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Conception and features of bacillary layer detachment

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[Abstract] The separation of the outer retinal photoreceptors in patients with toxoplasmosis chorioretinitis was first named bacillary layer detachment (BALAD). It manifests as a split at the level of the photoreceptor inner-segment myoids, creating a distinctive intraretinal cavity on optical coherence tomography. Subsequently, BALAD has been reported by many researchers in different diseases. In the outer retina, the myoid is a relatively weak structure in the photoreceptor inner segments. When the outward force that promotes the attachment of the photoreceptors to the retinal pigment epithelium exceeds the tensile strength of the photoreceptor inner-segment myoids, the myoid zone splits and BALAD occurs. BALAD has unique multimodal imaging characteristics, and its identification can facilitate the diagnosis, detection, and treatment of ocular diseases. This paper reviews the development of BALAD nomenclature, its anatomical structure, pathophysiological mechanism, and multimodal image features.

[Key words] Retinal cone photoreceptor cells; Retinal rod photoreceptor cells; Optical coherence tomography; Retinal detachment; Bacillary layer detachment
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In recent years, with the development of optical coherence tomography (OCT) and the further study of retinal stratification, the hyporeflective area between the external limiting membrane (ELM) and the ellipsoid zone (EZ) has been considered the imaging manifestation of the photoreceptor myoids and named the myoid zone (MZ).[1] Bacillary layer detachment (BALAD) is a new term recently proposed based on OCT images. It is characterized by the separation of the photoreceptor inner-segment myoids, forming a cystic fissure.

In 2018, Mehta et al.[2] proposed an accurate definition of BALAD for the first time in patients with toxoplasma chorioretinitis. Subsequently, BALAD was found in more than 200 eyes with blunt ocular trauma,[3-4] neovascular age-related macular degeneration (nAMD),[5-8] central serous chorioretinopathy (CSC),[9] tuberculous choroidal granuloma,[10] Vogt–Koyanagi–Harada syndrome (VKH),[11-14] acute posterior multifocal placoid pigment epitheliopathy,[15-19] acute idiopathic maculopathy (AIM),[20-21] peripapillary pachychoroid syndrome,[22] macular telangiectasia type 2,[23] bilateral diffuse uveal melanocytic proliferation,[24-25] preeclampsia,[26] and other diseases. Due to the lack of detailed understanding of this new term, more occurrences of BALAD may be undiscovered. The purpose of this review is to clarify the accurate definition of BALAD by analyzing its anatomical basis, pathophysiological mechanism, and multimodal imaging characteristics, in order to deepen the understanding of ophthalmologists and guide clinical work.

1 Development of BALAD nomenclature

When Polyak analyzed the microanatomical structure of the retina, the term bacillary layer was first used to describe the inner and outer segments of photoreceptors.[27] Since then, this terminology has been widely accepted. In 2011, observed on OCT, Spaide et al.[28] corresponded the bacillary layer with some bands of the outer retina containing the MZ, EZ, and outer segments of photoreceptors. Since photoreceptors are long cells spanning multiple anatomical layers—including the outer plexiform layer, Henle's fiber layer, and the outer nuclear layer—the bacillary layer is a more appropriate term than the photoreceptor cell layer, and the preferred term to describe the inner and outer segments.

In 2004, Maruyama et al.[29] first reported the detachment of photoreceptors shown on OCT in patients with VKH and termed it intraretinal fluid in the outer retinal. Subsequently, this pathological imaging feature has been described by different terms in various diseases, but there has been no unified name. For example, Yamaguchi et al.[30] named it subretinal septum in VKH in 2007, Ishihara et al.[31] described it as cystoid space in VKH in 2009, Liakopoulos et al.[32] introduced the term atypical outer retinal fluid in nAMD in 2013, Ouyang et al.[33] named it the huge outer retinal cystoid space, while Lujan[34] termed it subretinal fluid in ocular toxoplasmosis in 2014. In 2018, Mehta et al.[2] reported this imaging manifestation in a case of toxoplasmosis chorioretinitis and defined that the separation occurred at the level of the photoreceptor inner-segment myoids, from which it was named bacillary layer detachment. Subsequently, the new term was widely accepted and abbreviated as basal laminar deposits (BLDs) or BALAD. Ramtohul et al.[27] recommended the abbreviation of BALAD to distinguish it from BLDs; BLDs present in eyes with age-related macular degeneration (AMD).

