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SYSTEMATIC REVIEW ARTICLE

Cardiac Complications Attributed to Hydroxychloroquine: A Systematic Review of the Literature Pre-COVID-19

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Abstract: ***Introduction*:** Hydroxychloroquine has been used for rheumatological diseases for many decades and is considered a safe medication. With the COVID-19 outbreak, there has been an increase in reports associating cardiotoxicity with hydroxychloroquine. It is unclear if the cardiotoxic profile of hydroxychloroquine is previously underreported in the literature or is it a manifestation of COVID-19 and therapeutic interventions. This manuscript evaluates the incidence of cardiotoxicity associated with hydroxychloroquine prior to the onset of COVID-19.

***Methods*:** PubMED, EMBASE, and Cochrane databases were searched for keywords derived from MeSH terms prior to April 9, 2020. Inclusion eligibility was based on appropriate reporting of cardiac conditions and study design.

***Results*:** A total of 69 articles were identified (58 case reports, 11 case series). The majority (84%) of patients were female, with a median age of 49.2 (range 16-92) years. 15 of 185 patients with cardiotoxic events were in the setting of acute intentional overdose. In acute overdose, the median ingestion was 17,857 ± 14,873 mg. 2 of 15 patients died after acute intoxication. In patients with long-term hydroxychloroquine use (10.5 ± 8.9 years), new onset systolic heart failure occurred in 54 of 155 patients (35%) with median cumulative ingestion of 1,493,800 ± 995,517 mg. The majority of patients improved with the withdrawal of hydroxychloroquine and standard therapy.

***Conclusion*:** Millions of hydroxychloroquine doses are prescribed annually. Prior to the COVID-19 pandemic, cardiac complications attributed to hydroxychloroquine were uncommon. Further studies are needed to understand the impact of COVID-19 on the cardiovascular system to understand the presence or absence of potential medication interactions with hydroxychloroquine in this new pathophysiological state.

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1. INTRODUCTION

The antimalarial medication Hydroxychloroquine (HCQ) has an established safety profile in the long-term use for rheumatological conditions, such as Systemic Lupus Erythematosus (SLE) and Rheumatoid Arthritis (RA) [1]. For connective tissue disorders, HCQ has been demonstrated to improve clinical symptoms, decrease SLE renal involvement, and decrease the rate of thrombotic events in patients [1, 2]. It has also been commonly prescribed for the prevention and treatment of malaria in adults and children who are planning on traveling to areas where malaria transmission occurs [3]. HCQ has been approved by the United States Food and Drug Administration (FDA) since 1955 [3]. Although HCQ has been in widespread use for decades, recent retrospective database reviews of HCQ administration in patients infected with the novel coronavirus (SARS-CoV-2) has cast doubt on the safety profile of HCQ [4, 5].

Given the recent outbreak of the novel severe respiratory distress syndrome coronavirus (SARS-CoV-2) and early reports of the possible therapeutic benefit of HCQ in its management, a national spotlight has been shone on the medication. Until recently, HCQ-related cardiac adverse events (AE) have been infrequently reported, despite its regular usage for about seven decades. Since the onset of the SARS-CoV-2 pandemic, there is speculation regarding the acuity of side effects, with some publications suggesting the incidence of arrhythmias in the acute to a sub-acute setting, requiring closer monitoring [6].

However, it currently remains unclear if there is a specific cumulative dosage, duration of therapy, or vulnerable patient population at risk of toxicity with HCQ administration. The aim of this study is to perform a systematic review of

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the literature regarding the cardiac AE related to HCQ prior to the onset of SARS-CoV-2 to help elucidate if HCQ was previously not well-studied or if SARS-CoV-2 is exacerbating cardiac manifestations with HCQ use.

