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Ocular blood flow alterations in autoimmune diseases: insights from optical coherence tomography angiography

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Abstract

The global incidence of autoimmune diseases is on the rise, leading to a corresponding increase in ocular complications associated with these conditions. Autoimmune pathologies can directly target ocular tissues via inflammatory mediators or induce endothelial dysfunction and microvascular alterations that compromise ocular integrity. These mechanisms give rise to a broad spectrum of ocular manifestations. In this review, optical coherence tomography angiography (OCTA)-detected changes in the eye are examined in the context of systemic autoimmune and inflammatory diseases. These imaging biomarkers show promise as novel indicators for identifying relapse risk and subclinical inflammatory stages in systemic autoimmune disorders.

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Introduction

Autoimmune diseases[1] represent a category of multisystem disorders characterized by immune dysregulation, aberrant production of autoantibodies, and subsequent damage to the host's own cells, tissues, and organs. The etiopathogenesis of these conditions remains incompletely elucidated due to their considerable complexity. A multifaceted interplay among genetic susceptibility, immune dysfunction, environmental exposures, hormonal influences, and infectious agents collectively contributes to the breakdown of immunological tolerance and the initiation of autoimmune pathology.

As a common target of systemic autoimmunity, the eyes can manifest a wide array of pathologies, from mild discomfort to blinding lesions, though such manifestations are frequently overlooked and their clinical importance underappreciated[2]. Autoimmune diseases can directly attack the eyes by stimulating the production of inflammatory cytokines and amplifying the inflammatory cascade. Inflammatory factors can also damage the eyes by contributing to the endothelial dysfunction and altering the small vessels of the microcirculation.

The directly observable nature of retinal blood vessels makes them a unique bridge connecting local microcirculation with systemic physiological conditions, demonstrating significant practical value in disease prediction, early diagnosis, dynamic monitoring, and public health screening. The choroid is a highly vascularized stromal tissue with a unique three-dimensional architecture, composed of a spongy vascular connective tissue, abundant extracellular matrix, and resident immune cells[3]. It exhibits the highest tissue-specific perfusion rate in the human body. This distinctive vascularity and immune cell presence have consequently positioned the ocular blood vessels as potential imaging biomarkersfor systemic autoimmune diseases.

Objective

The objective of this study was to review the literature regarding the changes of eyes through OCTA in systemic autoimmune and inflammatory diseases.

Areas covered

A comprehensive literature search was conducted across multiple databases, including PubMed, Web of Science, and ScienceDirect. The search utilized keywords 'OCTA' along with the names of major systemic autoimmune inflammatory diseases (including systemic lupus erythematosus, Vogt–Koyanagi–Harada disease, Behçet's disease, rheumatoid arthritis, ankylosing spondylitis, psoriasis, dermatomyositis, and Sjögren's syndrome), The search encompassed articles published up to the date of October 25, 2025. Studies that did not focus on retinal or choroidal vascular flow changes were excluded.

Optical coherence tomography angiography

Leveraging principles of optical coherence and interferometry, Optical Coherence Tomography Angiography (OCTA) enables non-invasive, high-resolution three-dimensional visualization of dynamic microvascular networks in the retina and choroid[4,5]. This technology provides depth-resolved imaging of retinal and choroidal vasculature over extensive areas, allowing detailed evaluation at the capillary level and supporting computational quantification of vascular parameters. Currently, OCTA is extensively applied in the assessment of diseases such as diabetic retinopathy and retinal vein occlusion, where it aids in monitoring disease onset and progression by detecting structural and circulatory changes in the retinal-choroidal complex[6,7].

Systemic diseases

Ankylosing spondylitis

Disease characteristics and ocular involvement

Ankylosing spondylitis (AS)[8] is a chronic inflammatory rheumatic disease and is often considered a clinical outcome of other subtypes of spondyloarthropathies. The condition primarily affects axial joint

structures, with significant involvement of the sacroiliac joints being particularly prominent. Among extra-articular manifestations, ocular involvement is relatively common, especially uveitis, and there are also rare case reports of optic neuritis.

Systemic and ocular vascular dysfunction

Current studies indicate that AS patients exhibit impaired microvascular function, which is associated with disease activity. This conclusion is primarily based on nailfold capillaroscopy and structural observations of the skin vascular bed[9]. The chronic systemic inflammatory state is considered a significant contributor to microvascular dysfunction. Furthermore, multiple studies have confirmed a substantially increased risk of cardiovascular disease in AS patients[10].

Regarding ocular manifestations, although there are reports of occasional retinal vasculitis and cilioretinal artery occlusion due to posterior scleritis in AS patients[11], research on the impact of the disease on the ocular vascular system remains relatively limited. A recent study using ultra-widefield fluorescein angiography (FFA) revealed a 42.2% incidence of retinal vascular leakage in AS patients with uveitis, with diffuse leakage observed in six eyes (13.3%) and peripheral leakage in 13 eyes (28.9%). This study further confirmed the presence of ocular vascular pathology in AS patients from an angiographic perspective[12].

OCTA findings and cardiovascular correlations

Although research on the impact of AS on the ocular vascular system remains relatively limited, emerging evidence from non-invasive OCTA technology has begun to provide crucial insights. Studies demonstrate that retinal vascular assessment can detect microvascular alterations at the microscopic level in AS patients.

Van Bentum et al.[13] conducted a study with an 'eye-to-heart' approach, aiming to investigate the correlation between microvascular changes and cardiovascular disease in AS patients through non-invasive retinal vascular assessment. This cross-sectional study compared retinal images from 59 AS patients and 105 healthy controls. The results showed that AS patients overall exhibited significantly reduced retinal arteriolar tortuosity (β = −0.1, 95% CI [−0.2; −0.01], p = 0.02) and increased vessel density (VD) (β = 0.5, 95% CI [0.1; 0.9], p = 0.02). Notably, gender-stratified analysis revealed a significant decrease in the arteriolar-to-venular ratio (β = −0.03, p = 0.04, 95% CI [−0.05; −0.001]) only in male AS patients, while no difference was observed in female patients compared to the control group. The study concluded that AS patients, particularly males, exhibit specific retinal microvascular alterations associated with cardiovascular disease. It confirmed that retinal imaging technology could serve as a potential novel tool for assessing cardiovascular risk in AS patients.

