OCTA-Based Detection of Retinal Microvascular Alterations in JIA Patients Without Ocular Involvement: Unveiling Subclinical Manifestations

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ABSTRACT

Purpose: This study evaluates retinal microvascular alterations in Juvenile Idiopathic Arthritis (JIA) patients without uveitis using Optical Coherence Tomography Angiography (OCTA) and compares findings with healthy controls.

Methods: A cross-sectional study included 42 JIA patients without uveitis and 48 age- and sex-matched controls. OCTA assessed superficial and deep capillary plexus vessel density, peripapillary retinal nerve fiber layer (PPRNFL) thickness, radial peripapillary capillary vessel density (RPC-VD), and foveal avascular zone (FAZ) area. Statistical analyses included independent samples t-test and Mann-Whitney U test for group comparisons.

Results: JIA patients showed reduced foveal vessel density in the superficial (20.33 ± 6.46% vs. 24.63 ± 6.46%, p = 0.003) and deep plexuses (37.00 ± 5.70% vs. 41.41 ± 5.70%, p = 0.002). PPRNFL thickness (120.62 ± 15.44 µm vs. 110.77 ± 15.44 µm, p = 0.001) and RPC-VD (51.76 ± 3.05% vs. 49.85 ± 3.05%, p = 0.002) were elevated. FAZ area showed a non-significant trend toward enlargement (p = 0.102). Multivariate regression confirmed these findings were independent of age, sex, and disease duration.

Conclusion: OCTA reveals subclinical retinal microvascular changes in JIA, suggesting early inflammatory effects. These findings support OCTA as a non-invasive tool for detecting vascular involvement, though a multimodal approach is recommended. Longitudinal studies are needed to assess progression and its clinical impact.

Keywords: Juvenile Idiopathic Arthritis, Optical Coherence Tomography Angiography, Retinal Microvasculature, Systemic Inflammation, Subclinical Ocular Changes.

INTRODUCTION

Juvenile Idiopathic Arthritis (JIA) is the most common pediatric rheumatic disorder, characterized by chronic joint inflammation beginning before 16 years of age.[1] Beyond articular manifestations, systemic inflammation affects multiple organs, including the eye. While anterior uveitis affects 10-20% of JIA patients and has been extensively studied, subclinical microvascular changes may occur even without overt ocular inflammation.[2,3]

The pathophysiology involves inflammatory cascades mediated by cytokines including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which induce endothelial activation and vascular permeability changes.[4] These processes may manifest as retinal microvascular alterations preceding clinically detectable signs.

Optical Coherence Tomography Angiography (OCTA) provides non-invasive, high-resolution visualization of retinal microvasculature using split-spectrum amplitude-decorrelation angiography (SSADA) algorithms. OCTA quantifies vessel density (VD), flow area, and foveal

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avascular zone (FAZ) parameters, demonstrating utility in detecting microvascular alterations in various ocular pathologies.[5,6]

Despite growing OCTA applications in inflammatory diseases, its role in evaluating subclinical changes in JIA patients without uveitis remains underexplored. Previous studies primarily focused on anterior segment manifestations or included heterogeneous cohorts with and without uveitis, limiting understanding of isolated systemic inflammation effects on retinal microvasculature.[7,8]

This study investigates retinal microvascular alterations in JIA patients without uveitis using OCTA, comparing findings with healthy controls. We hypothesize that despite absent clinical uveitis, JIA patients exhibit significant retinal microvascular parameter alterations reflecting systemic inflammatory burden.

MATERIALS AND METHODS

Study Design and Participant Selection

We conducted a cross-sectional study at a tertiary university hospital following Declaration of Helsinki principles. The Institutional Review Board approved the protocol, and written informed consent was obtained from all participants or guardians.

Two cohorts were established: JIA patients (without uveitis history) and healthy controls. The JIA group comprised patients aged 4-16 years with confirmed JIA diagnosis according to International League of Associations for Rheumatology (ILAR) criteria.[9] Controls were age- and sex-matched healthy children without systemic or ocular diseases.