2. METHODS

2.1. Data Sources and Literature Searches

A systematic review was conducted by performing a systematic electronic search in MEDLINE of PubMed, EMBASE, and Cochrane Databases, including all articles published prior to April 9, 2020. This search utilized a variety of relevant keywords according to the MeSH terms for heart disease with subcategories such as cardiomyopathy, heart failure, cardiac toxicity, and arrhythmias ("hydroxychloroquine"[MeSH]) AND ("QT"[MeSH] OR "QT prolongation" [MeSH] OR "arrhythmia" [MeSH] OR "torsades" [MeSH] OR "ventricular tachycardia" [MeSH] OR "ventricular fibrillation" [MeSH] OR "atrioventricular block" [MeSH] OR "cardiomyopathy" [MeSH] OR "heart failure" [MeSH] OR "cardiac toxicity" [MeSH]). After the removal of duplicate studies, one author (DDW) initially screened the potentially relevant manuscripts based on titles and abstracts, with the final selection of articles performed independently by two authors (GF, KM) based on the full-text evaluation. The consensus was obtained between the authors to resolve any article discrepancies.

2.2. Study Selection

As demonstrated in Fig. (**1**), all studies reporting patients who experienced cardiac complications while taking hydroxychloroquine, were included. Duplicate articles were excluded after review, as were articles that did not describe cardiac adverse effects. Articles were also excluded that described the use of HCQ as therapy for autoimmune conditions with cardiac manifestations.

2.3. Data Extraction

Using a standardized data form, two reviewers independently extracted study characteristics from the remaining articles. The data collected for each patient included: age, sex, median daily dose, median cumulative dose, the median duration of therapy, underlying disease, cardiac disorders, ECG and echocardiogram findings, and evolution of clinical course over time.

2.4. Statistical Analysis

Qualitative variables were described by frequencies. Quantitative variables were expressed as the mean with standard deviation, median, and minimum/maximum values.

3. RESULTS

The literature search identified a total of 69 manuscripts for review, attributing HCQ to cardiac disorders. Of these articles, 11 were case studies and 58 were case reports [7-73]. Baseline patient characteristics are summarized in Table **1**. The majority of patients were female (84%), with a median age of 49.2 ± 18.7 years old (range 16–92 years old). Most patients were being treated for SLE, representing 52% of our review, with rheumatoid arthritis (RA) the next most frequent entity treated.

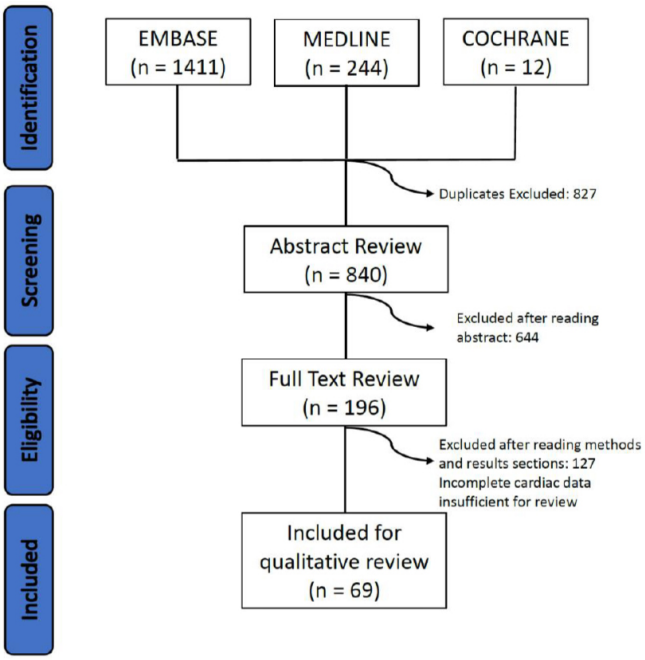

Fig. (1). Article review.

