

# Use of Imaging Modalities in Vogt-Koyanagi-Harada Disease: An Overview

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## SUMMARY

**Aims:** Vogt-Koyanagi-Harada (VKH) disease is an autoimmune disorder affecting multiple systems and characterized by bilateral granulomatous uveitis, frequently together with neurological, auditory, and integumentary manifestations. Prompt diagnosis and intervention are a must to prevent irreversible vision loss and possible systemic complications. Fundus imaging plays a pivotal role in the management of VKH, not only to reach the diagnosis but also to monitor disease activity and response to treatment. Fundus photography, fundus autofluorescence imaging, fluorescein angiography, indocyanine green angiography, and optical coherence tomography (OCT) are among the imaging modalities. These methods provide invaluable insights into the different phases of the disease. In addition, OCT angiography enables clinicians to visualize associated retinal and choroidal microvascular changes. Continuous advances in imaging technology assist ophthalmologists to comprehend the VKH pathophysiology and to facilitate its differential diagnosis. Thus, clinical outcomes are improving with timely interventions and personalized treatment approaches. This mini-review provides an overview of VKH disease, with a particular focus on the fundus imaging techniques utilized in its management.

**Key words:** fluorescein angiography, indocyanine green angiography, melanocyte-associated antigens, optical coherence tomography, optical coherence tomography angiography, panuveitis, Vogt-Koyanagi-Harada disease

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## INTRODUCTION

Vogt-Koyanagi-Harada (VKH) disease is a rare autoimmune condition that primarily targets the ocular structures. However, it can also significantly impact various other systems, such as the auditory, central nervous, and integumentary systems (skin and hair) [1–6]. Individuals of pigmented races, such as Asians, Hispanics, Indians, Native Americans, or those of Mediterranean origin, are more prone to develop VKH disease, accounting for 7% to 22.4% of uveitis referrals [2–4]. In addition, females are more susceptible to this disease when compared to males [3].

VKH pathogenesis is believed to involve an autoimmune mechanism in which T-cells target the tyrosinase family peptides (TYR, TRP1, and TRP2) of melanocytes. The specific trigger remains unclear, although instances of cutaneous injury or viral infections have been iden-

tified as potential contributors in some cases [3,4,7]. Moreover, specific HLA genotypes, such as HLA-DR1 and HLA-DR4 (subtypes 0405 and 0410), have been linked to the increased susceptibility to VKH disease, implying a genetic predisposition [3,4,7]. The immune response leads to a cascade of inflammatory events that ultimately results in damage to melanocyte-rich tissues in the eyes, auditory system, central nervous system, skin, and hair [2,3,7].

Patients with VKH disease typically present with bilateral granulomatous panuveitis, frequently accompanied by additional symptoms, including tinnitus, hearing loss, meningismus, and vitiligo. The disease often manifests with a preceding influenza-like illness, followed by a constellation of symptoms such as headache, ocular pain, and dizziness. Ocular pain is frequently followed by the rapid onset of vision loss in one or both eyes, either simultaneously or sequentially [1–4,7]. The ocular features

include serous retinal detachments, choroidal thickening, and optic disc swelling. Furthermore, the disease may exhibit extraocular manifestations, such as hearing loss, skin depigmentation, and hair loss, which may occur prior to or subsequent to ocular symptoms and contribute to the systemic nature of the disease [3]. VKH disease is characterized by four distinct clinical phases [1–3]. In the prodromal phase, patients exhibit neurological and auditory symptoms, in addition to lymphocytic pleocytosis in the cerebrospinal fluid. The acute uveitic phase is defined by diffuse choroiditis, which can result in papilledema and exudative retinal detachment, with the potential progression to panuveitis. Following the resolution of overt intraocular inflammation, the convalescent phase may manifest as subclinical choroidal inflammation, fundus depigmentation and integumentary changes, such as vitiligo, alopecia and poliosis [1–3]. The above manifestations may occur if prompt and adequate treatment is withheld during the acute uveitic phase. In the chronic recurrent phase, the disease exhibits an uncontrolled chronic recurrent granulomatous anterior uveitis. In some cases, this is accompanied by relapsing exudative retinal detachment [1–3].

The diagnosis of VKH is based on the clinical criteria. These include a combination of ocular and systemic findings [7]. Ancillary tests, including fundus autofluorescence (FAF), fluorescein angiography (FA), indocyanine green angiography (ICGA), optical coherence tomography (OCT), OCT-angiography (OCTA), and cerebrospinal fluid analysis, may be employed in order to further elucidate the diagnosis or the differential diagnosis. It is of paramount importance to distinguish VKH from other etiologies of uveitis and meningitis, as this is the key for optimal management [3,4,7]. The primary therapeutic approach to VKH is the administration of systemic steroids to control inflammation. In some instances where corticosteroids are inadequate or contraindicated, immunosuppressant agents, such as cyclosporine, azathioprine, or mycophenolate mofetil, can be administered. Moreover, initiation of immunosuppressive and/or biological agents is mandatory for the prevention of recurrences after the initial systemic steroid therapy [3]. It has been demonstrated in numerous studies that prompt initiation of treatment is crucial for effective management of initial-onset VKH [1–4,7]. The term “window of therapeutic opportunity” in the context of immune-mediated diseases refers to the period immediately following the disease onset, during which appropriate intervention can significantly alter the disease’s course and potentially achieve remission [1]. It can be postulated that the critical period for VKH may be between two to four weeks after onset, though it is probable that the optimal window is closer to two weeks in more severe cases. Thus, prompt and aggressive treatment is crucial to prevent complications and to preserve visual function [1–4,7].

