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# Retinal Nerve Fiber Layer Thickness and Contrast Sensitivity in Human Immunodeficiency Virus patients

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**Abstract**

Objective: To evaluate retinal nerve fiber layer (RNFL) thickness, measured by optical coherence tomography (OCT), and contrast sensitivity (CS) in HIV-positive patients compared to HIV-negative controls at the Yaounde University Teaching Hospital.

Patients and Methods: This prospective, comparative study included 51 HIV-positive patients (group 1) and 51 HIV-negative controls (group 2). All participants underwent peripapillary RNFL OCT and CS testing. The following variables were analyzed: age, gender, HIV disease duration, antiretroviral (ARV) therapy duration, lowest CD4 T-cell count, highest viral load, recent viral load (within the last 6 months), RNFL thickness, and CS score.

Results: The mean age was similar in both groups (50.4 ± 7.1 years for group 1 and 50.2 ± 7.1 years for group 2). The median HIV disease duration in group 1 was 13 years (IQR: 9-18 years), and 60% had a CD4 cell count below 200 cells/ml during the disease course. All patients had undetectable viral loads within the last 6 months. HIV-positive patients had significantly thinner mean RNFL thickness (106.6 ± 12.2 µm) compared to controls (117 ± 7.2 µm) ($p < 0.001$). Similarly, the mean CS score was significantly reduced in the HIV-positive group (1.3 ± 0.2) compared to the control group (1.51 ± 0.7) ($p < 0.001$). In the HIV-positive group, RNFL thickness increased with CD4 T-cell count ($r = 0.7$, $p < 0.001$) and decreased with peak viral load ($r = -0.50$, $p = 0.007$). Contrast sensitivity was inversely proportional to peak viral load ($r = -0.4$, $p = 0.022$), and there was a statistically significant positive correlation between CS and mean RNFL thickness ($r = 0.33$, $p = 0.018$).

Conclusion: HIV infection is associated with reduced RNFL thickness and contrast sensitivity. Low CD4 T-cell count and high peak viral load are correlated with greater reductions in RNFL thickness and contrast sensitivity.

**Keywords:** Human Immunodeficiency Virus; Retinal Nerve Fiber Layer Thickness; Contrast Sensitivity; Optical Coherence Tomography

**Abbreviations:** RNFL: Retinal Nerve Fiber Layer; OCT: Optical Coherence Tomography; CS: Contrast Sensitivity; ARV: Antiretroviral; HIV: Human Immunodeficiency Virus; ART: Antiretroviral Therapy; VA: Visual Acuity; YUTH: Yaoundé University Teaching Hospital; FMSB: Faculty Of Medicine and Biomedical Sciences

## Introduction

Human Immunodeficiency Virus (HIV) infection remains a global pandemic, affecting an estimated 38.4 million people worldwide in 2021, with over two-thirds residing in Africa. It continues to pose a significant public health challenge. HIV primarily targets the immune system, weakening the body's defenses against opportunistic infections and certain cancers [1]. During HIV infection, multi-organ involvement is common, with ocular manifestations occurring in over 50% of cases [2-4]. These can arise from opportunistic infections or cancerous processes [3,4]. While antiretroviral therapy (ART) has decreased the prevalence of retinal opportunistic infections [5,6], HIV-associated neuroretinal disorders (HIV-NRD) have emerged as a concern despite effective viral suppression. These disorders are characterized by alterations in retinal nerve fiber layer (RNFL) thickness [7-9], primarily thinning, resulting from direct neural damage by HIV, the host's immune response, and accelerated degenerative processes [5,9,10]. Specifically, chronic inflammation and persistent immune activation may cause neurodegeneration, leading to RNFL thinning.

HIV-NRD are associated with visual function abnormalities, including reduced contrast sensitivity (CS), impaired color vision, and visual field defects [8,9,11]. Diminished CS is as debilitating as visual field loss and more so than reduced visual acuity (VA), impacting everyday activities such as reading, facial recognition, night driving, and mobility [12-14]. In Cameroon, approximately 2.7% of the population is living with HIV [15]. A study in Douala revealed a 63.2% prevalence of ocular complications among HIV-infected individuals in Cameroon [4]. However, the specific impact of HIV on RNFL in our context remains poorly understood. Therefore, this study aims to evaluate RNFL thickness and contrast sensitivity in patients living with HIV in Cameroon."

