Brief Communication

A Case of Full-thickness Parafoveal Hole Associated with Chorio-retinal Atrophy

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ABSTRACT

Background: An idiopathic full-thickness parafoveal hole (PFH) in the absence of trauma or intraocular surgery is a rare finding.

Case: A 60-year-female who did not gain good vision following an uneventful phacoemulsification with intraocular lens (IOL) implantation in the right eye (RE) consulted a retina specialist, one year after her cataract surgery. There was no history of trauma, radiation exposure, reduced scotopic vision, or any other intraocular surgery. Her personal and family history were unremarkable for any systemic or ocular diseases. Routine blood investigations, an electrocardiogram, and a detailed ocular examination were done.

Observation: She had the best corrected visual acuity (BCVA) of LogMAR 1.0 (20/200; 6/60) in the right eye. The right eye had an axial length (AL) of 23.50 mm and an intraocular lens power of 21.0 dioptres. The ultrawide field fundus examination saw parafoveal chorio-retinal atrophy without significant peripheral myopic degeneration. On optical coherence tomography (OCT), a central foveal thickness of 138 microns with foveal scarring was noticed. There was a full-thickness parafoveal hole between the fovea and optic disc having a height of 198 microns; base diameter of 240 microns; arm lengths of 203 microns and 206 microns; and a minimum linear dimension of 42 microns. The optical coherence tomography angiography scan showed a reduced vessel density in the superficial and deep retina; and increased visibility of choroidal vessels in outer retina chorio-capillaries, chorio-capillaries, and choroid slab at the parafoveal hole The ultrasound B scan was anechoic and there was no posterior vitreous detachment (PVD).

Conclusion: The axial length, intraocular lens power and fundus examination did not indicate pathological myopia. As there was no preceding posterior vitreous detachment or retinal surgery, the underlying retino-choroidal atrophy most probably caused the full-thickness parafoveal hole.

Key words: Non-foveal macular hole, Optical coherence tomography angiography, Parafoveal hole; Parafoveal macular hole.

Financial Interest : Nil
Conflict of Interest : Nil

Received : 20.11.2022
Accepted : 25.12.2022

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Access this article online
Website: www.nepjol.info/index.php/NEPJOPH
DOI: https://doi.org/10.3126/nepjoph.v15i1.51624

ISSN: 2072-6805, **E-ISSN:** 2091-0320

INTRODUCTION

The early event leading to an idiopathic macular hole (MH) is the persistent adherence of cortical vitreous to the fovea with adjoining vitreoretinal separation. The consequent foveal traction causes foveal detachment and foveal tissue dehiscence, finally resulting in the formation of a full-thickness macular hole (FTMH) (Hussain et al, 2003; Yeh et al, 2010). Even after a complete posterior vitreous detachment (PVD), tangential traction on the fovea from the epiretinal membrane (ERM) can also lead to forming a FTMH (Yeh et al, 2010).

Procedures like an internal limiting membrane (ILM) and ERM peeling may give rise to parafoveal macular holes (PFMH) through Muller cell damage, structural breakdown and retinal weakening (Rush et al, 2014;Yeh et al, 2007; Steven et al, 2006). Other reasons can be traction in a fibrovascular proliferation of diabetic retinopathy (Lai et al, 2015). Remarkably, the paracentral holes have been seen located temporal to the fovea (Hussain et al., 2018; Rush et al., 2014; Yeh et al., 2007;Steven et al., 2006).

We report a case of a parafoveal hole (PFH) located nasal to the fovea in the absence of any vitreoretinal procedure, PVD, trauma, pathological myopia, or any other ocular or systemic disease.

CASE

A 60-year-female presented to the Ophthalmology department with an immature senile cataract in both eyes (BE). She had the best corrected visual acuity (BCVA) of LogMAR +1.2 (20/320; 6/95) in the right eye (RE) and LogMAR +0.6 (20/80; 6/24) in the left eye (LE). She underwent phacoemulsification with intraocular lens (IOL) implantation in BE and cataract surgeries were uneventful. However, she did not gain good vision in RE. There was no history of trauma, radiation exposure, reduced scotopic vision, or any other intraocular surgery. Her personal and family history was unremarkable for any systemic or ocular disease.

