Bacillary layer detachment in a patient with serpiginoid choroiditis

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Serpiginous choroiditis is posterior uveitis involving the retinal epithelium pigment, the choriocapillaris, and the choroid. It presents a geographic pattern of choroiditis, extending from the juxtapapillary choroid and intermittently spreading in a centrifugal fashion. The cause and pathogenesis are unknown. Some authors suggest a degenerative disorder[1] and others an inflammatory cause. Secchi *et al*.[2] suggested the presence of autoantibodies; however, similar clinical profiles have been described in relation to infectious microorganisms, prominent among which is *Mycobacterium tuberculosis*, which has been termed tuberculous serpiginoid choroiditis.

Bacillary layer detachment (BLD) is a novel optical coherence tomography (OCT) finding that was first introduced by Mehta *et al*.[3] Although the first descriptions of this tomographic image were made in patients with ocular toxoplasmosis, BLD appears more frequently in Vogt–Koyanagi–Harada (VKH) syndrome.[4] It has also been described in other inflammatory ocular conditions, such as posterior scleritis, systemic lupus erythematosus, and primary inflammation of the choriocapillaris, as well as in acute posterior placoid pigment epitheliopathy, choroidal metastasis, osteoma, and tuberculous choroidal granuloma.[5-7] However, to date, a BLD has never been described in a case of tuberculous serpiginoid choroiditis.

A 62-year-old woman with tuberculous serpiginoid choroiditis who had received treatment with antituberculosis drugs presented inflammatory outbreaks after finishing the treatment. The diagnosis had been established via the clinical appearance of the lesions (multifocal and sparing the juxtapapillary area), a positive interferon-gamma release assay (QuantiFERON® TB Gold), and a chest radiograph with pleural effusion that supported the diagnosis. No other microbiological tests such as a polymerase chain reaction or cultures of intraocular fluids were performed. Oral corticoid treatment was therefore administered; however, she continued to suffer inflammatory outbreaks, which required immunosuppressive treatment with azathioprine and cyclosporine. While the patient was taking 50 mg/12 h of azathioprine, 50 mg/12 h of cyclosporine, and 5 mg/24 h of prednisone, she reported a 4-day evolution of central scotoma in the right eye (OD). Best-corrected visual acuity (BCVA) in OD was 6/30 (previously 6/6) and 6/6 in the left eye (OS). In a fundus examination of the OD, atrophic parapapillary dendritic areas were seen and an exudative detachment was detected in the posterior pole [Fig. 1]. Fundus autofluorescence (FAF) images did not show alterations at the level of the exudative detachment [Fig. 1]. A spectral-domain optical coherence tomography (SD-OCT) was performed that showed intraretinal fluid at the level of the photoreceptor layer with the splitting of the ellipsoid zone, forming a BLD with interspersed septations within the lesion suggestive of a detachment of the bacillary layer, together with a thickened subfoveal choroid (308 µm thick) as shown in Fig. 2.

Consequently, an intravitreal dexamethasone implant was injected urgently, which resulted in the resolution of the intraretinal fluid and a decrease in subfoveal choroidal thickness, which went from 308 µm to 225 µm. In addition, as shown in Fig. 2, the results remained stable 1 month later. At that moment, BCVA was 6/6 and SD-OCT showed disruption of the ellipsoids and thickening of the retinal pigment epithelium [Fig. 2]. It was considered that the immunomodulatory therapy that the patient was receiving up to that moment was not effective and azathioprine was replaced with adalimumab. After 3 months' monitoring, BCVA is 6/6 in both eyes, the patient has not shown recurrence of the disease and is receiving treatment with cyclosporine 50 mg/12 h, prednisone 5 mg/24 h, and adalimumab 40 mg/2 weeks.

Discussion

The OCT image of the BLD has been described as a cystic cupuliform retinal space with amorphous hyperreflective matter along the outer retinal surface and a faint line of reflectivity following the ellipsoid area. It has been suggested that this finding could be a division of the photoreceptors at the myoid level, between the outer limiting membrane and the ellipsoid of the inner photoreceptor segment, pointing to

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an inherent weakness in the structure of the photoreceptors at that level as part of the pathogenesis of the disease.[3] Recent histological findings demonstrating the possibility that individual cones in the human macula could detach from their inner segments in neovascular age-related macular degeneration[8] were consistent with this definition.

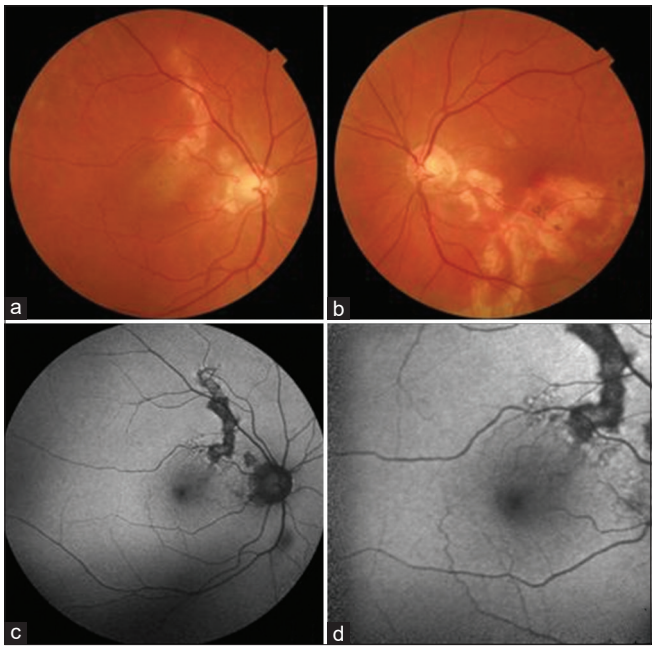

Figure 1: Retinographies of both eyes (a and b) and fundus autofluorescence (FAF) of the right eye (OD) (c and d) at baseline. The retinographies show atrophic areas with a parapapillary dendritic appearance, and an exudative detachment in the posterior pole of the OD (a) can just be seen. In the FAF images of the OD (c) and at higher magnification (d) no alterations are seen at the level of the exudative detachment