The aim of this mini-review is to provide an overview of VKH disease, with a particular focus on the fundus imaging techniques employed in its management.

## METHODS

A comprehensive search of the relevant literature was conducted on the PubMed/MEDLINE database up to 2025, using the key words “Vogt-Koyanagi-Harada disease,” “melanocyte-associated antigens,” “panuveitis,” “fundus autofluorescence,” “optical coherence tomography,” “fluorescein angiography,” “indocyanine green angiography,” “optical coherence tomography angiography” and “retromode infrared scanning laser ophthalmoscopy” to identify the studies investigating the use of various fundus imaging modalities both in the diagnosis and monitoring of patients with VKH disease. Relevant studies were identified in the initial search, and further articles were selected by carefully examining the references in the included studies. It is important to note that the final selection of articles for this review was limited to those that were published in the English language.

## RESULTS

An extensive analysis of 45 articles selected from the initial search and examination of the reference lists of the included studies was performed. These publications were evaluated by three senior ophthalmologists (O.K., Z.A., A.O.S.), who carefully reviewed all selected articles. This was followed by a discussion of these studies and a comparison of their results.

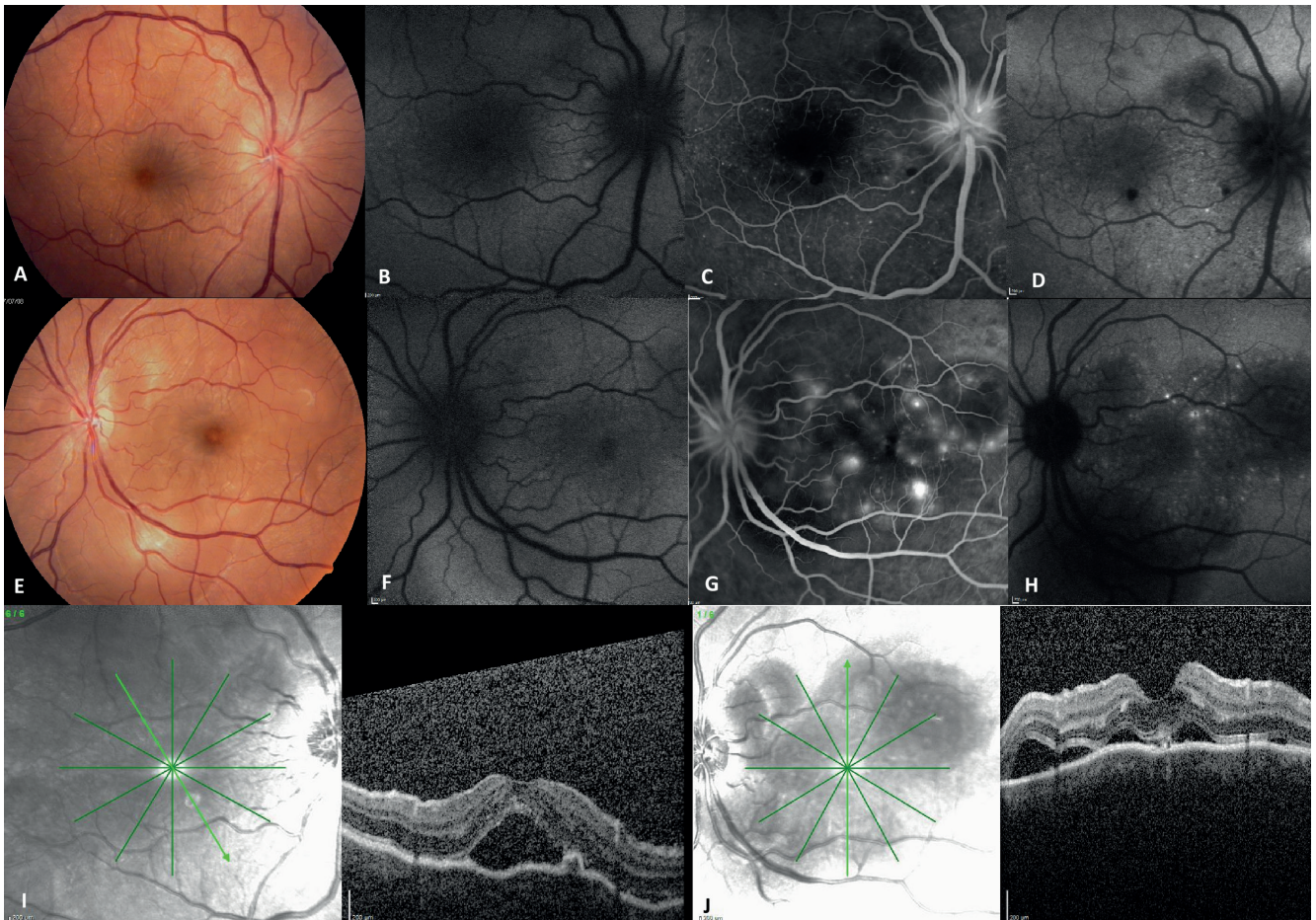
## DISCUSSION

Multimodal fundus images that may be obtained in various stages of VKH disease are outlined for each imaging method in detail, under the subheadings seen below. Examples of multimodal fundus images depicting the acute and chronic disease stages are shown in Figures 1 and 2.

### Current Fundus Imaging Techniques in Patients with Vogt-Koyanagi-Harada Disease

#### A. Color Fundus Images

In the prodromal phase, fundus examination is usually unremarkable and does not provide any diagnostic information. However, during the acute uveitic phase, up to 70% of patients may develop sudden onset bilateral granulomatous uveitis, characterized by subretinal fluid accumulation and choroidal thickening [3]. Initially, choroidal thickening occurs as multifocal inflammatory segments, leading to the disruption of the retina pigment epithelium (RPE) and serous retinal detachment (sRD). Other clinical signs may include retinal edema, optic disc swelling, and hyperemia [3]. The convalescent phase typically occurs several weeks or months after the acute uveitic phase. This phase may last for several months. It is characterized by the presence of Dalen-Fuchs-like nodules and



