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REVIEW

The revival of an old term with optical coherence tomography: Bacillary layer detachment

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Abstract

Bacillary layer detachment (BALAD) is a relatively new optical coherence tomography (OCT) finding described by the utilization of OCT and characterized by the separation within the photoreceptor inner segment. The most common ocular diseases associated with BALAD include Vogt-Koyanagi-Harada's disease, neovascular age-related macular degeneration, and central serous chorioretinopathy, and it is frequently observed in inflammatory, infectious, traumatic, and mass lesions of the choroid. It usually has a benign course and can resolve spontaneously or with treatment in a very short time, accompanied by an increase in visual acuity. However, its prognostic significance is still contradictory, as studies with long-term follow-up have shown that BALAD is related with subretinal fibrosis. In this review, the anatomical definition, multimodal imaging findings, course, and prognosis of BALAD in various ocular diseases are discussed.

Keywords: Bacillary detachment; bacillary layer detachment; neovascular age-related macular degeneration; optical coherence tomography; Vogt-Koyanagi-Harada's disease.

The layer of photoreceptors (PRs), "stratum bacillorum et conorum," was originally described by van Leeuwenhoek in 1722.[1] Then, in 1941, a neuroanatomist called Polyak identified the bacillary layer as the arrangement of PR inner and outer segments (IS and OS).[2] The OS consists of stacked disks and is located adjacent to the retinal pigment epithelium (RPE). The IS next to the external limiting membrane (ELM) is separated into two zones: The myoid zone (MZ), which is rich in ribosomes and endoplasmic reticulum, and the ellipsoid zone (EZ), which has high reflectivity owing to the abundant mitochondria it contains. An optical coherence tomography (OCT) and a schematic diagram of retinal layers are shown in Figure 1a and b, respectively. Figure 1c shows a detailed illustration of the PR IS and OS structures.

Anatomical definition of the retinal layers has improved in relation to the recent advancements in ultra-high-resolution OCT imaging. This has led to the identification of many lesions that guide in diagnosing posterior segment diseases and predicting the disease prognosis. One of the most recent of these is bacillary layer detachment (BALAD), which was described as the separation of the bacillary layer from the other retinal layers caused by an intraphotoreceptor split immediately posterior to the ELM within the PR IS myoids.

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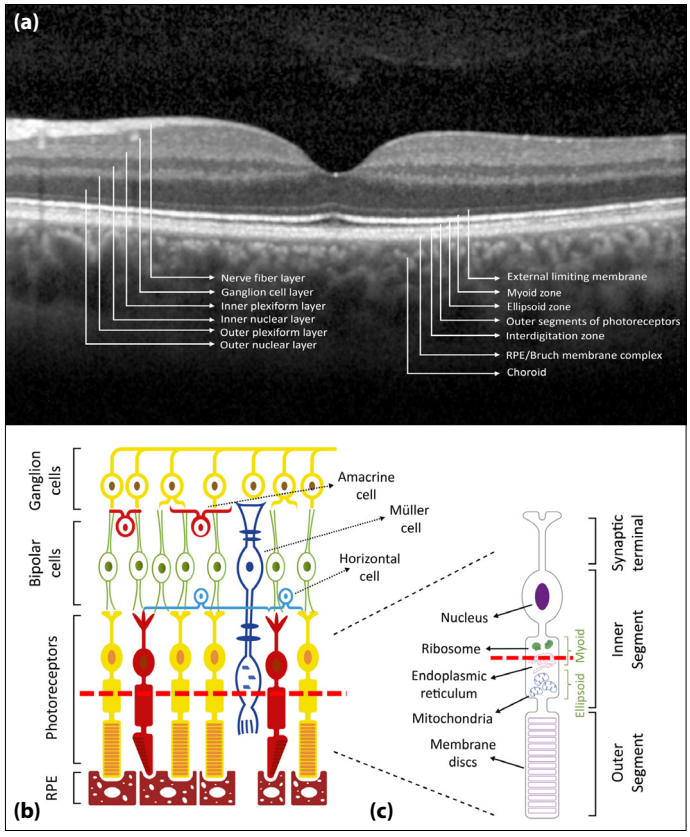

