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Case Report

Newly Diagnosed Heart Failure with Renal Insufficiency – Cardiac MRI-Guided Clinical Decision Making

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Abstract

A 63 year-old female with multiple atherosclerotic risk factors presented with newly diagnosed heart failure. Clinical presentation and initial work-up studies (e.g. laboratory findings, ECG, and echocardiography) were indeterminate for ischemic or non-ischemic etiology. As she developed contrast-induced nephropathy, coronary angiography was initially deferred and cardiac MRI was performed instead. Cardiac MRI elegantly demonstrated non-ischemic cardiomyopathy that was subsequently confirmed by invasive coronary angiography. This case emphasizes the important role of cardiac MRI in establishing the etiology of cardiomyopathy, ultimately altering the clinical management of the patient with newly diagnosed heart failure.

Key words: heart failure; cardiac magnetic resonance imaging; ischemic cardiomyopathy; cardiomyopathy

Introduction

A 63 year-old female with hypertension, hypercholesterolemia and active cigarette smoking presented with orthopnea and dyspnea on exertion that had worsened over the previous 2 months. Pulmonary embolism was ruled out with chest CT angiogram. ECG showed new T-wave inversion in the lateral wall when compared to an old tracing (**Figure 1**).

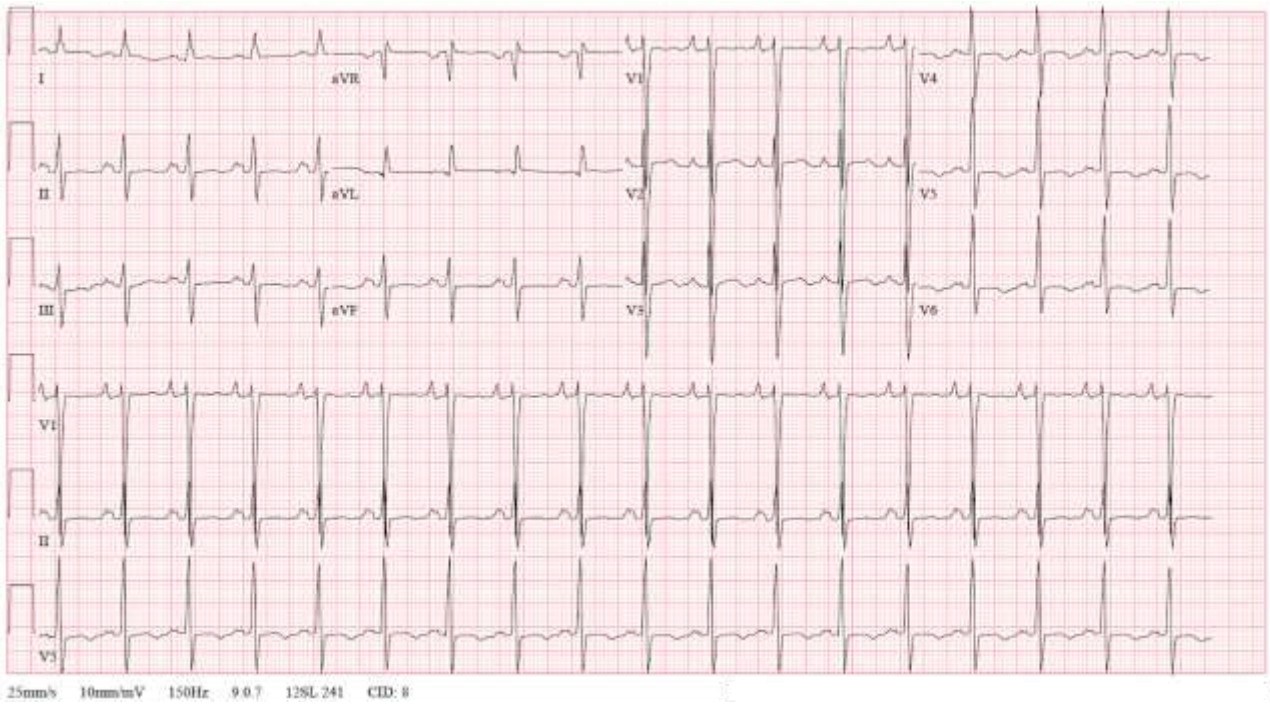

Figure 1: *ECG on admission.*

Laboratory data showed severely elevated pro-BNP (23,615 pg/mL). Initial troponin T was undetectable. Her renal function worsened secondary to contrast-induced nephropathy (Cr 1.5 mg/dL [baseline 0.9 mg/dL], eGFR 42 ml/min/1.73 m^2 [baseline 71 ml/min/1.73 m^2]). Echocardiography showed biventricular dilatation and decreased systolic function (LV EF 20%) with regional wall motion abnormalities (**Video 1**).

Video1_anno.avi

Video 1: *Echocardiography of apical (A) 4-chamber, (B) 2-chamber, and (C) 3-chamber views. The left ventricle was mildly dilated with severely decreased function. The right ventricular function was moderately decreased. The left ventricular wall motion was globally severely hypokinetic with regional variations. Inferior and inferoseptal segments were akinetic, whereas the wall motion of basal anterior and lateral segments were rather preserved. The findings were suggestive of ischemic cardiomyopathy.*

Cardiac MRI cine imaging showed severely dilated LV with severely decreased systolic function (LVEF 17%) with global hypokinesis (**Video 2**).

