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Hydroxychloroquine-associated retinal toxicity in juvenile-onset systemic lupus erythematosus: A single-center experience

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Abstract

Hydroxychloroquine (HCQ) is a fundamental therapy in juvenile-onset systemic lupus erythematosus (SLE), yet data on HCQ-related retinal toxicity in children are limited and primarily extrapolated from adult studies. Understanding pediatric-specific timing, risk factors, and reversibility of toxicity is essential for optimizing monitoring strategies. This study aimed to characterize HCQ-associated retinal toxicity in children with SLE and to evaluate exposure-related risk patterns. This retrospective cohort included 87 patients diagnosed with SLE before age 18 and treated with HCQ for at least 6 months at a tertiary pediatric rheumatology center between June 2016 and June 2025. Demographic features, HCQ dosing, exposure duration, ophthalmologic screening intervals, and toxicity outcomes were extracted from electronic records. Retinal toxicity was defined by ophthalmologic assessment, primarily based on automated visual field testing, with structural imaging (OCT/FAF) reviewed when available. The cohort was predominantly female (82.8%). Median age at diagnosis and HCQ initiation was 13 years (IQR 10–15). Median HCQ exposure was 80 months (IQR 52–100.5). Retinal toxicity occurred in 16.1% of patients and typically manifested as isolated functional abnormalities without structural changes on OCT or FAF. Toxicity developed after a median of 37.5 months (IQR 13.25–64.25), with cumulative risks of 8.2%, 9.5%, and 12.2% at 36, 48, and 60 months, respectively. Doses >5 mg/kg/day were significantly associated with toxicity (p=0.00045). Following HCQ withdrawal, 64.3% of affected patients showed clinical or functional improvement. Hydroxychloroquine-related retinal toxicity in juvenile-onset SLE may appear earlier than suggested by adult-based models and often begins as reversible functional impairment. Accurate weight-based dosing and vigilant ophthalmologic monitoring—potentially at intervals shorter than adult guidelines—may help detect toxicity at a reversible stage and protect long-term visual outcomes in children.

Keywords: Hydroxychloroquine, lupus erythematosus, systemic, pediatrics, retinal toxicity

Introduction

Hydroxychloroquine (HCQ) remains a cornerstone in the long-term management of systemic lupus erythematosus (SLE), providing benefits in reducing disease activity, preventing flares, improving survival, and exerting steroid-sparing and antithrombotic effects [1–3]. Owing to its favorable safety profile compared with other immunomodulatory therapies, HCQ is routinely recommended for nearly all pediatric and adult patients with SLE. However, despite its overall safety, long-term HCQ use is associated with the risk of retinal toxicity, which may present as early macular changes or progress to the classic bull's-eye maculopathy [4–6].

Hydroxychloroquine (HCQ)-induced retinal toxicity is clinically important because it can be irreversible and may continue to progress even after drug discontinuation, particularly when detected at an advanced stage [4,5]. Early toxicity often manifests with subtle parafoveal outer retinal alterations, loss of the ellipsoid zone, or macular pigment irregularity on optical coherence tomography (OCT), while late-stage disease involves retinal pigment epithelium (RPE) damage and characteristic ring-like parafoveal atrophy [5,7,8]. Modern imaging modalities, particularly spectral-domain OCT, fundus autofluorescence (FAF), and multifocal electroretinography (mfERG), have significantly improved early detection, allowing clinicians to identify pre-symptomatic changes before visual acuity declines [4,7,9].

Although the majority of available data originate from adult cohorts, juvenile-onset SLE patients differ in several important

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aspects: they often require longer treatment duration, may receive higher weight-adjusted doses, and have a longer projected lifetime cumulative exposure, potentially increasing toxicity risk [3,10]. However, the true burden of HCQ-related retinal toxicity in children remains poorly defined. The few available pediatric studies are small, heterogeneous, and mainly focus on screening adherence or early functional changes rather than providing robust incidence estimates or detailed phenotypic characterization [10,11]. Current recommendations on HCQ dosing (≤5 mg/kg/day) and ophthalmologic screening intervals are therefore largely extrapolated from adult data and expert consensus rather than pediatric evidence [4,7].

Given these uncertainties, additional data from pediatric cohorts—especially from real-life clinical settings—are crucial to clarify the frequency, risk factors, and clinical course of HCQ-related retinal toxicity in juvenile-onset SLE.