**Figure 1.** Multimodal fundus images of a 45-year-old female patient presenting at the acute stage of Vogt-Koyanagi-Harada disease. Color fundus photograph of right (A) and left (E) eyes revealing the optic disc edema and multiple foci of serous retinal detachment (SRD) at the posterior pole. Fundus autofluorescence imaging of the right (B) and left (F) eyes depicting the hypoautofluorescence in regions corresponding to the SRD and hyperautofluorescence in the adjacent regions. Fluorescein angiography of right (C) and left (G) eyes showing multiple pin-point hyperfluorescent leaks, pooling, and disc hyperfluorescence. Indocyanine green angiography of right (D) and left (H) eyes showing numerous hypofluorescent dark dots, and hyperfluorescent pinpoints at posterior pole. Optical coherence tomography of right (I) and left (J) eyes illustrating subretinal fluid compartments delineated by septa, and choroidal/retinal pigment epithelium folds

choroidal depigmentation [3,4]. Choroidal depigmentation typically occurs within two to three months and the bright orange-red hue of the fundus is commonly referred as the “sunset glow” appearance. Sunset glow fundus (SGF) has a 94.5% positive predictive value and an 89.2% negative predictive value for diagnosing VKH in patients presenting with uveitis [3]. The interval between the initial manifestations and the subsequent SGF appearance is notably shortened in patients with chronic inflammation, when compared to those without. In some patients, chronic, recurrent intraocular inflammation develops [3]. This is characterized by episodes of granulomatous anterior uveitis that is often resistant to systemic steroid therapy. Typically, this chronic recurrent phase occurs between six to nine months following the initial presentation and is associated with various complications, including RPE proliferation, subretinal fibrosis, and macular neovascularization (MNV). In addition, relapses may be accompanied by exudative retinal detachments [3].