Fig. 1. **(a)** Spectral-domain optical coherence tomography image from the macula of a normal eye. The vitreous, retinal layers, and choroidal layers are visible. **(b)** Schematic diagram of the cellular organization of the retina. Retinal neurons are arranged into specialized layers. Photoreceptors and RPE cells are found in the outer retina, while horizontal cells, bipolar cells, amacrine cells, and ganglion cells which are downstream neurons of the neural excitation pathway of photoreceptors constitute the inner retina. Müller cells are long glial cells spanning all layers of the retina. The dashed red line represents the presumed place of BALAD. **(c)** Photoreceptor structure exemplified by a rod. Photoreceptors have specialized subcellular compartments consisting of an outer segment with tightly packed membrane disks, an inner segment containing the organelle structures necessary for energy and protein synthesis, a cell body containing the nucleus, and a synaptic terminal. The inner segment is further divided into two regions: The myoid zone, which is rich in ribosomes and endoplasmic reticulum, and the ellipsoid zone, which contains abundant mitochondria. The dashed red line depicts the presumed place of BALAD (BALAD, bacillary layer detachment).

This term was introduced by Mehta et al.[3] in a case of ocular toxoplasmosis, although similar findings were previously reported as "photoreceptor elongation or splitting along with huge outer retinal cysts" by Ouyang et al.[4] and "fibrin septum in the subretinal fluid (SRF)" by Lujan[5] in the same disease. Thereafter, a number of authors have used this OCT term in a variety of case reports of Vogt-Koyanagi-Harada (VKH) disease, acute posterior multifocal placoid pigment epitheliopathy (APMPPE), acute idiopathic maculopathy (AIM), ocular trauma, and peripapillary pachychoroid syndrome.[6–10] Although BALAD is a recent term, it may reflect previous naming such as "photoreceptor delamination"[11] in neovascular age-related macular degeneration (nAMD), "subretinal septae,"[12] and "membranous structure"[13] in VKH disease as mentioned in older studies.

Mechanism of Balad Formation

Histological studies provide important insight into why BALAD originates in the MZ. Retinal detachment is a well-known histological artifact in cadaveric eyes in postmortem studies.[14] It can be seen as either a true detachment of the PR OS from the RPE or as a detachment of the MZ itself, as in BALAD. In the subretinal space, the physical, biochemical, and electrical attraction forces generated by the RPE's ensheathment of the OS strengthen the floor. Müller cells, on the other hand, form tight connections with each other and with the PR ISs on the ceiling. Therefore, splitting may be seen in the inner segment MZ, a relatively weak space between the floor and the ceiling. A BALAD-like appearance was also seen in postmortem histological examination of eyes with AMD.[15,16]

The exact mechanism of BALAD formation is not yet known, but several theories exist. One of the pathophysiological processes in BALAD development, similar to exudative retinal detachment, may be the breakdown of the outer blood-retinal barrier, which has been supported by late-phase fluorescein angiography (FA) demonstrating dye pooling inside the BALAD lesion.[17] Furthermore, the higher incidence of SRF and the rarity of intraretinal fluid in BALAD cases support the idea that the key problem is at the level of the outer blood-retinal barrier. Another hypothesis is that BALAD is caused by choroidal ischemia, which reduces blood flow to the PR layer.[7] The majority of BALAD patients had an increase in choroidal thickness. Increased choroidal thickness has been associated with ischemic, inflammatory, infiltrative, and neovascular choroid disorders, including VKH, sympathetic ophthalmia (SO), APMPPE, and choroidal malignancies. Recent OCT angiography (OCTA) studies have revealed choroidal and choriocapillaris flow abnormalities associated with these diseases.[18] The regression of BALAD in cases where the inflammation ceased and the choroidal thickness returned to normal following pulse steroid or plasmapheresis treatment also supports this theory.[6,13,17,18]