Inclusion and Exclusion Criteria

JIA group inclusion criteria: (1) age 4-16 years; (2) confirmed JIA diagnosis per ILAR criteria; (3) adequate cooperation for OCTA examination. Control inclusion criteria: (1) age-matched to JIA cohort; (2) absence of systemic inflammatory conditions; (3) normal ocular examination.

Exclusion criteria for both groups: (1) uveitis history; (2) prior ocular surgery/trauma; (3) refractive error exceeding ±6.00 diopters; (4) media opacities compromising image quality; (5) inadequate fixation; (6) other ocular diseases. Patients receiving biologic agents were noted but not excluded.

Clinical Assessment Protocol

All participants underwent comprehensive masked ophthalmic evaluation including:

1. Best-Corrected Visual Acuity (BCVA) using Snellen charts converted to logMAR
2. Slit-lamp biomicroscopy with anterior chamber assessment per Standardization of Uveitis Nomenclature (SUN) criteria
3. Intraocular pressure (IOP) measurement via Goldmann applanation tonometry
4. Axial length (AL) determination using IOLMaster (Carl Zeiss Meditec, Jena, Germany)
5. Central corneal thickness (CCT) evaluation with Canon TX-20P tonometer
6. Spherical equivalent (SE) calculation following cycloplegic refraction
7. Dilated fundus examination
8. Spectral-Domain OCT for peripapillary RNFL thickness, central macular thickness (CMT), and subfoveal choroidal thickness using Heidelberg Spectralis

For JIA patients, additional parameters included JIA subtype, disease duration, current medications, and Juvenile Arthritis Disease Activity Score (JADAS).[10]

Optical Coherence Tomography Angiography Protocol

OCTA imaging used the Optovue AngioVue system (RTVue XR Avanti) with SSADA algorithm. Following pupillary dilation and 5-minute rest, three consecutive scans were acquired, selecting the highest quality scan based on signal strength index (SSI) and motion artifact absence.

Macular Microvascular Assessment

The 6 × 6 mm macular scanning protocol evaluated:

- Superficial capillary plexus (SCP): internal limiting membrane to 10 μm above inner plexiform layer

- Deep capillary plexus (DCP): 10 µm above to 10 µm below outer plexiform layer

Vessel density was calculated for whole image, foveal (1mm diameter), parafoveal (1-3mm ring), and perifoveal (3-6mm ring) regions.

Optic Nerve Head Assessment

The 4.5 × 4.5 mm optic disc protocol evaluated radial peripapillary capillaries (RPCs) within 2-4mm annular regions of interest.

Foveal Avascular Zone Analysis

Automated FAZ delineation measured area, perimeter, and foveal density within 300µm (FD-300).

Quality Control

OCTA scans were excluded if: SSI < 7/10, significant motion artifacts, segmentation errors, or media opacities affected quality. Two independent masked graders performed measurements, averaging results or using median values for >10% discrepancies.

Statistical Analysis

Statistical analyses used SPSS version 25.0. Sample size calculation based on previous OCTA inflammatory condition studies aimed to detect 10% vessel density differences with 80% power at $p<0.05$ significance.

Normality was assessed using Shapiro-Wilk test. Descriptive statistics presented as mean ± standard deviation for normal distributions and median (interquartile range) for non-normal distributions.

Group comparisons used independent samples t-test for normally distributed variables and Mann-Whitney U test for non-normally distributed variables. Chi-square test evaluated categorical variables.

Correlation analyses between OCTA parameters and clinical variables used Pearson's or Spearman's coefficients. Multivariate linear regression models identified independent predictors, adjusting for age, sex, and axial length. ROC curve analysis evaluated OCTA parameter diagnostic performance. Two-tailed p-values <0.05 were considered significant, with Bonferroni correction for multiple comparisons when appropriate.

RESULTS

Clinical Characteristics

The study included 42 JIA patients without uveitis and 48 healthy controls with comparable sex distribution ($p = 0.114$). Baseline ocular parameters including spherical equivalent, IOP, CCT, and CMT showed no significant differences (all $p > 0.05$) (Table 1).

