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Review Article

The Role of Hydroxychloroquine in Chronic Management of Systemic Lupus Erythematosus: A Review

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ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that presents with a variety of clinical symptoms and unpredictable flare-ups. Hydroxychloroquine (HCQ), an immunomodulatory antimalarial drug, is crucial for the long-term treatment of SLE. Examining the pharmacology, therapeutic role, safety profile, mechanism of action, and novel applications of HCQ in adult and paediatric SLE populations, including during pregnancy, is the aim of this review. A detailed literature review was carried out using PubMed and indexed sources to evaluate clinical trials, pharmacologic studies, and expert recommendations related to HCQ in SLE. HCQ offers metabolic and cardiovascular benefits, reduces disease activity, avoids flare-ups, and enhances pregnancy outcomes. It has a great safety profile, notwithstanding retinal toxicity and subtherapeutic exposure problems. Blood level monitoring and dosage modifications are essential for ensuring safety and efficacy, especially in unique groups. Because of its many advantages and safety, hydro chloroquine is still a mainstay treatment for SLE. However, more study is required in the areas of combination therapy, paediatric pharmacokinetics, individualized dosing, and therapeutic drug monitoring.

INTRODUCTION

A complicated autoimmune disease, systemic lupus erythematosus (SLE), is characterized by persistent inflammation and involvement of multiple organ systems. SLE can affect any organ and presents with a wide range of immunologic and clinical symptoms. Its natural course is marked by flare-ups or relapses that alternate with periods of remission, and the outcome is highly unpredictable, ranging from death to lifelong remission. A better understanding of the pathogenetic pathways and prognostic factors of SLE, along with the use of immunosuppressive treatments, may explain the current rise in morbidity and mortality. [1] Most affected

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individuals are women of reproductive age. African and Asian women are particularly vulnerable and may experience severe symptoms. [2] It has a variable prognosis, with severity ranging from moderate to life-threatening, and is characterized by the presence of anti-nuclear antibodies (ANA). The main goals of SLE treatment are to achieve remission or low disease activity, prevent inflammation-induced damage, and improve quality of life. When managing the condition, it is essential to minimize drug side effects. [7]

From mild skin involvement to severe organ damage, including kidney failure, pulmonary hypertension, and heart failure, this illness exhibits a wide spectrum of clinical features. Laboratory tests and clinical assessments are used to diagnose SLE. The American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) have updated their classification standards. [3] The symptoms, organ involvement, severity of the disease, and medication availability all influence the treatment plan, duration of treatment, mode of administration, and dosage of medication. [8]

For many years, hydroxychloroquine (HCQ), a proven immunomodulatory and antimalarial medication, has been widely used for treating SLE. Its benefits in reducing disease activity, preventing flare-ups, and improving long-term outcomes are well known. It is distinguished by the addition of a hydroxyl group to the chloroquine side chain, resulting in fewer adverse reactions. Due to its improved safety profile, it was adopted for SLE treatment in the 1950s after gaining recognition as an immunomodulator. HCQ is commonly prescribed for autoimmune diseases because of its excellent safety record. Additionally, HCQ offers various therapeutic advantages, including photoprotection, glycaemic control, cholesterol level improvement, cardiovascular risk reduction, and anti-thrombotic effects.[4] Hydroxychloroquine reduces lupus activity during pregnancy without harming the foetus, prevents lupus flares, and enhances longevity across all demographic groups. [5] Moreover, hydroxychloroquine may prevent thrombosis, bone loss, and permanent organ damage.[6]

Classification of SLE: the Systemic Lupus International Collaborating Clinics (SLICC)

