Acute Myocarditis and Cardiac Magnetic Resonance Imaging

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A thirty-two-year-old female presented with acute myocarditis which was confirmed with Cardiac Magnetic Resonance Imaging (CMRI) where endomyocardial biopsy sampling was not feasible. The patient was managed initially with dual anti-platelet therapy and heparin; the patient was discharged home after four days on 75 mg aspirin.

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Myocarditis is an inflammatory disorder of the myocardium with multiple etiologies and variable clinical presentations. The inflammation can be secondary to viral infections, autoimmune mechanisms and a reaction to certain medications and toxins. The disease itself is well recognized for its ambiguous clinical presentation, which ranges from no symptoms to unexpected death. Physicians have struggled for decades attempting to achieve early diagnosis due to the diversity of the symptomology and lack of specific diagnostic techniques[1,2].

The diagnosis of myocarditis is frequently made empirically by clinical presentation, ECG changes, elevated cardiac enzymes and lack of coronary artery disease[3]. The gold standard investigation is endomyocardial biopsy (EMB). Although highly specific, it lacks sensitivity, as the disease can affect the myocardium heterogeneously. In addition, it is an invasive technique with associated risks; a high level of suspicion is needed prior to its performance. The diagnosis could be achieved by Nuclear Imaging and Cardiovascular Magnetic Resonance Imaging (CMRI), the latter has an increasing role in the diagnostic process[1]. Until recently CMRI was employed primarily as an adjunct to EMB. CMRI is gaining special importance in solely diagnosing acute myocarditis.

Studies have been performed to compare the equivalence of the standard endomyocardial biopsy to the innovative CMRI in the diagnosis of acute myocarditis[4]. It has been concluded that the high spatial resolution it offers, safety, accessibility and limited contraindications makes CMRI the ideal alternative[5].

The aim of this presentation is to report a case of a young female successfully diagnosed with acute myocarditis subsequent to CMRI.

THE CASE

A thirty-two-year-old female with no known cardiovascular risk factors presented with acute chest pain. The patient had no history of recent illness or infection. The patient was admitted on 30 June 2016. During the examination, the patient developed sudden constant retrosternal non-radiating chest pain, but the vitals were stable. On auscultation, heart sounds were normal and breathing was vesicular.

The investigations revealed elevated cardiac enzymes with peak values of CK, CK-MB and troponin (1161 IU/L, 96 ng/mL and 10ng/ml, respectively). There were no other signs of inflammation (WBC of 8.29x10^9/L, C-reactive protein of 1.06mg/L, Erythrocyte sedimentation rate of 4 mm/hour); renal and liver function tests were normal. Echocardiography revealed no left ventricular regional wall motion abnormalities, with an ejection fraction (EF) of 60%. There was no evidence of pericardial thickening or effusion.

Chest X-ray was normal and an electrocardiogram revealed sinus rhythm and 1 mm convex ST-segment elevation in the limb leads II, III and AVF, see figure 1. Troponin I was 8.1 ng/mL and potential diagnosis of acute inferior wall myocardial infarction was made.

The patient was managed with dual antiplatelet therapy and heparin. Angiography was performed on the same day of admission, which revealed normal epicardial coronary arteries and the patient was subsequently transferred to the CCU for further observation, see figures 1 and 2.

On the first and second days of admission, the patient experienced two episodes of chest pain which responded promptly to analgesics. Her state was deemed stable, and she was transferred to the ward with continuous telemetry. There was no evidence of arrhythmias during her hospital stay and the ST segment changes normalized on day 3, see figure 4.

In the absences of coronary pathology, a diagnosis of acute myocarditis was suspected; CMRI was performed on 4 July 2016. The CMRI showed mild hypokinesia in mid to inferior

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apical wall and inferior septum, with EF of 58% and on late enhancement images there was sub-epicardial uptake of gadolinium (Gad) in the mid to apical inferior wall and inferior septum, features supporting acute myocarditis, see figure 5 (A and B).