Her blood pressure was 120/80 mmHg and investigations including blood sugar (93 mg/dl), kidney function tests (blood urea of 28 mg/dl and serum creatinine of 0.86 mg/dl), and liver function tests (serum glutamic oxaloacetic transaminase of 25 U/L and serum glutamic pyruvic transaminase of 35 U/L) were within normal limits. The electrocardiogram showed a normal sinus rhythm.

One year after cataract surgery, she was referred to the Retina specialist with a complaint of difficulty in seeing with RE. The ocular examination showed BCVA of LogMAR 1.0 (20/200; 6/60) in RE with a refractive correction of -0.75 Dcyl at 70 degrees and LogMAR 0.2 (20/32; 6/9) in left eye (LE) with -1.50 Dcyl at 100 degrees. She had an intraocular pressure of 18 mmHg and axial length (AL) of 23.50 mm in BE. The IOL power was 21.0 dioptres in RE and 21.50 dioptres in LE. The ultrawide field fundus examination showed clear ocular media with an attached retina and optic disc having a cup-to-disc ratio of 0.3:1 in BE. In RE, there was parafoveal chorio-retinal atrophy temporal to the fovea but the retinal periphery did not show degeneration. The simultaneously captured scan of ultrawide field optical coherence tomography showed a central foveal thickness of 138

microns with foveal scarring in RE; and of 267 microns with normal foveal contour in LE. There was no other abnormality like subretinal fluid, neurosensory retinal cystic changes, intra-retinal edema, epiretinal membrane, or vitreo-macular adhesion. The parafoveal chorio-retinal atrophy showed staining on fundus fluorescein angiography. The swept source OCT showed a full-thickness PFH located at a point lying 2.75 mm nasal to the fovea and 3.08 mm temporal to the optic disc in RE. The PFH had a height of 198 microns; a base diameter of 240 microns; arm lengths of 203 microns and 206 microns; and a minimum linear dimension of 42 microns **(Figures 1a and 1b).**

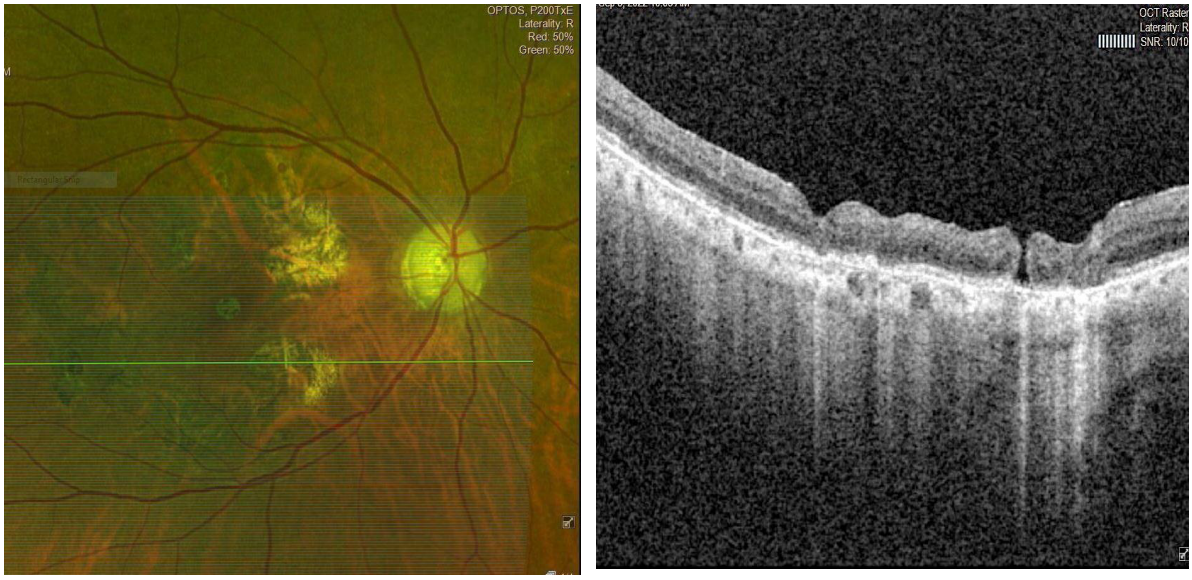

Figure 1a: An ultrawide field fundus photo with optical coherence tomography of the right eye.