Vessel Density Analysis

Foveal Vessel Density Changes

Quantitative assessment revealed significantly reduced foveal vascular density in JIA patients:

- SCP-VD at fovea: 20.33 ± 6.46% (JIA) vs. 24.63 ± 6.46% (controls), ($p = 0.003$)
- DCP-VD at fovea: 37.00 ± 5.70% (JIA) vs. 41.41 ± 5.70% (controls), ($p = 0.002$)

Table 1. *Baseline Clinical and Demographic Characteristics of the Study Cohort*

Parameter	JIA (n = 42)	Control (n = 48)	p-value
Male, n (%)	19 (45.2%)	14 (29.2%)	0.114 (Chi-square test)
Female, n (%)	23 (54.8%)	34 (70.8%)	
Spherical Equivalent (SE)	–0.69 ± 1.23	–0.88 ± 1.23	0.410*
Intraocular Pressure (IOP, mmHg)	17.93 ± 3.51	18.71 ± 3.51	0.317*
Corrected IOP (cIOP, mmHg)	17.29 ± 3.28	18.02 ± 3.28	0.341*
Central Corneal Thickness (CCT) (µm)	549.14 ± 35.29	554.08 ± 35.29	0.505**
Central Macular Thickness (CMT, µm)	241.02 ± 21.28	242.21 ± 21.28	0.607*
Choriocapillary Blood Flow Area (CBFA)	0.973 ± 0.477	0.830 ± 0.477	0.152*
Juvenile Arthritis Disease Activity Score (JADAS)	10.82 ± 10.41	–	–

*Mann–Whitney U test; **Independent samples T test.

No significant differences occurred in parafoveal or perifoveal vessel densities (all p > 0.05) (Tables 2 and 3).

Peripapillary Structural and Microvascular Changes

Analysis demonstrated:

- Mean PPRNFL thickness: 120.62 ± 15.44 µm (JIA) vs. 110.77 ± 15.44 µm (controls), (p = 0.001)
- PPRNFL thickness increases in superior (p = 0.006) and inferior (p = 0.001) quadrants
- Mean RPC-VD: 51.76 ± 3.05% (JIA) vs. 49.85 ± 3.05% (controls), (p = 0.002), with significant superior and inferior quadrant increases (p = 0.001 for both) (Table 4)

Table 2. *Evaluation of Superficial Capillary Plexus Vessel Density (SCP-VD)*

Region	JIA (Mean ± SD)	Control (Mean ± SD)	p-value
Fovea (%)	20.33 ± 6.46	24.63 ± 6.46	**0.003**
Parafovea (%)	53.39 ± 3.63	53.67 ± 3.63	0.856*
Temporal (%)	52.89 ± 4.16	53.59 ± 4.16	0.552*
Superior (%)	54.51 ± 4.01	54.40 ± 4.01	0.129**
Nasal (%)	52.35 ± 4.40	52.45 ± 4.40	0.562**
Inferior (%)	53.82 ± 4.07	54.23 ± 4.07	0.234**
Perifovea (%)	51.87 ± 2.60	51.57 ± 2.60	0.407*
Temporal (%)	48.22 ± 3.38	48.87 ± 3.38	0.324*
Superior (%)	51.93 ± 2.74	51.13 ± 2.74	0.081*
Nasal (%)	55.23 ± 2.67	54.94 ± 2.67	0.462*
Inferior (%)	52.13 ± 3.00	51.42 ± 3.00	0.273*

*Mann–Whitney U test; **Independent samples T test.
Bold p-values indicate statistically significant differences.