This single-center study aims to address this gap by characterizing HCQ-associated retinal toxicity, its timing, clinical spectrum, and outcomes in a cohort of juvenile-onset SLE managed at a tertiary pediatric rheumatology center.

Material and Methods

Study Design and Ethical Approval

This retrospective observational study was conducted at the Pediatric Rheumatology Clinic of the University of Health Sciences Ümraniye Training and Research Hospital and included patients diagnosed with juvenile-onset SLE between June 2016 and June 2025. The study adhered to the principles of the Declaration of Helsinki and was approved by the Scientific Research Ethics Committee of the University of Health Sciences Ümraniye Training and Research Hospital (Approval No: B.10.1.TKH.4.34.H.GP.0.01/32; Date: 15 January 2026). As the study involved retrospective review of electronic medical records without direct patient contact, the requirement for informed consent was waived.

Study Population

Patients were eligible for inclusion if they met all of the following criteria:

- Diagnosis of SLE before 18 years of age, fulfilling at least one established classification system appropriate for the diagnostic year (SLICC 2012 [12] or EULAR/ACR 2019 [13]) together with clinical judgment by pediatric rheumatologists.
- HCQ therapy for ≥6 months.
- Follow-up duration ≥6 months after diagnosis.
- At least one baseline ophthalmologic examination performed before or at the initiation of HCQ therapy.
- Availability of complete electronic medical records, including serial ophthalmologic evaluations (visual field testing and/or retinal imaging such as OCT or FAF).

Patients were excluded if they:

- Had an uncertain SLE diagnosis or were later reclassified under an alternative condition,
- Had incomplete or insufficient clinical, laboratory, or ophthalmologic documentation,
- Had a total follow-up duration of <6 months,
- Lacked baseline ophthalmologic assessment at or prior to HCQ initiation.

Data Collection

Demographic and clinical data were extracted from electronic medical records, including sex, age at SLE diagnosis, and age at initiation of HCQ, and total follow-up duration. Treatment-related variables collected for each patient comprised the daily HCQ dose (mg/kg/day), whether the prescribed dose exceeded 5 mg/kg/day, and the total duration of HCQ exposure (months).

Routine HCQ whole-blood level monitoring was not performed during the study period, and dosing decisions were based on weight-adjusted calculations and clinical evaluation. HCQ dosing was prescribed according to actual body weight (mg/kg/day) at treatment initiation and was routinely recalculated during follow-up visits in accordance with patient growth and weight changes.

Ophthalmologic monitoring parameters included routine screening intervals during HCQ therapy and, for patients who developed toxicity, intensified follow-up intervals after toxicity detection. Ophthalmologic evaluations were conducted by ophthalmologists within our tertiary care center during the study period, and in some cases by ophthalmologists at referring institutions. All retinal toxicity assessments were based on formal written ophthalmologic reports documented in the patients' medical records.

Retinal toxicity variables included the presence or absence of toxicity, the time to toxicity from HCQ initiation (months), and the type of toxicity documented. Toxicity findings were categorized as functional abnormalities detected by visual field testing (visual field defect, macular scotoma, or central sensitivity loss) and structural abnormalities on OCT or FAF, when available. Patient-reported symptoms at the time of toxicity (asymptomatic, blurred vision, or scotoma) were recorded. Management after toxicity included documentation of HCQ discontinuation, the presence of clinical or functional improvement following drug withdrawal, and the time to improvement (months).

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 27 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for distribution and summarized as median and interquartile range (IQR), while categorical variables were presented as frequencies and percentages. Comparisons between patients with and without retinal toxicity were conducted using the Mann–Whitney U test for continuous variables and Fisher's exact test for categorical variables. Time-

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to-toxicity was evaluated using Kaplan–Meier survival analysis, and cumulative incidence at 36, 48, and 60 months was calculated as 1–S(t). Median time-to-toxicity was reported when estimable. To account for zero-event cells in dose–toxicity comparisons, a Haldane–Anscombe correction was applied to calculate odds ratios with 95% confidence intervals. A two-sided p value <0.05 was considered statistically significant.

Results

A total of 87 children with juvenile-onset SLE receiving HCQ were included. The cohort was predominantly female (72/87, 82.8%). The median age at SLE diagnosis was 13 years (IQR 10–15), which was identical to the median age at HCQ initiation as treatment was started at diagnosis. The median duration of HCQ exposure was 80 months (IQR 52–100.5). Four patients (4.6%) received a daily HCQ dose exceeding 5 mg/kg/day. The routine ophthalmologic screening interval for the whole cohort was a median of 12 months (IQR 6–12).

