Abstract: P576

15 minutes after Gadolinium two diagnoses appear

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Clinical Case: A 52-year-old female was admitted after an episode of chest tightness on exertion. The admission ECG demonstrated ST and T-wave changes suggestive of myocardial ischaemia and peak Troponin T (TnT) was 331ng/L. Invasive angiography demonstrated only mild luminal irregularities in the distal LAD and a cardiac MRI (CMR) was therefore obtained.

The CMR showed a non-dilated left ventricle (LV), an akinetic LV apex and a likely apical LV thrombus (Figure). The right ventricle (RV) also demonstrated regional wall motion abnormalities including 2 small aneurysmal segments in the free wall of a non-dilated RV with normal ejection fraction. Elevated values for T2 (up to 87msec) and T1 (up to 1237msec) were detected in the LV apex.

Three patterns of late gadolinium enhancement (LGE) were present (Figure). Subendocardial apical LGE was most suggestive of a small acute myocardial infarction (AMI) with an adherent small mural thrombus. A separate area of nearly transmural LGE (sparing endocardium) was evident in the mid-anterolateral segment which was associated with increased LV wall thickness. The third pattern consisted of thin mid-wall LGE in the basal to mid inferior segment. Inpatient CT FDG-PET imaging was suggestive of acute inflammation in the apex and mid anterolateral segments, but without extra-cardiac activity amenable to biopsy. The patient declined myocardial biopsy.

Following symptomatic improvement, serial decline in TnT and cessation of non-sustained ventricular tachycardia, she was discharged on oral anticoagulation, beta blockade, ACE-inhibition and antiplatelet therapy – but without immunosuppression. Serum TnT remained elevated 4 months following discharge (70ng/L) when repeat CMR demonstrated partial resolution of mid-anterolateral LGE to a more clearly epicardial distribution but with persistent segmental T2 elevation (69msec).

In this complex case, CMR detected multi-focal myocardial injury to 5 discrete LV and RV sites, with evidence that these have different mechanisms and ages. The clinical hypothesis is of presentation with a small apical AMI with an additional incidental diagnosis of very likely acute (anterior) and old (inferior and RV freewall) cardiac sarcoidosis.
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