Acute MI and Cardiac Amyloidosis

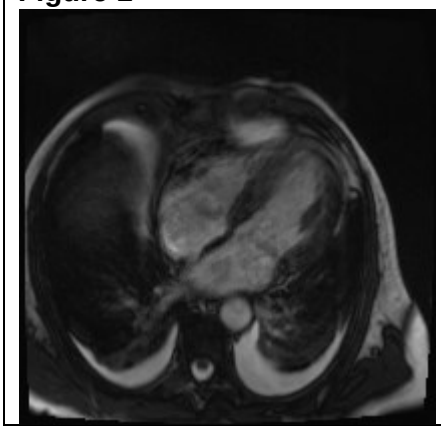
Author: Dr Swamy Gedela, Department of Cardio-thoracic Radiology, Cardiac MRI Unit, Essex Cardio-thoracic Centre

Clinical History

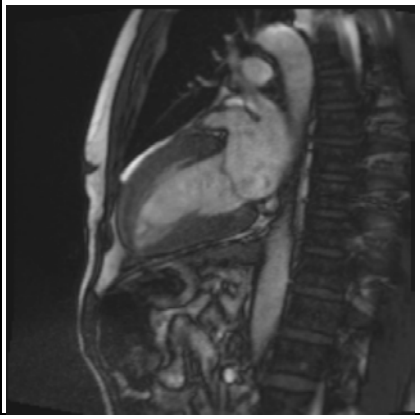
An 80-year male with an acute coronary syndrome had a primary PCI to his LAD coronary artery. Subsequent echocardiogram showed anterior regional wall motion abnormalities but also concentric LVH (left ventricular hypertrophy) with a speckled appearance. Patient referred for CMR (cardiac MRI) to assess for an infiltrative cause of LVH.
As patient was in atrial fibrillation a prospective gated study was performed.

CMR Findings: SSFP Cine images show a left ventricle with severe systolic impairment (LVEF 36%), poor long axis function, severe concentric LVH (maximum LV wall thickness 2cm), inter-atrial septal thickening, bi-atrial enlargement and bilateral pleural effusions. (Figure 1 and movie 1).

Figure 1



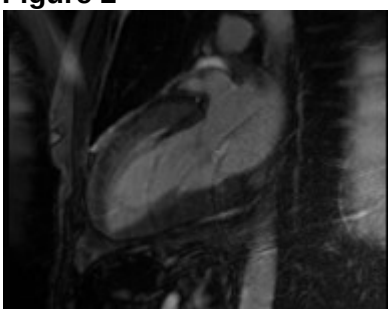
Movie 1



SSFP T2 weighted images

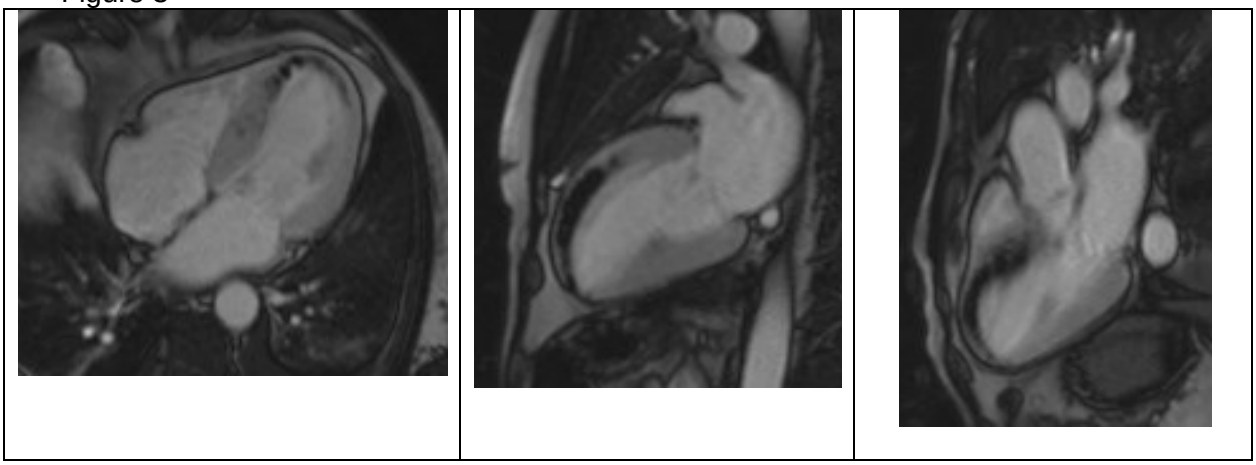
High signal at the anterior wall in keeping myocardial oedema. (Figure 2)