Classification Criteria

CLINICAL CRITERIA	IMMUNOLOGICAL CRITERIA
• Acute cutaneous lupus erythematosus (including "butterfly rash") • Chronic cutaneous lupus erythematosus (e.g., localized or generalized discoid lupus erythematosus) • Oral ulcers (on palate and/or nose) • Non-scarring alopecia • Synovitis (≥ 2 joints) or tenderness on palpation (≥ 2 joints) and morning stiffness (≥ 30 min) • Serositis (pleurisy or pericardial pain for more than 1 day) • Renal involvement (single urine: protein/creatinine ratio or 24-hour urine protein, >0.5 g) • Neurological involvement (e.g., seizures, psychosis, myelitis) • Haemolytic anaemia • Leukopenia (<4000/μL) or lymphopenia (<1000/μL) • Thrombocytopenia (< 100,000/μL	• ANA level above the laboratory reference range • Anti-dsDNA antibodies • Anti-sm antibodies • Antiphospholipid antibodies (anticardiolipin and anti-β 2-glycoprotein I [IgA-, IgG- or IgM-] antibodies; false-positive VDRL [Venereal Disease Research Laboratory] test) • Low complement (C3, C4, or CH50) • Direct Coombs test (in the absence of haemolytic anaemia)

MECHANISM OF ACTION

Inhibition of lysosomal function

In addition to its alkalescency and hydrophobicity, HCQ spreads swiftly across cell membranes into acidic lysosomes. It raises the pH of acidic subcellular compartments by stabilizing cell membranes, accumulating in lysosomal compartments, and inhibiting the activity of acidic proteases. Changes in acid enzyme activity, on the other hand, decrease the generation and distribution of major histocompatibility class II complex components, which suppresses autoimmune reactions and antigen presentation. Additionally, HCQ interferes with autophagy, a lysosome-dependent process that breaks down damaged organelles and cytoplasmic components to preserve cell homeostasis. [9]

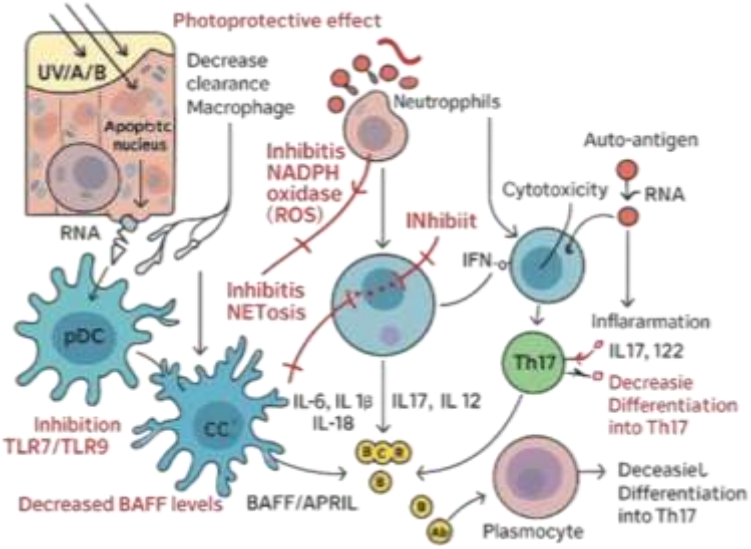

Figure 1: Mechanism of action of Hydroxychloroquine

Inhibition of toll-like receptor (TLR)-mediated innate immune response

HCQ suppresses a number of common inflammatory pathways, especially TLR-mediated signalling. Important receptors in the innate immune system, TLRs are mostly produced by immune cells and mediate the stimulation of genes that produce inflammatory cytokines. The innate immune system's TLR3, TLR7, and TLR9 are found in intracellular spaces like endosomes. Through TRIF/IRF3, TLR3 primarily stimulated the production of type I interferon (IFN) and IFN-inducible genes. TLR7, which was sensitive to synthetic imidazoquinolinone-like compounds, activated the production of type I IFN and inflammatory cytokines through pathways that were reliant on nuclear factor-kappaB (NF-κB) and myeloid differentiation primary-response protein 88 (MyD88). DNA in immune cells' endosomes activates TLR9, which results in a strong IFN production. TLR3, TLR7, and TLR9 are the receptors that HCQ admires to engage with; it selectively prevents the activation of these receptors, which mediate inflammatory pathways and cytokines. As a result, it stops these receptors from being activated, which mediates inflammatory pathways and cytokines.[10]