Table 3. *Evaluation of Deep Capillary Plexus Vessel Density (DCP-VD)*

Region	JIA (Mean ± SD)	Control (Mean ± SD)	p-value
Fovea (%)	37.00 ± 5.70	41.41 ± 5.70	**0.002**
Parafovea (%)	55.56 ± 4.70	56.49 ± 4.70	0.378*
Temporal (%)	56.11 ± 5.14	57.76 ± 5.14	0.234*
Superior (%)	55.36 ± 4.92	56.81 ± 4.92	0.139*
Nasal (%)	56.84 ± 5.32	55.87 ± 5.32	0.186*
Inferior (%)	53.86 ± 5.37	55.53 ± 5.37	0.073*
Perifovea (%)	52.44 ± 6.53	54.91 ± 6.53	0.061*
Temporal (%)	54.78 ± 5.84	56.83 ± 5.84	0.120*
Superior (%)	51.73 ± 7.29	54.63 ± 7.29	0.060*
Nasal (%)	51.41 ± 7.29	53.92 ± 7.29	0.056*
Inferior (%)	52.11 ± 7.35	54.23 ± 7.35	0.131*

*Mann–Whitney U test; **Independent samples T test.
Bold p-values indicate statistically significant differences.

Table 4. *Peripapillary RNFL Thickness, FAZ Area, and RPC Parameters*

Parameter	JIA (Mean ± SD)	Control (Mean ± SD)	p-value
FAZ area (mm²)	0.291 ± 0.081	0.253 ± 0.081	0.102*
PPRNFL mean (µm)	120.62 ± 15.44	110.77 ± 15.44	**0.001**
PPRNFL superior (µm)	120.29 ± 17.07	111.52 ± 17.07	**0.006**
PPRNFL inferior (µm)	121.79 ± 17.00	110.02 ± 17.00	**0.001**
RPC-VD mean (%)	51.76 ± 3.05	49.85 ± 3.05	**0.002**
RPC-VD superior (%)	52.20 ± 2.46	49.89 ± 2.46	**0.001**
RPC-VD inferior (%)	52.50 ± 2.32	49.82 ± 2.32	**0.001**
RPCID (%)	51.39 ± 3.78	52.47 ± 3.78	0.128*

*Mann–Whitney U test; **Independent samples T test.
Bold p-values indicate statistically significant differences.

Foveal Avascular Zone and Central Macular Thickness

- FAZ area showed non-significant enlargement trend in JIA (0.291 ± 0.081 mm² vs. 0.253 ± 0.081 mm², p = 0.102)
- CMT was comparable between groups (p = 0.607)

Correlation Analysis

FAZ area negatively correlated with CMT (r = -0.41, p < 0.05), SCP-VD (r = -0.37, p < 0.05), and DCP-VD (r = -0.39, p < 0.05). No significant associations occurred between vessel densities and disease duration (all p > 0.05).

Multivariate Analysis

Multivariate linear regression adjusting for age, sex, and disease duration confirmed PPRNFL thickness and RPC-VD remained significantly elevated in JIA patients (adjusted p < 0.01). DCP-VD reduction remained significant (adjusted p = 0.004).

Diagnostic Performance

ROC analysis showed CMT had highest AUC (0.675), followed by superior SCP-VD (AUC = 0.640) and superior PPRNFL thickness (AUC = 0.640). No OCTA parameter achieved statistical significance for disease classification (all p > 0.05) (Figure 1).

DISCUSSION

This study provides compelling evidence of subclinical retinal microvascular alterations in JIA patients without uveitis, demonstrating that systemic inflammation impacts ocular vasculature even without overt inflammatory signs. Using OCTA, we identified reduced vessel density in the deep capillary plexus of the fovea, increased peripapillary RNFL thickness, and elevated radial peripapillary capillary vessel density. These findings extend beyond earlier structural OCT studies that focused on macular abnormalities in JIA patients with active uveitis[11], shifting toward detecting subclinical vascular changes in uveitis-free patients.

