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Hydroxychloroquine Cardiac Toxicity in a Systemic Lupus Erythematosus Patient

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Learning Objectives

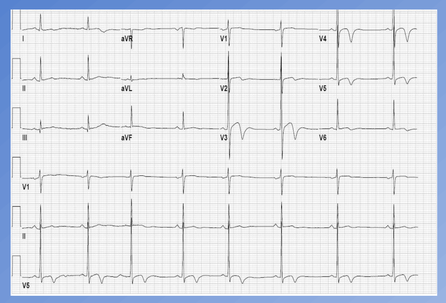
- **Identifying patients at risk of Hydroxychloroquine toxicity**
- **Diagnosing Hydroxychloroquine cardiac toxicity**

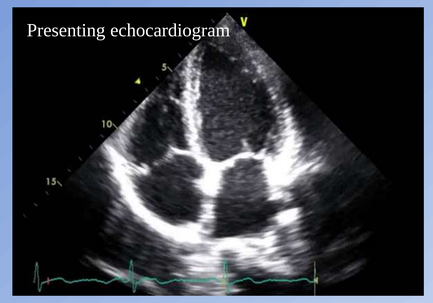
Background

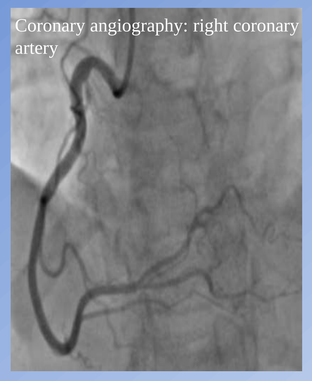
Prior literature details Hydroxychloroquine (HCQ) toxicities including retinopathy, neuromyopathy and cardiomyopathy. HCQ cardiac toxicity is an underdiagnosed condition and failure to withdraw the offending drug can lead to preventable adverse effects. A strong index of suspicion is required for patients on long-term therapy with HCQ to prevent this potentially fatal condition. We report a case of a patient with systemic lupus erythematous (SLE) found to have early HCQ cardiac toxicity.

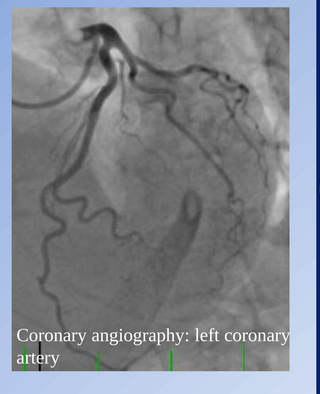
Case

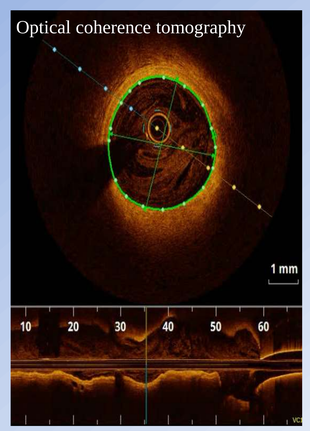
59 year old female with a 30 year history of SLE stable on HCQ 200 mg twice a day since diagnosis presented with sub-acute onset of atypical chest pain. Initial work-up revealed an **ECG** with biphasic T waves in anterior leads and a slightly elevated cardiac troponin I (**cTnI**) level of 0.16 ng/ml, with stable serial measurements. Creatinine phosphokinase was mildly elevated at 275 IU/L. Inflammatory markers were normal. An **echocardiogram** (TTE) was normal except for a significant increase in estimated LV mass of 205 g. Cardiac **catheterization** and coronary angiography along with optical coherence tomography (**OCT**) showed non-obstructive coronary artery disease and no evidence of spontaneous coronary artery dissection. Cardiac magnetic resonance imaging (**cMR**) was done which was unremarkable.

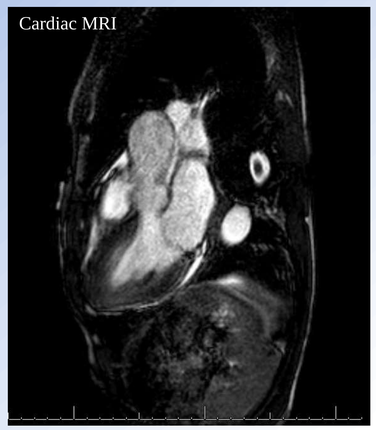






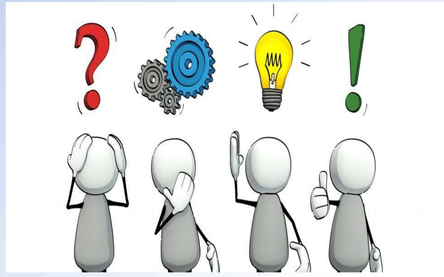




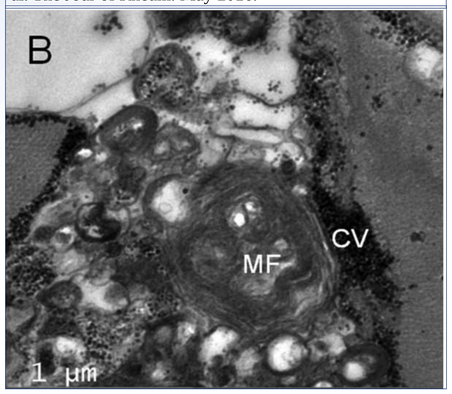


Decision Making

There was continued clinical signs of cardiac injury, including persistent chest pain along with mild cTnI elevation upon follow up in clinic. She also admitted to slowly worsening exercise capacity and strength for the past several months. A **skeletal muscle biopsy** was performed. Pursuing a muscle biopsy rather than cardiac biopsy was considered because of clinical signs of chronic progressive myopathy (decreased physical activity) and less invasive with lower risk. Results showed myopathic changes and several collections of curvilinear bodies (diagnostic), largely consistent with HCQ toxicity. **HCQ therapy was discontinued.** On follow up ~4 months later, she denied chest pain and reported improvement in strength. Her **cTnI** level was decreased to 0.07 ng/ml. ECG was unchanged. **TTE** showed an estimated LV mass of 165 g, decreased from prior.



Electron microscopy of a skeletal muscle biopsy revealing curvilinear bodies. Taken from Heart Involvement in a Woman Treated with Hydroxychloroquine for Systemic Lupus Erythematosus Revealing Fabry Disease. Chatre et al. The Jour of Rheum. May 2016.



Graphic representation of cTnI level over time

Discussion

Our case demonstrates that **ECG** and **cTnI** might be useful screening tools in patients on long-term HCQ therapy. **LV mass** on TTE might be a sensitive marker of early HCQ-cardiac toxicity. Additionally, with the clinical context taken into consideration, an EMB can be forgoed by pursuing other less invasive tissue-organ **biopsies** if clinically suspected to be involved.

Conclusion

We report HCQ-induced cardiac toxicity in a patient with a high cumulative dose of HCQ diagnosed via peripheral skeletal muscle biopsy in the setting of: biphasic anterior T wave changes on ECG; mild elevation of cTnI levels; and no abnormalities on TTE and cMR other than elevated LV mass. Our report highlights the importance of routine follow-up and screening along with a high index of suspicion in patients undergoing long-term HCQ therapy.