## Materials and Methods

### Study Population

This prospective, cross-sectional, comparative study was conducted at the Yaoundé University Teaching Hospital (YUTH) between October 2022 and October 2023. The study included 51 adult HIV-positive patients (group 1) and 51 HIV-negative adult controls (group 2). Exclusion criteria included: diabetes, hypertension, glaucoma, any condition causing media opacity, vitreoretinal pathology, or use of known oculotoxic medications (e.g., chloroquine, ethambutol) within the preceding year. The study was performed in accordance with the Declaration of Helsinki. The institutional ethics committee of the Faculty of Medicine and Biomedical Sciences (FMSB) at the University of Yaoundé I approved the protocol (number 0131/UYI/FMSB/VDRC/DAASR/CSD), and written informed consent was obtained from all participants after a thorough explanation of the procedures. Cases (group 1) consisted of HIV-positive patients receiving care at the YUTH and Central Hospital of Yaoundé. Controls (group 2) were HIV-negative individuals presenting for consultation at the ophthalmology department of the YUTH, matched to cases based on age and gender.

### Data Collection

All participants underwent a comprehensive ophthalmological examination, including visual acuity measurement and slit-lamp biomicroscopy of the anterior segment. Clinical and biological HIV-related parameters were collected from patient medical records: disease duration, antiretroviral therapy duration, lowest CD4+ T-cell count, peak viral load, and viral load within the past six months. Peripapillary RNFL thickness was measured using optical coherence tomography (OCT) with a Topcon Maestro 3D device. After entering patient details (identification number, age, sex, race [African]), participants were positioned comfortably with their chin and forehead stabilized. Images were acquired using the optic nerve head assessment mode with a green cross as an internal fixation target. The device's integrated mapping software was used for analysis. Monocular contrast sensitivity (CS) was assessed with best-corrected visual acuity in a well-lit room using the Compact Acuity Tester Vision device, following the manufacturer's instructions. Participants were seated 3 meters from a screen displaying optotypes (letters) in seven rows with decreasing contrast (2% to 100%; CS score: 0-1.69). Each row contained five optotypes sized according to the Snellen acuity scale. A descending method was used, and the contrast threshold was defined as the lowest contrast level at which the participant correctly identified at least four of the five letters in a row. A CS score below 1.5 was considered abnormal [10].

## Statistical Analysis

Data were analyzed using SPSS version 26.0. Categorical variables are presented as frequencies and percentages, and continuous variables as mean ± standard deviation or median with interquartile range (IQR), as appropriate. Comparisons of continuous variables were performed using Student's t-test or Mann-Whitney U test, and comparisons of categorical variables using the chi-squared test or Fisher's exact test. Correlations were assessed using Pearson's or Spearman's correlation coefficients. Statistical significance was defined as $p < 0.05$.

## Results

The analysis included 102 participants (204 eyes): 51 HIV-positive patients (group 1; 102 eyes) and 51 HIV-negative controls (group 2; 102 eyes), comprising 68.6% men and 31.4% women. Sixty percent of HIV-positive patients (30/51) had a nadir CD4+ T-cell count below 200 cells/µL during the disease course. The median peak viral load among HIV-positive patients was 17,767 copies/mL (IQR: 180-172,269 copies/mL). All HIV-positive patients had an undetectable viral load within the past six months. The mean peripapillary RNFL thickness was significantly lower in group 1 (106.6 ± 12.2 µm) than in group 2 (117 ± 7.2 µm; $p < 0.001$). (Figures 1 and 2) illustrate the mean RNFL thickness per quadrant for groups 1 and 2, respectively. (Table 1) presents a comparison of mean and quadrant RNFL thickness between the two groups . The mean thickness differences in RNFL per quadrant between the two groups are summarized in (Figure 3).