2 Anatomical basis of BALAD

In the outer retina, the retinal pigment epithelium (RPE) layer, the interdigitation zone (IZ), the photoreceptor outer segments, the EZ, MZ, and ELM are arranged from outside to inside,[28] and the detachment usually occurs in the structurally weak areas relative to the adjacent layers. In the subretinal space, the microvilli of RPE cells tightly adhere to the outer segments of photoreceptors through complicated mechanisms, and the photoreceptor matrix composed of glycosaminoglycans between the outer segments also enhances this adhesion.[35] The individual photoreceptor cell is highly compartmental: the MZ contains organelles involved in protein production such as ribosomes, endoplasmic reticulum, Golgi complex, and few mitochondria, while the EZ has dense mitochondria.[36] Moreover, Müller cells adhere to the photoreceptor inner segments and together form the ELM, which provides a support structure for photoreceptors

and acts as a semi-permeable membrane barrier.[37] By contrast, the structure of the photoreceptor inner-segment myoids is not as strong as that of the ELM and the mitochondria-rich EZ on its sides, which is an inherently weak structure of photoreceptors.

Mehta et al.[2] reported that splitting photoreceptors at the myoid level was a typical histopathological image in AMD eyes. Litts et al.[38] also demonstrated that cones might fall out of inner segments in late-stage nAMD, which was part of the cone degeneration process. The above findings suggest that the inner segments of the photoreceptors are relatively weak structures. In addition, this conclusion is further supported by the consistent observation that the split is located at the MZ level in the OCT images of BALAD.

3 Pathophysiological mechanisms of BALAD

The similar imaging features of BALAD in different diseases suggest an overlap in the pathogenesis of these diseases. Despite the differing etiologies among diseases presenting BALAD, they all mainly involve the choroid and RPE.[20] Therefore, we speculate that choroidal inflammation, trauma, infiltration, or RPE destruction comprise the pathological basis of BALAD formation.

The anatomical basis of BALAD formation is the structural weakness of the inner segments of the photoreceptors; the myoids.[6] When the outward force promoting attachment of the photoreceptor to the RPE exceeds the tensile strength of the myoids, the MZ splits and BALAD occurs. We speculate that the force to split the myoids is derived from the following three aspects. First, since photoreceptors receive nutrients from choroidal capillaries, choroidal hypoperfusion may lead to pressure and the split of the bacillary layer.[15,20] Venkatesh et al.[20] found that fundus fluorescein angiography (FFA) and indocyanine green angiography showed an early choroidal filling defect; therefore, it was speculated that in the acute phase of the disease, impaired choroidal perfusion leads to the division of the bacillary layer, and once the inflammation subsides and choroidal perfusion improves, BALAD disappears. Moreover, our hypothesis was confirmed by OCT angiography (OCTA) findings showing that AIM eyes with BALAD showed a significant reduction in choroidal blood flow signal in the early stage, and the anatomical recovery of the outer retina was correlated with the gradual improvement of blood flow signals inside the choroid.[21] Second, when exudative fluid penetrates the damaged RPE-Bruch's membrane complex, and rapidly flows into the spatial region between the ELM and the ellipsoids, the shear stress generated by sudden intense exudation may cause the split of photoreceptors resulting in BALAD.[6, 8, 39] Ledesma-Gil et al.[9] reported an eruptive increase in subretinal fluid with subfoveal BALAD in patients with CSC after half-dose photodynamic therapy, speculating that the fulminant onset of hyperacute choroidal exudation could not be blocked by the RPE-Bruch's membrane complex and therefore entered the subretinal space. The resulting shear stress exceeded the tensile strength of the photoreceptor inner segments, tearing the MZ or separating it from the EZ. Jung et al.[6] also observed similar intense exudation in patients with BALAD and type 2 macular neovascularization (MNV). Third, shear stress from subretinal hyperreflective material (SHRM) or intrabacillary layer hemorrhage may also cause detachment within the photoreceptor inner segments.[5] The high-pressure hemorrhage caused by the rupture of the neovascularization structure may tear the bacillary layer at the level of the inner-segment myoids and leak into the separated space, resulting in hemorrhagic BALAD.[7] Venkatesh et al.[5] found that SHRM and intrabacillary layer hemorrhage were significantly reduced, and BALAD subsequently disappeared, after anti-vascular endothelial growth factor (VEGF) treatment in patients with nAMD and BALAD, suggesting a potential link between the two.

Furthermore, Ramtohul et al.[8] suggested that pathological adhesion of the photoreceptor outer segments to RPE may be involved in the formation of BALAD, especially when BALAD is associated with subretinal hemorrhage. The presence of hemorrhage stimulates fibrin deposition and interlacing between RPE and the outer segments of photoreceptors, creating a secure adhesion between above two layers, thereby enhancing the outward force of photoreceptor outer segments to attach to the RPE.