Rheumatoid arthritis

Disease characteristics and ocular manifestations

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disease characterized primarily by symmetric inflammatory arthritis, commonly affecting the small joints of the wrists, hands, and feet[14]. Beyond articular symptoms, RA can lead to various extra-articular manifestations and comorbidities, including rheumatoid nodules, rheumatoid vasculitis, interstitial lung disease, as well as neurological, cardiovascular, renal, and hematological impairments.

In terms of ocular manifestations, keratoconjunctivitis sicca is the most common ocular comorbidity in RA patients[15]. Other ocular abnormalities include episcleritis, scleritis, and peripheral ulcerative keratitis (PUK)[16]. Foster et al.[17] noted in a retrospective study that the presence of necrotizing scleritis or PUK may be an important indicator of underlying life-threatening systemic vasculitis.

Pathogenic mechanisms and systemic vascular implications

Regarding pathogenesis, RA promotes the production of inflammatory cytokines and amplification of the inflammatory cascade, which upregulates endothelial chemotaxis and proliferation, ultimately leading to pathological angiogenesis in synovial tissue[18]. These inflammatory factors are also considered major contributors to endothelial dysfunction, causing structural and functional alterations in the microcirculation of small vessels[19]. Furthermore, inflammatory cytokines disrupt the balance among coagulation, anticoagulation, and fibrinolysis systems. Under the combined influence of endothelial dysfunction and RA-related medications, this can further induce a hypercoagulable state[20].

Notably, RA patients exhibit a significantly elevated risk of cardiovascular disease and overall mortality compared to the non-RA population. However, as highlighted by Weber et al.[21] some RA patients may exhibit coronary vasomotor dysfunction due to impaired microvascular reactivity—even in the presence of mild calcified plaque burden—rather than typical diffuse atherosclerotic lesions.

OCTA evidence of retinal microvascular alterations

Substantial evidence indicates that RA, as a systemic autoimmune disease, intrinsically damages the retinal microcirculation, independent of hydroxychloroquine (HCQ) treatment.

Multiple cross-sectional studies[22,23] consistently report that RA patients show significant reductions in vessel density in both the SCP and DCP of the macula compared to healthy controls. Importantly, this reduction in microvascular density has been observed even in treatment-naïve RA patients. This suggests that impaired retinal microcirculation may be an early biomarker of systemic vascular pathology in RA, preceding overt clinical ocular symptoms. Ayar et al.[23] also found that vessel density in the radial peripapillary capillary (RPC) plexus was negatively correlated with the RA disease activity score (DAS-28) (Rho = −0.272, p = 0.006), indicating that higher disease activity may be associated with greater ocular microvascular involvement.

The impact of HCQ on retinal microvasculature in RA patients presents a complex picture. A study by Iacono et al.[24] showed that RA patients receiving short-term HCQ therapy did not exhibit a statistically significant reduction in SCP or DCP vessel density, suggesting that HCQ's microvascular toxicity may not be prominent in early treatment. In contrast, Ozek et al.[25] focused on 'high-risk' patients with more than five years of HCQ use and identified focal reductions in DCP vessel density (48.13% ± 8.5%, p = 0.041), particularly in the temporal and inferior quadrants. These changes were detectable even when visual field testing remained normal—a finding of high clinical relevance, since early characteristic changes in HCQ retinopathy typically begin in the temporal macula. However, Abdeltawab et al.[26] found that some visual field defects and associated reductions in vessel density observed in the HCQ group were also present in RA patients not treated with HCQ. This suggests that at least some of the observed microvascular alterations may stem from RA's underlying pathology rather than HCQ toxicity.

Clinical implications and OCTA monitoring strategies

Therefore, when evaluating a patient's ocular status, it should be recognized that RA itself can lead to reduced retinal microvascular density. In the context of RA, OCT angiography (OCT-A) can detect deep capillary changes undetectable by conventional perimetry in patients on long-term HCQ, serving as a useful supplementary monitoring tool. However, given that the specificity and sensitivity of OCT-A for early HCQ retinopathy screening are not yet fully established, the current cornerstone of clinical monitoring should remain regular and sequential OCT structural scans to assess retinal

integrity, with OCT-A vessel density analysis serving as an ancillary reference.

Behçet's disease

Disease characteristics and ocular involvement

Behçet's Disease (BD) is a chronic, relapsing systemic inflammatory disorder pathologically characterized by occlusive vasculitis[27]. It presents with diverse clinical manifestations, typically featuring recurrent oral and genital ulcers, cutaneous and ocular lesions, and may involve multiple organ systems, including vascular, neurological, gastrointestinal, musculoskeletal, pulmonary, and renal systems.

Among the various organs affected in BD, ocular involvement is particularly common, with its hallmark presentation being non-granulomatous panuveitis, frequently accompanied by retinal vasculitis[28].

OCTA findings and disease dynamics

Extensive research has confirmed that even in BD patients without any clinical ocular symptoms, OCTA has detected widespread, statistically significant microvascular abnormalities. This reveals that BD, as a systemic vasculitis, involves the ocular microvasculature far earlier than clinical manifestations appear.

Multiple analytical studies have indicated that, compared to healthy controls, non-ocular BD patients exhibit significant reductions in VD in both the SCP and DCP of the macular region, as well as an enlargement of the foveal avascular zone (FAZ) area[29–32]. The most consistent and prominent finding is the reduction in SCP VD in the parafoveal region, a discovery corroborated by meta-analyses conducted by Fan et al.[33], Goker et al.[34], and Ji et al.[35]. Microvascular alterations are not confined to the macular retina. Studies by Küçük et al.[31] and Karalezli et al.[36] found that the radial peripapillary capillary (RPC) density and the VD inside the optic nerve head (ONH) were also significantly reduced. Findings regarding the choroid are heterogeneous. Studies by Küçük et al.[31] and Fan et al.[33] found no significant change in the choriocapillaris (CC) flow area, whereas Çömez et al.[37] reported a decrease. Simsek et al.[38] introduced the more sensitive Choroidal Vascularity Index (CVI) and found it was significantly reduced in non-ocular BD patients(the macula sectors $p = 0.009$, the temporal sectors $p = 0.002$, the nasal sectors $p = 0.010$, and the inferior sectors $p = 0.008$), while choroidal thickness remained unchanged, suggesting that choroidal hypoperfusion may precede structural changes. CVI may represent an earlier and more robust indicator of choroidal hypoperfusion and ischemia. In contrast to retinal microvascular reduction, Xiong et al.[39] observed increased conjunctival vessel density in BD patients (1.60 ± 0.06; $p < 0.001$). The authors attributed this 'retina-conjunctival vascular dichotomy' to their fundamental anatomical differences: retinal vessels, protected by the blood-retinal barrier, predominantly exhibit occlusive vasculitis leading to decreased VD, reflecting cumulative damage; conjunctival vessels, lacking a tight barrier, display dilative inflammation leading to increased VD, indicating current inflammatory activity. This complementary pattern offers a unique clinical perspective for simultaneously assessing both long-term prognosis and current disease activity.