Retinal toxicity was identified in 14 of 87 patients (16.1%). Among these, 13 (92.9%) were female. The median age at HCQ initiation did not differ between patients with and without toxicity (13 years [IQR 10.25–15] vs. 13 years [IQR 10–15], p=0.749). Toxicity developed after a median HCQ exposure of 37.5 months (IQR 13.25–64.25). A time-to-event (Kaplan–Meier) analysis demonstrated cumulative toxicity risks of 8.2% at 36 months, 9.5% at 48 months, and 12.2% at 60 months of treatment. Median time-to-toxicity was not reached because fewer than 50% of patients developed toxicity during follow-up (Table 1 and Figure 1).

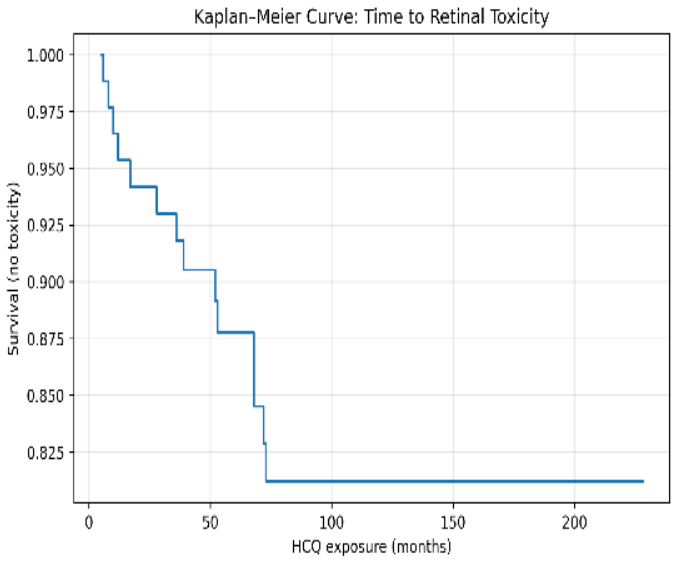

Figure 1. Kaplan–Meier survival curve demonstrating the cumulative probability of remaining free of retinal toxicity among patients with juvenile-onset systemic lupus erythematosus (SLE) receiving hydroxychloroquine (HCQ). Time to event was defined as the number of months of HCQ exposure until toxicity onset; patients without toxicity were censored at their last documented month of HCQ use. Median time-to-toxicity was not reached because fewer than 50% of participants developed toxicity during follow-up.

Table 1. Key outcomes from the Kaplan–Meier time-to-toxicity analysis

Time point (months)	Survival probability S(t)	Cumulative incidence 1–S(t)
36 months	0.918	8.2%
48 months	0.905	9.5%
60 months	0.878	12.2%

Survival probability S(t) represents the proportion of patients who remained free of retinal toxicity at each time point. Cumulative incidence (1–S(t)) reflects the estimated proportion of patients who had developed toxicity by that time. Median time-to-toxicity was not reached because fewer than 50% of patients developed toxicity during follow-up

Patients receiving >5 mg/kg/day had a markedly higher toxicity rate (4/4, 100%) than those receiving ≤5 mg/kg/day (10/83, 12.0%) (Fisher's exact p=0.00045) (Table 2 and Figure 2). A Haldane–Anscombe corrected odds ratio was 63.0 (95% CI 3.16–1255.93), supporting a strong dose–toxicity signal despite the small high-dose subgroup. The follow-up interval after toxicity detection was shortened to a median of 3 months (IQR 3–3), consistent with intensified monitoring.