In the cohort of patients admitted for HCQ overdose after acute intoxication, 15 patients presented with adverse cardiac events. Adverse cardiac events in any patient were defined as sudden cardiac death, atrioventricular heart block, new bundle branch block, atrial fibrillation, atrial flutter, QT prolongation, ventricular tachycardia, ventricular fibrillation, torsades de pointes, QRS prolongation, new-onset left or right heart failure, or new-onset valvular disorder. Acute overdose was defined as the intentional ingestion of multiple tablets with purposeful intent to go beyond standard FDA approved dosing regimens. The median dose ingested during acute intoxication prior to hospital presentation was 17,857 ± 14,873 milligrams (mg), with minimum-maximum of 4,000 – 60,000 mg, with details described in Table **2**.

There were no documented episodes of sudden cardiac death after an acute intentional overdose of HCQ. Seven patients had a manifestation of QTc prolongation, three patients developed unstable ventricular tachycardia requiring attempted defibrillation, three patients developed ventricular fibrillation, one developed a prolonged QRS, and one patient developed torsades de pointes. Two patients expired after arrival to the emergency department, one developed ventricular fibrillation and the other pulseless ventricular tachycardia.

Table 1. General characteristics.

Sex n, (%)	**Results**
Female	156 (*84*)
Male	29 (*16*)
Median age, years ± SD	49.2 ± 18.7
Age range, years (min - max)	16-92
Diseases involved, *n (%)*	-
Systemic lupus erythematosus	94 (*52*)
Rheumatoid arthritis	39 (*20*)
Discoid lupus	3 (*2*)
Sjogren syndrome	4 (*2*)
Mixed connective tissue disease	2 (*1*)
Scleroderma	4 (*2*)
Other	45 (*24*)

Abbreviations: SD – standard deviation.

Amongst patients whose intoxication was after long-term chronic usage, the median duration of therapy with HCQ was approximately 10.5 years, with a range of 0.25 – 31 years. The median lifetime cumulative dose was approximately 1,493,800 mg with a range of 16,000 – 3,212,000 mg.

Compared to the acute intoxication arm, patients with long-term HCQ cumulative usage over 10 years, had more literature documentation of arrhythmias and cardiomyopathies. Incidence of atrial fibrillation, bundle branch block, QT prolongation, and left-sided heart failure were more pronounced in long-term intoxication case series compared to acute intoxication case reports.

The data revealed the most common cardiac AE associated with acute HCQ overdose was QT prolongation (7/15 patients). There were no cardiomyopathies reported in acute HCQ overdose. In patients who were using HCQ in the 10.5 ± 8.9-year long-term setting, the most common cardiac adverse event was left-sided heart failure. Table **3** summarizes findings of cardiac adverse events.

4. DISCUSSION

A meta-analysis was performed to evaluate the cardiac adverse events profile of the HCQ pre-COVID pandemic. The findings demonstrated time-dependent variable and cumulative dosage response to the manifestation of adverse cardiovascular outcomes. In acute HCQ overdose, the most common adverse cardiac events were QT prolongation (7 patients), followed by ventricular tachycardia or ventricular fibrillation (3 patients each, respectively). Of the 7 out of 15 patients presenting with adverse cardiac events following acute intoxication, the presence of ventricular tachycardia or ventricular fibrillation is suggestive of an underlying mechanism of significant QT prolongation secondary to massive overdose. Acute HCQ intoxication was successfully treated in the majority of reported cases.

Long-term hydroxychloroquine use in publications demonstrate less common arrhythmic events and more association with structural cardiac abnormalities. After over 10 years of HCQ usage, with cumulative dosage levels exceeding 1,400,000 milligrams, there were sixty reports of new left-sided heart failure felt to be secondary to HCQ administration and 7 reported cases of right-sided heart failure.

To date, it is not well understood if the cardiomyopathy identified with chronic decade use of HCQ is attributable to the medication or progression of underlying autoimmune and connective tissue disease. When HCQ toxicity is suspected, it is standard of care to withdraw the medication and initiate pharmacotherapy for the specific cardiovascular process. There is evidence of high rates of recovery, replicated in our review, when this treatment regimen is enacted. However, further complicating the diagnosis of HCQ-related cardiotoxicity, the underlying inflammatory disorder may cause similar disease processes, independent of HCQ usage [74, 75]. This may lead to poor patient outcomes due to the unnecessary withdrawal of HCQ when toxicity is suspected. Although there are well-established pharmacotherapies for the most common cardiac complications, the underlying autoimmune disease may progressively worsen with the discontinuation of HCQ.