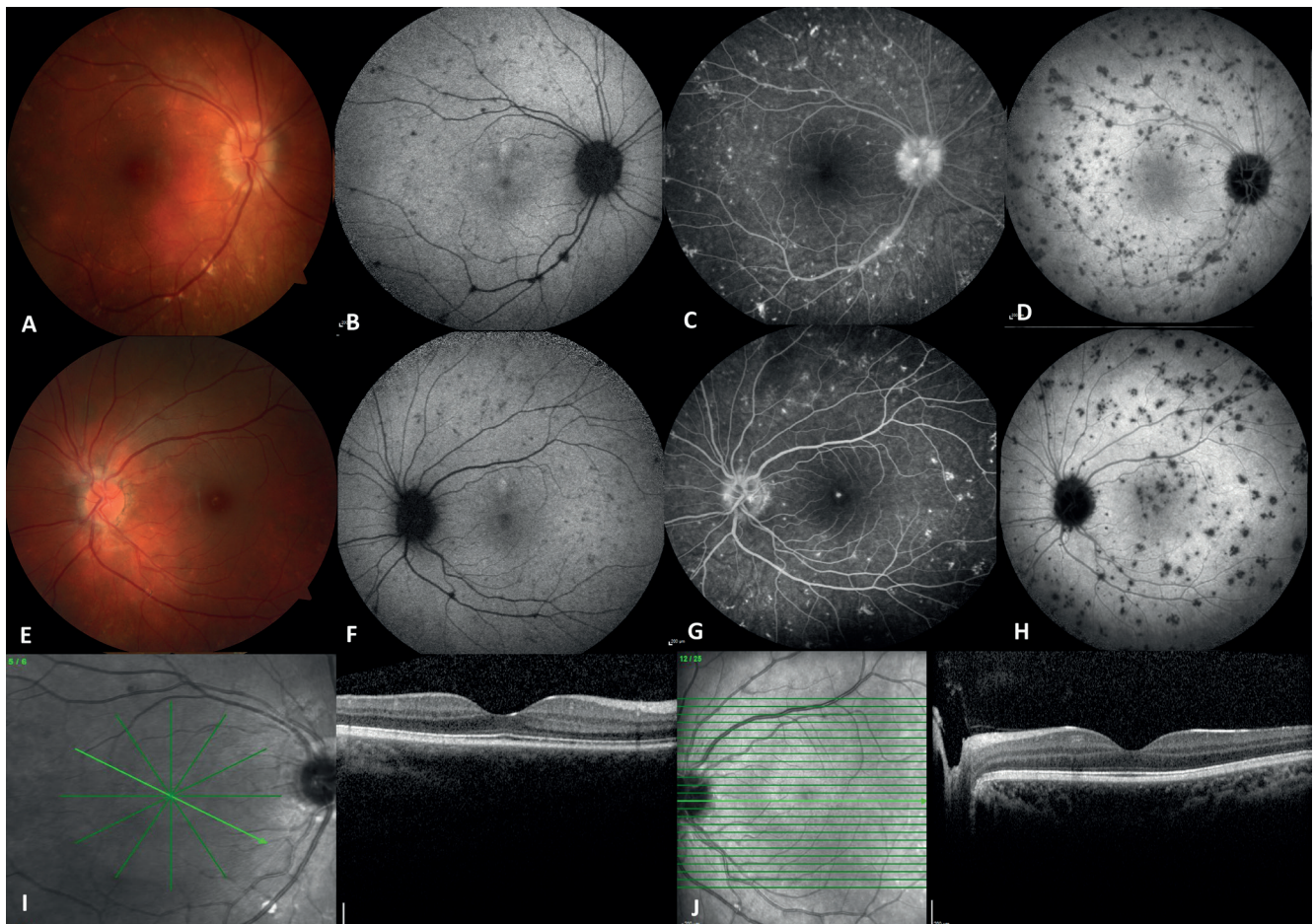
## B. Fundus Autofluorescence

FAF offers a promising and essential method for non-invasive, in vivo visualization of the RPE and outer retina [8,9]. FAF provides insights into the functional and metabolic changes within the RPE, highlighting the presence of lipofuscin (blue light-adapted fundus autofluorescence; BL-FAF) and melanin and its derivatives (near-infrared fundus autofluorescence; NIR-FAF) [10]. The FAF signal in VKH disease reflects the lipofuscin and other fluorophore accumulation within the RPE and outer retina. This distinctive quality enables the functional evaluation of the RPE layer, offering insights that transcend the scope of traditional imaging modalities, such as fundus photography, FA, and OCT [9]. FAF imaging offers the potential for enhanced demonstration of atrophic retinal areas and improved visualization of subclinical adjacent RPE injury. This may be of benefit in situations where there is an early indication of injury, prior to development of visible lesions [3,9]. In the

acute uveitic phase, FAF imaging often reveals diffuse hyperautofluorescence, with occasional interruption caused by the areas of blockage resulting from the presence of subretinal fluid [3]. These abnormalities typically resolve within six months following the administration of high-dose intravenous steroids. Nevertheless, patients presenting weeks after the initial symptoms display a mixed autofluorescence pattern of diffuse and mottled fluorescence, interspersed with regions of hypoautofluorescence, corresponding to the areas affected by exudative retinal detachments. By six months post-treatment, these areas typically exhibit hypoautofluorescent dots [3,11]. In a study by Koizumi et al. [11], it was observed that in ten eyes of five patients with acute VKH disease, the hypoautofluorescent regions identified through both BL-FAF and NIR-FAF imaging corresponded to the regions of serous retinal detachment. Following the resolution of subretinal fluid, FAF revealed the presence of placoid hyperautofluorescent regions at the macula and peripapillary area, which

correlated with the hypofluorescence observed on ICGA. Patients who received initial pulse corticosteroid therapy demonstrated the resolution of hyperautofluorescent areas, whereas those with delayed treatment exhibited persistent macular hyperautofluorescence, potentially due to changes which occurred in melanin and lipofuscin distribution [11].