When BD manifests with clinical ocular involvement (primarily posterior uveitis), the microvascular damage revealed by OCTA is more severe and correlates with disease activity, duration, and visual function. Studies by Karaca et al.[40], and Smid et al.[41] showed that patients with ocular involvement had significantly lower VD in both SCP and DCP across all regions compared to those without ocular involvement and healthy controls. Research by Khairallah et al.[42], Somkijrungroj et al.[43], and Emre et al.[44] consistently emphasize the predominance of DCP involvement throughout the disease course. The extent and severity of VD loss, non-perfused areas, and capillary morphological abnormalities (e.g., dilation, shunts) in the DCP far exceed those in the SCP. This explains why vision can be impaired in BD due to severe DCP ischemia, even when the SCP remains relatively intact.

OCTA reveals distinct features across the dynamic evolution of the disease. A longitudinal study by Wassef et al.[45] highlighted differences between active and remission phases: during remission, SCP VD showed a degree of reversible recovery ($1.81\% \pm 3.57\%$, $p = 0.025$), whereas DCP damage and FAZ enlargement were more irreversible, making them better indicators for assessing long-term prognosis. The study by Cheng et al.[46] further linked microvascular damage to cumulative disease burden, finding that patients with more frequent attacks (≥ 5 episodes) had larger FAZ areas, and that deep retinal microvascular damage was closely associated with disruption of the outer retinal structure. Furthermore, Yan et al.[47] found that BU patients had significantly reduced RPC density in the peripapillary region, particularly inferiorly, which correlated with decreased best-corrected visual acuity (BCVA) ($r = -0.692$, $p = 0.013$), indicating that peripapillary ischemia is a significant factor in visual dysfunction in BU patients.

Diagnostic and prognostic value of OCTA

In summary, OCTA objectively delineates a continuum of microvascular pathology in BD, from subclinical to clinical stages. Quantifiable alterations in the retinal, peripapillary, and choroidal microvasculature are already present in the stage without ocular involvement; the clinical uveitis stage is characterized by severe and often irreversible damage to the DCP. These findings establish the indispensable value of OCTA in the early diagnosis, monitoring of disease activity, and prognosis assessment of BD.

Systemic sclerosis

Disease characteristics and ocular manifestations

Systemic Sclerosis (SSc) is a rare multisystem autoimmune disease characterized by distinctive pathological features, including vascular-abnormalities, inflammatory responses, and widespread fibrosis[48]. In terms of ocular manifestations, SSc patients can present with a range of symptoms such as eyelid skin involvement, dry eye disease, iris transillumination defects, glaucoma, and uveitis[49,50].

Pathogenic mechanisms of vascular involvement

The immune-mediated inflammatory response in this disease can trigger endothelial cell damage, intimal hyperplasia, and perivascular inflammatory infiltration, leading to structural and functional endothelial abnormalities and impairing normal angiogenesis[51,52].

OCTA findings and systemic microvascular correlations

A series of studies based on OCTA have now established that SSc can lead to significant alterations in the posterior segment ocular microvasculature. These changes are closely associated with disease stage, clinical subtype, and the status of systemic microcirculation.

Research indicates that choroidal microcirculation is affected earlier and more severely than the retina. Microvascular damage can be detected by OCTA even in the early stages of the disease. Mihailovic et al.[53] found that patients with Very Early Diagnosis Of Systemic Sclerosis (VEDOSS) already exhibited a significant reduction in choriocapillaris flow density (VEDOSS: 107.14% [103.77%, 113.42%], healthy controls: 116.96% (114.19%, 118.74%), $p = 0.008$), while retinal microvascular changes might still be undetectable at this point. This suggests the choroid may be the first site of ocular microcirculatory involvement. A precise layered study by

Ranjbar et al.[54] revealed significantly reduced blood flow perfusion and marked thinning in the choriocapillaris, Sattler's layer, and Haller's layer in SSc patients.

In diagnosed patients without clinical ocular symptoms, multiple studies (El-Hameed et al.[55]; Alahmadawy et al.[56]; Hekimsoy et al.[57]) consistently report widespread reductions in vessel density in both the superficial and deep retinal capillary plexuses, alongside enlargement and irregularity of the Foveal Avascular Zone (FAZ) (Küçük et al.[58]), fully demonstrating OCTA's sensitivity in identifying subclinical pathology.

As the disease progresses, microvascular damage tends to become more diffuse. The study by Rommel et al.[59] revealed that patients with a longer disease duration (e.g., over five years) exhibited not only impaired retinal microcirculation but also a concurrent significant reduction in perfusion across all choroidal layers (including Sattler's and Haller's layers), with the severity of damage in both compartments correlating positively ($\rho = 0.672$; $p < 0.001$). Particularly noteworthy, Cutolo et al.[60] found that patients with diffuse cutaneous SSc (dcSSc), compared to those with limited cutaneous SSc (lcSSc), demonstrated more severe choroidal perfusion reduction yet exhibited a compensatory increase in choroidal thickness—a phenomenon potentially linked to local inflammation and matrix deposition.

