RESEARCH LETTER

Enhanced Vitreous Imaging Technique With Spectral-Domain Optical Coherence Tomography for Evaluation of Posterior Vitreous Detachment

Vitreous degeneration, gradual separation of the posterior vitreous cortex (PVC), and eventual development of posterior vitreous detachment (PVD) are important in vitreoretinal interface diseases. However, determination of the stages of PVD with biomicroscopy or ultrasonography is not sufficiently sensitive and specific.\(^2\,^7\) Since the advent of spectral-domain (SD) optical coherence tomography (OCT), PVD detection has improved and new concepts regarding the evolution of PVD have emerged.\(^2\,^3\) However, SD-OCT remains underused, possibly owing to general low awareness of its potential to image the vitreoretinal interface and the vitreous itself.\(^4\,^5\) We aim to demonstrate the usefulness of an enhanced vitreous imaging (EVI) OCT technique in evaluating PVD.

Methods | Six radial scans with a scan width of 30° centered at the fovea were obtained on the Heidelberg Spectralis (Heidelberg Engineering), in addition to a single 30° line scan taken with a 7° tilt, traversing both the optic disc and fovea simultaneously (Figure 1). All scans were the product of 45 to 70 averaging images by automatic real-time function, pulling back to focus on the vitreous. The retinal layers were positioned inferiorly on the image screen to obtain a maximum imaging depth of 1.5 mm into the vitreous, and the image brightness was increased slightly to improve visualization of the vitreous.

Results | Using EVI-OCT, we were able to demonstrate various stages of PVD. In posterior vitreous attachment, there is persistent attachment of the PVC over the entire posterior pole including the fovea and optic disc (Figure 2A). Incomplete PVD may begin with detachment of the PVC at perifoveal areas but persistent attachment over the fovea and persistent attachment at the optic disc (Figure 2B and C). With gradual release of the persistent attachment over the fovea, there is progression of the PVD, although it is incomplete due to persistent attachment at the optic disc (Figure 2D and E). Disruption of the posterior wall of the premacular bursa\(^4\) was well demonstrated with EVI-OCT (Figure 2F and G). The EVI-OCT displayed vitreous opacities and laminar structures of the posterior vitreous in the context of vitreoschisis (Figure 2G).\(^6\) Complete PVD could be appreciated as an optically empty vitreous cavity seen central to the retinal layers, and often an intact PVC was seen (Figure 2H). During the development of perifoveal PVD, strong vitreomacular adhesion may lead to macular hole formation with an operculum or vitreomacular traction syndrome (Figure 2I and J).

Discussion | Studies using SD-OCT for the posterior vitreous have previously been limited to vertical and horizontal scans on the Cirrus OCT machine.\(^4\,^5\) This article illustrates the use of radial scans on the Spectralis SD-OCT. Inclusion of both the optic disc and the fovea is critical to unequivocally stage posterior vitreous separation. With EVI-OCT, we were able to achieve adequate image resolution and depth into the vitreous by using built-in functions including defocus, scan tilt, and automatic real-time function. In our experience, the “sweet spot” for automatic real-time function lies between 45 and 65 scans, as small saccades during the lengthy scanning process lead to vitreous movement and consequent apparent doubling of the PVC (Figure 2J). This
problem is obviated on swept-source OCT machines owing to the much faster image acquisition. The use of EVI-OCT may enhance patient counseling by demonstrating visible vitreous floaters and PVC separation to symptomatic patients, improving patient understanding and promoting greater patient satisfaction. Keeping in mind that only the posterior vitreous is visualized, supplementing with B-scan ultrasonography may be prudent to establish peripheral vitreous face status.

In summary, EVI-OCT enables more precise characterization of the PVD process, which is essential in this era of pharmacologic vitreolysis. This article aims to heighten...
awareness of this vitreous imaging technique and encourage its use in the appropriate clinical setting.

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**OBSERVATION**

**Development of Bilateral Acquired Toxoplasmic Retinochoroiditis During Erlotinib Therapy**

*Toxoplasma gondii* retinochoroiditis is the most common cause of posterior uveitis in North America. Immunocompromised patients are particularly susceptible, and bilateral acquired toxoplasmic retinochoroiditis is thought to be rare without immunosuppression. We describe a woman who developed bilateral acquired toxoplasmic retinochoroiditis while undergoing treatment with erlotinib hydrochloride, an epidermal growth factor receptor tyrosine kinase inhibitor, in the absence of other immunomodulatory agents or neutropenia. When considered in conjunction with a recent report detailing similar findings in a patient treated with a related biologic therapeutic, the association of erlotinib with this unusual presentation suggests that inhibition of epidermal growth factor receptor and the downstream Janus kinase (JAK)—signal transducers and activators of transcription (STAT) pathway may be an underrecognized host risk factor for toxoplasmic retinochoroiditis.

**Report of a Case** A woman in her early 60s with metastatic lung adenocarcinoma undergoing treatment with erlotinib presented with bilateral floaters and decreased vision. She regularly consumed wild venison. Best-corrected visual acuity was 20/50 OD and 20/20 OS. There were bilateral fine keratoprecipitates and 1+ anterior chamber cells. Dilated ophthalmoscopy revealed 2+ vitritis bilaterally with 3 peripapillary patches of retinitis in the right eye and a single focus of retinitis in the temporal perifoveal region of the left eye (Figure 1). No scars consistent with previous retinitis were detected. Infection workup revealed strongly positive serum *T gondii* IgM, IgG, IgA, and IgE titers, a pattern highly suggestive of acute infection. Notably, the patient recalled prolonged fever and influenza-like illness affecting herself and her husband following consumption of undercooked venison 3 months prior, possibly representing initial *Toxoplasma* infection. Results of the remainder of her workup (blood cultures, serum syphilis, tuberculosis skin testing, and aqueous humor polymerase chain reaction for herpes simplex virus, varicella-zoster virus, and cytomegalovirus) were negative. Testing of her husband, who consumed the same venison, demonstrated similarly positive *Toxoplasma* serology, although no ocular disease was detected. Treatment of the woman was initiated with oral atovaquone, 750 mg 4 times daily; subsequently, vitritis cleared bilaterally and vision returned to that at baseline. Postmortem examination 2 months after initiating atovaquone showed full-thickness retinal necrosis in the areas of retinitis noted on clinical examination (Figure 2A) and 1 *T gondii* cyst (Figure 2B).

**Discussion** The development of bilateral acquired toxoplasmic retinochoroiditis in this patient suggests susceptibility due to immunosuppression from metastatic cancer, erlotinib treatment, or a combination of the two. Epidermal growth factor receptor resides on cell membranes and helps regulate cell proliferation and survival; overexpression in certain neoplasms makes it a desirable target. Erlotinib inhibits epidermal growth factor receptor-induced activation of STAT, which may impair host resistance to *T gondii*. The secretion of interferon γ by host cells following *T gondii* exposure results in activation of the JAK-STAT pathway with downstream synthesis of indoleamine 2,3-dioxygenase, an enzyme in the pathway responsible for tryptophan degradation. Tryptophan is essential for *Toxoplasma* tachyzoite replication, and it is believed that interferon γ stimulation of tryptophan degradation is an important mechanism for host resistance to *T gondii*. Inhibition of the JAK-STAT pathway by erlotinib and related agents may thus increase disease susceptibility. Goldberg et al recently reported a similar case of bilateral acquired toxoplasmic retinochoroiditis in a patient treated with ruxolitinib, a JAK-1 and