4 Multimodal image features of BALAD

In recent years, the disease spectrum of BALAD has expanded, including infectious, inflammatory, vascular, and malignant diseases. However, a comprehensive and detailed understanding of the lesion characteristics of BALAD is still lacking. By multimodal imaging techniques including color fundus photography, OCT, OCTA, FFA, and other examinations, the morphological structure and pathological characteristics of BALAD can be understood more clearly from many aspects to deepen the understanding of its pathogenesis. According to our observations, BALAD has similar imaging features in multimodal imaging of different eye diseases.

On OCT, BALAD shows remarkable characteristics (Figure 1). It is located in the fovea or parafovea, and its incidence in the fovea is much higher than that in the parafovea. Due to the weak structure of the photoreceptor inner-segment myoids, the split is located at the MZ level, forming a unique intraretinal cystic space. The top of BALAD is composed of continuous ELM, which remains intact throughout the disease course. At the base of BALAD, there is a thickened hyperreflective band, which is contiguous with the EZ of the adjacent retina. The EZ, IZ, and photoreceptor outer segments still adhere to the RPE-Bruch's membrane complex. The reflectivity of BALAD cavity contents is higher than that of adjacent subretinal fluid, probably because the BALAD cavity contains detached or regenerated photoreceptor fragments and inflammatory products such as fibrin. BALAD is often accompanied by subretinal fluid. Furthermore, choroidal thickening and loss of standard choroidal vascular structure can also occur, which may be caused by choroidal inflammation and trauma. In addition to similar BALAD manifestations, patients with blunt ocular trauma often show hemorrhage in the bacillary layer simultaneously, which manifest as hyperreflective substances in the BALAD cavity and correspond to dark red hemorrhage lesions shown in fundus color photos.[3-4] Almost all nAMD eyes with BALAD are accompanied by SHRM in the cavity of BALAD, which may comprise components such as effusion, fibers, blood, and neovascularization, suggesting a potential link between the presence of SHRM and the development of BALAD.[5, 7-8]

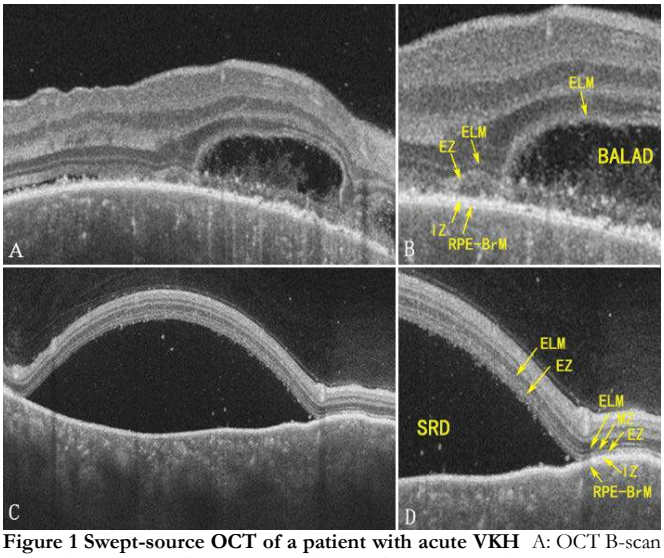

Figure 1 Swept-source OCT of a patient with acute VKH A: OCT B-scan shows the presence of BALAD with hyperreflective material in its cavity B: A magnified view of A shows that BALAD occurs at the level of the hyporeflective MZ, where the ELM above it remains continuous, and the lower EZ, IZ, and photoreceptor outer segments remain adherent to the RPE-Bruch's membrane complex C: OCT B-scan shows a large cystic cavity called SRD D: A magnified view of C shows that all retinal zones remain intact, and fluid accumulates between the retinal neuroepithelium layer and RPE-Bruch's membrane complexes. ELM: external limiting membrane; EZ: ellipsoid zone; IZ: interdigitation zone; RPE: retinal pigment epithelium; MZ: myoid zone; BALAD: bacillary layer detachment; SRD: serous retinal detachment

Ophthalmoscopy or color fundus photography usually shows the presence of subretinal fluid in BALAD eyes, which is manifested as round or irregular yellow lesions at the posterior pole of the retina. The fundus autofluorescence is characterized by a low central autofluorescence with a peripheral hyperautofluorescence ring in the lesion due to exudation and/or RPE destruction. Near-infrared reflection images show a hyperreflective ring at the edge of BALAD, which corresponds to the edge of the lesion demonstrated by ophthalmoscopy or color fundus photography, the hyperautofluorescence ring of fundus autofluorescence, and the BALAD boundary on OCT. En-face OCT images, similar to near-infrared reflection, also show a hyperreflective ring corresponding to the edge of BALAD. FFA is characterized by hypofluorescence in the early stage, indicating poor perfusion. In the late stage of FFA, a large area of hyperfluorescence leakage in the BALAD cavity suggests that there is increased vascular permeability of the retinal and choroidal vessels due to inflammation, trauma, degeneration, or other causes. Indocyanine green angiography shows early hypofluorescent lesions, which may be due to choroidal malperfusion. In addition, OCTA results of patients with nAMD and type 2 MNV suggest subretinal neovascularization,[6,23] while OCTA of eyes with toxoplasmosis chorioretinitis, acute posterior multifocal placoid pigment epitheliopathy, and AIM show reduced capillary blood flow signals,[2, 19, 21] which may be related to the pathological characteristics of the diseases.

