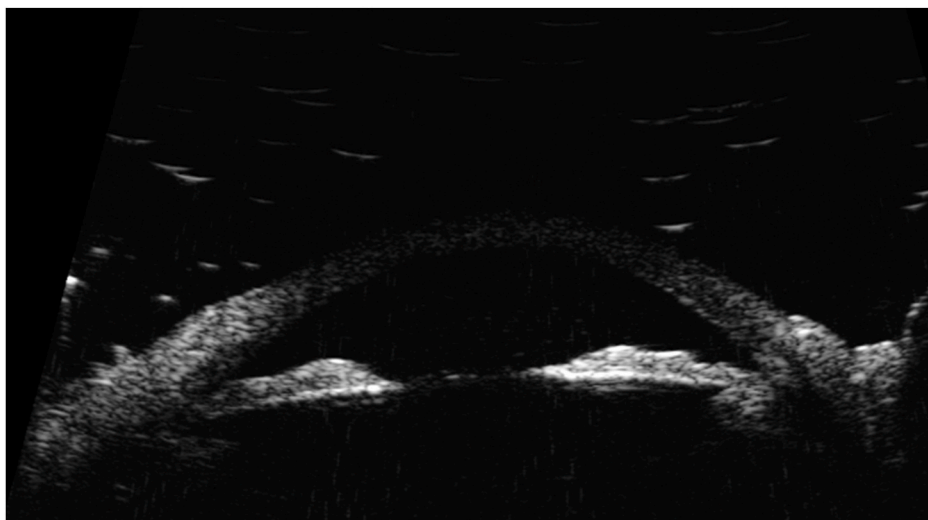


Fig. 3. Ultrasound biomicroscopy (UBM) of the right eye demonstrating a normal ciliary body.

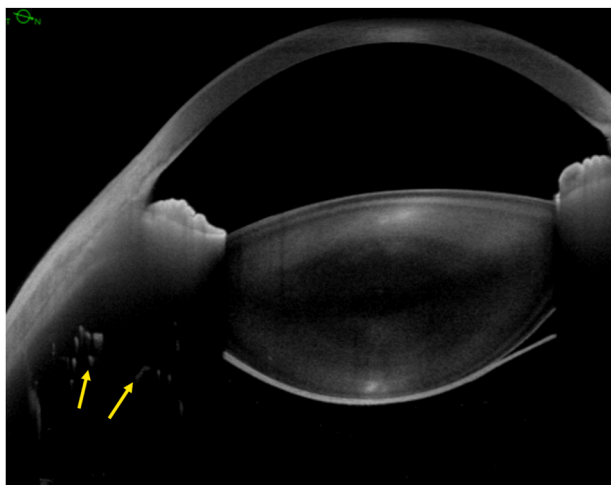


Fig. 4. Anterior segment optical coherence tomography (AS-OCT) of the right eye revealing an intact thickened posterior capsule with a dense hyperreflective membrane attached to it posteriorly which most probably represents the neovascular tissue complex. Separation of these two layers is visible in the periphery. Additionally, few hyperreflective dots are also visible behind the ciliary body region (yellow arrows). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

the vascularization (Fig. 4). An allergic reaction to a test dose of sodium fluorescein precluded us from performing fundus fluorescein angiography for the patient. Routine blood investigations, including ANA titres, Tuberculin skin test, and the carotid doppler ultrasonography were within normal limits. Physician evaluation did not reveal any cardiovascular pathology. Considering the excellent visual acuity and asymptomatic nature of the neovascularization, a posterior lens capsule biopsy was deferred. The patient is under close observation and continues to be monitored with a well-maintained BCVA, IOP, and unchanged nature of the posterior lenticular surface vascularization at 6-month follow-up.

