Acute myocarditis often is demonstrated on cardiac MRI (CMR) as increased signal on T2-weighted imaging and late gadolinium enhancement (LGE) in either a midwall or a subepicardial distribution. Previous reports have demonstrated a similar pattern of enhancement on delayed postcontrast CT.1–3 We present 2 cases of edema demonstrated on arterial phase CT coronary angiography (CTCA) in patients with clinical features that suggest acute myocarditis as confirmed by CMR.

CT examinations were performed on a dual-source 64-detector CT scanner (SOMATOM Definition; Siemens Medical Solutions; Forchheim, Germany). Iomeron 350 (Iomoprol 71.4%; Bracco-Eisai; Tokyo, Japan) was injected at 6.5 mL/second into the cubital fossa through an 18-gauge cannula followed by a 50-mL saline flush. Scans were performed in the arterial phase, with triggering from the ascending aorta with a threshold of 100 Hounsfeld units. Both studies were acquired with dose-modulated, retrospective ECG gating. The tube voltage was set at 120 kV (peak) for the first case and 100 kV for the second case.

CMR was performed with a 1.5-T GE Signa Twinspeed system (GE Healthcare; Milwaukee, WI) with an 8-element cardiac phased-array coil. LGE imaging was performed using a segmented inversion recovery fast-gradient echo sequence between 10 and 20 minutes after intravenous administration of 0.2 mmol/kg of gadolinium-diethylene triamine pentaacetic acid.

**Case 1**

A 27-year-old man with no significant medical history presented with chest pain. Troponin I level was elevated at 4.4 \( \mu \text{g/L} \), and the ECG showed a sinus bradycardia. Echocardiography was normal. CTCA showed normal epicardial coronary arteries. There was a small area of subepicardial and midwall low attenuation within the mid anterolateral left ventricular wall (Figure 1A and 1B). CMR demonstrated high signal on both steady-state free precession imaging and fat-suppressed T2-weighted imaging (Figure 1C), with LGE corresponding to the areas of low attenuation on CT (Figure 1D). A diagnosis of myocarditis was made on the basis of the MRI and clinical findings in the presence of normal coronary arteries. Estimated dose from CTCA was 8.3 mSv.

**Case 2**

A 24-year-old male smoker with no significant medical history presented with sudden onset of sharp chest pain radiating to the left arm that was not pleuritic in nature. Troponin I level was elevated at 8.0 \( \mu \text{g/L} \); other laboratory results were normal. There were nonspecific inferolateral ECG changes. CMR demonstrated midwall LGE within the basal septum and adjacent inferior wall (Figure 2B). Corresponding high signal was demonstrated on both steady-state free precession imaging and fat-suppressed T2-weighted imaging (Figure 2A). CTCA was performed to evaluate the coronary arteries, which were normal. Decreased attenuation was demonstrated within the interventricular septum and inferior wall corresponding to the areas of abnormal signal intensity on CMR (Figure 2C and 2D). Estimated dose from CTCA was 2.45 mSv.

A third patient later presented with chest pain and elevated troponin I level (24.0 \( \mu \text{g/L} \)) after a viral illness. CTCA demonstrated areas of subepicardial and midwall hypoattenuation corresponding to the areas of LGE on CMR, and the diagnosis of myocarditis was made.

We subsequently reviewed 233 cases of myocarditis imaged with CMR since 2006. Thirteen also had CTCA performed. Of these, 2 further cases were found with similar findings to the three cases described here.

**Discussion**

Acute myocarditis is a relatively uncommon disease but may well be underdiagnosed. The utility of CMR, particularly with T2-weighted imaging and LGE, is well established. Previous studies and case reports have demonstrated delayed enhancement on ECG-gated cardiac CT in a similar pattern to that seen on MRI.1–3 There has been only 1 previous report in the literature of hypoattenuation on early phase CT, but there...
was no correlation with MRI. Focal edema due to acute myocarditis on CTCA has not been previously reported to our knowledge.

The cause of the focal myocardial hypoattenuation in these cases is presumed to be edema, with increased water-reducing attenuation. CMR signal characteristics of high signal intensity on fat-suppressed T2-weighted imaging are consistent with this interpretation. The other possibility would be a localized reduction in perfusion; however, this is less likely. The inflammation associated with myocarditis would be expected to increase vascularity and, hence, perfusion. The differential diagnosis for the CTCA appearance would include areas of fibrosis from old insult (including myocarditis); metastasis; sarcoma; and infiltrative processes, such as sarcoidosis or Fabry disease.

Visualization of subtle differences in myocardial attenuation was enhanced by using thicker reconstructions (5 to 8 mm), narrow window settings and not using a maximum intensity projection technique. This may explain why the phenomenon has not been identified previously.

**Disclosures**

None.

**References**


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