5 Clinical significance and the prospect of BALAD

As a unique OCT feature, BALAD can be used to identify, diagnose, and treat ocular diseases. First, BALAD needs to be accurately distinguished from serous retinal detachment (SRD), which has similar OCT features, but different pathological manifestations involving different retinal layers (Figure 1). BALAD describes an intraretinal fluid formed by the detachment of the myoids, leaving the detached myoid fragments, the adjacent EZ, and the rest of the outer segments adhering to the RPE, while the ELM extends forward to form the top of the BALAD. By contrast, SRD is characterized by the accumulation of fluid within the subretinal space.[40] In addition, the reflectivity of the BALAD cavity on OCT is higher compared to the adjacent SRD and is accompanied by suspended hyperreflective particles, which suggests a differing composition in these two fluid compartments.

Agarwal et al.[11] proposed that BALAD could be used as one of the distinguishing features of VKH and CSC. Although these two diseases commonly affect the choroid and RPE, with similar multimodal imaging features, the incidence of BALAD in VKH is as high as 94.9%, much higher than that in CSC. In a study of patients with nAMD in a Korean population, Kim et al.[7] found that the incidence of BALAD in type 2 MNV was significantly higher than that of other types of MNV; therefore, the occurrence of BALAD has specific significance in the identification of different types of MNV. Jung et al.[6] found that intravitreal injection of anti-VEGF drugs could rapidly relieve BALAD and improve visual acuity in most patients with MNV. Ramtohul et al.[8] conducted a long-term visual follow-up of 30 patients with nAMD and BALAD, and found that at 3 months after intravitreal injection of anti-VEGF drugs, BALAD in all eyes had resolved, and the best corrected visual acuity improved significantly. However, after 4 years, the visual acuity returned to the baseline level, and the cumulative risk of subretinal fibrosis was as high as 77%. They speculated that risk factors associated with subretinal fibrosis included the presence of hemorrhagic BALAD and SHRM. Considering that ocular inflammation may play a role in the occurrence of BALAD, treatment with glucocorticoids may also contribute to the early recovery of BALAD and prevent the development of subretinal fibrosis, thereby promoting early and better visual recovery.[3] Agarwal et al.[11] studied 112 eyes of patients with VKH and BALAD, and found that BALAD disappeared rapidly after receiving high-dose glucocorticoid treatment for an average of 3.4 days, and the best corrected visual acuity of the affected eyes improved from 0.96 to 0.4. Given that most current studies are observational and retrospective, more extensive prospective studies may be needed to discuss the exact correlation between anti-VEGF or glucocorticoid therapy, BALAD resolution, and visual acuity.

In conclusion, the identification of BALAD has tremendous clinical significance. The high incidence of BALAD in patients with VKH helps to identify VKH more quickly and accurately, which can provide valuable information for clinical and differentiated diagnosis. Intravitreal injection of anti-VEGF drugs and the use of glucocorticoids may be helpful for the rapid recovery of BALAD and improvement of visual acuity. This finding can guide ophthalmologists in the administration of drug treatment for BALAD. Moreover, hemorrhagic BALAD and SHRM may be risk factors for subretinal fibrosis, suggesting a poor visual prognosis. In addition, the rapid functional improvement and structural recovery of the bacillary layer on multimodal images imply that the photoreceptor inner segments

are able to regenerate rapidly, further guiding the exploration of the photoreceptor regeneration pathway.

With the development of OCT technology, a more precise and intuitive understanding of the morphological characteristics of BALAD has been obtained. However, due to the retrospective nature of BALAD studies, there is still a lack of direct histopathological correlation. In addition, the mechanism of BALAD formation and its relationship with disease progression have not been fully elucidated. Future animal models and the improvement of OCT technology may further help gain a clearer understanding of the retinal changes in BALAD eyes and the specific mechanism of their formation. In conclusion, this paper briefly introduced BALAD as a new OCT term, and elucidated its possible pathophysiological mechanism and multimodal imaging characteristics. However, its relationship with the development of disease needs to be further studied for better clinical application.

Conflict of interests None declared.

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