Suppression of other signalling pathways

Many proinflammatory signalling cascades contain nicotinamide adenine dinucleotide phosphate (NADPH) oxidases (NOXs), which are unique enzyme complexes in the generation of reactive oxygen species (ROS). It has been

demonstrated that HCQ lowers NOX-mediated TNF-α and IL-1β production by directly preventing gp91phox from translocating. Because excessive formation of ROS is the primary cause of vascular endothelial dysfunction, it also protects the endothelium by inhibiting NADPH and activating extracellular signal-regulated kinase 5 (ERK5). By disrupting intracellular Ca2+ mobilization, HCQ reduces the production and release of immunological cytokines and factors in T- and B-cells with respect to second messenger-mediated signal transduction.[11]

Pharmacology of hydroxychloroquine

The cytochrome P450 (CYP) 3A4, 3A5, 2D6, and 2C8 enzymes primarily break down HCQ into three metabolites in the liver: didesethylchloroquine (DDCQ), desethylchloroquine (DCQ), and the active metabolite desethylhydroxychloroquine (DHCQ). These three metabolites plus HCQ are likewise competitive, reversible CYP2D6 inhibitors.[12] Following oral treatment, absorption was quick and usually consistent; nevertheless, the overall apparent volume of distribution was quite large (>100 L/kg), indicating significant tissue binding. It's possible that the stated average terminal phase elimination half-lives for hydroxychloroquine (about 54 days) and chloroquine (about 38 days) don't differ much. Therefore, rather than medication clearance, distribution processes define the initial plasma or whole-blood concentration profile in the treatment of acute sickness.[13]

Protonated HCQ enters the cell, and its concentration is inversely correlated with pH. Consequently, it builds up in acidic organelles, such as Golgi vesicles, lysosomes, and endosomes, raising their pH.[14] When taken as HCQ sulphate, it exhibits outstanding oral absorption and bioavailability. With a lengthy mean residence period of 1,300 hours and a huge distribution volume, HCQ is a weak base. About 62% of drug metabolites have unaltered renal clearance, whereas 21% have changed renal clearance.[15]

HCQ in Disease Control and Flare Prevention

In incomplete or newly diagnosed SLE, HCQ may inhibit early mediators such as interferon (IFN) and B cell activating factor (BAFF), reducing the levels of IFN-γ-induced protein 10 (IP-10). This finding supports the idea that HCQ may have an impact on the course of the disease.(16) Managing SLE disease activity itself is the most important effect of HCQ. This includes reducing active clinical involvements, lowering serum markers, lowering activity scores, preventing flare-ups of the disease, and achieving sustained remission after long-term use. Consequently, a reduction in disease activity(17), prevention of disease flares, and improvement of proinflammatory cytokine profiles have been highlighted with HCQ.(18)

The frequencies of flares were comparable for the HCQ decrease and cessation cohorts. The mean dosages of those flaring and those not flaring were comparable among those who decreased HCQ. Individual variations in medication metabolism or even the quantity of HCQ retained in bodily tissues could be the cause of this.(19)

HCQ and Pregnancy Outcomes

SLE causes a number of adverse pregnancy outcomes (APOs) in women who are of childbearing age. Intrauterine growth restriction (IUGR), spontaneous abortion, thromboembolic disease, postpartum infection, and illness flare are among the issues that pregnant women with SLE are more likely to have.(20) Flares are significantly more common during pregnancy and the first three months after giving birth.(21) The process of aPL

antibodies being partially reversed by HCQ treatment may lessen trophoblastic invasion and migration. This enhancement of placental function lowers the prevalence of pathological pregnancies and helps prevent foetal death.[22] According to clinical research, pregnant women who continue using HCQ medication have much lower levels of lupus activity. It appears that taking HCQ continuously lowers the risk of flare-ups both during and after pregnancy. After adjusting for factors like age, race, antibody status, corticosteroids, and prior cardiac-neonatal lupus risk, the study found no evidence of HCQ as an independent protective factor for neonatal lupus, even though neonatal lupus cases were less common in pregnancies treated with HCQ.[24,25]