It has been hypothesized that BLD develops in the context of processes characterized by an abnormally acute thickening of the choroid, associated with a massive flow of fluid into the neuroretina, which would cause splitting in the photoreceptor layer related to shear stress caused by vigorous and sudden exudation.[6] This situation is very frequent in VKH disease and, in fact, as mentioned, the presence of BLD is common in this disease. Tuberculous serpiginoid choroiditis is a type of posterior uveitis caused by *M. tuberculosis*. Usually, diagnosis is based on the clinical appearance of the lesions (multifocal and sparing the juxtapapillary area, as opposed to classic or autoimmune serpiginous choroiditis, in which lesions arise from the optic disc and are contiguous) and the demonstration of latent tuberculosis using a Mantoux test and/or an interferon-gamma release assay. Regarding the treatment of tuberculous serpiginoid choroiditis, the COTS group recommends antitubercular treatment with positive results from even 1 immunological test, even if there were no radiological features suggestive of tuberculosis.[9] Our patient fulfilled this criterion and hence received antitubercular treatment. In tuberculous serpiginoid choroiditis, the inflammation is thought to occur primarily at the level of the choriocapillaris and, therefore, a significant increase in choroidal thickness would not be expected.[4,10] However, in this case, an increase in choroidal thickness was observed, and we believe that this fact could have been responsible for the appearance of BLD in this patient.

In conclusion, BLD has been described in uveitis, mainly in cases of VKH and also in tubercular choroidal granuloma; however, as far as we know, there are no cases described in the literature with this type of detachment in tuberculous serpiginoid choroiditis.

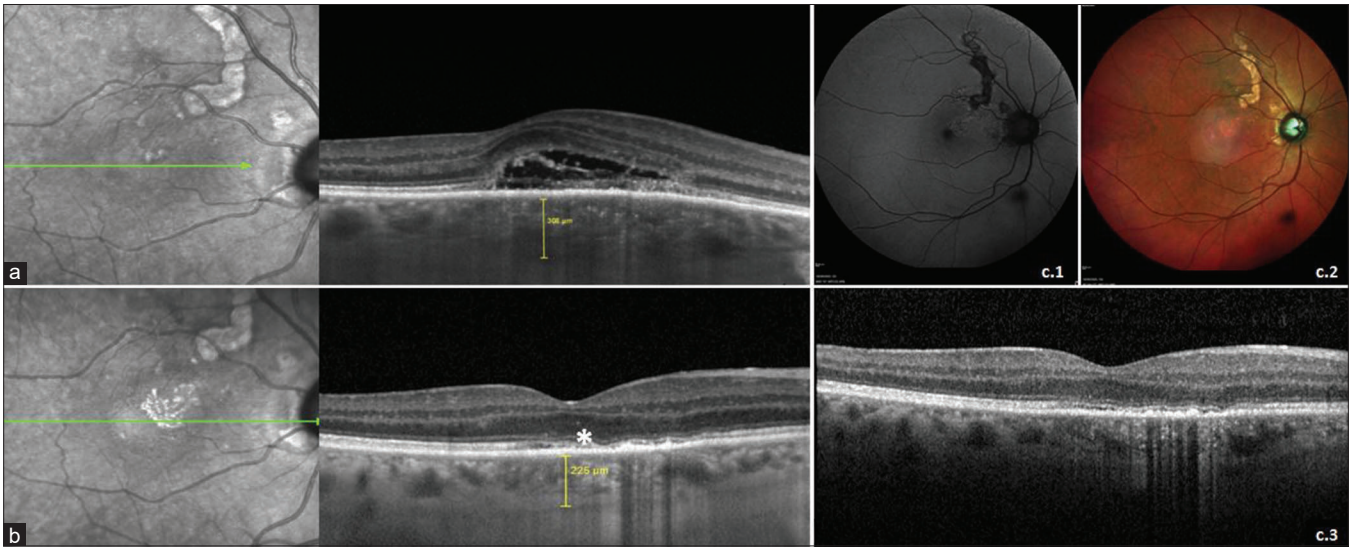

Figure 2: (a) Spectral-domain optical coherence tomography (SD-OCT) of the right eye (OD) showing intraretinal fluid at the level of the photoreceptor layer, suggestive of detachment of the bacillary layer (BD). A thickened subfoveal choroid can also be observed. (b) SD-OCTs 1 week after the intravitreal dexamethasone implant injection.(*) Disappearance of the subretinal fluid with disruption of the ellipsoids and thickening of the retinal pigment epithelium are shown, together with a decrease in the subfoveal choroidal thickness. (c) FAF (c. 1), multicolor image (c. 2) and SD-OCT (c), of the OD 1 month after treatment with dexamethasone implant. In the FAF (a), hyperautofluorescence is seen in the area of the BD, which is seen as an atrophic area in the multicolor image (b). SD-OCT (c), shows disruption of the ellipsoids and thickening of the retinal pigment epithelium

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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