Besides the underlying inflammation, it is thought that the sudden increase in hydrostatic pressure induced by the rapid accumulation of fluid in the outer retina may contrib-

ute to the development of BALAD by triggering PR layer breakdown.[11,19,20] This phenomenon, which is named as retinal acute fluid accumulation by Agarwal et al.,[20] is reported in VKH disease, SO, hyperacute stage of APMPPE, and AMD, all of which have been shown to develop BALAD.[6,7,21,22] Acute exudative maculopathy (AEM) following photodynamic treatment (PDT) is considered to be caused by a similar mechanism.[23] The hyperacute choroidal exudation is assumed to be too fulminant in these cases to be retained in the subretinal space, thus it penetrates into the PR IS layer, exceeds the tensile force, and creates a dissection plane at the myoid and/or myoid-ellipsoid level.[24] Fernández-Vigo et al.[23] found that the rate of subretinal fibrin development in the group with BALAD was significantly higher than the group without BALAD in AEM cases. Fibrin interdigitation between PR OS has been hypothesized to improve adhesion of OS to RPE microvilli, resulting in a split in MZ level. This process has also been observed in experimental subretinal hemorrhages as well as in hemorrhagic BALAD.[19,25] Therefore, the rapidity and speed of fluid accumulation in the outer retina may be important for the development of BALAD.

Aside from the aforementioned mechanisms, there may be contributing factors related to the primary ocular disease in the formation of BALAD. In exudative AMD, the development of BALAD may be related to insufficient choroidal perfusion, which leads to stress and splits the PR layer, or to a fast inflow of exudative fluid into the region between the ELM and the EZ zone.[22] Subretinal hyper-reflective material (SHRM) or hemorrhage in the bacillary space, which creates a shearing force, might accompany the formation of BALAD. The BALAD's disappearance following choroidal neovascular membrane (CNV) regression after treatment or when the SHRM or intrabacillary hemorrhage stops further supports this theory.[26] In cases of post-laser BALAD, RPE damage and pump failure, as well as thermal damage to the PR and RPE layer, may contribute.[23] In cases of ocular trauma, the shearing force due to trauma causes separation of the MZ. If there is hemorrhage in the subretinal space, this may further compromise perfusion of the PR layer, leading to BALAD.[27] Choroidal rupture in ocular trauma cases may be another contributory factor.[9,27] Although cone PR degeneration has been proposed as an additional pathogenetic mechanism, this hypothesis does not explain why BALAD was found outside the posterior pole in a few cases.[3,28,29]

Multimodal Imaging Features of Balad

BALAD is frequently identified as a circular, well-circumscribed, yellow-grayish foveal elevation surrounded by a hypopigmented parafoveal ring resembling SRF in color fundus images. The foveal zone is the most common location for BALAD, followed by the parafoveal region, and then the peripapillary region.

On OCT, BALAD appears as an intraretinal cystic space secondary to a split of the hyporeflective MZ with a hyper-reflective granular band as the anterior border, and a line of variable thickness and reflectivity, which is the continuation of the EZ as the posterior border. The granularity of the anterior border or "ceiling" may result from regenerating PR IS and OS. Below the posterior border or "floor" of BALAD, a second hyper-reflective band continuous with the IZ may be prominent. This hyper-reflectance has been attributed to disordered mitochondria of PR ISs attached to the underlying RPE, residual myoid fragments or outer PR segments; or signal attenuation of the overlaying fluid.[17] ELM is seen anterior to the ceiling of BALAD.

BALAD is frequently associated with subretinal and, in rare cases, intraretinal fluid. It may be accompanied by intralesional hyper-reflective particles, and the reflectivity of intra-BALAD space is higher compared with that of SRF, presumably due to fibrin, PR debris, or inflammatory material it contains.[6,18] In certain cases of ocular trauma or CNV, intrabacillary layer hemorrhage is evident and appears as a hyper-reflective material with or without underlying shadowing.[19,27] In some cases, vertical septae within the BALAD cavity are visible.[7] It is hypothesized, based on histologic evidence, that the these septae may reflect potentially migrating mitochondria.[28] The great majority of patients also have increased subfoveal choroidal thickness.