**Table 1:** Comparison of mean and quadrant RNFL thickness between the 2 groups.

| Variables | HIV+ (N=51) | HIV- (N=51) | P |
|---|---|---|---|
| | Mean ± SD | Mean ± SD | |
| RNFL mean thickness | 106.6 ± 12.2 | 117 ± 7.2 | < 0.001 |
| RNFL superior thickness | 135 ± 17.4 | 151 ± 13 | < 0.001 |
| RNFL inferior thickness | 143.6 ± 20 | 153,1 ± 1.7 | 0.005 |



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| | | | |
|---|---|---|---|
| RNFL temporal thickness | 6.6 ± 15 | 73,5 ± 8.9 | < 0.001 |
| RNFL nasal thickness | 81.2 ± 14 | 89.9 ± 8 | < 0.001 |

HIV: Human Immunodeficiency Virus; RNFL : Retinal Nerve Fiber Layer; SD: Standard Deviation

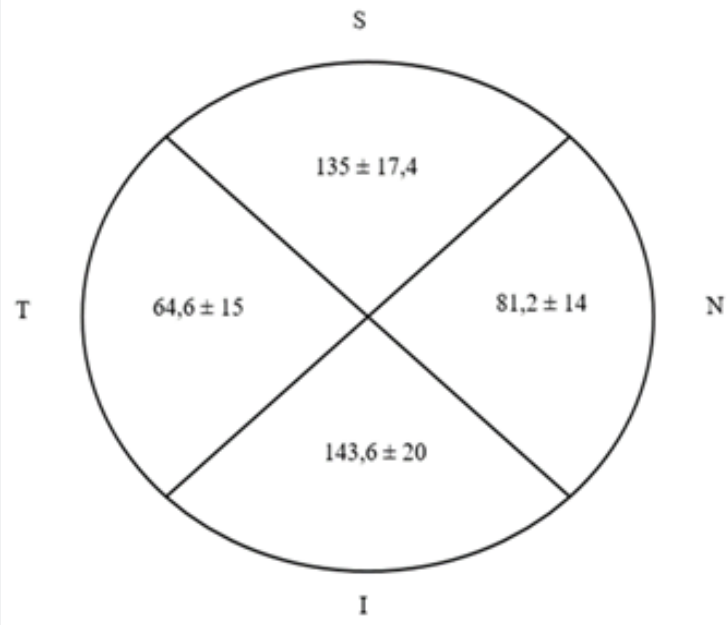


**Figure 1:** Mean RNFL thickness per quadrant for group 1 patients ( S=Superior, I=Inferior, T=Temporal, N=Nasal, Unit=µm).

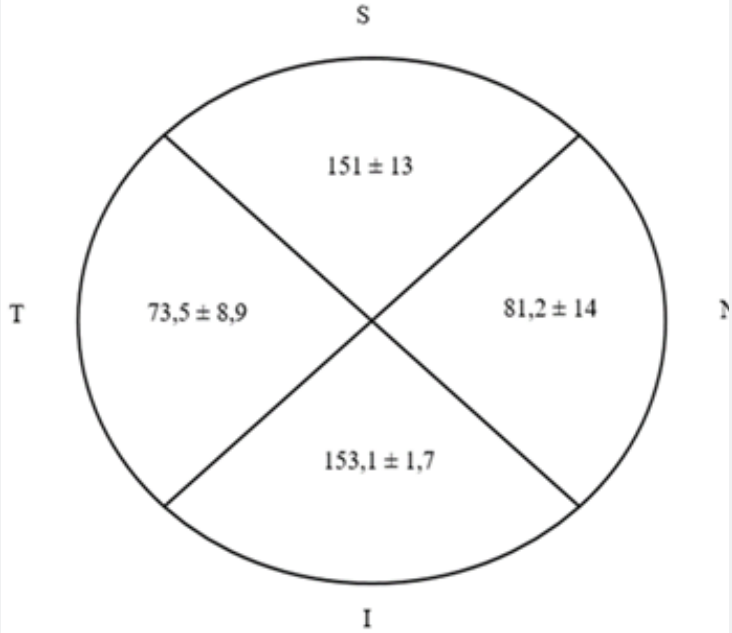


**Figure 2:** Mean RNFL thickness per quadrant for Group 2 patients (1=Internal 2=External, S=Superior, I=Inferior, T=Temporal, N=Nasal).



