Right ventricular (RV) involvement after acute myocardial infarction (MI) carries an adverse prognosis, but in the majority of patients RV function recovers fully over time. It is believed that the lower oxygen requirement of the RV and higher collateral flow make the RV more resistant to ischemia. In the absence of infarction, myocardial ischemia leads to intracellular and subsequently extracellular edema, which may contribute to reversible RV dysfunction after acute MI.

Cardiovascular magnetic resonance (CMR) is the optimal tool to image the RV because it is not constrained by imaging windows or insufficient spatial resolution. CMR can assess RV function, volume, and infarction. Furthermore, tissue edema can be depicted in vivo using T2-weighted CMR methods, but there are no reports to date describing RV edema by T2-weighted CMR.

A 56-year-old man presented to our institute with an acute inferoposterior MI and underwent primary percutaneous coronary intervention. Coronary angiography demonstrated a dominant right coronary artery that was occluded proximally with TIMI (Thrombolysis in Myocardial Infarction) grade 0 flow. The vessel was reopened by direct stenting using a single bare metal stent. Although the ST changes improved, only TIMI grade II flow was achieved.

A CMR study was performed on day 3 after MI using a 1.5-T scanner (Philips Medical Systems, Best, The Netherlands). T2-weighted imaging with a short $\tau$ inversion recovery (STIR) spin-echo sequence demonstrated myocardial edema in the inferior and posterior walls of the left ventricle (LV) and the RV (Figure 1). Balanced steady-state free precession (SSFP) cine images demonstrated LV and RV hypokinesia corresponding with the edematous regions (Movie 1). Late gadolinium-enhanced imaging

**Figure 1.** T2-weighted imaging demonstrates hyperintense signal (arrows) in the myocardium of the inferior and posterior walls of the left ventricle (LV) and right ventricle (RV). This probably represents myocardial edema.
using an inversion-recovery gradient echo sequence revealed transmural inferior LV wall infarction but no evidence of RV infarction (Figure 2). A repeat CMR study was performed at 3 months. This demonstrated almost complete resolution of the edema in the LV and RV (Figure 3). Although LV systolic function improved, contractility in the inferior wall remained slightly reduced with evidence of infarction on late gadolinium-enhanced images. Right ventricular systolic function had recovered fully (Movie II).

This case illustrates the differences in response to acute myocardial ischemia between the left and right ventricles. In this case, a proximal right coronary artery occlusion resulted in transmural LV infarction but no RV infarction. However, in the acute phase, CMR demonstrated RV edema and associated contractile abnormalities, both of which had normalized by 3 months. Edema must be considered as the most likely mechanism for the transient RV dysfunction in the absence of infarction. This mechanism has previously been shown to be the cause of acute stunning in the LV. As yet, it is not known whether the presence of RV edema carries any additional prognostic significance or whether there are any pharmacological means to reduce the extent of edema.

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None.

**References**

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