3. Discussion

We hereby describe a case that presented with abnormal posterior lenticular surface neovascularization as an isolated finding. Though there are three reports of lens capsular neovascularization published in the literature, our case is unique because there was no evidence of any anterior segment neovascularization or posterior segment pathology. Gupta et al. reported capsular neovascularization in a blind eye with closed funnel retinal detachment.⁴ In another case Gupta et al. reported a case of capsular proliferation in a young patient with UBM proven localized ciliary body thickening and inflammation.⁵ They hypothesized that ciliary body inflammation probably led to a breach of the lens capsule thus aiding sequestration of the inflamed mediators into the lens and precipitating neovascularization. Walkden et al.⁶ reported anterior lens neovascularization in a 53-year-old female with a history of nanophthalmos, peripheral iridotomy (PI), and argon laser iridoplasty with good vision. The crowded anterior chamber, the previous laser iridoplasty treatment-induced lens capsule breach, or laser-induced segmental inflammation and/or ischemia of the iris segment could be the trigger for neovascularization in that case.

Our case had no known antecedent trauma or inflammation nor did he have an inflammatory or established vascular pathology. Though fibro-proliferative scaffold behind the iris was ruled out on UBM, reflective signals correlating to possible neovascular elements or inflammatory cells were seen posterior to the ciliary body on AS-OCT. We believe that an unexplained microbreach of the capsule could have triggered neovascularization which could have extended posteriorly. It

is known that the lens capsule secretes anti-endothelial cell inhibitory factors inhibiting neovascularization even in the presence of concurrent iris neovascularization.⁷ In the event of a breach, however the heparin-sulphate proteoglycans that are expressed by the lens capsule act as a potential reservoir of vascular endothelial growth factor (VEGF)-188 isoform to permit proliferation of new vessels.⁸ Also regression of such abnormal neovascularization has been reported after anti-VEGF therapy.⁹ Another possible hypothesis could be the presence of chronic low-grade cyclitis which can trigger sequestration of inflammatory material in the Berger's space. Such benevolent long-standing inflammation may incite the development of inflammatory membrane with secondary neovascularization. This theory is loosely supported by the presence of suspicious hyperreflective signals on AS-OCT, which may represent inflammatory cells and the thickened membrane associated with the posterior capsule that can correspond to the inflammatory neovascular tissue complex.

Persistent fetal vascular (PFV) is an important differential for our case. Although we found no evidence of posterior PFV,¹⁰ such as Bergmeister Papilla, retinal folds, or a hyaloid stalk, the presence of an isolated anterior PFV was possible. However, no iridohyaloid blood vessels, anterior chamber shallowing, persistent pupillary membrane, Mitten-dorf Dot, cataract, or the classic retrolental membrane described with anterior PFV were observed.¹⁰ Furthermore, the AL was normal and the neovascular complex lacked any discernible fibrous element, as evidenced by the retroillumination image (Fig. 1B), making anterior PHV very unlikely. Besides which, in the presence of PFV-related retrolental vascularization, we can invariably expect meaningful visual decline, which was not observed in our case.

Performing a fundus fluorescein angiography would have provided vital information regarding the vascular status of the peripheral retina. Any retinal vascular pathology can trigger raised VEGF levels in the vitreous cavity that can potentially incite posterior capsular vascularization. Nonetheless, a thorough clinical evaluation at baseline and up to 6 months of follow-up did not reveal any such posterior segment vascular pathology. A posterior lens capsular biopsy would give us better insight into the histopathological aspect of this novel finding. This may also provide us a clue into the etiology of neovascularization. However, on account of the asymptomatic and non-progressive nature of the condition and with the patient maintaining excellent visual acuity, performing such an invasive procedure was withheld. Nevertheless, the patient is under closed monitoring and will undergo appropriate and timely surgical procedures if the neovascularization shows any signs of progression or is complicated by intraocular hemorrhage or cataract development.

4. Conclusion

In conclusion, posterior lenticular surface neovascularization can occur in the absence of any associated ocular or systemic pathology, which has not been reported earlier. Regular follow-up and closed monitoring are essential for these patients to foresee the development of any complications. Histological studies of the tissue, including the posterior lens capsule and the ciliary body, are needed to shed more light on its etiopathogenesis.

Patient consent

The patient consented to publication of the case in writing.

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Authorship

All authors attest that they meet the current ICMJE criteria for

Authorship.

Declaration of competing interest

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