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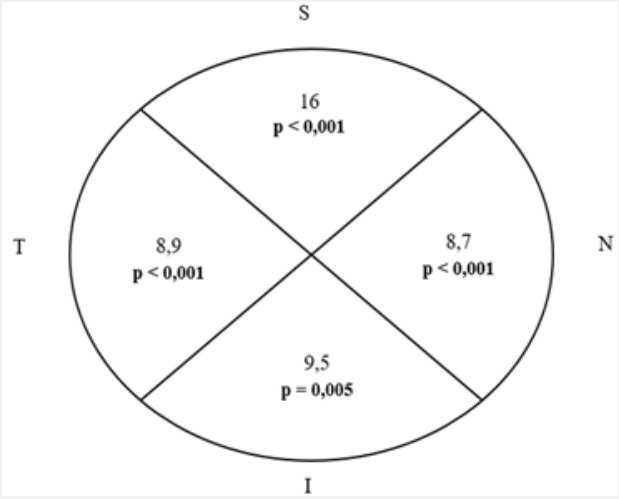


**Figure 3:** Mean RNFL thickness differences per quadrant between the 2 groups (1=Internal 2=External, S=Superior, I=Inferior, T=Temporal, N=Nasal).

The frequency of abnormal contrast sensitivity (CS) scores was significantly higher in group 1 (45.1%) than in group 2 (3.9%; p < 0.001). Contrast sensitivity scores of the two groups are compared in (Table 2). In the HIV-positive group, mean RNFL thickness showed a significant positive correlation with CD4+ lymphocyte count (r = 0.7, p < 0.001; (Figure 4) and a significant negative correlation with peak HIV viral load (r = -0.5, p = 0.007; (Figure 5). Similarly, contrast sensitivity was significantly negatively correlated with peak viral load (r = -0.4, p = 0.022; (Figure 6). Contrast sensitivity showed a positive correlation with mean RNFL thickness (r = 0.33, p = 0.018) and temporal RNFL thickness (r = 0.39, p = 0.005) (Figure 7). (Table 3) summarizes the associations between mean RNFL thickness, CD4+ lymphocyte count, and peak HIV viral load in group 1. No significant correlations were found between peripapillary RNFL thickness and age, HIV disease duration, or ARV therapy duration.

**Table 2:** Comparison of contrast sensitivity scores between the 2 groups.

| Variables | HIV + | HIV - | P |
|---|---|---|---|
| | Mean ± SD | Mean ± SD | |
| Mean CS Score | 1.3 ± 0.2 | 1.51 ± 0.7 | < 0.001 |
| CS Score | Frequency | Frequency | |
| | N=51 | N=51 | |
| Score de SC < 1.5 | 23 (45.1) | 2 (39) | < 0.001 |
| Score de SC ≥ 1.5 | 28 (54.9) | 49 (96.1) | |

CS: Contrast Sensibility; HIV: Human Immunodeficiency Virus; SD: Standard Deviation

**Table 3:** Correlation between the mean RNFL thickness of group 1 patients and variables such as age, duration of HIV disease, duration of ARV treatment, CD4 cell count, viral load.

| | Age | CD4 Cell Count | HIV Duration | Duration of ARVT | Hiv viral Load |
|---|---|---|---|---|---|
| RNFL Mean Thickness | | | | | |
| R | -0.2 | 0.7 | -0.1 | -0.2 | -0.5 |
| P | 0.221 | < 0.001 | 0.424 | 0.216 | 0.007 |
| RNFL Superior Thickness | | | | | |
| R | -0.2 | 0.4 | -0.2 | -0.3 | -0.1 |
| P | 0.146 | 0.002 | 0.124 | 0.04 | 0.453 |
| RNFL Inferior Thickness | | | | | |
| R | -0.1 | 0.6 | -0.2 | -0.2 | -0.3 |
| P | 0.332 | < 0.001 | 0.189 | 0.112 | 0.166 |