Video2_anno3b.avi

Video 2: *Cardiac MRI steady-state free precession (SSFP) cine imaging of long axis views: (A) 2-chamber, (B) 3-chamber, and (C) 4-chamber views. Cardiac MRI cine imaging showed severely dilated left ventricle with severely decreased systolic function (ejection fraction 17%). The left ventricular wall is globally severely hypokinetic with minimal regional variations. There were prominent trabeculations in the anterior wall from base to apex that did not meet the criteria for non-compaction. The right ventricle was severely dilated with moderately decreased systolic function.*

On late gadolinium enhancement imaging, there was patchy myocardial fibrosis in the anterolateral papillary muscle and a small portion of the mid lateral wall, mid-wall fibrosis in the basal anteroseptal segment, and fibrosis at both anterior and inferior right ventricular insertion sites (**Figure 2A**). Cardiac MRI findings were compatible with non-ischemic cardiomyopathy. Invasive coronary angiography performed due to subsequent troponin elevation revealed normal coronaries (**Figure 2B**).

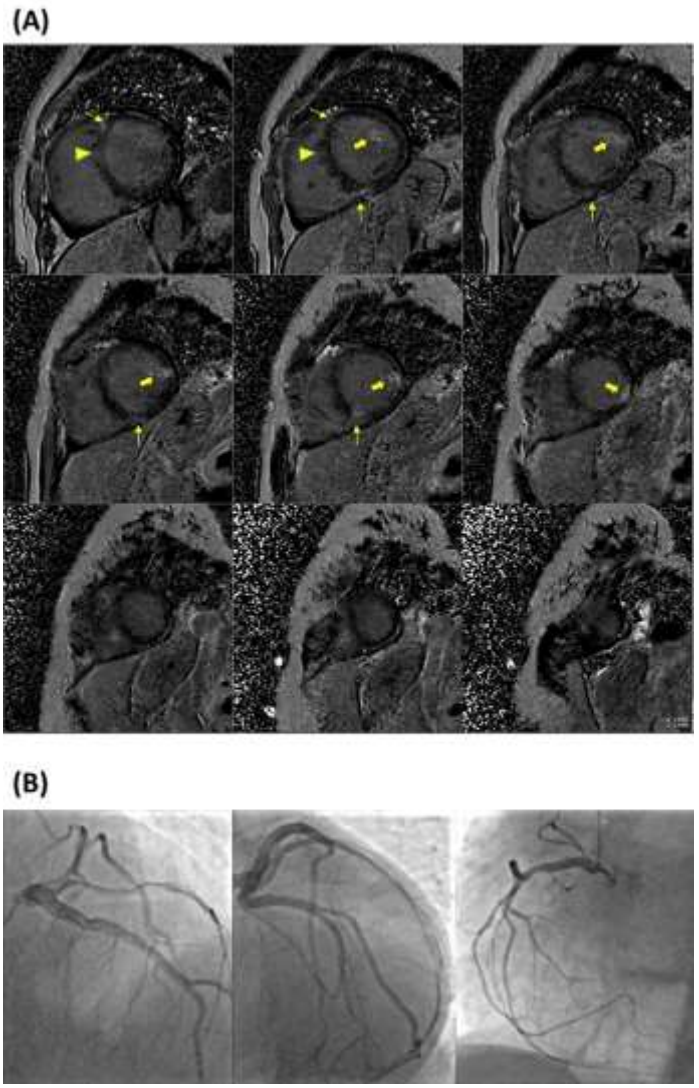

Figure 2: (A) *Cardiac MRI: late gadolinium enhancement imaging (PSIR) in short axis stack. There is patchy myocardial fibrosis in the anterolateral papillary muscle and a small portion of the mid lateral wall (thick arrows). There is mid-wall fibrosis in the basal anteroseptal segment (arrow head). Additionally, there is fibrosis at both anterior and inferior right ventricular insertion sites (thin arrows). The pattern of myocardial fibrosis is compatible with non-ischemic cardiomyopathy.* ***(B)*** *Coronary angiography: (left) RAO cranial of LCA, (middle) LAO caudal view of LCA, and (right) LAO view of RCA. There was no significant coronary artery disease.*

Antiplatelet therapy was discontinued. She was discharged home with low dose furosemide, carvedilol, losartan, isosorbide dinitrate, and hydralazine. Her symptomatic status improved and furosemide was discontinued. Losartan was then switched to sacubitril/valsartan. Subsequent dose up-titration of her regimen was well tolerated and she remained in NYHA class I. Follow-up echocardiography showed normalization of LV size along with improvement of the systolic function (LVEF 45 %) 4 months after her initial presentation.

Coronary artery disease is the most common cause of systolic heart failure in Western countries. Ischemic cardiomyopathy is associated with decreased survival when compared to non-ischemic cardiomyopathy [1]. If feasible, surgical coronary revascularization improves survival when compared to medical management [2]. This case emphasizes the important role of cardiac MRI in establishing the etiology of cardiomyopathy, ultimately altering the clinical management of the patient with newly diagnosed heart failure.

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