En face SD-OCT reveals hyper-reflective circles containing hyper-reflective fluid within BALAD cavities. On cross-sectional and en face OCTA, the BALAD core has a variable degree of reflectivity, whereas the BALAD border is hyper-reflective.[29]

Fundus autofluorescence reveals a hypoautofluorescent BALAD due to RPE autofluorescence masking caused by intra-BALAD exudation and RPE disruption.[29] The near-infrared reflectance of BALAD lesions shows a hyporeflective zone surrounded by a hyper-reflective ring.

Fluorescein angiography (FA) demonstrates hyperfluorescent dye pooling within the BALAD cavity, which is comparable to the pattern seen with serous pigment epithelial detachment. Late-phase hypofluorescent boundaries on FA correspond to the yellow-colored border in fundus pictures and a hyper-reflective margin on near-infrared reflectance.[29,30] There is no permeability or leakage on FA, such as cystic degeneration or a macular cyst and retinal schisis;

therefore, FA is a valuable tool for differential diagnosis. BALAD has no distinctive features on indocyanine green angiography, but it gives findings of primary pathology in which BALAD is seen together.

Ocular Pathologies with Balad and the Clinical Course

In their retrospective literature review published in November 2021, Ramtohul et al.[17] documented 164 cases of BALAD associated with 22 distinct ocular diseases. They identified the most prevalent three of these diseases as VKH (47%), APMPEE (11%), and SO (7.3%). According to this article, 41 distinct OCT terminologies are used to describe BALAD-like lesions. Rapid regression of BALAD was found in 77 eyes during the course of 10 (1–48) days on average. In 99 eyes with extended follow-up, progressive restoration of the EZ followed by the interdigitation zone was noted. All eyes had permanent focal disruptions of the ellipsoid and interdigitation zones, although they had no functional importance. With careful inspection of OCTs, the prevalence of reported BALAD instances has grown since its discovery, notably in AMD and central serous chorioretinopathy (CSCR) patients. Based on the existing literature published until July 20, 2022, Table 1 shows a list of ocular disorders related to BALAD.

Table 1. Ocular diseases with BALAD and the number of patients and affected eyes reported in the current literature*

Disease	Patients (n)	Eyes (n)
Vogt-Koyanagi-Harada disease	93	147
Age-related macular degeneration	68	68
Central serous chorioretinopathy	15	15
Ocular trauma	7	7
APMPPE	7	7
Uveal melanocytic proliferation	3	5
Acute idiopathic maculopathy	4	4
Serpiginous choroiditis	4	4
Toxoplasma retinochoroiditis	2	2
Macular telangiectasia type 2	2	2
Choroidal metastases	2	2
Drug induced	2	2
Pachychoroidal diseases	2	2
Choroidal granuloma	2	2
Sympathetic ophthalmia	2	2
Posterior scleritis	1	1
Endophthalmitis	1	1
Choroidal osteoma	1	1
Pathologic myopia	1	1
Carotid-cavernous fistula	1	1
Preeclampsia	1	1
Retinal vein occlusion	1	1

APMPPE: Acute posterior multifocal placoid pigment epitheliopathy, *includes original articles, case reports, and case series published until July 20, 2022.