The reduction in foveal DCP-VD suggests that deeper retinal capillaries, with their high metabolic demand, are particularly vulnerable to systemic inflammatory mediators characteristic of JIA pathophysiology. Pro-inflammatory cytokines such as IL-6, TNF-α, and IL-17, elevated in JIA patients, can induce endothelial dysfunction and capillary rarefaction through increased oxidative stress and impaired angiogenic signaling.[12] The increased PPRNFL thickness and RPC-VD likely represent an inflammatory response characterized by subclinical optic disc edema or hyperemia, driven by angiogenic factors including VEGF and angiopoietin-2 that are known to be altered in JIA-associated ocular inflammation.[13]

Our findings show important similarities and differences compared to previous JIA OCTA studies. Tuğan et al.[14] reported decreased deep parafoveal vessel density in oligoarticular JIA patients without uveitis, corroborating our results regarding DCP involvement. However, our study identified this reduction at the foveal center, whereas their study focused on the parafoveal region. This difference may reflect variations in JIA subtypes or OCTA

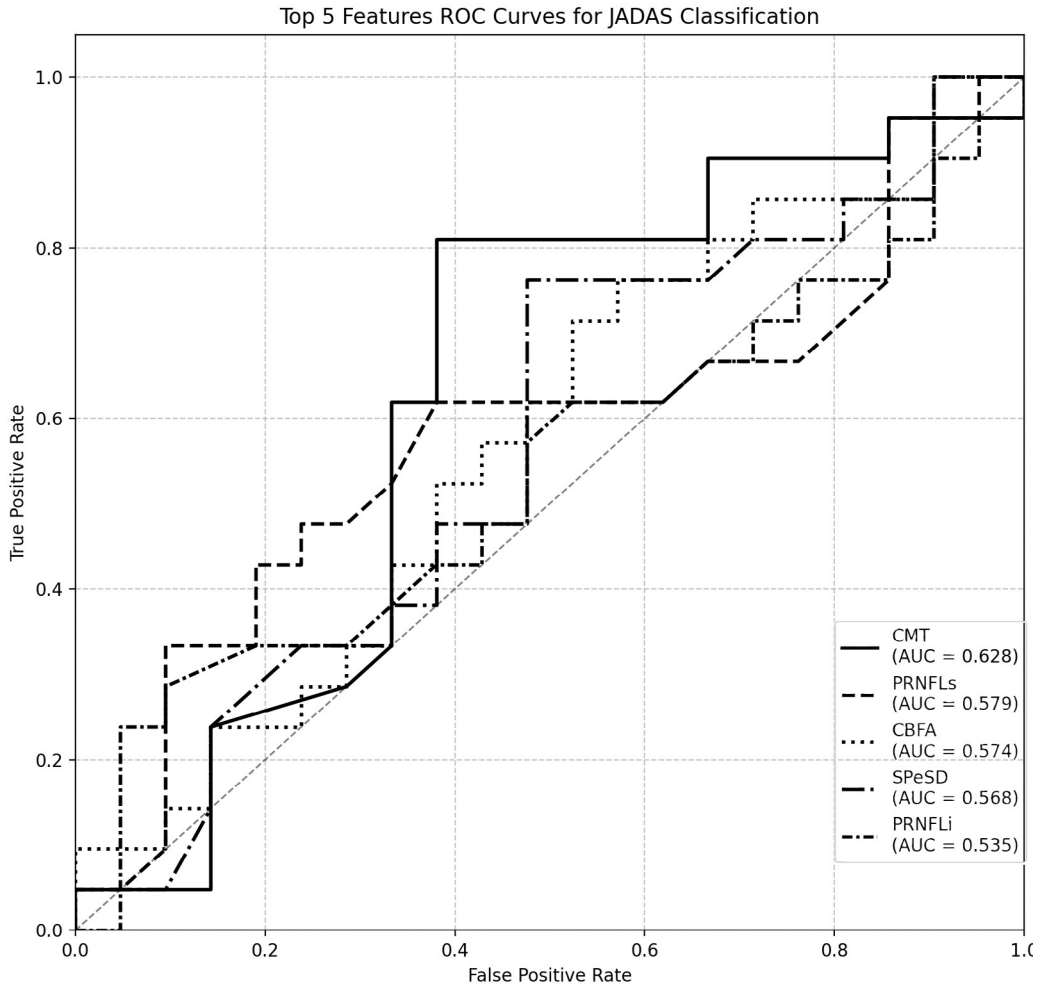

Figure 1. *Receiver Operating Characteristic (ROC) Curves Analysis of Key Ophthalmic Parameters for Discriminating Disease Activity in JIA Patients*

Legend: ROC curves illustrate the diagnostic performance of five key ophthalmic measurements in differentiating disease activity levels in JIA patients ($p > 0.05$ for all). Central macular thickness (CMT) exhibited the highest discriminative ability (AUC = 0.628), followed by superior peripapillary retinal nerve fiber layer (PPRNFL) thickness (AUC = 0.579), superior SCP-VD (AUC = 0.568), choroidal blood flow area (CBFA) (AUC = 0.574), and inferior PPRNFL thickness (AUC = 0.535). The diagonal dashed line represents random chance (AUC = 0.5).