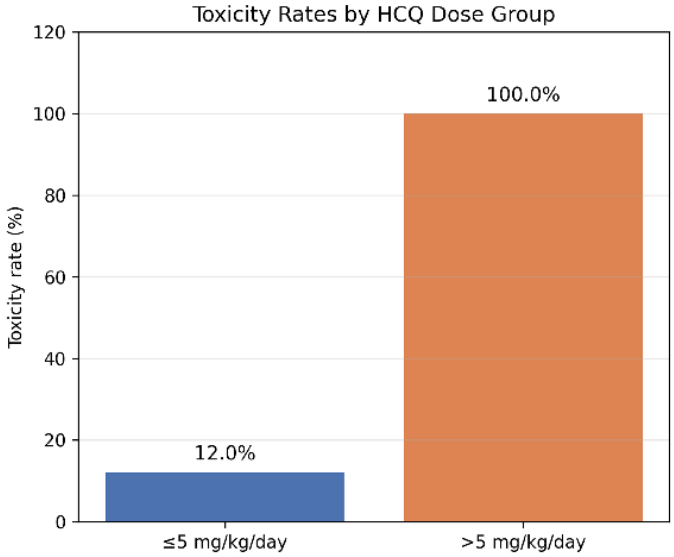

Figure 2. Bar chart comparing the proportion of retinal toxicity among juvenile-onset SLE patients receiving different daily hydroxychloroquine (HCQ) doses. Toxicity occurred in 12.0% of patients receiving ≤5 mg/kg/day (10/83), whereas all patients receiving >5 mg/kg/day (4/4) developed toxicity. These findings highlight a strong dose–toxicity association consistent with current safety recommendations.

All toxicity cases exhibited functional abnormalities on visual field testing. The most common finding was visual field defects (12/14, 85.7%), followed by central sensitivity loss (1/14, 7.1%) and macular scotoma (1/14, 7.1%). No structural macular damage (e.g., OCT or FAF changes suggestive of early or late-stage HCQ retinopathy) was documented. Most patients were asymptomatic (11/14, 78.6%), while three patients (3/14, 21.4%) reported visual symptoms, including blurred vision (2/14, 14.3%) and scotoma (1/14, 7.1%).

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Table 2. Summary of clinical characteristics, hydroxychloroquine exposure, screening data, and retinal toxicity outcomes

Parameter	Value
Population	
Juvenile-onset SLE patients, n (%)	87 (100%)
Female sex, n (%)	72 (82.8%)
Age at SLE diagnosis, years, median (IQR)	13 (10–15)
HCQ Exposure	
Age at HCQ initiation, years, median (IQR)	13 (10–15)
Total HCQ exposure, months, median (IQR)	80 (52–100.5)
Patients receiving >5 mg/kg/day, n (%)	4 (4.6%)
Ophthalmologic Screening	
Routine screening interval, months, median (IQR)	12 (6–12)
Post-toxicity screening interval, months, median (IQR)	3 (3–3)
Retinal Toxicity Outcomes	
Retinal toxicity, n (%)	14 (16.1%)
Female among toxicity cases, n/N (%)	13/14 (92.9%)
Time to toxicity, months, median (IQR)	37.5 (13.25–64.25)
Symptoms at presentation, n/N (%)	• Asymptomatic (11/14, 78.6%) • Blurred vision or scotoma (3/14, 21.4%)
Toxicity phenotypes, n/N (%)	• Visual field defects (12/14, 85.7%) • Central sensitivity loss (1/14, 7.1%) • Macular scotoma (1/14, 7.1%) • No structural OCT/FAF abnormalities
HCQ discontinued after toxicity, n/N (%)	14/14 (100%)
Symptomatic/functional improvement after discontinuation, n/N (%)	9/14 (64.3%)
Time to improvement, months, median (IQR)	4 (3–6)
Toxicity after >5 mg/kg/day HCQ, n/N (%)	4/4 (100%)
Toxicity after ≤5 mg/kg/day HCQ, n/N (%)	10/83 (12.0%)
p-value (Fisher's exact test)	0.00045

Values are presented as median (interquartile range, IQR) or number (percentage). Functional toxicity was defined by visual-field abnormalities consistent with hydroxychloroquine (HCQ) retinopathy. No structural macular changes were observed on optical coherence tomography (OCT) or fundus autofluorescence (FAF). Abbreviations: SLE, systemic lupus erythematosus; HCQ, hydroxychloroquine; IQR, interquartile range; mg/kg/day, milligrams per kilogram per day; OCT, optical coherence tomography; FAF, fundus autofluorescence

HCQ therapy was discontinued in all patients who developed toxicity. Clinical or functional improvement was documented in 9 of 14 patients (64.3%) following drug withdrawal, occurring after a median of 4 months (IQR 3–6). A detailed summary of demographic characteristics, HCQ exposure parameters, screening intervals, and toxicity outcomes is provided in Table 2.