The overwhelming majority of cases of HCQ-related cardiotoxicity occurred at very high cumulative doses of the medication, with once or twice daily dosing over several years. In these chronic-HCQ patients, intoxication often presented with progressive onset of symptoms over many months. As stated above, excluding acute intoxication events, the average duration of therapy of patients with cardiotoxicity was greater than 10 years, with an average cumulative dose of over 1,400 grams. SLE and RA are widely prevalent diseases, with SLE estimated to affect 1.4 million people in North America alone, and RA having a worldwide incidence estimated at 16 million [76, 77]. HCQ is often first- or second-line therapy in a large number of these patients, yet there are only a few reports of cardiac AE. Our data reinforces that patients taking their medication appropriately are at very low risk of experiencing cardiac AE, particularly with short term administration and at the onset of therapy. Of the 69 manuscripts included in this review, only three identified patients experienced cardiotoxicity within

Table 2. Cardiac complications with all HCQ-toxicity.

Dose	Acute Intoxication *(n=15)*	Long-term Intoxication *(n=170)*
Median daily (mg±SD)	-	377 ± 98.2
Median cumulative (mg±SD)	17,857 ± 14,873	1,493,800 ± 995,517
Median duration (years±SD)	-	10.5 ± 8.9
Post-withdrawal change, *n*	-	-
Improvement	13	77
Death	2	35
Stable	-	6
Pacemaker placement	-	9
Heart transplant	-	1
Cardiac disorder, *n*	-	-
1st or 2nd degree AV block	-	9
Complete AV block	-	7
New left or right BBB	1	23
Atrial fibrillation/ flutter	-	4
QTc prolongation	*7	14
Ventricular tachycardia	3	^4
Ventricular fibrillation	**3	1
Torsades de pointes	*1	^2
Widened QRS	1	-
Sudden cardiac death	-	1
New left sided heart failure	-	60
LVEF	-	-
< 40%	-	45
40 – 60%	-	21
> 60%	-	7
New right sided heart failure	-	7
New valve disorder	-	5

Note:* patient had QT prolongation and torsades de pointes.
** case 1 occurred in setting of HCQ, bromazepam, zolpidem, & paroxetine overdose.
** case 2 occurred in setting of rapid correction of hypokalemia.
^ patient had torsades that degenerated into ventricular tachycardia.
Abbreviations: SD – standard deviation.

one year of therapy initiation, making it difficult to elucidate any pattern of susceptible patient populations.

Acute intoxication certainly poses a separate yet equally important threat of toxicity to the cardiovascular system. The most common finding in acutely intoxicated patients appears to be QT prolongation, possibly leading to other, more life-threatening arrhythmias. The average dose of patients presenting with acute HCQ-toxicity was ingestion of approximately 17,000 milligrams, demonstrating a high dosage needed in a short timeframe to cause these life-threatening abnormalities.

While there are well-established guidelines for monitoring of more common HCQ-related AE, there remains debate over periodic cardiac monitoring in patients taking the medication. This literature review did not reveal a single study which incorporated a formal EKG screening protocol in any patient that was initiated on the medication or long term follow up. In 2018, Chatre *et al.* proposed monitoring intervals for cardiotoxicity, with heart screening occurring one month after onset of therapy and biennially thereafter [1]. To date, there is no widespread adoption of monitoring guidelines for HCQ-related cardiotoxicity.

