

# Treating Acquired Optic Disc Pit Maculopathy with an Ocular Hypotensive Agent

## Abstract

**Purpose:** To describe a case of glaucomatous optic disc pit maculopathy that resolved after bimatoprost treatment.

**Observations:** A 63-year-old African American female with a history of type 2 diabetes mellitus presented to the clinic after 3 months of blurry vision in the right eye and was found to have an asymmetric cup-to-disc ratio, and right intraretinal and subretinal fluid without leakage on fluorescein angiography concerning for optic disc pit maculopathy. A trial of intravitreal anti-vascular endothelial growth factor (VEGF) injection failed to resolve the fluid. Optical coherence tomography of the ganglion cell complex showed thinning of the ganglion cell complex concerning for previously undiagnosed glaucoma, and the patient was started on bimatoprost. The previously noted fluid resolved, and the patient has been quiescent for 4 years on ocular hypotensive medication.

**Conclusion:** In untreated or poorly controlled glaucoma, progressive cupping of the optic nerve head may lead to the development of an acquired optic disc pit, which can produce maculopathy. This maculopathy can improve under ocular hypotensive treatment.

## Introduction

A 63-year-old African American female was referred for evaluation of possible central serous chorioretinopathy in the right eye. The patient noted blurred vision in the right eye for 3 months without interval improvement. Past medical history was significant for type 2 diabetes mellitus treated with metformin. She denied any previous known ocular history.

Best corrected visual acuity was 20/100 in the right eye and 20/30 in the left eye. Intraocular pressure was 14 mmHg in each eye (Goldmann applanation tonometry). There was no relative afferent pupillary defect (RAPD). Anterior segments were both unremarkable with no signs of significant ocular surface diseases, or corneal edema. Mild nuclear sclerotic cataracts were noted bilaterally. The fundus of the right eye compared to the left eye was notable for asymmetry in the cup-to-disc (C:D) ratio and subretinal fluid nasal to the fovea (Figure 1). The optical coherence tomography (OCT) performed through the right eye demonstrated the elevation nasal to the foveal center, and the cross-sectional view was notable for subretinal fluid and intraretinal fluid with macular schisis and deep cupping (Figure 2). Fluorescein angiography did not show leakage or filling defects.

At this time, the differential diagnoses for the subretinal fluid involving the macula and the optic nerve head included optic nerve pit, pathologic myopia, morning glory deformity, and papillorenal syndrome. OCT of the ganglion cell complex was obtained (Figure 3). The images were notable for thinning of the nerve fiber layer (NFL), ganglion cell complex (GCC), and inner plexiform layer (IPL) more pronounced in the right eye than the left eye. This suggested that the patient may have had a previously undiagnosed glaucoma, and the decision was made to start bimatoprost. The patient followed up 3 months later while on bimatoprost. The visual acuity improved



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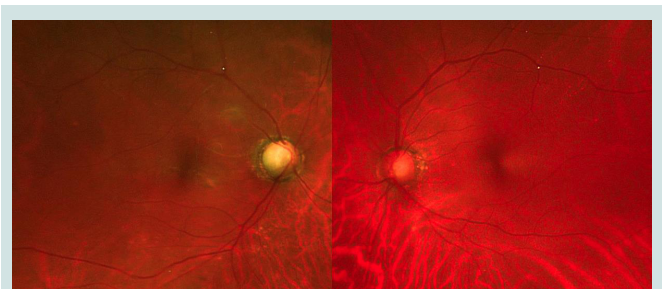
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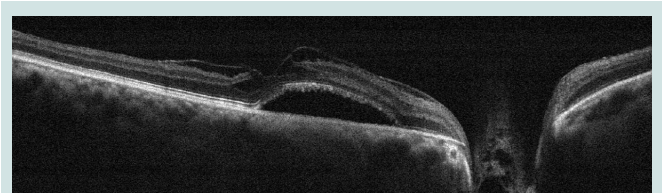
to 20/50 and OCT showed resolution of the subretinal fluid and improvement in macular schisis and intraretinal fluid (Figure 4). Therefore, given the clinical course, our diagnosis for the patient was glaucomatous optic disc pit maculopathy that subsequently resolved after starting a prostaglandin analog. Throughout the clinical course, the corneal clarity remained clear.

