Elvira Bangert et al., J Clin Exp Cardiolog 2014, 5:3
http://dx.doi.org/10.4172/2155-9880.S1.020

OMICS Group Conferences
Accelerating Scientific Discovery

4th International Conference on
Clinical & Experimental Cardiology
April 14-16, 2014 Hilton San Antonio Airport, TX, USA

Takotsubo cardiomyopathy triggered by necrotizing myopathy: The role for cardiac magnetic resonance imaging

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Objectives: This is a case presentation to 1) illustrate the utility of cardiac magnetic resonance imaging (CMR) in diagnosing Takotsubo cardiomyopathy (TC); 2) demonstrate that necrotizing myopathy (NM) can trigger TC.

Background: Approximately 2-3% of patients presenting with acute coronary syndrome (ACS) have TC. CMR is an emerging noninvasive gold-standard method for diagnosing TC. Typical TC triggers include severe emotional or physical stress, and, in some situations, an illness.

Methods and Results: A 61-year-old female presented with retrosternal chest pain, shortness of breath, and diaphoresis. She had a 1-day history of proximal muscle weakness and fever. She reported no recent stressful events. Electrocardiography showed ST segment elevation in leads V2-V5 and anterolateral Q waves. Serum troponin I and creatine kinase (CK) were slightly elevated. One-day post-admission, troponin I decreased but CK rose. Angiography did not show any significant coronary obstruction. Ventriculography revealed anterolateral, apical, and inferoapical akinesis with hyperdynamic basal constrictors. Echocardiography demonstrated left-ventricular ejection fraction (LVEF) of 30 %. On CMR, there was high apical signal on edema-sensitive sequences but no enhancement on late gadolinium enhancement (LGE) sequences. Electromyography and muscle biopsy confirmed NM. Muscle weakness improved and CK level normalized soon after initiation of prednisone and azathioprine treatment. One month post-discharge, LVEF was 63%.

Conclusions: Echocardiography, angiography, and CMR findings were consistent with the 2008 Mayo Clinic TC criteria. It was important to rule out myocarditis as it could

present with features of ACS in the setting of unobstructed coronary arteries and LV apical ballooning. CMR is useful in distinguishing TC from myocarditis, which may also present with clinical and ECG features of ACS in the setting of unobstructed coronary arteries and LV apical ballooning. Myocarditis may also be a feature of some inflammatory myopathies. CMR findings of a regional apical elevated signal on edema-sensitive pulse sequence imaging, combined with the absence of LGE, argue against myocarditis or myocardial infarction, and represent a typical pattern for TC. Similar to our case, the reversibility of both clinical symptoms and LV systolic dysfunction is also characteristic of TC. To our knowledge this is the first report of TC triggered by NM.

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