Since its creation, HCQ has had multiple mechanisms of action, allowing its versatility as a medication primarily blocking toll-like receptors in addition to the prevention of stimulation of B cell antigen receptors, thus acting as an anti-inflammatory agent [78]. The proposed mechanism of action of HCQ, as it relates to SARS-CoV-2, involves its ability to prevent membrane fusion, disrupting the viruses' ability to enter cells and begin replication [79, 80]. The underlying pathophysiology of SARS-CoV-2 contributes to cardiac complications in the population it infects, with estimates ranging from 20-40% incidence [81, 82]. Proposed SARS-CoV-2 mechanisms of injury involve a direct injury *via* viral infiltration into the myocardial tissue, leading to inflammation [83]. Additionally, respiratory failure may lead to systemic inflammation, causing further damage to the myocardium. While SARS-CoV-2 patients will be at high risk of cardiotoxicity, this is due to the inherent nature of the virus, and the question remains if the addition of HCQ to their therapy is associated with an increased risk of cardiac AE [84].

Table 3. Acute intoxication case characteristics.

Age (Years) [Ref]	Gender	Dose Ingested (mg)	Pills Ingested (*n*)	Adverse Event	Therapy	Survival
16 [53]	Female	12,000	60	QTc 600 ms	BCB, vasopressor	Yes
16 [50]	Female	20,000	100	QTc 520 ms	BCB, vasopressor, inotropic support, KCl	Yes
16 [40]	Female	*	*	RBBB	IVF, dopamine, GL, KCl	Yes
17 [69]	Female	22,000	110	V-tach	BCB, IVF, GL, AC, MgS	Yes
18 [32]	Female	20,000	100	QTc 564 ms	GL, AC, vasopressor, KCl, MgS,	Yes
19 [41]	Female	6,000	30	V-fib	KCl **	Yes
20 [19]	Female	36,000	180	QTc 563 ms	AC, BCB, MgS, KCl	Yes
20 [47]	Female	60,000	300	QTc 600 ms, TdP	GL, KCl, MgS, ILE	Yes
28 [72]	Female	20,000	100	V-tach	IVF, diazepam, vasopressor	No
29 [73]	Female	14,000	70	V-fib	GL, AC, BCB, MgS, diazepam, vasopressor	No
30 [18]	Male	4,000	20	V-tach	GL, vasopressor **	Yes
39 [43]	Female	12,000	60	V-fib	GL, AC, vasopressor, ECMO	Yes
47 [73]	Female	4,000	20	QRS prolongation	IVF, vasopressor, clonazepam, atropine	Yes
49 [37]	Male	8,000	40	QTc 492 ms	Vasopressor, KCl	Yes
55 [16]	Female	12,000	60	QTc 474 ms	ILE, vasopressor, KCl	Yes

Note:* case reported "handful of 200 mg HCQ tablets", unable to determine exact final dose; ** incomplete treatment regimen provided.
Abbreviations: ms - milliseconds, RBBB - right bundle branch block, GL - gastric lavage, BCB - bicarbonate infusion, KCl - potassium chloride, IVF - intravenous fluids, V-Tach - ventricular tachycardia, AC- activated charcoal, MgS- magnesium sulfate, V-fib - ventricular fibrillation, TdP - torsades de pointes, ILD - intravenous lipid emulsion, ECMO - extra-corporeal membrane oxygenation, [Ref] - Reference number.

Several recent publications have mentioned the use of HCQ at variable doses without significant cardiac toxicity. The RECOVERY trial administered a total of 2400mg of HCQ in the first 24 hours, followed by 800mg per day thereafter for 9 days for a total of 9600mg HCQ in the study arm [85]. A total of 1542 patients were randomized to hydroxychloroquine and compared with 3132 patients randomized to usual standard of care, and upon an urgent internal review of the trial by their safety committee following the publication of the now-retracted LANCET manuscript [4, 86], the Committee found no reasons to suspend the trial due to safety concerns over HCQ [85]. Subsequent analysis by the investigators found no significant difference in 28-day mortality between patients in the HCQ arm *versus* the usual care arm (p=0.10) [85]. Publications by Didier *et al.* have demonstrated efficacy in the treatment of COVID-19 patients with early HCQ + Azithromycin + Zinc therapy [84, 87]. When administered within 72 hours of symptom onset in COVID-19 patients, there was a decreased risk of hospitalization, shorter duration of viral shedding, decreased transfer to the ICU, and lower incidence of death. Out of 3,737 patients, 25 (0.67%) experienced QT prolongation requiring cessation of HCQ in 3 cases. The duration and cumulative dosing of HCQ therapy for SARS-CoV-2 patients did not exceed the thresholds described in our review when cardiotoxic risk begins to become prominent.