Patients who present during the late acute phase without prior treatment may display varying patterns on FAF imaging, including areas of hyperautofluorescence, hypoautofluorescence, and lattice-like patterns [3,9,12,13]. These patterns have been observed in the chronic phase, where they can exhibit patterns of increased, decreased, or normal autofluorescence. The pattern of decreased autofluorescence is indicative of RPE loss and involvement of the outer retina in the course of disease progression. The peripapillary atrophy and nummular chorioretinal scars typically manifest as areas of decreased fluorescence. Conversely, an increased autofluorescence pattern is associated with the development of cystoid



**Figure 2.** Multimodal fundus images of a 21-year-old female patient at the chronic stage of Vogt-Koyanagi-Harada disease. Color fundus photographs of the right (A) and left (E) eyes showing a sunset glow fundus appearance with a widespread peripheral scattered yellowish-gray spot-like aggregates. Fundus autofluorescence imaging of the right (B) and left (F) eyes exhibiting the patterns of increased and decreased autofluorescence. Fluorescein angiography of the right (C) and left (G) eyes showing the staining of spot-like lesions without leakage corresponding to the aggregates seen on color fundus photographs. Indocyanine green angiography of the right (D) and left (H) eyes demonstrate the widespread hypofluorescent dark dots. Optical coherence tomography of the right (I) and left (J) eyes illustrating a normal appearance with preservation of the foveal contour

macular edema, subretinal fibrosis, and the presence of areas of RPE proliferation. However, the presence of a SGF does not necessarily correlate with the abnormalities observed on FAF. Given its correlation with RPE health, FAF is an effective modality in assessing recurrent and subclinical inflammation, as supported by the use of wide-field imaging [3,9,14].

### C. Fluorescein Angiography

Although FA is unable to detect choroidal inflammation during the acute exudative phase, it can reveal inflammation spreading from the choroid into the retina and optic disc [4]. Two distinct angiographic patterns can be visualized in patients with VKH disease. The first pattern is indicative of active inflammation, whereas the second pattern is indicative of the subsequent scarring phase [15,16].

In the acute stage of VKH disease, two notable angiographic findings are exudative retinal detachment and optic disc hyperfluorescence. The following signs are characteristic of this stage: focal areas of delayed choroidal perfusion and choroidal folds, which manifest as hypofluorescent lines emanating from the optic nerve. Multiple hyperfluorescent pinpoint indicating leakage, progressive subretinal pooling delineating the extent of exudative retinal detachment, optic disc hyperfluorescence, and vascular hyperfluorescence are among the common angiographic findings [17,18]. These angiographic patterns correspond to the presence of papillitis, choroiditis, and serous retinal detachment, which overall define the acute inflammatory stage of VKH syndrome. In this stage, the diffuse choroidal inflammation typically spares the choriocapillaris, while involving the inflammation of medium-sized vessels [4,15–19]. In the hyperacute phase, peripapillary hyperfluorescent pinpoints may be detected. In the absence of this sign, it may be inferred that the FA was conducted at a subsequent phase of the disease, necessitating a course of immunosuppressive therapy that should be more serious and prolonged. The occurrence of pinpoint peripapillary hyperfluorescence is more frequent in eyes that subsequently show resolution, than in eyes that progress to chronic disease [4,15].

In the chronic phase, FA reveals window defects in areas where RPE cells are lost, or a fluorescence-blocking effect in areas of pigment clumping from destroyed RPE cells. FA can clearly delineate the boundaries of the exudative detachments during the acute phase following the reattachment, that are known as “high water marks”. These manifest as hyperpigmented lines and disc hyperfluorescence [4,13]. This phase is distinguished by a “salt and pepper” fundus appearance, which is characterized by spotted hyperfluorescence and hypofluorescence. This phenomenon indicates the onset of chorioretinal scarring processes [16]. Furthermore, FA can assist in the diagnosis of various complications, including arteriovenous and retinochoroidal anastomoses, as well as neovascularization in the optic disc and choroid [4,16]. In

addition to conventional angiography, ultra-wide-field fluorescein angiography (UWF-FA) can provide valuable data in patients with VKH disease. In a recent study, Kim et al. [19] described the findings of UWF-FA in acute VKH disease. UWF-FA identified three distinct abnormal findings in the central fundus area (CFA) compared to the peripheral fundus area (PFA). Focal leakage was observed in 92.3% of cases in the CFA versus 76.9% of cases in the PFA. Pooling with a dark rim was noted in 84.6% of cases in the CFA versus 53.8% of cases in the PFA. Retinal vascular leakage occurred in 0% of cases in the CFA versus 46.2% of cases in the PFA. Consequently, UWF-FA may potentially yield a more extensive dataset than conventional FA. This enhanced capability may offer deeper insights into the underlying pathogenic mechanisms, rendering UWF-FA a valuable tool for assessing treatment responses and prognosis [19].