Changes in ocular microcirculation are closely linked to peripheral microcirculatory alterations. Cutolo et al.[60] also found that patients with active digital ulcers typically exhibited more pronounced ocular microcirculatory impairment. Studies by Elsayed et al.[61] and Mihailovic et al.[53] using Nailfold Capillaroscopy (NFC) found a significant positive correlation between nailfold capillary density and retinal/choroidal flow density measured by OCTA, confirming that SSc microvasculopathy is systemic, with ocular changes serving as a 'window' to the systemic microcirculatory status. Furthermore, Elsayed, SA[61] found a negative correlation between retinal microvascular density and the modified Rodnan Skin Score (mRSS).

The in-depth research by Zirtiloglu et al.[62] further revealed strong links between optic disc blood flow and systemic markers: as antinuclear antibody (ANA) titers increased, Radial Peripapillary Capillary (RPC) density significantly decreased in multiple regions, and patients positive for anti-Scl-70 antibodies had significantly lower RPC density inside the optic disc compared to antibody-negative patients. Notably, pharmacologic treatment might improve optic disc microcirculation, as this study found that patients using hydroxychloroquine (HCQ) and calcium channel blockers had significantly higher RPC density than non-users.

Clinical implications and monitoring strategies

In summary, OCTA, as a non-invasive *in vivo* imaging technique, clearly reveals progressive ocular microvascular damage in SSc starting from the very early stages. This damage highly correlates with disease subtype, activity, severity, and systemic microcirculatory status. It is recommended to introduce OCTA examination at the initial diagnosis of SSc to detect subclinical pathology, and to consider its integration into the comprehensive disease assessment and treatment monitoring framework, thereby providing crucial evidence for the precise management of SSc.

Vogt-Koyanagi-Harada disease

Disease characteristics and clinical spectrum

Vogt-Koyanagi-Harada (VKH) disease is a multisystem granulomatous autoimmune disorder, with its core pathological mechanism being T-cell-mediated destruction of melanocytes. The clinical course of the disease can typically be divided into two stages: the initial prodromal phase presents with headache, fever, confusion, and meningeal signs; this is followed by the later phase, characterized by systemic manifestations including auditory symptoms (such as tinnitus, dysacusis, and vertigo) and integumentary changes (such as vitiligo and poliosis). Due to the high concentration of melanocytes in choroidal tissue, it becomes a vulnerable site for inflammation. Clinically, this most often manifests as bilateral granulomatous uveitis (posterior or panuveitis), accompanied by exudative retinal detachment, and may progressively evolve into the characteristic 'sunset glow' fundus appearance[63].

OCTA findings across disease stages and treatment response

Recent advancements in wide-field OCTA have provided a powerful tool for the *in vivo*, non-invasive, and quantitative assessment of microcirculatory changes in VKH patients across different disease stages, significantly deepening our understanding of its pathophysiology, diagnosis, treatment, and prognosis.

During the acute phase, OCTA consistently reveals extensive perfusion impairment centered on the choriocapillaris. Wintergerst et al.[64] suggested that perfusion impairment is most pronounced in Sattler's layer. The study by Geng et al.[65] found that treatment-naïve, active patients exhibited significant reductions in both large- and medium-caliber choroidal vessel density (LMCV-VD) in the mid-periphery and macula, as well as in macular retinal and choriocapillaris densities. Studies by Ding et al.[66] and Luo et al.[67] utilizing wide-field OCTA, further delineated characteristic acute-phase findings: multiple 'flow void areas' in the retinal capillary layers, choriocapillaris, and Sattler's layer. Luo et al.[67] also observed another abnormal pattern characterized by hyperreflective foci surrounded by increased flow. Jia et al.[68], through quantitative analysis, further found that acute-phase patients had significantly lower vessel density in the SCP, DCP, and choriocapillaris (CC) compared to healthy controls, and a correlation might exist between reduced choriocapillaris flow density and retinal detachment (RD).

When the disease enters the chronic or inactive phase, acute inflammation subsides, but microvascular damage is not fully repaired, manifesting as irreversible microvascular rarefaction. Research by Karaca et al.[69] indicated that even during inactivity, patients had an enlarged FAZ area and reduced macular capillary perfusion compared to healthy controls, suggesting permanent microcirculatory loss. Moreover, prolonged disease duration is a critical factor exacerbating microvascular damage. The study by Guo et al.[70] clearly demonstrated that chronic patients with a disease duration exceeding 24 months had significantly lower vessel density in all retinal layers (SCP, DCP) and choroid (from capillaries to large/medium vessels), as well as reduced choroidal volume and vascular index, compared to those with a shorter duration (≤ 24 months). Research by Fan et al.[71] confirmed that inactive patients with a 'sunset glow fundus' (SGF) exhibited significantly reduced vessel density in the macular SCP and DCP. These chronic microcirculatory disturbances are significantly associated with the development of irreversible fundus complications, such as choroidal neovascularization and chorioretinal atrophy.

OCTA can sensitively capture dynamic microcirculatory changes corresponding to disease activity before and after treatment. Longitudinal observation by Geng et al.[65] showed that retinal and choroidal parameters began to improve significantly as early as two weeks post-treatment, with spatial heterogeneity in the recovery process: the macular and temporal areas recovered preferentially over the nasal area. Furthermore, the recovery status of mid-peripheral LMCV-VD at two weeks was identified as a potential biomarker for predicting the subsequent development of SGF ($p = 0.044$, OR = 2.695, 95% CI = 1.027 ~ 7.056). An intriguing phenomenon was

observed in the study by Fayed et al.[72] upon transitioning from the active to the remission phase, choroidal thickness decreased significantly, yet the choroidal vascular index paradoxically increased, potentially resulting from the resolution of inflammatory infiltration and recanalization of vascular lumens. Research by Jiang et al.[73], focusing on the optic disc, found that patients with pre-treatment disc edema had a higher prevalence of reduced blood flow density in the radial peripapillary capillary (RPC) plexus and retinal plexuses post-treatment, suggesting more severe inflammatory impact on local microvessels. VKH-associated uveitis also impacts the microvasculature; Liang et al.[74] found that patients with a history of recurrent anterior uveitis had lower choriocapillaris density during the recovery phase, and this parameter could be used to assess uveitis status.