Figure 2



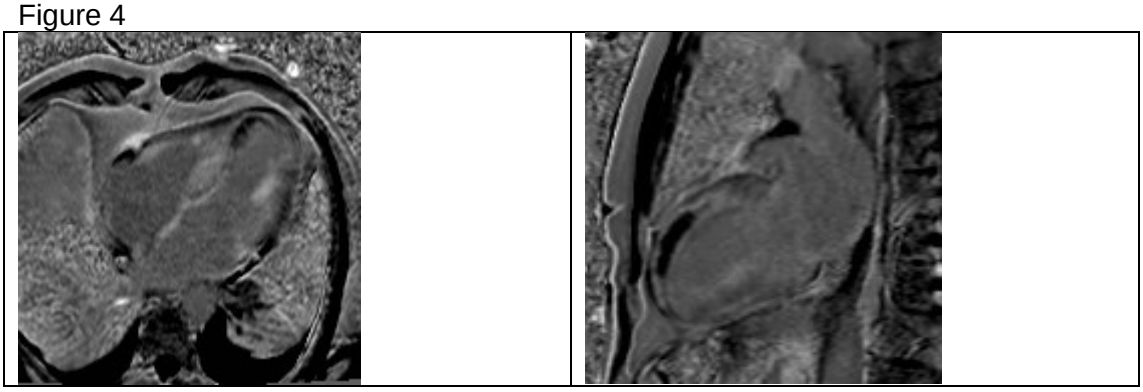
Early gadolinium phase:
In the early phase after gadolinium a core of hypointensity is seen at the mid to apical cavity anterior and antero-septal segments of the left ventricle which is characteristic of early MVO (microvascular obstruction). (Figure 3)