There are multiple prospective, randomized clinical trials evaluating the safety of hydroxychloroquine. The United Kingdom's "Solidarity Trial" is an adaptive prospective randomized trial comparing different treatments against COVID-19 syndrome, of which one arm evaluates hydroxychloroquine [88]. This trial was initially stopped on May 27th, 2020, following safety concerns surrounding hydroxychloroquine raised by Mehra *et al.*'s LANCET publication, and then restarted on June 4th, 2020, after the LANCET study by Mehra *et al.* was retracted by the authors due to serious concerns on data integrity [4, 86, 89]. A second randomized clinical trial, the "COPCOV" study by the Mahidol Oxford Tropical Medicine Research Unit, is ongoing and prospectively enrolling 40,000 frontline healthcare workers and staff, evaluating if chloroquine or hydroxychloroquine is able to prevent COVID-19 in the healthcare setting [90]. To date, three smaller randomized clinical trials investigating hydroxychloroquine for outpatient use as pre-exposure prophylaxis, post-exposure prophylaxis, and early treatment have completed data safety monitor board review and reported no ventricular arrhythmias and no sudden deaths [91].

COVID-19 syndrome has been established to trigger a biphasic response in patients infected with SARS-CoV-2, an early viral replication stage, and a subsequent hyperimmune response, cytokine storm. Cardiac complications of cytokine storm have been well documented to involve fatal cardiac dysrhythmias and acute systolic heart failure [92, 93]. Prior to the COVID-19 pandemic, this systematic review demonstrates no literature for acute intoxication of HCQ and sudden cardiac death. From April 2020, till present, numerous publications have been released, describing electrocardiographic QT prolongation in COVID-19 patients attributed to the use of HCQ. Most recently, Kawasaki's disease has been reported in adult and pediatric survivors of COVID-19 [94, 95]. As the world's supply of personal protection equipment starts to get replenished, the implementation of cardiac imaging studies is allowing increased discovery of pericardial effusions, new cardiomyopathies, stent thrombosis, and myocarditis in the setting of acute COVID-19 infection, independent of HCQ administration [92, 93, 96-100].

CONCLUSION

The cardiotoxic AE of HCQ, including atrial and ventricular arrhythmias, ECG changes, and HF, is not a common occurrence. It most often presents with high cumulative doses after many years of therapy or in the setting of a purposeful acute intoxication. It is exceedingly rare for patients to develop cardiac AE while taking recommended doses within the first year of therapy, particularly under the supervision of a cardiologist. There is a divergence in the proliferation of literature pre and post COVID-19 pandemic on the safety of HCQ in patients. The question remains, did the medication change after 70 years, or could there be a different pathophysiology associated with COVID-19 warranting more scientific evaluation of the impact of SARS-CoV-2 on cardiac structure and function. It is important to study and understand the 3 distinct COVID-19 patient groups in the evaluation of the efficacy of medical interventions, such as hydroxychloroquine. In summary, the reported increased arrhythmic events in the COVID-19 era appear to be more related with the direct inflammatory effect of the virus (myocarditis) or the concomitant administration of multiple drugs capable of prolonging QT intervals rather than to hydroxychloroquine itself [91, 101]. Given the dynamic pathophysiology of the COVID-19 syndrome, larger studies are needed to understand the role and impact of hydroxychloroquine in pre-exposure, early post-exposure, and late post-exposure therapeutics.

CONSENT FOR PUBLICATION

Not applicable.

STANDARD OF REPORTING

PRISMA guidelines & methodologies were followed.

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CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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Declared none.

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