The timing of treatment initiation is crucial for determining the extent to which the microcirculation can be preserved. The study by Huang et al.[75] directly compared an early treatment group (within two months of onset) and a late treatment group (after two months from onset). Results clearly showed that the early treatment group had significantly superior outcomes on a series of OCTA parameters—including flow area in the SCP and DCP, choriocapillaris flow area, and three-dimensional choroidal vascular volume (CVV)—compared to the late treatment group, and their parameters were closer to healthy levels. These superior microvascular parameters correlated significantly with better final best-corrected visual acuity (BCVA). Thus, early and aggressive intervention is essential to optimize both microvascular structure and visual function.

OCTA parameters combined with machine learning algorithms show great potential in the objective staging of VKH. The study by Xiao et al.[76], utilizing WFSS-OCTA parameters combined with machine learning, achieved high accuracy exceeding 96% in distinguishing healthy controls, acute-phase, and convalescent-phase VKH patients, with logMAR BCVA and whole-field choriocapillaris vascular perfusion density (whole FOV CC-VPD) identified as the most important discriminatory features.

Clinical implications and future perspectives

In summary, OCTA technology clearly delineates the microvascular evolution in VKH disease: the acute phase is characterized by ischemia and edema, while the chronic phase culminates in microvascular rarefaction and remodeling. Its dynamic changes sensitively reflect treatment response, with early parameters like LMCV-VD at two weeks[65] holding potential value for predicting long-term complications such as SGF. Both treatment timing[75] and disease duration[70] collectively determine the final extent of microcirculatory damage. In the future, AI-enhanced OCTA analysis is expected to play a more central role in the precise staging, activity assessment, visual prognosis prediction, and guidance of individualized therapy for VKH disease.

Psoriasis

Disease characteristics and ocular involvement

Psoriasis is an immune-mediated dermatological disease, typically characterized by scaly erythematous skin plaques and frequently associated with psoriatic arthritis (PsA). This condition can affect multiple ocular structures, increasing the risk of orbital myositis, blepharitis, conjunctivitis, uveitis, and retinopathy[77].

Pathogenesis and subclinical ocular manifestations

Existing research suggests that patients with psoriasis may exhibit subclinical ocular involvement even in the absence of overt ocular inflammatory manifestations. One study observed peripheral vascular leakage (PVL) in patients with moderate-to-severe psoriasis, serving as supporting evidence. Recent studies further indicate a significantly elevated incidence of vascular leakage[78] in psoriasis patients without typical ocular symptoms, suggesting inflammatory damage and dysfunction of the blood-retinal barrier[79,80].

The pathogenesis of psoriasis is currently believed to involve complex interactions between vascular endothelial growth factor (VEGF), hypoxia-inducible factors, angiopoietins, and pro-angiogenic cytokines (such as tumor necrosis factor and specific interleukins)[81].

OCTA findings and associations with disease course and comorbidities

OCTA provides a crucial perspective for revealing the impact of psoriasis, as a systemic disease, on the retinal microcirculation.

Multiple studies consistently demonstrate quantifiable abnormalities in the retinal microvasculature of psoriasis patients, even in those without any history of ocular inflammation or clinical symptoms. A multicenter study by Castellino et al.[82] found significantly reduced VD in both the whole image and parafoveal regions of the SCP and DCP in patients. This suggests that retinal vascular alterations may precede clinically detectable posterior segment ocular inflammation, representing an early subclinical marker of systemic inflammation in the eye.

Disease severity and comorbidities significantly influence the extent of microvascular changes. Alkan et al.[83], stratifying patients by disease severity, found that those with moderate-to-severe psoriasis had a significantly enlarged FAZ area and significantly reduced VD in the parafovea DCP. These changes correlated significantly with the Psoriasis Area and Severity Index (PASI) score. Research by Baris et al.[84] further confirmed that patients with severe psoriasis had significantly lower VD in both the SCP and DCP compared to healthy controls. Furthermore, the presence of PsA was significantly associated with increased central retinal thickness, enlarged FAZ area, and reduced parafoveal SCP VD, indicating that concomitant arthritis exacerbates retinal microcirculatory impairment.

Additionally, metabolic syndrome (MetS) is considered a key additional factor that increases the risk of retinal ischemia. A comparative study by Tolba et al.[85] found that patients with psoriasis alone already exhibited reduced VD in both the SCP and DCP. However, when psoriasis coexisted with MetS, the vascular damage was more extensive and severe, with the DCP VD being significantly affected early on, suggesting that ischemic changes may originate here. This study emphasized that the presence of MetS significantly increases the additional risk of vision loss due to retinal ischemia in these patients.

Clinical implications and risk stratification

In summary, OCTA, as a non-invasive imaging tool, can sensitively detect early retinal vascular abnormalities associated with psoriasis. These findings not only deepen the understanding of the systemic nature of this disease but also suggest the significant potential value of OCTA in monitoring systemic complications and performing early risk stratification for psoriasis.

Sjogren's syndrome

Disease characteristics and systemic involvement

Sjögren's Syndrome (SS) is a chronic autoimmune disease characterized primarily by progressive dysfunction of the exocrine glands. Due to lymphocytic infiltration and exocrine gland dysfunction, the most common clinical manifestations in patients are xerostomia and keratoconjunctivitis sicca. Beyond glandular involvement, the disease

can also affect multiple non-glandular organs such as the skin, lungs, and kidneys[86]. The presence of Raynaud's phenomenon and cutaneous vasculitis typically indicates systemic vascular involvement.

OCTA findings and treatment response

The application of OCTA in the evaluation of ocular microvasculature in SS has, in recent years, revealed characteristic patterns of alteration.

Multiple cross-sectional studies have confirmed significant reductions in retinal microvascular density and structural damage in SS patients. The study by Liu et al.[87] indicated that compared to healthy controls, SS patients had significantly reduced SCP in multiple subfields of the macular region, accompanied by localized retinal thickness (RT) thinning, particularly in the outer nasal sector. Further analysis revealed a negative correlation between retinal thickness in this sector and serum IgG levels, suggesting that microvascular and structural tissue changes may be associated with systemic immune-inflammatory activity. The findings by Yener et al.[88] further deepened this understanding; they reported a significant decrease in DCP density in SS patients, while no significant difference was found in the superficial plexus, indicating layer-specific microvascular involvement. Furthermore, capillary density across retinal layers negatively correlated with disease duration, highlighting a trend of worsening microvascular damage with disease progression.

