Antimalarial-induced cardiomyopathy: retrospective case series

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Background

Antimalarial drugs chloroquine and hydroxychloroquine represent an important therapeutic option in autoimmune diseases. Rare cases of potentially lethal antimalarial-induced cardiomyopathy (AMIC) have been described. Pattern of minimal changes with wall hypertrophy, followed by restrictive and end-stage dilative cardiomyopathy was proposed in AMIC.

Purpose

Our aim was to identify patients with definite AMIC and characterize their clinical and imaging findings.

Methods

Retrospective analysis of admissions to Cardiology department from January 2010 to January 2019 was done to identify patients with definite diagnosis of AMIC based on positive endomyocardial biopsy. Clinical, echocardiographic, and cardiac magnetic resonance (CMR) imaging findings were reviewed if available.

Results

Three patients with biopsy proven diagnosis of AMIC were identified. Two patients, 62 and 76-year-old females, presented with signs of congestive heart failure, while the third, a 41-year-old man presented with chest pain and cardiac conduction abnormalities necessitating pacemaker implantation. All were treated with chloroquine phosphate. Both females had a history of pacemaker due to atrioventricular block. All had positive troponin and coronary artery disease was excluded.

Echocardiography in the first case showed a severely dilated left ventricle (LV) with global hypokinesia and severe systolic dysfunction (LVEF 30%). CMR confirmed dilative cardiomyopathy, furthermore subepicardial late gadolinium enhancement (LGE) of the inferolateral LV wall and of the right ventricle free wall was present. In the second case a restrictive cardiomyopathy pattern was observed with mild biventricular systolic dysfunction and biatrial enlargement. CMR of this case was not available. Echocardiography in the third case showed a normal sized LV with mild diastolic and systolic dysfunction (LVEF 50%), along with septal hypokinesia. CMR showed patchy subepicardial and mid-wall LGE of the septum that was falsely attributed to myocarditis. Additionally, a chemical shift artefact suggestive of myocardial fatty infiltration (Image 1) was visible in the apical septum.

After discontinuation of chloroquine two patients improved, while the first patient deteriorated and eventually succumbed to refractory heart failure.

Conclusions
Our three cases confirm high diversity of cardiac imaging findings in AMIC. Even mild non-specific findings such as LV systolic dysfunction or wall motion abnormalities in patients on antimalarial drugs should therefore be thoroughly investigated. CMR with non-ischaemic LGE pattern may aid in diagnosis, however definite diagnosis is currently possible only with endomyocardial biopsy. Novel imaging techniques, such as T1 mapping, have a potential to increase the diagnostic yield given the known pathohistological similarities between AMIC and Anderson-Fabry disease.