Discussion

In this single-center cohort of patients with juvenile-onset SLE receiving HCQ, we identified clear evidence of retinal toxicity despite routine ophthalmologic monitoring and weight-based dosing. Most cases reflected early, functional abnormalities without structural retinal involvement, emphasizing that HCQ-related toxicity in children often begins subtly and requires systematic surveillance for timely detection. Notably, many cases demonstrated clinical or functional improvement after drug withdrawal, suggesting that early, pre-structural toxicity may retain meaningful reversibility in pediatric patients—an observation that contrasts with adult data, where progression may continue despite discontinuation [5]. The Kaplan–Meier analysis demonstrated a gradual, time-dependent increase in cumulative toxicity risk over the first several years of therapy, indicating that even though the overall risk remains relatively low, clinically relevant events can occur earlier than expected from adult-based assumptions. Importantly, our findings revealed a clear dose-dependent risk pattern, with HCQ doses exceeding 5 mg/kg/

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day—an adult-derived safety threshold [4] often extrapolated to pediatric practice—strongly associated with the development of toxicity. This highlights the importance of accurate weight-based dosing when HCQ is used in children. Given the scarcity of pediatric studies—most of which are small, cross-sectional, or focused on screening adherence rather than longitudinal risk—this cohort represents one of the most comprehensive follow-up series in the pediatric HCQ literature and is among the first to delineate a child-specific, time-dependent and dose-dependent risk profile. By integrating long-term ophthalmologic follow-up with detailed exposure data, this study helps bridge a major gap between adult-derived recommendations and the realities of pediatric clinical practice.

Findings from adult cohorts consistently show that the risk of HCQ retinopathy is relatively low during the early years of therapy but increases markedly with prolonged exposure, with long-term observational data reporting cumulative prevalences of approximately 8.6–11.5% after 15 years and a clear rise in risk beyond 5–10 years of continuous use [14]. Accordingly, adult-based guideline recommendations advise a baseline ophthalmologic evaluation followed by annual screening after five years of therapy—earlier if additional risk factors are present—and emphasize adherence to weight-based dosing (≤5 mg/kg/day) to mitigate toxicity risk [4,14,15]. The 2023 European League Against Rheumatology (EULAR) recommendations similarly reaffirm the central role of HCQ in SLE management and endorse a target dose of 5 mg/kg actual body weight/day, taking into account individual risk for retinal toxicity; however, they do not provide pediatric-specific screening intervals, highlighting a persistent gap in guidance for children [16]. In contrast, our pediatric cohort demonstrated that toxicity may occur earlier in the course of therapy, with a notable proportion of cases emerging within the first 3–4 years of exposure. This suggests that pediatric patients may require more frequent ophthalmologic monitoring than adult-derived screening schedules would imply. Pharmacokinetic differences, growth-related fluctuations affecting weight-adjusted dosing, and a longer projected lifetime cumulative exposure likely contribute to this discrepancy. Taken together, these observations indicate that adult-based screening intervals may not fully capture early risk in children and may need to be adapted to pediatric treatment dynamics—an approach aligned with our own clinical practice, in which routine annual screening was implemented and follow-up intervals were further shortened after toxicity detection.

Daily dose is one of the most important determinants of HCQ retinopathy risk in adults, where exceeding the commonly recommended threshold of 5 mg/kg/day is consistently associated with a markedly increased likelihood of toxicity. Modern guidelines emphasize weight-based dosing and regular ophthalmologic surveillance to mitigate this risk, yet these recommendations are based almost entirely on adult data [4,17]. Contemporary reviews and population-based analyses further reinforce that higher daily dosing and prolonged cumulative exposure substantially amplify toxicity risk, while advanced imaging modalities improve detection of early functional change [15,18-20]. Although recent EULAR recommendations reaffirm the 5 mg/kg/day target dose, they do not provide pediatric-specific dosing or screening intervals, leaving clinicians to extrapolate adult thresholds for use in children [16]. In our cohort, although HCQ dosing was routinely recalculated according to actual body weight during follow-up, transient periods above 5 mg/kg/day may have occurred due to growth-related weight fluctuations or temporary weight loss during phases of higher disease activity, as well as individualized treatment decisions consistent with adult recommendations. In this study, the adult-derived cutoff appeared highly relevant, as all children receiving >5 mg/kg/day developed toxicity, whereas those maintained within recommended dosing limits had markedly lower rates. These findings suggest that pediatric patients—whose weight, pharmacokinetics, and cumulative exposure trajectories differ fundamentally from adults—may be particularly vulnerable to inadvertent exceedance of weight-based thresholds, highlighting the need for careful, repeated recalculation of mg/kg dosing throughout growth.