The frequency of BALAD was found to be 94.9% in the study by Agarwal et al.,[6] which included eyes with acute VKH disease. BALADs were totally resolved in all eyes after 3.4±1.3 days of high-dose intravenous methylprednisolone administration. The SRF took longer to dissolve. At the end of the follow up period, ELM and RPE integrity had been restored in all eyes, and that thinning and granularity of the EZ/IZ had been detected in 57.2% of the eyes. As there is no permanent functional damage to the PRs, which preserve the capacity to repair the outer segment by producing new disc membranes, the recovery in visual acuity (VA) is reported to be rapid.[13] In the study by Atas et al.,[31] BALAD was detected in 57% of 85 eyes with VKH disease and was found significantly more common in patients with a SRF height of over 500 µm at baseline. The VA was significantly lower in patients with both BALAD and SRF than in the ones without BALAD at the beginning, but the final VA was not different between these two groups. Although not statistically significant, EZ attenuation and loss of RPE integrity were more common during the final visit in patients who had BALAD at baseline. In a case report of VKH disease, BALAD recovered within 2 weeks, but it took 6 weeks for SRF to completely resolve.[32] Figure 2 shows the multimodal imaging findings of a patient with VKH disease with bacillary detachment.

In the study by Yordi et al.[33] that revealed the results of the post hoc analysis of the ORPSREY trial, 7.4% of 81 eyes with nAMD were found to have BALAD. Compared to eyes without bacillary detachment, these eyes had higher fluid volumes, increased central subfield thickness, EZ attenuation, and increased sub-RPE volume at baseline. In a recently published cohort study of 442 patients, the incidence of BALAD in patients with nAMD was 4.5%.[34] In this study, all BALAD lesions completely resolved following anti-VEGF treatment. In addition, the visual results of BALAD patients was generally favorable, with 80% exhibiting increased or maintained VA. Ramtohul et al.[29] observed that BALAD disappeared in all eyes after anti-VEGF therapy in 30 eyes with treatment naive nAMD. In 83% and 50% of the patients, respectively, there was focal attenuation of EZ-IZ reflectivity and the absence of the ELM. Three (10%) individuals had recurrent BALAD, which resolved after a single intravitreal anti-VEGF injection. Jung et al.[22] diagnosed nAMD in 11 of 14 eyes with macular neovascularization (MNV) and BALAD, and anti-VEGF treatment resulted in regression of findings in all 11 eyes and enhanced VA in eight of them. The visual loss seen in three eyes could be attributed to the presence

