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Choriovitreal Neovascularization following resolution of Infectious Chorioretinitis

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Summary statement:

This report describes the first documented case of choriovitreal neovascularization (CVNV) occurring after infectious chorioretinitis, with OCT-angiography (OCT-A) demonstrating flow through the vitreal portion of the lesion, and response to anti-VEGF therapy.

Keywords

Choriovitreal neovascularization; optical coherence tomography angiography; OCT angiography; choroidal neovascular membrane; infectious chorioretinitis

Choriovitreal neovascularization (CVNV) is a rare condition where choroidal neovascularization (CNV) extends into the vitreous cavity. Although sporadic cases have been reported,[1, 2] CVNV often occurs as a result of iatrogenic damage to the retinal layers and Bruch's membrane from high-energy laser or cryotherapy[3] for treating various pathologies such as diabetic retinopathy, sickle cell disease,[4] retinal vein occlusions,[3] and tumors.[5] Prior reports of CVNV preceded the widespread use of anti-vascular endothelial growth factor (anti-VEGF) agents, and were associated with a poor prognosis.

A 56-year-old man with methicillin-sensitive *S. aureus* (MSSA) bacteremia and endocarditis presented with vitritis and focal chorioretinitis temporal to the fovea, which appeared as a hyperreflective lesion on OCT (Figure 1A) . A week after systemic penicillin treatment, OCT showed resolution of the chorioretinitis lesion with focal loss of most retinal layers and Bruch's membrane (Figure 1B). Two weeks later, a choroidal neovascular membrane (CNV) developed at the site of the prior lesion, which appear similar to the retinal infiltrate on OCT (Figure 1C), but was distinguished by the presence of hemorrhage on clinical exam. Monthly intravitreal bevacizumab led to improvement in the retinal hemorrhage, but persistence of the CNV. After adjunct treatment with half-fluence photodynamic therapy (PDT), the CNV decreased in size, but extended into the vitreous cavity along the posterior hyaloid (Figure 1D). OCT angiography (OCT-A) revealed vascular flow (red areas, Figure 2A) through both the base of the lesion in the avascular outer retinal slab (purple dashed lines, Figure 2A), and the apex of the lesion along the vitreoretinal interface (yellow dashed lines, Figure 2A),

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consistent with CVNV. The en face OCT-A images corresponding to these segmented slabs demonstrate the CVNV architecture at different layers (Figures 2B and 2C). Further treatment with bevacizumab resulted in decreased flow through the CVNV lesion, although the overall structure appeared overall unchanged (not shown).

This unique case demonstrates a CVNV lesion that developed through a full-thickness defect in the retina and Bruch's membrane caused by infectious chorioretinitis, with reduction of vascular flow on OCT-A in response to anti-VEGF treatment. The lesion developed after switching to PDT therapy, suggesting that anti-VEGF therapy may be more effective than PDT in suppressing CVNV. OCT-A may be helpful in diagnosing this condition and monitoring clinical response to therapy.

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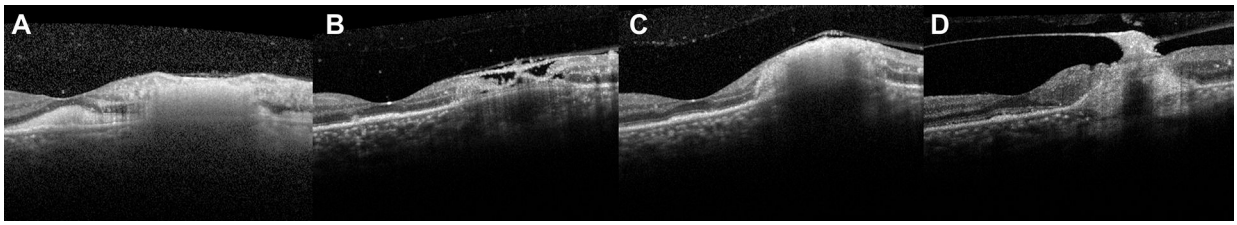

Figure 1. Progression of chorioretinitis to choriovitreal neovascularization (CVNV)

Optical coherence tomography (OCT) B-scans demonstrating focal chorioretinitis at presentation (A) and its resolution one week after systemic treatment, leaving a defect in most of the retinal layers and Bruch's membrane (B). A choroidal neovascular membrane developed 2 weeks later (C), with the CVNV forming 6 weeks after photodynamic therapy (D).

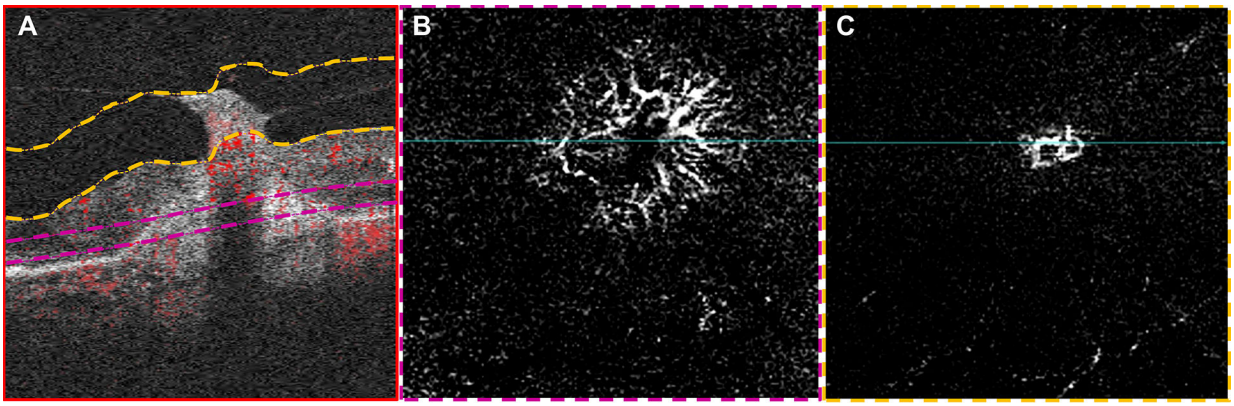

Figure 2. Optical coherence tomography angiography (OCT-A) of choriovitreal neovascularization (CVNV)

OCT-A B-scan through CVNV lesion (A), demonstrating vascular flow (red). Purple dotted lines show segmentation of the avascular layer with flow in the base of the lesion, which corresponds to the en-face projections (B). Yellow dotted lines highlight segmentation of vitreoretinal interface with flow in the apex of the lesion seen on en-face projections (C).