In our cohort, all cases of retinal toxicity presented with functional abnormalities on visual field testing in the absence of structural macular changes on OCT or FAF, indicating that pediatric HCQ toxicity may predominantly manifest as an early, functional-only phenotype. Notably, a substantial proportion of affected children experienced clinical or functional improvement after HCQ withdrawal, suggesting that early-stage toxicity in the pediatric population may have a greater potential for reversibility compared with the structural retinopathy typically reported in adults. Adult studies have shown that once structural damage—especially ellipsoid zone loss—occurs, progression may continue despite cessation of therapy, underscoring the importance of identifying toxicity before anatomical involvement develops [6,7,9]. The absence of structural findings and the observed improvement in many of our patients likely reflect earlier detection, shorter cumulative exposure compared with adults, and possibly unique pediatric retinal resilience. However, given that progression after drug discontinuation has been documented in adults [4,5], the long-term trajectory of these early pediatric functional abnormalities remains uncertain. These findings highlight the essential role of routine, sensitive functional assessment in pediatric patients and emphasize the need for prospective studies to determine whether early functional changes in children invariably stabilize, improve, or may in some cases progress over longer follow-up.

This study has several notable strengths. It represents one of the largest pediatric cohorts to date evaluating HCQ-related retinal toxicity with long-term, systematic ophthalmologic follow-up. The availability of detailed treatment exposure data enabled time-to-event analysis, allowing characterization of both dose-dependent and time-dependent risk patterns—an aspect rarely examined in pediatric populations. Additionally, the standardized

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clinical practice at our center, including regular visual field testing and consistent follow-up intervals, minimized variability in screening frequency and strengthened the reliability of the observed early functional toxicity profile.

Several limitations should also be acknowledged. The retrospective, single-center design may limit generalizability, and ophthalmologic assessments were not uniform across all patients; structural imaging modalities such as OCT and FAF were not performed in every case, restricting the ability to detect very early anatomical changes. Ophthalmologic evaluations were not conducted by a single examiner throughout the entire study period, which may have introduced inter-observer variability despite reliance on formal written reports. Advanced diagnostic tests (e.g., mfERG, color vision assessment, or genetic susceptibility markers) were not routinely available, which may have led to underrecognition of subtle or preclinical toxicity. HCQ whole-blood levels were not routinely measured, which precluded assessment of potential variability in systemic exposure related to adherence or pharmacokinetic differences. Additionally, although the cohort size is relatively large for juvenile-onset SLE, the number of toxicity cases—particularly those exposed to doses >5 mg/kg/day—was modest, resulting in wide confidence intervals for some estimates. Despite these constraints, the consistency of the findings across multiple analytic approaches supports the robustness of the observed dose- and time-related patterns.

Conclusion

In conclusion, this study demonstrates that HCQ-related retinal toxicity in juvenile-onset SLE may occur earlier in the treatment course than expected from adult-derived risk models and often manifests initially with isolated functional abnormalities in the absence of structural retinal damage. The observation that a substantial proportion of cases showed clinical or functional improvement following HCQ withdrawal suggests that early, pre-structural toxicity in children may retain meaningful reversibility, further underscoring the value of prompt detection. Both time-to-event and dose-stratified analyses highlighted a clear dose-dependent risk pattern, with >5 mg/kg/day emerging as a particularly vulnerable range even in pediatric patients. Taken together, these findings support the importance of meticulous weight-based dosing and reinforce the need for vigilant ophthalmologic surveillance in children receiving HCQ. Although optimal pediatric screening intervals remain undefined, our results suggest that monitoring strategies tailored to the pediatric treatment context—potentially more frequent than adult-based schedules—may help identify toxicity at a reversible stage and minimize long-term visual sequelae. Future prospective, multicenter studies with larger pediatric populations are needed to validate these findings and refine evidence-based screening recommendations.

Conflict of Interests

The authors declare that there is no conflict of interest in the study.

Financial Disclosure

The authors declare that they have received no financial support for the study.

Ethical Approval

This study was approved by the University of Health Sciences Ümraniye Training and Research Hospital (Approval No: B.10.1.TKH.4.34.H.GP.0.01/32; Date: 15 January 2026)

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