Beyond the fundus vasculature, microvascular abnormalities in SS also extend to the anterior segment. Using anterior segment OCTA, Ferrigno et al.[89] observed that SS patients had significantly higher conjunctival vessel density compared to healthy controls, which correlated with disease duration and complement C3/C4 levels (p = 0.03, r = −0.3 and p = 0.02, r = −0.3, respectively), reflecting pathological vascular proliferation driven by local chronic inflammation.

In terms of differential diagnosis, these microvascular alterations demonstrate disease specificity. In a comparative study by Wolf et al.[90] between SS and Relapsing-Remitting Multiple Sclerosis (RRMS) patients, although both groups exhibited a general reduction in SCP VD, rarefaction of the DCP was unique to SS. This change correlated with visual function impairment in patients, suggesting distinct pathological mechanisms between the two diseases and providing potential imaging evidence for differentiating SS from other neuroimmune disorders.

In the field of treatment monitoring, OCTA also shows significant value. Differing from observations in RA patients undergoing hydroxychloroquine (HCQ) treatment, a retrospective study by Yu et al.[91] revealed the potential impact of HCQ therapy on the fundus microvasculature in SS patients: SS patients receiving HCQ treatment exhibited a further significant reduction in retinal microvascular density compared to untreated SS patients. This finding suggests that while treating the underlying disease, HCQ may exacerbate damage to the retinal microcirculation. OCTA technology provides a sensitive tool for the non-invasive monitoring of such drug-associated microvasculopathy.

Clinical implications and diagnostic value

In summary, the ocular microvascular changes in SS revealed by OCTA—including the specific reduction in deep retinal vasculature, its correlation with disease activity and duration, and conjunctival vascular proliferation—not only deepen our understanding of the pathological mechanisms of SS-related eye disease but also demonstrate considerable potential for clinical translation in auxiliary diagnosis, disease differentiation, condition assessment, and treatment monitoring.

Systemic lupus erythematosus

Disease characteristics and ocular manifestations

Systemic Lupus Erythematosus (SLE) is a multisystem autoimmune disease whose chronic inflammatory pathogenesis is primarily mediated by immune complex deposition and type III hypersensitivity reactions[92]. The clinical manifestations of SLE are highly heterogeneous, often involving multiple organs and tissues such as the skin (e.g., malar rash, photosensitivity), joints (non-erosive polyarthritis), kidneys (lupus nephritis), and hematopoietic system (cytopenias)[92].

In terms of ocular involvement, SLE can present with various ocular surface disorders such as dry eye syndrome, scleritis, episcleritis, and conjunctivitis. It can also affect the posterior segment and optic nerve, leading to optic neuritis, ischemic optic neuropathy, retinal vasculitis, retinal obstructive vasculopathy, and choroidopathy[93].

OCTA findings and systemic correlations

In recent years, the application of OCTA has enabled *in vivo*, non-invasive, and quantitative assessment of ocular microcirculation in SLE patients, thereby providing a new window into understanding the systemic and heterogeneous nature of this disease.

In SLE patients without clinical ocular symptoms, OCTA has revealed prevalent subclinical microvascular abnormalities. The retina is the most extensively studied area. Multiple studies (e.g., Chen et al.[94]; Ferreira et al.[95]; Liu et al.[96]) consistently report significantly reduced vessel density (VD) in both the SCP and DCP of SLE patients. This reduction follows a 'pan-retinal' pattern, encompassing the foveal, parafoveal, and peripheral regions. Notably, involvement of the DCP often occurs earlier and is more pronounced (Arfeen et al.[97]; Ermurat & Koyuncu[98]), suggesting it may be the initial target of SLE microvascular injury. Furthermore, enlargement and increased perimeter of the FAZ (An et al.[99]; Basiony et al.[100]) and decreased microvascular network complexity (e.g., reduced fractal dimension, Ferreira et al.[95]) are common features. The choroid is also involved; studies by Bayuk et al. and Ferreira et al.[95] found an increased choroidal vascularity index (CVI) alongside reduced choriocapillaris flow signal in SLE patients, indicating choroidal vascular abnormalities. In the optic disc region, Koyuncu & Emurat[101] and Çomçali et al.[102] observed reduced density in the radial peripapillary capillary (RPC) plexus. More cutting-edge OCTA studies of the conjunctiva (Chen et al.[94]; Shi et al.[103]) and iris (Pichi et al.[104]) have also confirmed decreased microvascular density or abnormal vascular proliferation, indicating that the microvasculopathy in SLE is a global event involving both the anterior and posterior segments.

In the specific population of juvenile-onset SLE (JSLE), studies (Xu & Zhang[105]; Tugan et al.[106]) have similarly confirmed widespread reductions in VD across the entire retina and choriocapillaris layer even in the absence of any ocular manifestations, emphasizing that microvasculopathy initiates early in the disease course.

When clinical ocular involvement occurs in SLE, OCTA can precisely quantify its severity. Meng et al.[107] using widefield OCTA, clearly demonstrated morphological changes such as vascular occlusion and non-perfusion areas in patients with lupus retinopathy.

Notably, these subclinical microvascular changes exhibit specific associations with systemic disease status. Although most studies, such as those by Bayuk et al.[108] and Koyuncu & Emurat[101] found no significant correlation between microvascular density and SLE disease activity indices; they are closely related to disease duration and cumulative damage. More importantly, ocular microvascular alterations are strongly linked to severe systemic complications.

Studies by multiple authors, including Conigliaro et al.[109], Basiony et al.[100], Wang et al.[110], and Yavuz et al.[111] have indicated that patients with concomitant lupus nephritis (LN) exhibit more severe retinal microvascular damage, particularly reduced DCP density, compared to those without nephritis.