### D. Indocyanine Green Angiography

ICGA is considered a gold standard for the evaluation and follow-up in VKH disease, due to its precision in assessing choroidal inflammation [4]. Currently, a considerable number of medical facilities employ the use of ICGA for the initial evaluation and monitoring in VKH patients [20–22]. Hypofluorescent dark dots (HDDs) are the most notable ICGA finding in VKH disease. This results from the impaired diffusion of the ICG molecule within the choroidal stroma, due to a consequence of space-occupying lesions. Their visibility is most pronounced during the intermediate and late phases of ICGA [20–22]. Correlation between the histopathological data from choroidal sections and the HDDs suggests that these images reflect the space-occupying lesions, or granulomas [20,22,23]. The granulomas may be of various sizes [20]. Some may be relatively small, not occupying the entire thickness of the choroidal stroma, while others may be extensive, filling the space from the sclera to the choriocapillaris. The presence of HDDs is a paramount indicator for the identification of subclinical choroidal disease and grading the response to treatment. It is crucial to acknowledge that the presence of dark dots does not invariably signify the presence of active granulomas. They may also be the sign of stromal scarring [20,22]. To ascertain whether persistent dark dots are related to active inflammation or scarring, a repeat ICGA can be conducted following a period of approximately three to four weeks, during which aggressive inflammation-suppressive therapy is administered. In the event that there is minimal or no change in the hypofluorescent areas, the dark dots are assumed probably to be due to stromal scarring [20,22]. Furthermore, additional reliable ICGA markers for the assessment and monitoring include hyperfluorescent choroidal vessels, fuzzy vascular patterns of large stromal vessels, late diffuse hyperfluorescence, and disc hyperfluorescence. These ICGA findings could be observed in nearly all new, untreated cases [22,24]. The presence of early hyperfluorescence and leakage from choroidal stromal vessels

indicates the presence of severe choroidal stromal inflammatory vasculopathy. This marker is commonly observed in the acute and severe stages of VKH disease and typically diminishes over time following the initial treatment or within the first three to six months of therapy, contingent on the severity of choroidal inflammation. This angiographic marker appears at an early stage and persists until it is replaced by diffuse late hyperfluorescence [20]. The fuzzy vascular patterns of large stromal vessels during the intermediate to late intermediate angiographic phase is an indication of diffuse inflammatory vasculopathy of the stromal vessels. In the late phase, this is followed by diffuse stromal hyperfluorescence [17,20]. Diffuse late stromal hyperfluorescence is frequently observed during the acute and subacute disease phases. Another important marker is disc hyperfluorescence. In the absence of pathological conditions, the optic disc remains dark and non-fluorescent in ICGA. However, the presence of disc hyperfluorescence is indicative of a particularly severe form of the disease. This sign typically regresses rapidly with the initiation of therapy, making it a valuable indicator for assessing the response to initial high-dose inflammation-suppressive treatment [20,22]. Other findings on ICGA include irregular filling patterns, which indicate a disturbance or delay in the early choriocapillaris circulation during the initial angiographic phase. Hyperfluorescent pinpoint and exudative subretinal hyperfluorescence are prominent features. Particularly in cases with acute and severe inflammatory disease, leakage points at the level of the RPE can be observed not only on the FA but also on ICGA. In some cases, subretinal fluid may also be identified on ICGA [20]. In patients with recurrent episodes of VKH disease, the same clinical signs may reappear that are observed in the initial phase of the disease. In contrast, in chronic smoldering disease, only HDDs and fuzzy, indistinct choroidal vessels indicate the presence of ongoing occult choroidal inflammation, with no other clinical signs present at this stage. This subclinical progression can explain the development of SGF due to suboptimal therapy. The SGF, which is often considered to be the natural course of the disease, is in fact the result of insufficiently treated inflammation. ICGA can be helpful during the subacute and convalescent stages to optimize therapy and thereby to prevent the development of HDDs [20,22].

### E. Optical Coherence Tomography

Spectral-domain OCT (SD-OCT) is a valuable imaging modality due to its noninvasive nature. Current OCTs detect even subtle exudative detachments located at the posterior pole extremely well, but in order to see changes extending beyond the posterior pole, ICGA can be a more suitable modality to view those changes [4,25].

In the acute phase of VKH disease, SD-OCT reveals several distinctive and diagnostic features, such as serous retinal detachment and the presence of subretinal fluid compartments, delineated by septa formed from the

inflammatory products such as fibrin. Furthermore, the presence of choroidal/RPE folds is frequently observed in patients with acute initial-onset VKH, which indicates a more severe disease state and prolonged inflammation in the choroidal level. These characteristics are of critical importance for the accurate diagnosis of VKH disease in its early stages [4,26]. These septa are typically regarded as hyperreflective membranous structures with OCT technology. They separate from the retina anterior to the RPE-Bruch's membrane complex and form a bridge with the retina. This anatomical feature observed on SD-OCT is referred to as "bacillary layer detachment" [4,26,27]. In the chronic inactive phase of VKH disease, structural alterations can be observed, including focal parafoveal outer nuclear layer atrophy, disruption of the ellipsoid and interdigitation zones, and an irregularly thickened RPE line [28]. SD-OCT is also capable of detecting signs of subclinical inflammation, including macular edema and early macular complications. In addition, SD-OCT is also very valuable in monitoring disease remission following treatment [4].