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| RNFL Temporal Thickness | | | | | |
|---|---|---|---|---|---|
| R | -0.2 | 0.5 | 0.1 | -0.1 | -0.5 |
| P | 0.22 | < 0.001 | 0.914 | 0.893 | 0.004 |
| RNFL nasal thickness | | | | | |
| R | 0 | 0,2 | 0,1 | 0,1 | -0,6 |
| P | 0.962 | 0.14 | 0.403 | 0.607 | 0.001 |

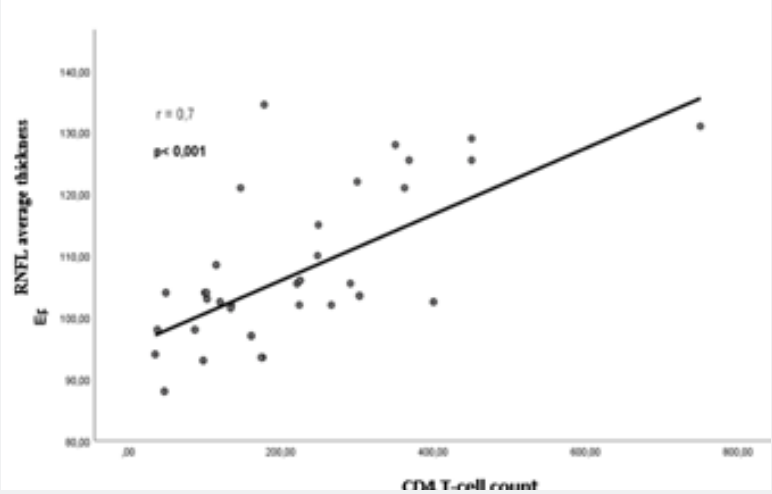


**Figure 4:** Correlation between mean RNFL thickness and CD4 T-cell count.

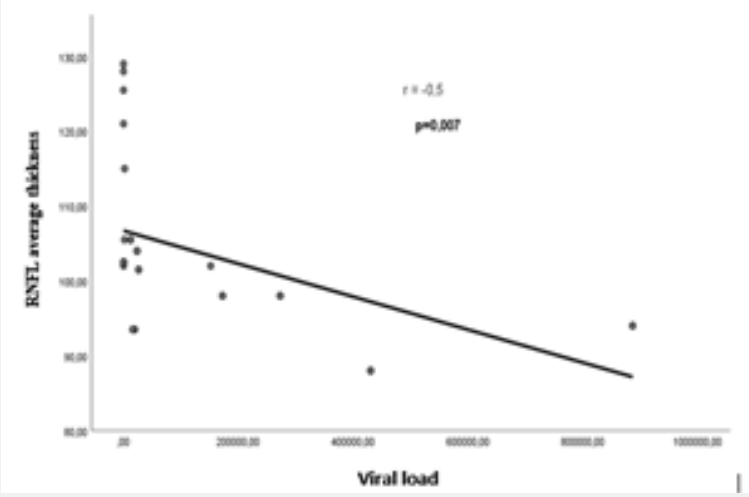


**Figure 5:** Correlation between mean RNFL thickness and HIV viral load.

## Discussion

The 51 HIV-positive patients in this study, all with undetectable viral loads and no other ocular complications, underscores the effectiveness of follow-up care and patient adherence to antiretroviral (ARV) treatment. The average age and female predominance in our study population align with the established epidemiology of HIV [11]. Sixty percent (30/51) of patients had a nadir CD4+ T-cell count below 200 cells/µL, indicating significant prior immunosuppression. Optical coherence tomography (OCT) is now a valuable tool for identifying structural changes in the RNFL of HIV-positive individuals, including both thinning and thickening. In the present study, OCT revealed a significant


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thinning of the mean RNFL thickness in HIV-positive patients (group 1) compared to HIV-negative controls (group 2). This thinning was observed across all quadrants, with the most pronounced changes in the superior, nasal, and temporal regions.