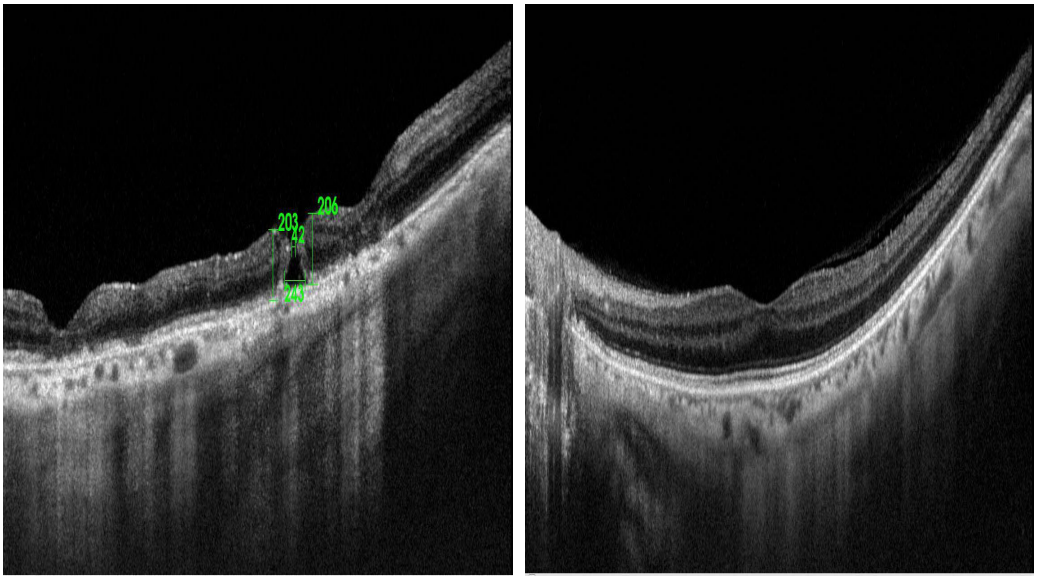

Figure 1b: Swept source optical coherence tomography showing full-thickness parafoveal hole in the right eye and normal foveal contour and macula in the left eye.

The optical coherence tomography angiography (OCTA) scan of RE showed a foveal avascular zone (FAZ) area of 0.14 mm^2, perimeter of 1.81 mm, and circularity index of 0.52. The 8 mm x 8 mm scan showed a reduced vessel density in the superficial and deep retina; and increased visibility of choroidal vessels in outer retina chorio-capillaries, chorio-capillaries, and choroid slab corresponding to the location of PFH **(Figure 2a).** The LE had a FAZ area of 0.02 mm^2, a perimeter of 0.59 mm, and a circularity index of 0.72.

The ultrasound B scan was anechoic and there was no posterior vitreous detachment (PVD) The LE showed some vitreous opacities. The 24-2 visual field (VF) examination of RE showed widespread field defect with a mean deviation (MD) of -12.27 and pattern standard deviation (PSD) of 9.49 while VF in LE had a mean deviation (MD) of -5.93 and pattern standard deviation (PSD) of 2.38 **(Figure 2b).**

The Amsler's grid showed metamorphopsia in RE while it was normal in LE. The retinal