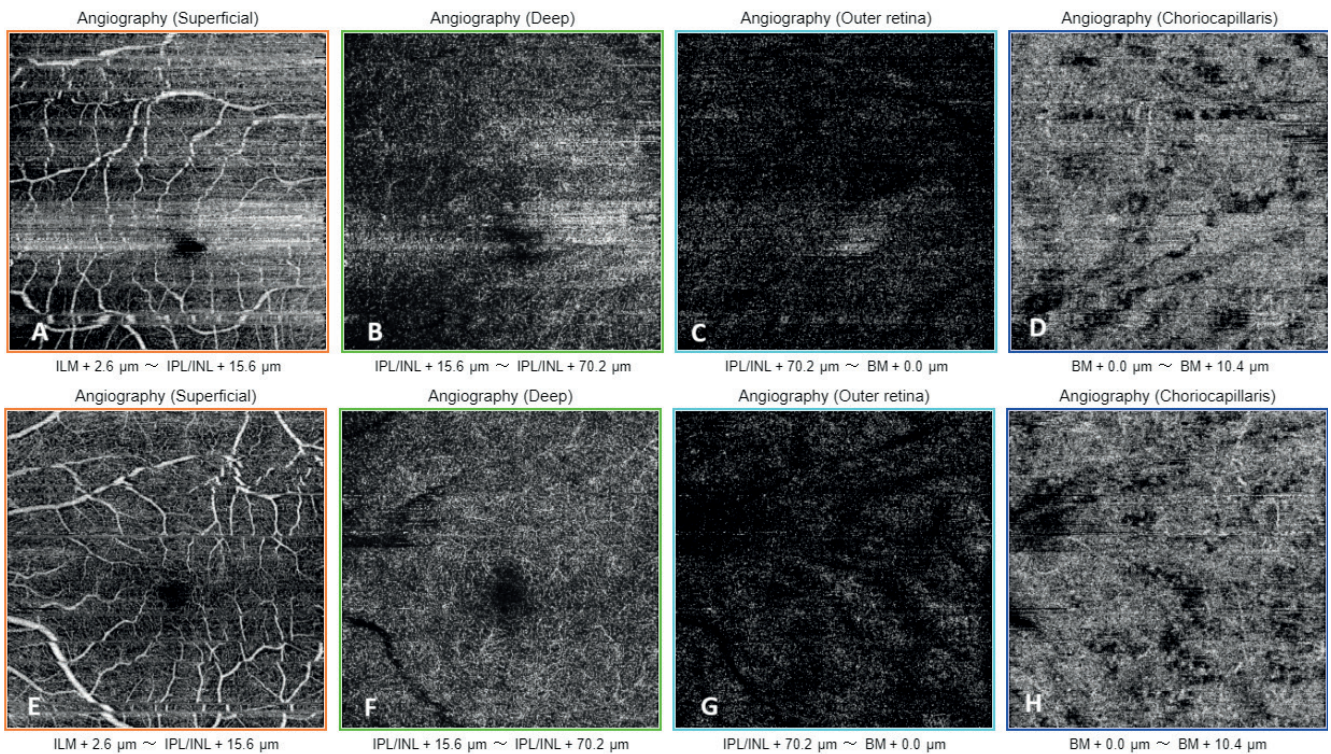
The Enhanced Depth Imaging Mode (EDI) of OCT (EDI-OCT) serves as an essential modality in VKH disease, offering significant insight into the choroidal thickness that indicates the severity of inflammatory infiltration [4]. Choroidal thickness fluctuates according to the disease stages. In the initial phase, choroidal thickening is pronounced, often exceeding the measurable limits. Proper treatment results in a gradual decrease in the choroidal thickness, making it a valuable parameter for assessing the treatment response. However, during the subacute phase, EDI-OCT provides less accurate information compared to ICGA, due to its limited imaging range focused on the posterior pole [25,29].

In the post-acute stage, choroidal thickness variations may reflect remission or disease reactivation, complicating its interpretation [30]. Nevertheless, studies indicate that EDI-OCT can detect subclinical reactivations before they are clinically obvious and can point out the development of an atrophic SGF [31]. Conversely, inadequate treatment and prolonged periods of uncontrolled disease can result in choroidal thinning due to atrophy. Many studies reveal a consistent finding of choroidal thinning during the convalescent or chronic phases of VKH, accompanied by disruption of the choriocapillaris layer in the disease's late stages [32–34].

### F. Optical Coherence Tomography Angiography

OCTA is a convenient and straightforward tool that enables the detailed visualization of the retinal and choroidal vasculature without the need for dye injection. OCTA provides both qualitative and quantitative insights into the vascular supply [35,36]. It allows for the visualization of inflammatory MNV and other retinal and choroidal changes in VKH patients [35].