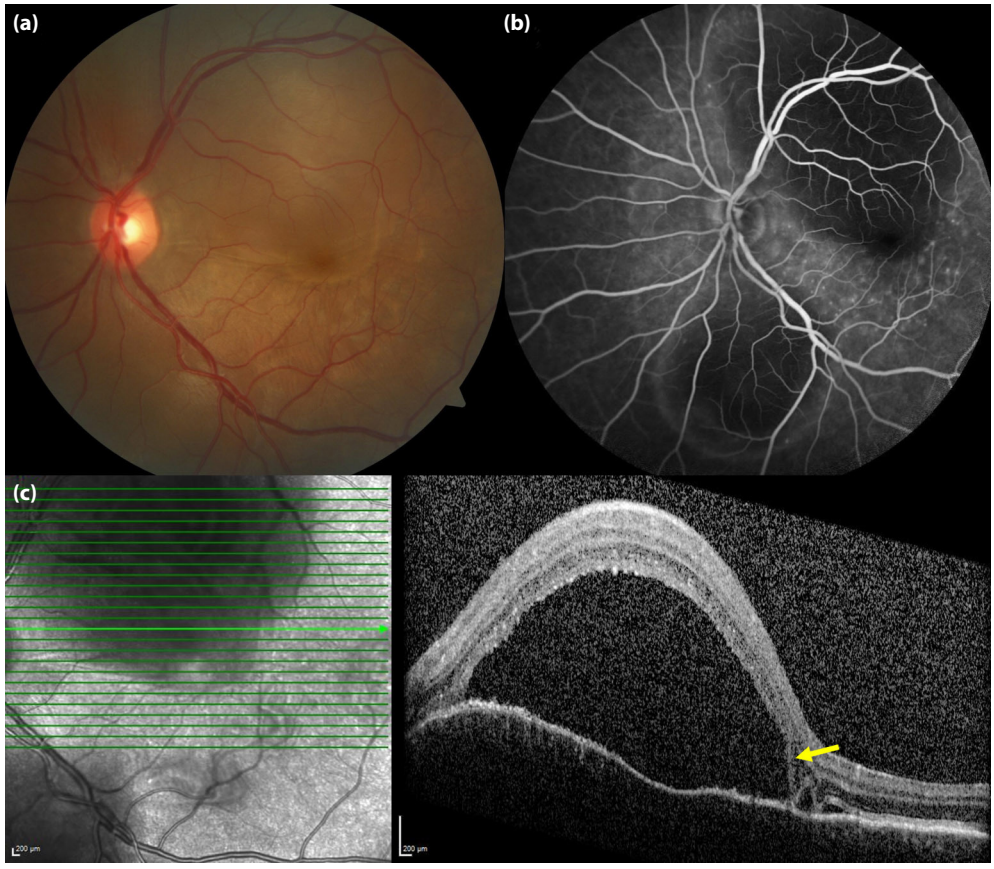

Fig. 2. Multimodal imaging in a patient with acute VKH. **(a)** The fundus photograph of the left eye reveals a hyperemic optic disk and deep yellow choroidal lesions with areas of the SRD in the peripapillary region. **(b)** The midphase FA of the left eye shows multiple hyperfluorescent pinpoint foci of leakage, and large hypofluorescent areas corresponding to the SRD. **(c)** A horizontal SD-OCT scan of the peripapillary region shows a large cystoid space. The ELM appears as a thin line of hyper-reflectivity near the cystoid space. Posterior the ELM, hyporeflective myoid zone appears to separate to form the BALAD (yellow arrow). (BALAD, bacillary layer detachment; ELM, external limiting membrane; FA, fluorescein angiography; SD-OCT, spectral domain optical coherence tomography; SRD, serous retinal detachment; and VKH, Vogt-Koyanagi-Harada's disease).

of persistent damage to the ELM and significant subretinal hemorrhage, leading to subretinal fibrosis. So far, it has been shown that BALAD lesions respond well to anti-VEGF treatment. However, Venkatesh et al.[26] found a regression in BALAD with anti-VEGF treatment in only one of the two nAMD cases. According to studies examining the prevalence of BALAD in nAMD, Type 2 MNV is present in the great majority of eyes, while Type 1 MNV is less common and Type 3 MNV occurs rarely.[33,34] However, Ramtohul et al.[29] reported that Type 1 MNV was shown to be the most prevalent subtype in their study. Jung et al.[22] hypothesized that the subretinal position of the Type 2 neovascular complex in close proximity to the outer retinal PRs may impact the formation of BALAD. Moreover, Type 2 MNV exudation may extend directly into the PR layer and split it.

Fernández-Vigo et al.[23] observed BALAD in 46% of chronic CSCR patients undergoing PDT treatment. They discovered that the rate of subretinal fibrin formation in the BALAD group was significantly higher than in the control group. However, no significant difference in VA change was identified across the groups. BALAD recovered completely and quickly in all cases within 1 month. Multimodal imaging findings of a patient with CSCR with BALAD are shown in Figure 3.

The hemorrhagic variant of BALAD was first reported in a case of MNV associated with macular telangiectasia Type 2 and referred to the presence of heme within the BALAD cavity.[19] Differentiation between hemorrhagic BALAD and a "true" submacular hemorrhage is important because the latter may require additional surgical treatment, such as pneumatic displacement.

Although the clinical course of BALAD varies depending on the accompanying disease, it is quite benign in most cas-