## Discussion

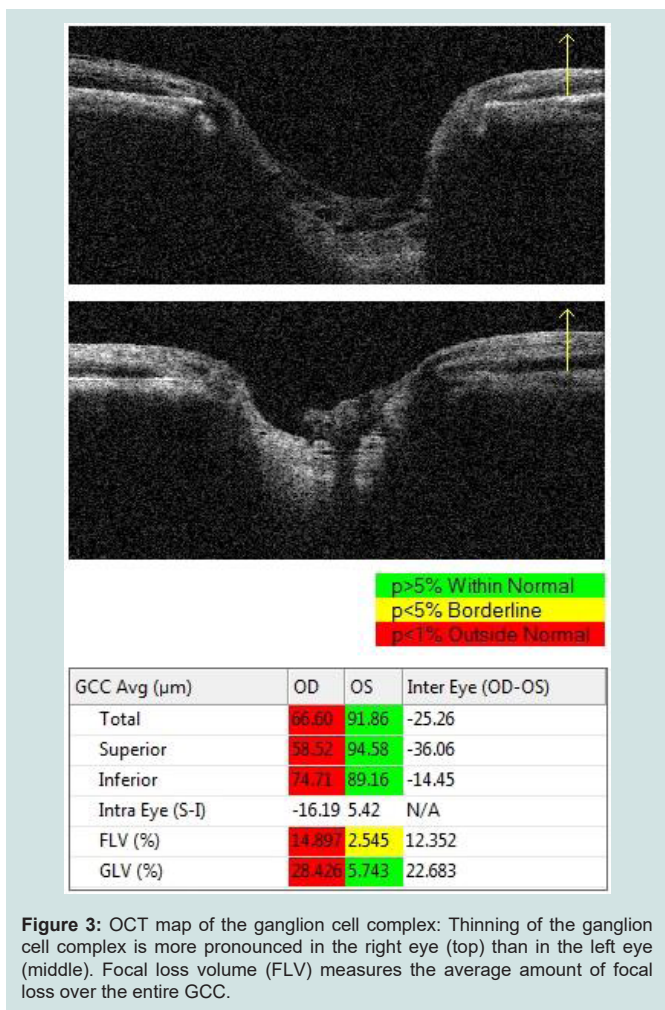
An optic disc pit (ODP) can be acquired as a result of localized glaucomatous damage [1]. A known complication of an ODP is maculopathy. It has been reported that an optic disc pit can resolve after starting ocular hypotensive medication and that ODP maculopathy may resolve spontaneous or after posterior vitreous detachment [1,2]. However, this case is the first to show a reversal of glaucomatous ODP associated maculopathy after topical ocular hypotensive medication,



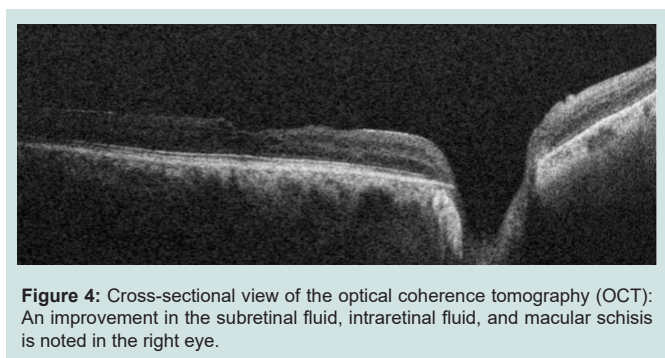
**Figure 1:** Color Fundus Photography of Both Eyes: It is notable for asymmetry in the cup-to-disc (C:D) ratio.



**Figure 2:** Cross-sectional view of the optical coherence tomography (OCT): Both subretinal fluid and intraretinal fluid with macular schisis are present with deep cupping of the optic nerve in the right eye.



**Figure 3:** OCT map of the ganglion cell complex: Thinning of the ganglion cell complex is more pronounced in the right eye (top) than in the left eye (middle). Focal loss volume (FLV) measures the average amount of focal loss over the entire GCC.



**Figure 4:** Cross-sectional view of the optical coherence tomography (OCT): An improvement in the subretinal fluid, intraretinal fluid, and macular schisis is noted in the right eye.

more specifically, a prostaglandin analog. The patient had previously documented posterior vitreous detachment, seen in the OCT images, which implied that changes in the vitreoretinal or vitreo-papillary interface did not have an effect.

Histologically, optic disc pit is a herniation of dysplastic retinal tissue through a defect in the lamina cribosa, extending posteriorly to the subarachnoid space, and may be congenital or acquired [3]. The major complication is optic disc pit maculopathy with fluid accumulating in the macula [4]. The pathogenesis of this phenomenon is not clear but there are three possible hypotheses. One is that

vitreous fluid enters the subretinal space through the defect. Two is cerebrospinal fluid enters from the subarachnoid space through the defect. Three is the blood vessel at the optic disc pit itself may leak fluid [4]. OCT will show a serous retinal detachment with a schisis cavity and a coexisting detachment of the outer layer of the retinal pigment epithelium with both IRF and SRF [4]

Once the maculopathy develops, it often begins with a progressive macular schisis, with fluid that extends through the external limiting membrane to cause a subfoveal macular detachment and acute vision loss. Classic treatment options include barrier laser, gas tamponade, and surgical treatment including PPV and macular buckling, all of which aim to close the communication between the pit and the subretinal or intraretinal space. No technique has been proven to be superior to one other and often multiple treatments are needed to achieve the reattachment of the retina [5].

The optic disc pit is not always congenital, but can also be acquired. This is thought to occur especially in patients with glaucoma where lamina cribosa is altered as part of the glaucomatous optic nerve damage, which then can lead to a focal formation of the optic disc pit [6-9]. While this is not the most common presentation for patients with glaucoma, there had been studies [10] that found a higher prevalence of acquired optic disc pit in people with normal-tension glaucoma, and successful reversal of those pits with IOP lowering drops [1,11,12]. In our case, we postulate that an increase in the uveoscleral outflow from bimatoprost may have altered the tissue remodeling through glial cell activation to facilitate the closure of optic disc pit as well as the resolution of the subretinal and intraretinal fluid.

**Conclusion**

In conclusion, subretinal fluid nasal to the fovea should involve an investigation of both the optic nerve and the macula. In untreated or poorly controlled glaucoma, progressive cupping of the optic nerve head may lead to the development of an acquired optic disc pit, which can produce maculopathy if untreated.

**Patient Consent**

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

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