In patients with active VKH, retinal and choroidal microvasculature changes are prominent, making OCTA a useful tool for disease monitoring. Compared to normal



**Figure 3.** Optical Coherence Tomography Angiography (OCTA) images of a 45-year-old female patient at the acute stage of Vogt-Koyanagi-Harada disease. Superficial, deep, and outer retinal slabs of the right (**A, B, C**) and left (**E, F, G**) eyes showing a normal appearance. The choriocapillaris OCTA slabs of the right (**D**) and left (**H**) eyes revealing patchy areas of flow void in the choriocapillaris

individuals and those in remission, vessel densities in the superficial capillary plexus (SCP) and deep capillary plexus (DCP) of the macula are reduced in active VKH. Moreover, the foveal avascular zone (FAZ) is larger in patients in remission than in healthy subjects [37]. In the convalescent period of VKH, OCTA findings reveal significant changes in vessel densities and vascular perfusion. Eyes with SGF show a marked reduction in vessel densities in the SCP and DCP, compared to healthy control eyes and VKH eyes without SGF. There is no significant difference in vascular perfusion between eyes without SGF and healthy control eyes [38]. In addition, VKH patients with SGF exhibit a slightly increased FAZ area and a reduced choroid flow area, compared to healthy controls. During the active stage of VKH, OCTA reveals multiple areas of choriocapillaris flow voids that correspond to the findings seen on ICGA. These inflammatory foci, indicative of choriocapillaris hypoperfusion, typically diminish in number and size following treatment [35,36]. Choriocapillaris vascular density (CC-VD) exhibits a notable decline along all stages of VKH. During the acute uveitis stage, CC-VD significantly declines, followed by an increase as inflammation subsides and a subsequent decline in the event of uveitis relapse. This indicates that choroidal circulation may be affected in response to the inflammation level. Therefore, CC-VD may serve as a valuable indicator of uveitis status and holds a promise for monitoring disease activity [39]. Furthermore, OCTA is a useful examination in the detection and post-treatment follow-up of VKH-as-

sociated inflammatory MNV [40]. Nevertheless, the utilization of OCTA in uveitis is constrained by a number of limitations. There is no standardized acclaimed terminology to describe OCTA findings in uveitic eyes [41,42]. In addition, the presence of vitritis or subretinal fluid can cause shadowing artifacts on choriocapillaris slabs that can compromise the accurate delineation of flow deficit (FD) areas. If these limitations can be overcome, FD findings on OCTA may have the potential to be utilized as an additional diagnostic criterion for early VKH disease and for early detection of its recurrence [41]. An OCTA image of a patient with acute VKH is shown in Figure 3.

Wide-field OCTA has been shown to be useful in the evaluation of VKH patients in addition to conventional OCTA. A recent study by Guo et al. [43] investigated the retinal changes corresponding to a 120° field of view in chronic VKH disease and the associations between these changes and irreversible fundus complications using wide-field swept-source OCTA. This study included 115 eyes of 69 patients with chronic VKH disease and showed that patients in the quiescent phase with disease duration >24 months had significantly greater reductions in vessel density (VD) in all retinal and choroidal layers, decreased choroidal volume, and sparse choroidal vascularity, compared to those with disease duration ≤ 24 months. The authors concluded that a longer duration of disease and a reduced VD in the large and medium-sized choroidal vessels are associated with irreversible complications in the fundus.

## G. Retro-mode Infrared Scanning Laser Ophthalmoscopy

Retro-mode infrared scanning laser ophthalmoscopy (RMI-SLO) has been found to be useful in the evaluation of patients with VKH, in addition to the aforementioned tests [44,45]. In a recent study, Yasuda et al. [45] evaluated six patients diagnosed with VKH using RMI-SLO and reported that in the acute phase of the disease, RMI-SLO revealed pseudo-three-dimensional (3D) areas and wavy patterns. The authors noted that these findings were consistent with areas of sRD and choroidal folds identified by OCT. In particular, it has been suggested that the detection of pseudo-3D areas with RMI-SLO may be more effective than color fundus photography in the detection of sRD. In addition, the authors reported the presence of hyperreflective lines within sRDs on RMI-SLO, corresponding to fibrinous membranes observed on OCT. They also noted the absence of prominent hyperreflective choroidal vasculature on RMI-SLO images, a finding similar to the fuzzy pattern of large choroidal stromal vessels seen on ICGA. In conclusion, this study suggests that RMI-SLO imaging may

serve as an alternative modality for pre- and post-treatment assessment of VKH disease and may have the potential to be a reduction in the need for retinal angiography.

## CONCLUSION

Fundus imaging techniques are invaluable in diagnosing, monitoring and assessing the treatment response in patients with VKH. These imaging modalities provide detailed insights into the different stages of VKH, varying from acute inflammation to chronic changes and potential recurrences. Despite challenges, including the standardization of terminology and the need to address potential artifacts, the advancement of imaging technology continues to refine our understanding of VKH pathophysiology. In future, further research and collaborative efforts are crucial to optimize the utilization of fundus imaging in the management of VKH. This will ensure the implementation of bespoke therapeutic strategies and improved clinical outcomes.

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