Regarding neuropsychiatric SLE (NPSLE), Koyuncu & Emurat[101] found that these patients had significantly lower peripapillary microvascular density and retinal nerve fiber layer thickness compared to non-NPSLE patients, suggesting a potential parallel between ocular and central nervous system microvascular injury. Concerning cardiovascular risk, the study by Ferrigno et al.[112] linked reduced deep retinal VD with carotid intima-media thickness (IMT) and QRISK3 cardiovascular risk scores, suggesting that OCTA parameters could serve as a window for assessing subclinical atherosclerosis and cardiovascular risk in SLE patients. Furthermore, Yu et al.[113] utilized a machine learning model integrating retinal nerve fiber layer thickness and VD parameters to successfully construct a high-accuracy model (AUC = 0.950) for diagnosing lupus nephritis, demonstrating the considerable potential of OCTA in assisting the diagnosis of systemic complications.

Regarding treatment effects, the impact of long-term drug exposure on the microvasculature cannot be ignored. Studies by Li et al.[114] and Mihailovic et al.[115] established that long-term (≥ five years) and high cumulative dose hydroxychloroquine (HCQ) use is associated with reduced superficial retinal VD and FAZ enlargement. A two-year follow-up study (Leclaire et al.[116]) further observed a decline in VD in the SCP and choriocapillaris over time in SLE patients, which was not directly linked to changes in HCQ cumulative dose, suggesting the disease itself is the primary driver of microvascular degeneration. Additionally, the report by Maitiyaer et al.[117] presented the novel finding that multiple Belimumab treatments might be independently associated with reduced DCP density, warranting further investigation.

Clinical implications and future perspectives

In summary, OCTA technology profoundly reveals that ocular microvascular injury in SLE is an early-onset, widespread systemic pathological manifestation closely linked to systemic status. It not only provides crucial biomarkers for SLE microvasculopathy but also demonstrates significant value in assisting the diagnosis and risk assessment of severe complications like LN and NPSLE. Future longitudinal studies are needed to clarify the evolution of these microvascular parameters and their prognostic significance, thereby establishing their standardized application value in clinical practice and pharmaceutical clinical trials.

Dermatomyositis

Disease characteristics and ocular manifestations

Dermatomyositis is an autoimmune disease involving multiple organs and systems. Dermatomyositis may affect any ocular structure, leading to manifestations such as edema, periorbital redness, iritis, uveitis, glaucoma, and episcleritis[118,119]. Previous case reports have documented fundus findings including cotton-wool spot[120,121], retinal hemorrhage[120,122,123], macular edema[124], and Purtscher-like retinopathy[125,126]. Fluorescein angiography (FFA)[127] has demonstrated findings such as delayed vascular filling, retinal vascular obstruction, and capillary non-perfusion, while optical coherence tomography (OCT)[121] has revealed macular edema. Collectively, these clinical and imaging findings point to underlying vascular pathology in dermatomyositis.

Pathogenic mechanisms and vascular pathology

The pathogenesis of dermatomyositis is complex, with one key mechanism being complement activation and deposition within capillaries, causing capillary ischemia, microinfarction, and hypoperfusion. Histopathologically[128], skin and muscle biopsy specimens demonstrate endothelial cell injury, capillary reduction, and perivascular inflammatory infiltration. Therefore, vascular pathology plays a central role in the disease process of dermatomyositis.

OCTA findings in juvenile and adult dermatomyositis

Yılmaz Tuğan et al.[129] were the first to apply OCTA to the ocular evaluation of JDM patients. Their study found that in JDM children without evidence of clinical ocular involvement, the VD of the DCP was significantly lower compared to healthy controls and showed a significant negative correlation with disease duration. No significant differences were observed in parameters such as the SCP, optic disc vessel density, or FAZ area. These results suggest that the DCP may be the earliest target of microvasculopathy in JDM, and its VD parameter could potentially serve as a sensitive indicator for assessing the subclinical status of JDM.

Huang et al.[130] focused on adult DM patients with comorbid interstitial lung disease, concurrently analyzing both retinal thickness and microvascular density. The results demonstrated that DM patients had significant thinning of the full-thickness and inner retina in the nasal and inferior quadrants. Concurrently, the SCP in the corresponding areas was also reduced. Receiver Operating Characteristic (ROC) curve analysis revealed that retinal thickness and SCP VD parameters in the inferior inner region possessed high diagnostic efficacy for distinguishing DM patients from healthy controls. This reveals that in adult DM, microvascular hypoperfusion and neuroretinal structural atrophy may coexist, and OCTA can precisely capture this dual 'structure-function' alteration.

Clinical implications and diagnostic value

In summary, current research confirms the unique value of OCTA in detecting subclinical retinal microvasculopathy in DM/JDM. Distinct differences exist in OCTA findings between JDM and adult DM: JDM is characterized by early, specific reduction in flow in the DCP, whereas in adult DM, perfusion deficits in the SCP and structural remodeling of the neuroretina may be more prominent. This disparity likely stems from the combined influence of factors such as disease subtype, duration, complications (e.g., interstitial lung disease), and age.

Conclusions

Shared patterns and disease-specific features of ocular microvascular alterations

Current evidence derived from OCTA reveals that although the etiology and core pathological mechanisms of various rheumatic and immune-mediated diseases differ, their effects on the ocular microvasculature demonstrate notable commonalities and specificities.

The shared characteristics are primarily reflected in the following aspects. First, subclinical microvascular pathology is universally present, indicating that the ocular microcirculation serves as an early and sensitive indicator of systemic inflammation and vascular injury. Second, enlargement and irregularity of the FAZ are common fundus changes observed across multiple diseases. Furthermore,

microvascular damage exhibits layer-specific heterogeneity, with the DCP demonstrating heightened vulnerability. Finally, ocular microvascular parameters are closely associated with systemic disease status—such as disease activity, duration, and specific complications—making them a valuable window for assessing overall disease condition.

In terms of specificity, the manifestations of different diseases are closely linked to their distinct pathophysiological mechanisms: microvascular alterations in AS are associated with cardiovascular risk; evaluation in RA requires careful distinction between disease-related effects and drug toxicity; BD presents with typical occlusive vasculitis and a retina–conjunctiva vascular dichotomy; SSc is characterized by early choroidal involvement and synchrony with systemic microcirculatory changes; the dynamic progression of VKH clearly reflects the transition from acute choroidal ischemia to chronic microvascular rarefaction; research in psoriasis highlights the additive effects of disease severity and comorbidities; SS manifests as specific involvement of the posterior DCP and increased conjunctival vessel density in the anterior segment; the severity of microvascular impairment in SLE is strongly correlated with major systemic complications; and dermatomyositis exhibits distinct age-specific OCTA phenotypes.