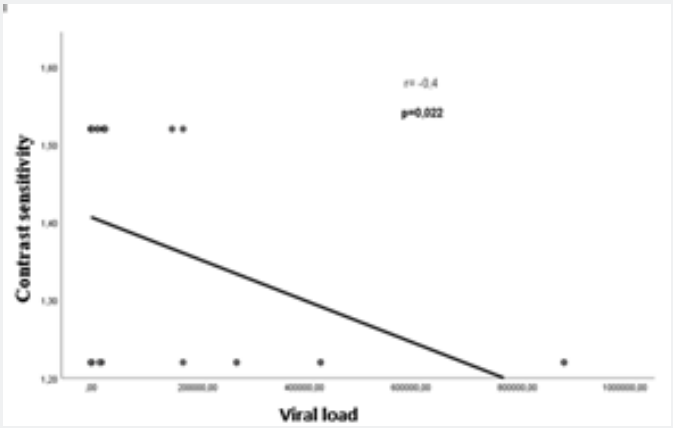


**Figure 6:** Correlation between contrast sensitivity and HIV viral load.

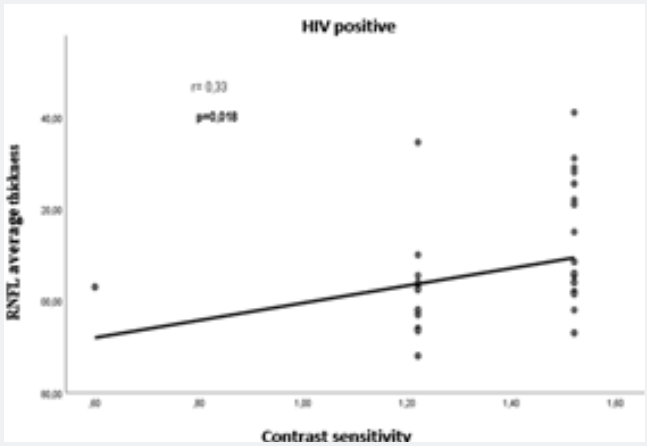


**Figure 7:** Correlation between mean RNFL thickness and contrast sensitivity for group 1.

RNFL thinning in HIV-positive patients has been reported in numerous studies. For instance, Kozak et al. (2005) found significant thinning of the peripapillary RNFL in the superior, inferior, and temporal quadrants in HIV-positive patients [3], and Paul et al. (2016) in India observed significant RNFL thinning in 21% of the eyes of HIV-positive patients [4]. Further supporting this, a comparative post-mortem study demonstrated a 40% reduction in optic nerve axons in all quadrants, along with severe degeneration of the remaining axons, in HIV-positive patients without opportunistic infection, suggesting direct degeneration of ganglion cell axons [12]. Conversely, Pathai et al. in South Africa found no overall difference in mean RNFL thickness between HIV-positive and HIV-negative patients, but did report thinning of the RNFL in the inferior quadrant of HIV-positive individuals [13]. The pathophysiology of the RNFL thinning in HIV patients is a multifactorial process involving direct viral toxicity, immune-mediated inflammation, damage to the retina and optic nerve caused by long-term microvasculopathy, accelerated degenerative processes. and potential effects of ART. These different mechanisms may be isolated or combined [4, 5,14].

These factors ultimately lead to retinal ganglion cell dysfunction and death, resulting in the loss of axons that comprise the RNFL. Maagaard et al. [15] reported that HIV infection and/or its treatment, particularly with certain nucleoside reverse transcriptase inhibitors, can impair mitochondrial function. Because the RNFL relies heavily on mitochondrial energy