The patient was discharged on 4 July 2016 in a hemodynamically stable condition and daily 75 mg of aspirin. During follow-up, Parvo-B19 virus antibodies IgG was positive. Other viruses were negative including Coxsackie antibodies. The patient complained of palpitation and Holter was requested but did not show significant arrhythmias except for few ventricular ectopic beats. A small dose of beta blockers was initiated.

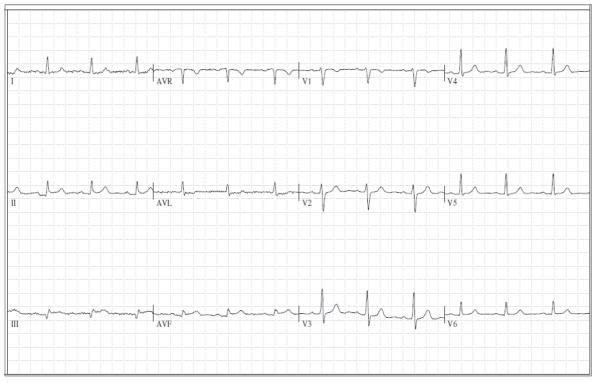

Figure 1: ECG on Presentation

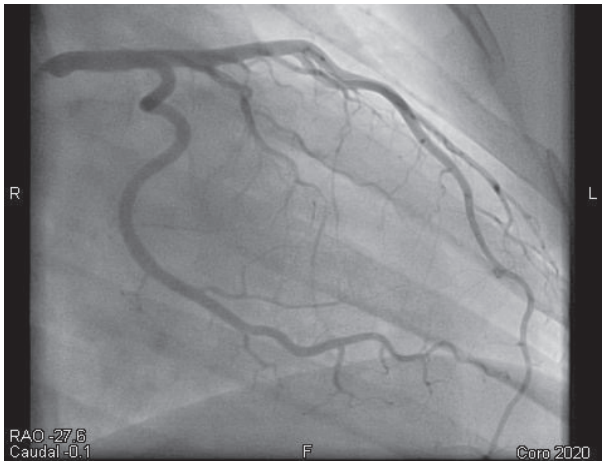

Figure 2: Normal Left Coronary Tree

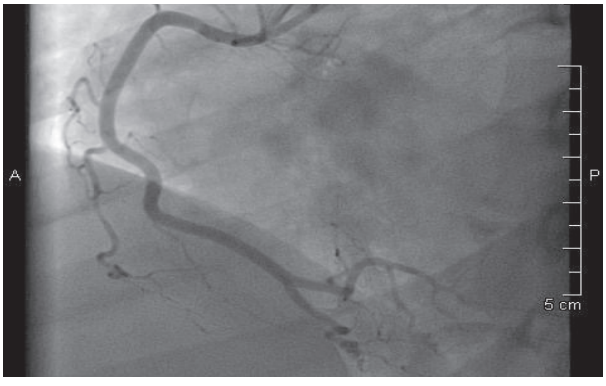

Figure 3: Normal Right Coronary Tree

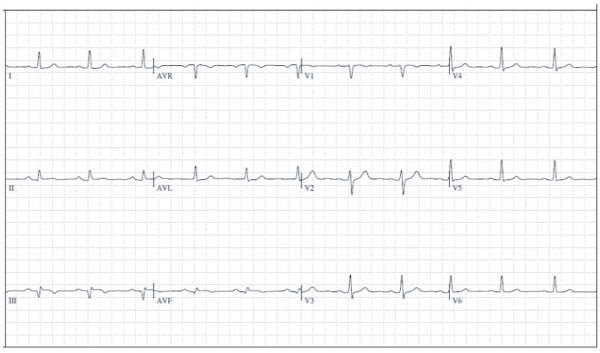

Figure 4: ECG on the Second Day of Admission

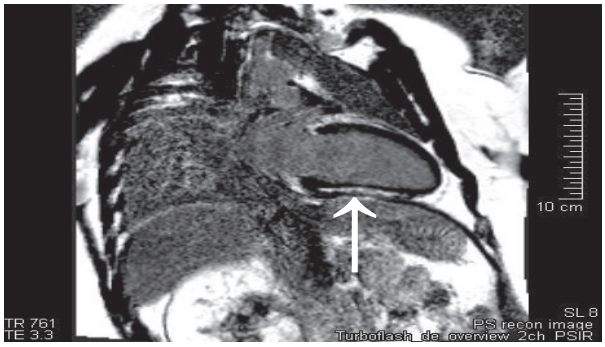

Figure 5 (A): Sub-epicardial Gad Uptake in the Inferior Wall of the Left Ventricle (Arrow)

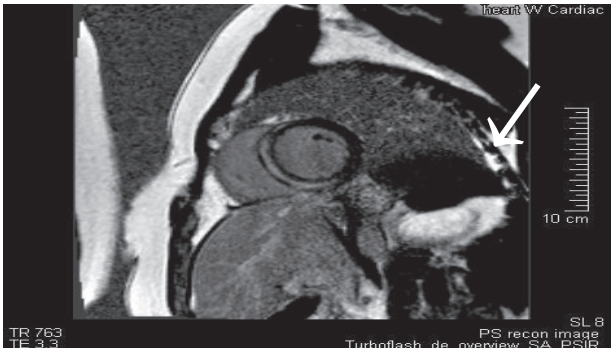

Figure 5 (B): Subepicardial Gad Uptake in the Mid Inferior and Inferoseptal Wall of the Left Ventricle (Arrow)

DISCUSSION

Multiple technical methods of CMRI could be implemented within a single examination in different combinations to evaluate the functional parameters of the left ventricle, the morphology and perfusion of the myocardium and the underlying myocardial damage.

CMRI has a unique potential to visualize tissue changes. In active myocarditis, the changes could be intracellular and interstitial edema, capillary leakage, hyperemia and in severe cases, cellular necrosis and fibrosis[6].

Myocardial Late Gadolinium Enhancement (LGE) reveals necrosis and fibrosis. The imaging uses an inversion pulse to decrease the signal response from normal myocardium;

therefore, revealing the increased accumulation of gadolinium as bright regions[5]. In myocarditis, the reported sensitivity of this method is 100% with a specificity of 90%[5]. Delayed contrast enhancement was also observed in 44%-90% of patients with irreversible myocardial injuries[5,7].