Abbreviations: AUC: Area Under the Curve; JIA: Juvenile Idiopathic Arthritis; CMT: Central Macular Thickness; PPRNFL: Peripapillary Retinal Nerve Fiber Layer; SCP-VD: Superficial Capillary Plexus Vessel Density; CBFA: Choroidal Blood Flow Area.

analysis protocols. Elnahry et al.[15] found reduced SCP and DCP densities in both macular and peripapillary regions, suggesting more widespread vascular involvement than our findings. Their study reported more extensive macular changes, which may be attributed to differences in disease activity levels or treatment regimens.

Recent evidence from Nedealcova et al.[16] reinforces that retinal vasculature peculiarities are consistent findings across JIA populations. Furthermore, Ağın et al.[17] demonstrated altered choroidal vascularity index in JIA patients, suggesting both retinal and choroidal vascular beds are affected by subclinical inflammation. These complementary findings may indicate multilayer vascular involvement in JIA pathophysiology.

The variations across studies may arise from differences in JIA subtypes, disease activity levels, treatment protocols,

OCTA devices, and analysis methods. Dingerkus et al.[18] highlighted OCTA's emerging role as a diagnostic tool in uveitis, emphasizing the need for standardized protocols across different inflammatory conditions. Our findings align with this broader context while specifically addressing the JIA population without clinical uveitis.

The clinical implications are significant for JIA patient management. The observed microvascular changes could serve as non-invasive biomarkers for monitoring subclinical disease activity. Reduced DCP-VD might indicate early ischemic damage requiring closer ophthalmologic follow-up, while increased PPRNFL thickness and RPC-VD could signal ongoing inflammation warranting treatment optimization. OCTA offers clear advantages over invasive methods like fluorescein angiography, particularly in pediatric populations where contrast-free imaging is preferred.[19]

However, our ROC analysis showed individual OCTA parameters had only moderate diagnostic ability, underscoring that a multi-parameter approach is necessary. Integration of OCTA findings with clinical disease activity scores, systemic inflammatory markers, and cytokine profiles is likely required for effective patient management and early detection of subclinical ocular involvement.

This study has limitations including its cross-sectional design preventing assessment of temporal changes, and relatively modest sample size limiting subgroup analyses. Future research should prioritize larger, multicenter longitudinal studies to track OCTA parameter changes over time, compare findings across JIA subtypes, and evaluate treatment impacts on retinal microvascular changes. Standardization of OCTA protocols across devices and centers is essential for comparability and clinical implementation.

In conclusion, OCTA reveals subclinical retinal microvascular changes in JIA patients without clinical uveitis, suggesting early inflammatory effects on ocular vasculature. These findings contribute to growing evidence that JIA-associated ocular involvement extends beyond clinically apparent uveitis. While OCTA shows promise as a monitoring tool, integration with clinical and laboratory parameters is essential for optimal patient management.

STATEMENTS & DECLARATIONS

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the Gaziantep University Gaziantep, Turkey, and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from all individual participants included in the study.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the authors used QuillBot tool to improve language and readability, with caution. After using this tool/service, the authors reviewed and edited the content as needed and took full responsibility for the content of the publication.

Author Contribution Statement

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by İEY, VD, MM, SAS, LD and GG. The first draft of the manuscript was written by İEY and all authors commented on previous versions of the manuscript. All authors read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of Interest

The authors declare no conflict of interest.

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All authors certify that this work is original and has not been published elsewhere, nor is it currently under consideration for publication anywhere.

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