Sudden Bilateral Inflammatory and Neovascular Lesions in Multifocal Choroiditis

Raquel Burggraaf-Sánchez de las Matas*, Laura Such-Irusta and Julián Zarco-Bosquet

1Hospital l’Esperit Sant, Barcelona, Spain
2Hospital of Sagunto, Valencia, Spain

Abstract

We report a 40-year-old Caucasian male presenting sudden bilateral Choroidal Neovascularization (CNV) along with Chorioretinal Lesions (CRL). He presented previous ocular history of relapsing idiopathic bilateral anterior uveitis.

Initial study of new signs included Fluorescein Angiography (FA), Spectral Domain Optical Coherence Tomography (SD-OCT) and OCT Angiography (OCT-A), reaching the diagnosis of multifocal choroiditis and panuveitis with bilateral CNV.

He started intravitreal injections of the anti-Vascular Endothelial Growth Factor (a-VEGF) Aflibercept. Apart from initial oral corticosteroids, immunosuppressive therapy was adjusted with Adalimumab and Cyclosporine. Lesions were analyzed and followed-up with both OCT-A en face and b-scans. While the Right Eye (RE) recovered VA to 20/20 and stabilized with 6 injections after 1 year-follow-up; left eye (LE) exhibited relapsing activity of his CNV with decreased VA not getting over 20/50.

OCT-A was a useful non-invasive tool for evaluating CNV activity and inflammatory lesions surveillance. CNV relapse was attributed to poor immunosuppressive control.

Keywords: Aflibercept; Bilateral choroidal neovascularization; Fluorescein angiography; Multifocal choroiditis and panuveitis; optical coherence tomography angiography

Copyright: © 2021 Burggraaf-Sánchez de las Matas R, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Received: May 24, 2021; Accepted: June 01, 2021; Published: June 08, 2021

Sudden Bilateral Inflammatory and Neovascular Lesions in Multifocal Choroiditis

A 40-year-old Caucasian myopic male consulted for bilateral acute central loss of vision. His ophthalmological records consisted in recurrent bilateral anterior uveitis, following a viral prodrome, without vitreous or posterior pole involvement. Due to relapsing attacks in spite of oral corticosteroids, treatment was initiated with Sulfasalazine (1500 mg daily). Nevertheless, it was switched to Infliximab (330 mg every 2 months) owing to a transient acute myopization event.

Ophthalmological examination at the time of new symptoms exhibited VA of 20/50 RE and 20/100 LE. There was a mild anterior chamber reaction. Fundoscopy revealed new yellowish Juxtafoveal lesions with blurred edges, and well delimited dots spread around the posterior pole and periphery (Figure 1A). Initial study included FA (Figure 1B&1C), SD-OCT (Figure 2) and OCT-A (Figure 3), elucidating the diagnosis of multifocal choroiditis and panuveitis with bilateral CNV.

Systemic steroids were initiated and maintenance treatment was adjusted to adalimumab (80 mg every 2 weeks). Both eyes received 2 Aflibercept injections, recovering VA to 20/20 and observing regression of CNV at month 2. Relapse occurred at month 4, presenting VA of 20/32 RE and 20/40 LE.

Figure 1: A-Retinographies: CNVs (red arrowheads) and perifoveal CRLs (white arrows). B-Arteriovenous phase of FA: CNVs visualized as hyperfluorescent loops which progressively stain (red arrowheads). Hypofluorescent CRLs become hyperfluorescent after venous filling (white arrows). C-Recirculation phase of FA: late leakage of CNVs (red arrowheads) and no dye diffusion of CRLs (white arrows).
Cyclosporine (400 mg daily) was added to systemic therapy but had to be interrupted at month 12 due to renal insufficiency and hypertension. RE received 4 additional doses, VA recovered to 20/20, and stabilized after 1 year-follow-up; while LE required further intravitreal management reaching 10 doses at month 14, and his VA did not get over 20/50.

Discussion

CNV is a common complication of MFC, nevertheless this case shows an acute bilateral presentation in a patient already receiving systemic immunosuppressive treatment for bilateral anterior chamber inflammation. This fact could have produced a delay in posterior pole manifestations.

Response of inflammatory CNV to α-VEGF agents has been demonstrated in literature [1,2]. This report shows rapid response and conversion from classic inflammatory type 2 lesions into type 1 CNV. We attribute reactivation of CNV and the large need of intravitreal injections in this patient to deficient systemic immunosuppressive treatment, which became challenging due to multiple intolerances [2]. Regarding imaging, OCT-A allowed us a non-invasive detailed evaluation for both initial characterization and follow-up of CNV activity [3,4].

Figure 2: We observe type-2 CNVs at week-1-visit (red asterisks), resulting from RPE disruption (red arrowhead). Conversion to type-1 inactive CNVs (white asterisks) takes place at month-2. RPE disruption (red arrowheads) reoccurs at month-4, producing mixed type-1/2 configuration active CNVs (yellow asterisks). From month-6 forward type-1 pattern remained stable.

Figure 3: A) Abnormal vascular loops (red arrowheads) correspond to type-2 CNVs, visualized within the avascular retina on en-face maps. These loops demonstrate intraretinal flow signal (IFS) at corresponding b-scans. Yellow arrowhead points at an artifact defect, which mimics IFS within an avascular CRL. B) Inactivity of type-1 CNVs with absence of IFS. C) New vascular networks (red surfaces) are observed, which exhibit IFS at b-scans (red arrowheads). D) Blurred neovascular areas at month 6 of follow-up. No activity within the RE is observed, but intraretinal flow signal at b-scan of the LE is still observed within type-1 CNV (red arrowhead).

Financial Support and Sponsorship

This report has received no funding.

Conflicts of Interest

The authors declare no conflicts of interest.

References


Submit Your Manuscript: https://www.heraldopenaccess.us/submit-manuscript