HYDROXYCHLOROQUINE-INDUCED CARDIOTOXICITY: A CASE SERIES

SC Heyliger (MD Candidate)[1], NL Altman (MD)[2], J Maloney (MD)[3]

[1]University of Colorado Anschutz Medical Campus, [2]Department of Medicine-Cardiology, University of Colorado Hospital, [3]Department of Medicine-Pulmonary Sciences & Critical Care, University of Colorado Hospital

Objective: Aid in determining the prevalence of hydroxychloroquine (HCQ)-induced cardiomyopathy through a literature review and presentation of a case series with the goal of creating guidelines for quick, non-invasive cardiac-screening before irreversible histopathologic damage occurs.

Background: Hydroxychloroquine (HCQ) has been used for decades to treat various rheumatologic diseases such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE); however, it gained name recognition following the COVID-19 pandemic in early 2020 as an unproven treatment. Although retinal toxicity is a widely recognized complication of HCQ use, cardiotoxicity has been less frequently reported in the literature, likely due to subclinical presentations as well as overlapping clinical signs, symptoms, and results that can often mimic other disease processes. Diagnosis of HCQ-induced cardiomyopathy consists of clinical findings of congestive heart failure and/or conduction abnormalities.

Methods: Over seven years (before 2020) the Univ. of Colorado heart failure team obtained endomyocardial biopsies on six patients taking HCQ for rheumatologic conditions. Endomyocardial biopsy was utilized when the cause of cardiomyopathy was unclear or suspected to be related to HCQ use. IRB approval was approved for this study and a subsequent retrospective chart review of previously identified patients was performed to obtain clinically significant data points.

Results: All six patients were biologically female and on HCQ for SLE or rheumatoid arthritis. HCQ treatment lengths ranged from 2-25 years (mean 17 years). Ages ranged from 35-76 years (mean 62 years). Baseline echocardiogram demonstrated ejection fractions (EF) of 22% to 71% with a mean of 36%. Mean baseline NYHA Classification while on HCQ was 3. After discontinuation of HCQ, the average NYHA Classification was 2.4. 50% of the patients had a right ventricular systolic pressure >40mmHg at baseline. Notably, on endomyocardial biopsy, 100% of the patients had cardiomyocyte degradation as evidenced by vacuolization with lysosomal inclusions. 5 out of the 6 patients had curvilinear bodies detected on endomyocardial biopsy, the pathognomonic finding of HCQ-induced cardiomyopathy.

Conclusions: Although rare, HCQ-induced cardiomyopathy is a serious, and potentially fatal, adverse event that is often under-recognized due to its mimicry of other disease processes such as SLE-induced myo/pericarditis, Fabry disease, or infiltrative cardiomyopathies such as sarcoidosis or amyloidosis. Accurate diagnosis can be achieved through careful correlation of clinical history, cardiac catheterization, and histopathologic examination using light and electron microscopy. Further studies are needed to determine the true prevalence of HCQ-induced cardiomyopathy and potential predictive factors such as duration of HCQ use and total dose exposure to develop standardized cardiac screenings for patients taking HCQ.