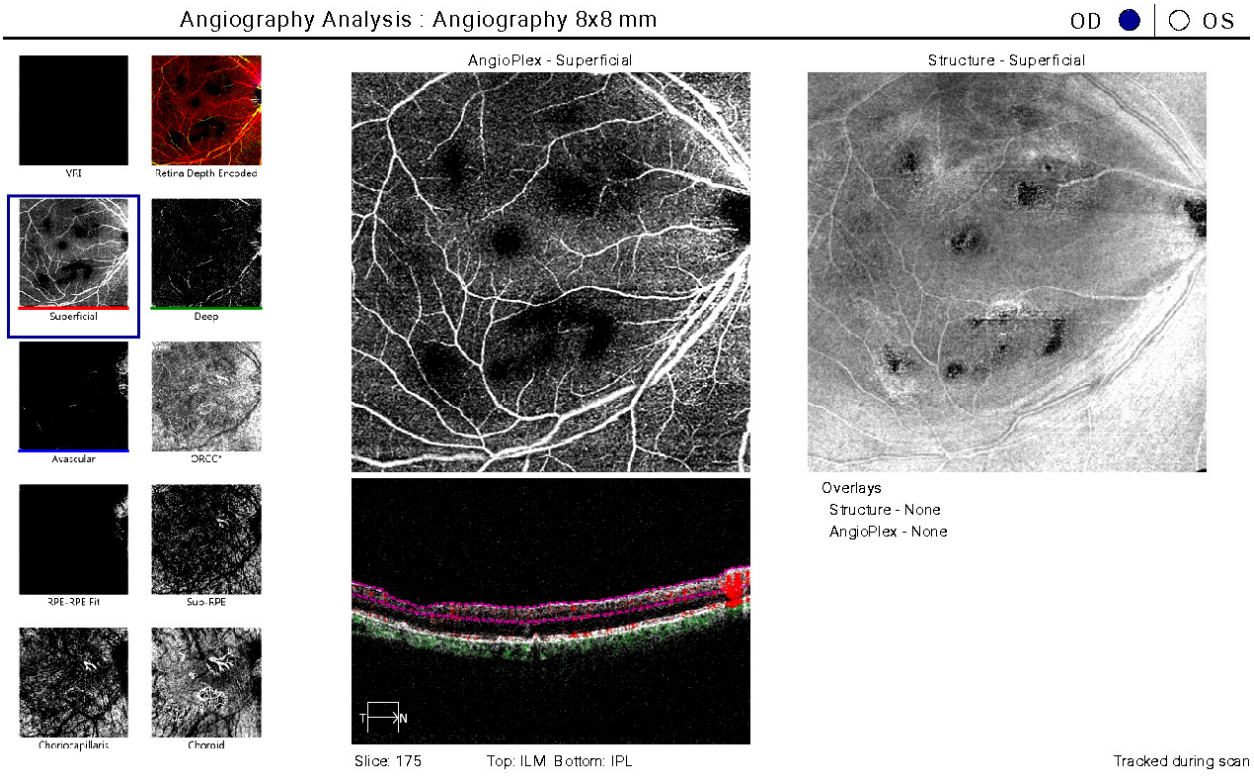

Figure 2a: Optical coherence tomography angiography (8 x 8 mm) scan showing a reduced vessel density in superficial and deep retina; and increased visibility of choroidal vessels in outer retina chorio-capillaries, chorio-capillaries and choroid slab corresponding to the location of parafoveal hole.