In summary, the main similarities and characteristics of ocular microvascular alterations across different disease types are summarized in Table 1.

OCTA's ability to reveal distinct, disease-specific vascular phenotypes across different rheumatic conditions underscores its potential for early diagnosis, detection of subclinical disease, monitoring of disease activity, prognostic evaluation, and treatment response assessment, positioning it as a promising tool in the management of systemic rheumatologic disorders.

Current limitations and challenges in clinical translation

The current research landscape in this field is constrained by several important limitations. Existing evidence remains largely cross-sectional, with a notable scarcity of large-scale, long-term prospective cohort studies. This gap impedes the establishment of causal relationships and limits the validation of the predictive value of microvascular alterations.

The application and interpretation of OCTA in systemic rheumatic diseases face multiple challenges. Firstly, technical standardization is lacking: variations in algorithms, scanning protocols, and resolution across devices from different manufacturers hinder direct comparison of quantitative parameters between studies. Moreover, there is an absence of unified, multicenter diagnostic criteria for defining normative ranges and pathological changes. Secondly, issues related to data quality and interpretation persist. Image acquisition is susceptible to artifacts arising from patient motion and optical media opacities, which can compromise analytical accuracy. Additionally, many studies fail to adequately control for systemic cardiovascular risk factors—such as hypertension and diabetes—which represent significant confounders in microvascular assessment and may obscure disease-specific alterations. Furthermore, the clinical translation of OCTA faces substantive bottlenecks. While the modality demonstrates high sensitivity, its diagnostic specificity for individual diseases remains uncertain. The overlap of OCTA findings across different rheumatic conditions and with non-inflammatory microvasculopathies constrains its utility in differential diagnosis. Consequently, most OCTA-derived insights remain confined to the research domain and have yet to be incorporated into routine diagnostic algorithms or clinical guidelines. How dynamic changes in OCTA parameters should inform therapeutic decision-making requires validation through further prospective studies.

Future research directions and technological advancements

Looking ahead, the advancement of OCTA in systemic rheumatic diseases is expected to evolve along four key directions. First, there is a critical need for large-scale prospective studies to establish clear correlations between OCTA-derived parameters and clinical endpoints—such as visual outcomes, systemic disease activity, and cardiovascular events—thereby laying an evidence-based foundation for its integration into clinical practice. Second, technological methodologies are poised for substantial innovation. The application of artificial intelligence will enable in-depth analysis of OCTA datasets, facilitating automated lesion identification and refined prognostic prediction. Concurrently, the development of integrated multimodal imaging platforms—combining OCTA with modalities such as structural OCT and fundus autofluorescence—will support a more comprehensive ophthalmic assessment. Furthermore, research efforts will focus on the discovery and validation of novel biomarkers. This includes the development of sophisticated metrics derived from flow characteristics and vascular architecture, which may

Table 1. Similarities and characteristics of ocular microvascular alterations.

Disease	Similarities	Characteristics
AS	Subclinical involvement DCP vulnerability FAZ alterations Systemic correlations	Associated with cardiovascular disease risk; Sex-specific disparity (More pronounced in males).
RA		Distinguishing disease-related microvasculopathy from HCQ toxicity.
BD		Occlusive vasculitis hallmark; Retina-conjunctival vascular dichotomy; Irreversible DCP damage: Critical for visual prognosis.
SSc		Early choroidal involvement (primary target from VEDOSS stage); Systemic microcirculatory synchrony (nailfold capillaroscopy & skin score); Choroidal perfusion-thickness dissociation(reduced perfusion with increased thickness due to fibrosis).
VKH		Stage-specific progression: From acute choroidal ischemia to chronic microvascular rarefaction/remodeling; Sensitive treatment response (Improvement evident within two weeks); Early response predicts long-term complications.
Psoriasis		Microvascular damage correlates with skin disease severity (PASI score); Comorbidity additive effect (Markedly worse with PsA or MetS).
SS		Anterior segment vascular abnormality (Local chronic inflammatory marker: Increased conjunctival vessel density, correlates with disease duration/complement levels).
SLE		Strong correlation with severe systemic complications (lupus nephritis, neuropsychiatric SLE, subclinical atherosclerosis)
DM		Juvenile DM (JDM): Early selective DCP involvement; Adult DM: Retinal atrophy with concomitant hypoperfusion.

enhance the discrimination of disease-specific phenotypes. Finally, clinical insights gained through OCTA are anticipated to propel mechanistic investigations forward. By elucidating distinct microvascular injury patterns associated with specific rheumatic conditions, OCTA has the potential to bridge clinical observation with pathological inquiry, fostering a translational research continuum from bedside to bench and back.

Evolving clinical applications and multidisciplinary integration

OCTA technology is poised to evolve from a research tool into a vital clinical asset. It has the potential to become a comprehensive ocular evaluation platform for systemic rheumatic diseases, establishing baseline microvascular profiles during initial diagnosis while simultaneously assessing drug toxicity and systemic microvascular status. This transition will advance precision medicine by enabling risk stratification through the identification of high-risk patients and dynamic monitoring of treatment response, thus supporting treatment optimization. In research, OCTA parameters could serve as sensitive surrogate endpoints for evaluating new drugs' vasculoprotective effects, accelerating therapeutic development. Importantly, this technology will foster multidisciplinary collaboration, making ocular microcirculation assessment a key connection between rheumatology, ophthalmology, and related specialties—ultimately improving holistic disease management.

In conclusion, OCTA has transformed our understanding of microvascular pathology in systemic rheumatic diseases. With ongoing technological advances and research, it is expected to progress from an auxiliary tool to a core component of disease management, providing crucial support for early detection, precise treatment, and improved patient outcomes.

Ethical statements

Not applicable.

Author contributions

The authors confirm their contributions to the paper as follows: study conception and design: Wei J; draft manuscript preparation: Tao L; writing – review and editing: Shen X. All authors reviewed the results and approved the final version of the manuscript.

Data availability

All research data referenced in this analysis are contained in the present article and the cited literature.

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Conflict of interest

The authors declare that they have no conflict of interest.

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