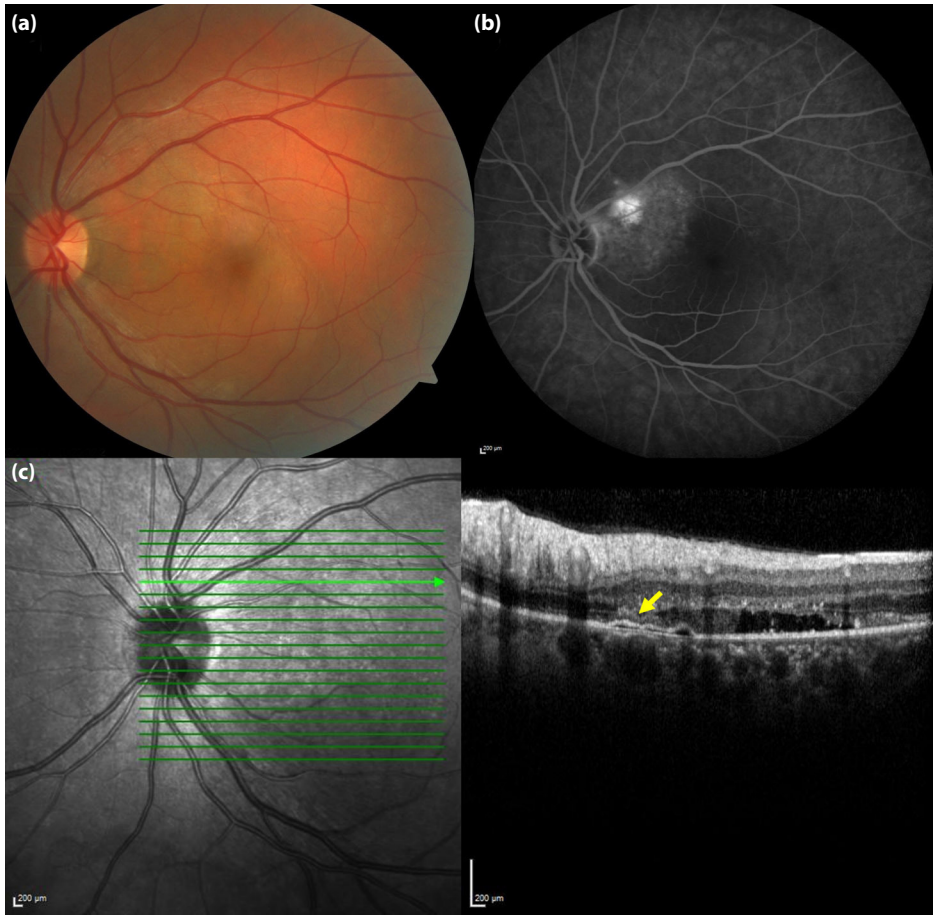

Fig. 3. Multimodal imaging in a patient with acute CSCR. **(a)** The fundus photograph of the left eye shows a relatively normal looking fundus. **(b)** Late phase of FA shows focal leakage from choroid to the subretinal space and oval area of SRD in the peripapillary region. **(c)** A horizontal SD-OCT scan of the peripapillary region shows an intraretinal cystoid space containing fluid admixed with the hyper-reflective material. The ELM is visible anterior to this cystoid space while BALAD (yellow arrow) is seen within it. (BALAD, bacillary layer detachment; CSCR, central serous chorioretinopathy; ELM, external limiting membrane; FA, fluorescein angiography; SD-OCT, spectral domain optical coherence tomography; and SRD, serous retinal detachment).