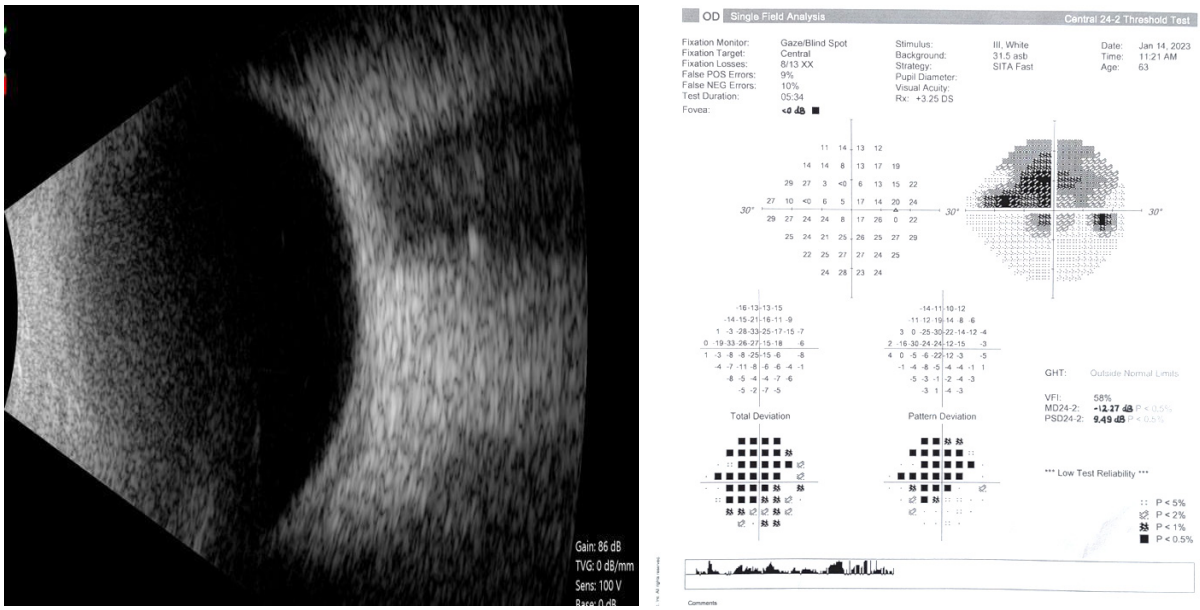

Figure 2b: Ultrasound B scan and visual field plot of the right eye.

nerve fibre layer (RNFL) was thinner in RE (90 microns in RE vs 106 microns in LE), and the ganglion cell complex (GCC) was lesser in thickness and volume in RE (224 microns in RE vs 254 microns in LE; and 6.32 microns in RE vs 7.17 microns in LE respectively). The optic nerve head parameters were affected in RE and showed an increased cup area (1.33 in RE vs 0.85 in LE); increased cup-to-disc ratio (0.51 in RE vs 0.32 in LE); reduced rim volume (0.07 in RE vs 0.16 in LE); and higher disc damage likelihood scale (DDLS) (7 in RE vs 5 in LE).

As there were no visual symptoms or visible traction on the hole, the patient was kept under observation.

DISCUSSION

From their retrospective review of patients after macular surgery, Sandali et al (2012) and Rush et al (2014) found the incidence for paracentral and eccentric MH to be 0.66% and 2.1% respectively. Steven et al (2006) found one to multiple paracentral holes three weeks to 16 months after an uncomplicated ILM removal irrespective of the use of triamcinolone acetonide and dyes like indocyanine green and trypan blue. Mason et al (2007) reported the development of four temporal, one superior, and one nasal eccentric flat FTMH about three months after ILM peeling. The indications of ILM peeling in these cases were cystoid macular edema, macular pucker, MH, and epi macular proliferation (Steven et al, 2006; Mason et al, 2007). Yeh et al (2007) reported a case of FTMH noticed two months after ERM surgery.

The retinal or retinal nerve fibre thinning, structural weakness, or dye toxicity cause secondary holes (Rush et al, 2014; Steven et al, 2006). These may have an associated intraretinal cystic change and minimal to no subretinal fluid

(SRF) (Hussain et al, 2018;Rush et al, 2014; Mason et al, 2007).

Generally, the parafoveal holes are small and were seen to be located temporally (Hussain et al, 2018; Rush et al, 2014; Yeh et al, 2007;Steven et al, 2006). These may remain asymptomatic or may cause visual distortion (Hussain et a, 2018; Lai et al, 2015; Rush et al, 2014;Yeh et al, 2007). Some of these holes were treated with endo photocoagulation (Hussain et al, 2018; Steven et al, 2006). In the absence of traction, only observation was done and the course was found uneventful for as long as 6 months (Steven et al, 2006).

As evident from AL, IOL power, and fundus examination, our patient did not appear to have pathological myopia. We feel that PFH most likely resulted from localized retino-choroidal atrophy. The hole located nasal to fovea over the papillomacular bundle could not be laser barraged. Additionally, the patient had pre-existing RNFL and GCC thinning and VF deterioration. As there was no underlying SRF or traction on the macula, it was decided to keep the patient under observation (Yeh et al, 2007; Steven et al, 2006).

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