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production for axonal transport, this toxicity is a likely contributor to RNFL thinning [16]. The relative contribution of each factor likely varies from patient to patient. Further research is needed to fully understand the complex interplay of these mechanisms and to develop targeted therapies to prevent or slow RNFL thinning in HIV-infected individuals. While some studies have reported RNFL thickening in HIV patients, our findings did not reflect this. Kalyani et al. [5], for example, observed increased RNFL thickness in newly diagnosed patients, and Demirkaya et al. [17] reported similar findings in HIV-positive patients with high viremia. This thickening has been attributed to "parainflammation," a low-grade adaptive tissue response aimed at maintaining homeostasis [18]. The hypothesis suggests that the RNFL undergoes an initial inflammatory phase in response to axonal damage before progressing to atrophy [18]. The absence of RNFL thickening in our study could be due to the relatively long median disease duration and the undetectable viral load in all our patients. This suggests that our patient population, with well-controlled HIV infection, may be beyond the initial inflammatory stage and already in a phase of chronic neurodegeneration.

Consistent with this, we found that RNFL thickness increased with CD4 T cell count, (r=0,7. p=< 0,001) while HIV viral load was inversely proportional to mean thickness in the temporal and nasal RNFL quadrants. These results align with studies reporting significant RNFL thinning in HIV-positive patients with a CD4 nadir below 100 cells per $mm^3$ [3,19]. However, some authors have observed a positive correlation between elevated viremia and RNFL thickening, and no significant association between CD4 lymphocyte count and RNFL thickness [13,20]. We found no significant correlation between patient age, disease duration, or duration of ARV therapy and peripapillary RNFL thickness which contrasts with Pathai et al. [13], who reported a negative correlation between ARV treatment duration and RNFL thickness. HIV infection is known to impair visual function, leading to reduced contrast sensitivity [6,10,21], impaired color vision [21,22], and peripheral visual field deficits [22-24]. In the present study, we observed a significant decrease in contrast sensitivity scores in group 1 compared to group 2 ($p < 0.001$).

Reduced contrast sensitivity is a common finding in HIV, with varying frequencies reported across studies [5,6,10]. While we found a 45.1% prevalence of abnormal contrast sensitivity scores in group 1 and 3.9% in group 2, other studies report different values: Pathai et al. [13] found 43.5% prevalence, while Freeman et al. [6] and Shah et al. [10] reported 12% and 7%, respectively. This reduction in contrast sensitivity can be attributed to retinal microvasculopathy, which leads to dysfunction of the outer retinal layers, particularly the photoreceptors and pigment epithelium [25]. Importantly, this reduction may persist even with improved immune function, possibly due to the continued transit of activated leukocytes in macular vessels, resulting in dynamic changes and damage to retinal tissue [26, 27]. Consequently, HIV patients may experience an impaired quality of life, with limitations in daily activities such as night driving, mobility, reading, and face recognition [7].

We found that contrast sensitivity was inversely proportional to the highest viral load during disease progression. These results are in agreement with those of Pathai et al. [13], but contrast with Mueller et al. [22], who found no significant correlation between contrast sensitivity and biological or clinical parameters. This discrepancy might be explained by our use of the highest viral load since diagnosis, rather than the most recent viral load as used by other authors. Similar to Shah et al. [10], we found a non-significant decrease in contrast sensitivity with increasing CD4 T-lymphocyte count. However, we did find a statistically significant positive correlation ($r = 0.33$, $p = 0.018$) between contrast sensitivity score and mean RNFL thickness, and a statistically significant positive correlation ($r = 0.39$, $p = 0.005$) between contrast sensitivity score and temporal RNFL thickness. This finding, corroborated by several studies [4,5,13], provides direct evidence linking impaired visual function to HIV-associated neuroretinal disorders. Our study is limited by its cross-sectional design and relatively small sample size. The lack of longitudinal data prevents us from tracking individual patients over time to observe the dynamic relationship between HIV disease progression, RNFL changes, and visual function decline. This also limits the generalizability of our findings and our power to detect subtle correlations.

## Conclusion

Peripapillary retinal nerve fiber layer thickness and contrast sensitivity are significantly reduced in HIV patients. Low CD4 T-cell count and high peak viral load appear to be predictive factors for these outcomes. These findings highlight the importance of routine ophthalmic examinations in HIV patients to detect early signs of visual impairment. Early detection and intervention may help preserve visual function and maintain quality of life.

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