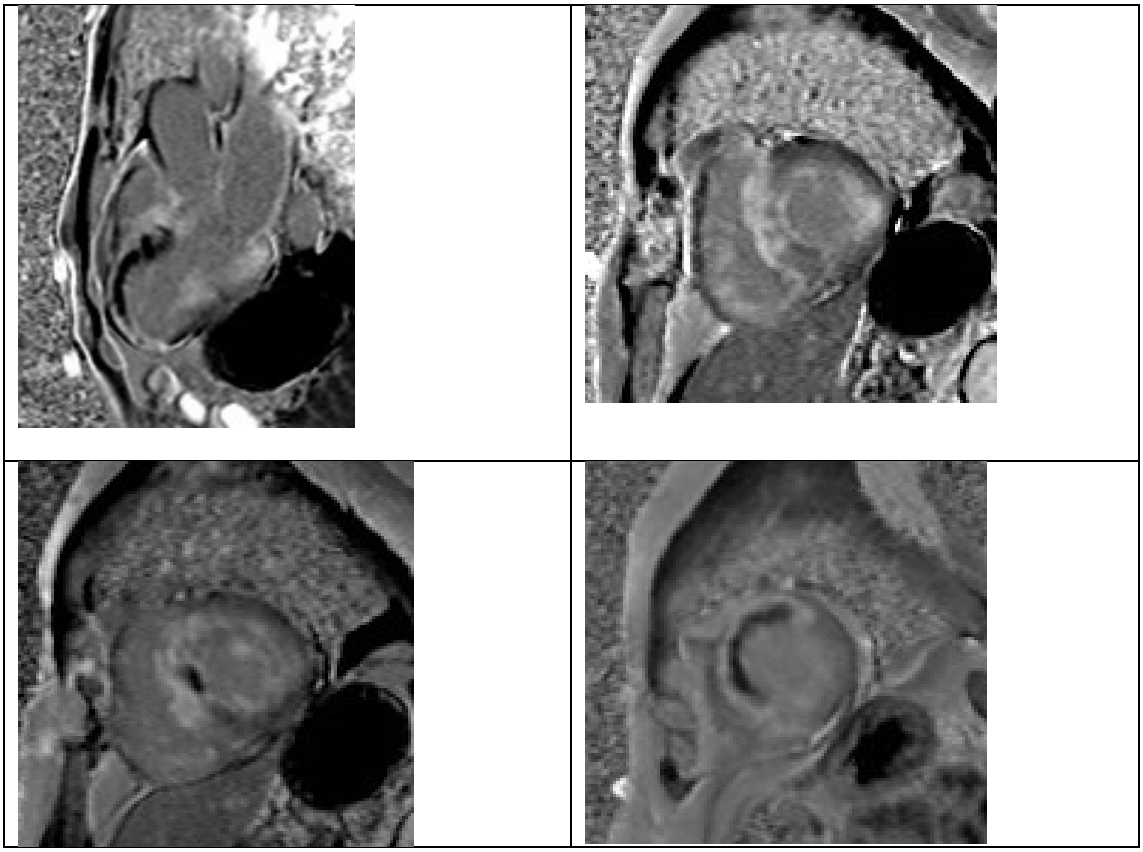
Figure 3



Late phase after Gadolinium injection: Diffuse left and right ventricle enhancement with a predominant subendocardial pattern. In addition there is transmural infarction at the mid to apical cavity anterior and antero-septal segments with late MVO. (Figure 4)
In cardiac amyloidosis CMR imaging demonstrates a characteristic pattern of global subendocardial late enhancement combined with abnormal myocardial and blood-pool kinetics.

Figure 4





Comment:
This case shows the value of CMR in diagnosing a dual pathology of both infiltrative cardiomyopathy Amyloidosis and myocardial infarction with microvascular obstruction.
Amyloidosis is caused by the extracellular deposition of pathological insoluble proteins. Cardiac involvement varies with the type of amyloidosis and is frequent in AL amyloidosis (cardiac light chain amyloidosis) and ATTR amyloidosis (amyloid deposits of unstable variant of transthyretin) (1). The prognosis is better with ATTR (typically 3-5 years) compared with < 12 months for AL amyloidosis (2,3).

CMR gives a specific type of late enhancement with Amyloidosis, which typically is diffuse, global and subendocardial (4).

Microvascular obstruction is associated with acute infarction and typically occurs as a manifestation of reperfusion injury. It is associated with poor outcome and adverse ventricular remodeling (5).

CMR delivers detailed and high-resolution imaging. It provides diagnostic information, as in this case of a new diagnosis of heart failure secondary to Cardiac Amyloidosis with acute myocardial infarction and microvascular obstruction.

References:

1. Falk RH, Comenzo RL, Skinner M. The systemic amyloi- doses. N Engl J Med 1997;337:898–909.

2. Dungu JN, Anderson LJ, Whelan CJ, Hawkins PN. Cardiac transthyretin amyloidosis. Heart 2012;98:1546–54.

3. Lebovic D, Hoffman J, Levine BM, et al. Predictors of survival in patients with systemic light-chain amyloidosis and cardiac involvement initially inel- igible for stem cell transplantation and treated with oral melphalan and dexamethasone. Br J Haematol 2008; 143:369–73.

4. Maceira AM, Joshi J, Prasad SK, et al. Cardiovascular magnetic resonance in cardiac amyloidosis. Circulation 2005; 111:186–93.

5. Gerber BL, Rochitte CE, Melin JA, et al. Microvascular obstruction and left ventricular remodeling early after acute myocardial infarction. Circulation 2000;101:2734–41.