Patients with active myocarditis might reveal focal signal increases typically localized in the subepicardial regions of the LV and through the ventricular wall. The LGE may be localized in the inferolateral and anteroseptal segments, but it might be multifocal or diffuse in distribution. The subendocardium is not involved in isolation, a distinguishing feature from ischemia-mediated injury[5].

T2-weighted imaging via the employment of a pulse sequence sensitive to an increase in both regional and global myocardial water is recognized as a well-established feature of the inflammatory response in acute myocarditis[7,8]. Myocardial edema appears as an area of high signal intensity in T2-weighted images. In myocarditis, it may be regional or global. In the absence of LGE, edema reflects reversible myocardial injury[5].

In patients with clinical signs suspicious of the diagnosis, the use of CMRI could determine the need for further investigations and the management plan. Early diagnosis of myocarditis is vital to avoid further morbidity and mortality. The significance of the imaging is not limited to the diagnosis; it demonstrates an equally imperative function in the follow-up of acute myocarditis and its succeeding stages by allowing a non-invasive reassessment of the myocardial response to treatment[5,9]. Furthermore, this method could improve the sensitivity of endomyocardial biopsy in acquiring a more accurate histological diagnosis[3].

CONCLUSION

Cardiac MRI is gaining a crucial role in the diagnosis of acute myocarditis. It could detect the tissue changes occurring in inflammation when other existing diagnostic tools proved unsatisfactory. It is achieved through the detection of three tissue markers: edema, hyperemia/ capillary leak and necrosis/fibrosis. The signal changes detected by the contrast enhanced magnetic resonance imaging reflect the existing inflammation.

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