HCQ in paediatric SLE

Early onset About 15% to 20% of SLE patients have cSLE, which is defined as disease onset in patients under the age of 18.[29] JSLE is more aggressive than adult-onset SLE, with higher disease activity and a higher medication burden (including corticosteroids and other immunosuppressive medicines), which increases the illness's morbidity and mortality.[30] A weight-based dosing paradigm for HCQ may result in suboptimal drug exposures, particularly for children with obesity.[32]

SAFETY, MONITORING AND TOXICITY

Within the first year of utilising HCQ, patients are advised by the American Academy of Ophthalmology(AAO) to have a fundus examination. Patients with maculopathy should have 10 2 AVF and SD-OCT tests performed. After five years of usage, or earlier if significant risk factors are present, such as an HCQ dose greater than 5 mg/kg real body weight, the existence of renal disease, the presence of macular disease, and concurrent use of tamoxifen, annual screening with SDOCT and 10-2 AVF testing is advised.[25] Screening tests advise routine primary screening to employ both automated visual fields and SD OCT. While SD OCT is objective, extremely specific, and generally sensitive for degrees of damage that may be visually noticeable, fields may be more sensitive, but they are subjective, and patients' reactions vary in dependability. Before poisoning is identified, at least one objective test should validate subjective observations unless toxic changes are severe and evident.[26] Higher HCQ dosages and the length of time spent on HCQ were risk factors for HCQ retinopathy development. Given its strong effectiveness in treating SLE and the possible overestimation of the danger of HCQ retinopathy in the past, HCQ is still a safe medicine to use.[27]

BLOOD MONITORING AND THERAPEUTIC DRUG LEVELS

HCQ blood level monitoring may be able to detect nonadherence and reduce the risk of lupus flare-ups and early mortality, per a global meta-analysis and other studies. All lupus patients, including those in groups with greater acute care rates, had decreased rates of acute care when their CQ blood levels are between 750 and 1,200 ng/mL. The causal relationship between HCQ level monitoring and acute care for lupus patients should be demonstrated by future clinical trials.[28] When compared to whole-blood data, HCQ in plasma displayed more variability, hence it is preferable to measure HCQ concentrations in whole blood rather than plasma.[31] The blood concentration limit for HCQ to harm other organs, however, was 500 ng/mL in a prospective trial. In SLE patients, the subtherapeutic HCQ concentration (<500 ng/mL) was significantly linked to disease activity and cumulative organ damage over time, and it was also linked to the development of new-onset

LN when compared to the therapeutic group (≥500 ng/mL).[32]

FUTURE DIRECTIONS

While hydroxychloroquine (HCQ) is still necessary for treating SLE, future approaches should concentrate on customized dosage, particularly for obese and paediatric patients. Although more extensive research is required to standardize this procedure, routine monitoring of HCQ blood levels may enhance adherence and disease control. Although more clinical research is necessary, there may be synergy when HCQ is combined with more recent biologics like belimumab. To lower the risk of retinal toxicity, safer medication formulations and enhanced screening methods are required. Furthermore, more studies on long-term pregnancy outcomes and paediatric populations will assist in clarifying the significance of HCQ in these unique populations.

CONCLUSION

Hydroxychloroquine's immunomodulatory properties, safety, and wide range of therapeutic advantages have kept it a mainstay in the treatment of systemic lupus erythematosus. It is an integral part of routine care because of its capacity to better long-term results, lessen illness flares, and offer extra vascular and metabolic protection. Optimising dosing strategies and preventing uncommon but severe side effects like retinal toxicity are still difficult despite its advantages, particularly in obese and paediatric patients. Further research into its usage during pregnancy and in conjunction with biologics, as well as the integration of therapeutic drug monitoring, will assist in improving its use in clinical practice. The function of hydroxychloroquine will continue to change as personalized medicine develops, highlighting the significance of continuous study to optimize its therapeutic potential while lowering hazards.

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