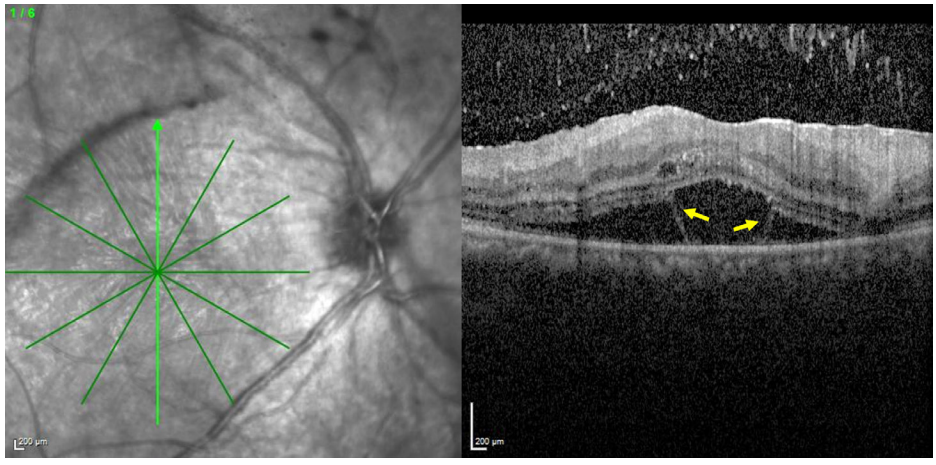

Fig. 4. A radial SD-OCT scan of a patient with Behçet's disease shows BALAD (yellow arrows) lesions formed by splitting of the hyporeflective myoid zone on both sides of the intraretinal cystoid space. (BALAD, bacillary layer detachment and SD-OCT, spectral domain optical coherence tomography).

es. According to the previous case reports, BALAD healed spontaneously within 1 week to 1 month in individuals diagnosed with APMPPE.[7,35–37] In patients with traumatic ocular injuries, BALAD recovered within 2–10 days, accompanied by an improvement in VA.[27] BALAD resolved within the 1st week following pulse steroid treatment in a patient with SO.[21] Although it is uncommon, there have been case reports of BALAD in AIM patients, with spontaneous recovery in all cases.[8,18,38] According to a new case report, BALAD can be observed not only in inflammatory and degenerative diseases of the choroid but also in central retinal vein occlusion, when the intraretinal hemostatic equilibrium is altered.[39] In this case report, BALAD spontaneously regressed in a short period of time.

As available OCT data are carefully examined retrospectively, it appears that BALAD will be detected in many more ocular diseases. Figure 4 shows a BALAD lesion on the OCT scan of a patient with Behcet's disease from our clinic.

The presence of BALAD may be important in differential diagnosis of diseases with similar phenotypes. Liu et al.[40] evaluated the OCT characteristics of VKH, CSCR, and posterior scleritis. The presence of BALAD provided more than 95% specificity and 97% positive predictive value in identifying acute VKH.

Differentiation of Balad From SRF

BALAD frequently accompanies SRF and these two entities can be distinguished by their position within the retinal layers and by the reflectivity of the fluid they contain. While the space where the SRF accumulates consists of the PR on the inside and the RPE on the outside; the BALAD occurs within the myoid layer. An amorphous hyper-reflective material, suggestive of fibrin mixed with debris from shed PR IS and OS, is present within the cystic BALAD cavities on OCT, which has a distinct appearance compared with the more hyporeflective SRF.[3,6,22,41]

Prognostic Importance of Balad

In many studies, the spontaneous disappearance of BALAD during follow-up or after treatment has resulted in an increase in VA.[6,41] In cases where there was no increase in VA, this was attributed to permanent changes in EZ, interdigitation zone, ELM, and RPE.[19,33]

According to Ramtohul et al.,[29] BALAD was associated with a relatively high rate of subretinal fibrosis, and the presence of SHRM or hemorrhagic BALAD was related to a greater risk of fibrosis development on long-term follow-up in nAMD patients. The presence of BALAD, with its high concentration of extracellular matrix proteins released in the subretinal area and frequent connection with intraretinal or subretinal hemorrhages, may promote subretinal fibrosis after the fluid is reabsorbed.

Missaka et al.[42] found that BALAD may be a potential biomarker of a worse prognosis in VKH disease as evidenced by changes in fundus such as chorioretinal atrophy, retinal fibrosis, longer EZ resolution time, and worse visual function at full-field electroretinogram in a study of 33 patients with VKH disease followed for at least 1 year.

Conclusion

BALAD is a relatively recent OCT finding and its frequency is increasing with more careful retrospective review of available OCTs in many ocular diseases. Although it is seen primarily in diseases affecting the choroid, it has been reported that it is also seen in conditions affecting mainly the retina. It is mostly benign and regresses spontaneously or rapidly with the treatment. However, more studies required to understand the prognostic significance of its presence.

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