Cardiac Imaging

Epistenocardiac Pericarditis

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A 72-year old male was admitted 8 hours after the onset of progressively worsening central chest pain at rest. Three years before, he had undergone coronary bypass surgery (left internal thoracic artery to left anterior descending artery and two venous grafts to the first diagonal and the first obtuse marginal branch) and aortic valve replacement with a bioprosthetic valve. Clinical examination on admission was unremarkable and the ECG showed inferior-lateral-posterior ST-elevation myocardial infarction (Figure 1A). The patient was immediately referred for primary angioplasty and a drug-eluting stent was successfully implanted at a stenotic (95%) thrombotic lesion in the right coronary artery. However, two days later the patient developed positional, respiro-phasic chest pain that radiated to the left shoulder. The pain was accompanied by a gradual increase in C-reactive protein values (136 mg/dL, normal limit<5 mg/dL), while fever was not detected. No further elevation of cardiac enzyme levels was noted. Cardiac auscultation revealed a to-and-fro sound in systole and in mid-diastole, suggestive of pericardial rub. Echocardiography showed normal pericardium, mild left ventricular (LV) global systolic dysfunction (ejection fraction, EF 50%) with akinesis of the lateral and hypokinesis of the inferior wall. Evaluation of contiguous ECGs showed atypical T-wave evolution with persistently (>72 hours) positive T waves and residual ST elevation in the inferolateral leads (Figure 1B). Given the clinical and laboratory evaluation the patient was diagnosed as having post-infarct pericarditis. We sought to unveil the presence of pericardial inflammation and evaluate the extension of the infarcted area by performing an early (8 days) post-infarct cardiovascular magnetic resonance (CMR) examination.

Functional analysis by steady-state free-precession cine sequences showed a moderately dilated LV with severe hypokinesia-akinesia of the inferolateral and inferior wall segments and mildly impaired global systolic function (LVEF 50%). Pericardial effusion was not present. Edema imaging by T2-weighted short-τ inversion recovery imaging showed a transmural area of edema in the inferior-inferolateral wall (Figure 2A). Late post-gadolinium administration imaging showed transmural enhancement in the aforementioned LV segments. Subsequent strong pericardial enhancement and increased pericardial thickness (4 mm) was evident mostly along the lateral LV border and established the clinical diagnosis of early infarct-associated (epistenocardiac) pericarditis (Figures 2B, C).

Although in our case the clinical presentation raised a high suspicion of early post-infarction pericarditis, the diagnosis of this condition still remains challenging

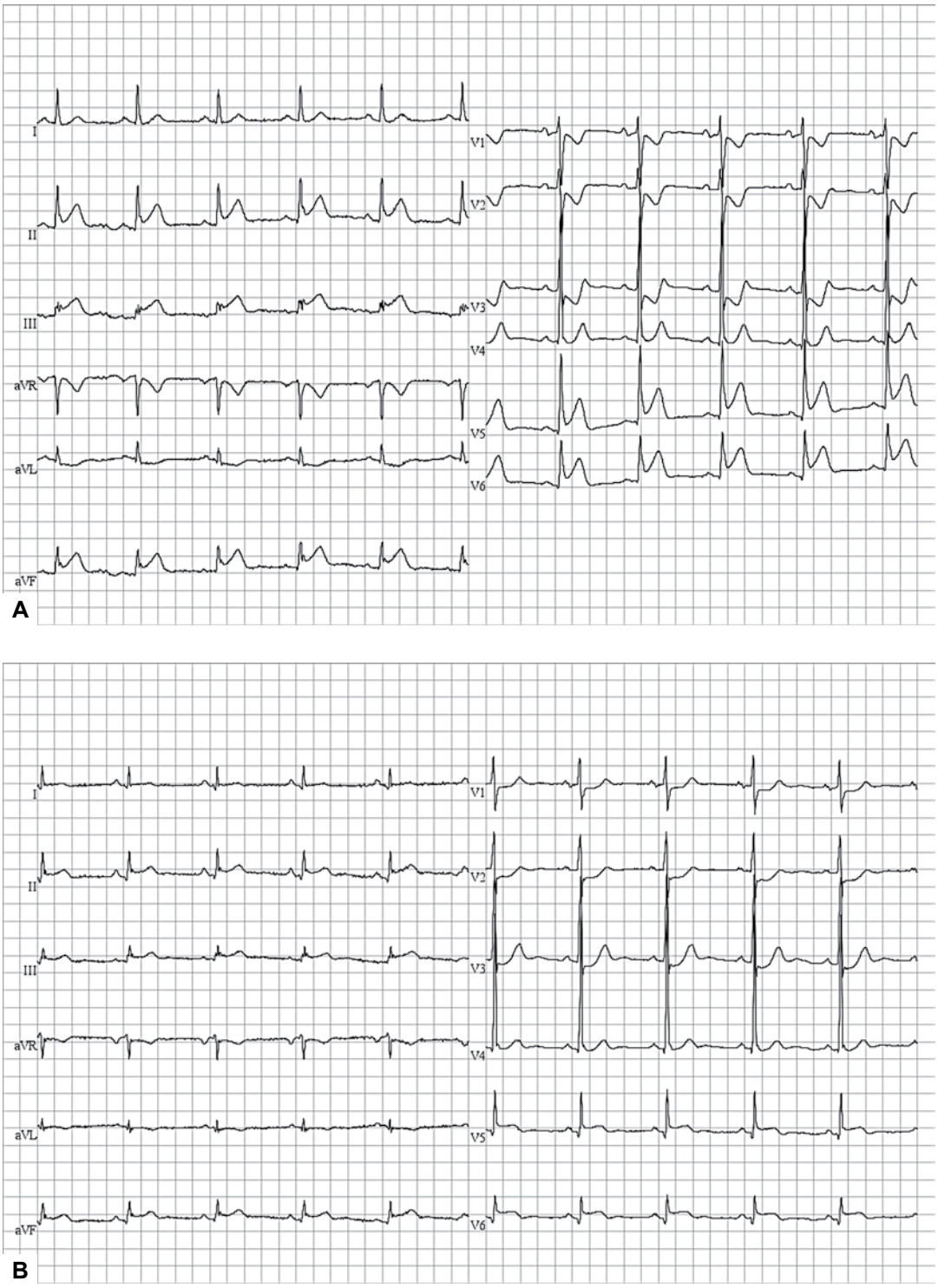

Figure 1. Electrocardiographic findings. Electrocardiograms (ECGs) recorded on admission (A) and 3 days later (B). The admission ECG (A) shows ST-segment elevation in leads II, III, aVF, V5 and V6, and ST-segment depression in leads V1-V3. The ECG on admission, along with the presence of positive T waves and an R/S ratio >1 in the same leads in the second ECG, are suggestive of inferior-lateral-posterior ST-elevation myocardial infarction. The second ECG (B) after the onset of positional-pleuritic chest pain shows residual ST-segment elevation and unchanged persistently positive T waves. Although such positive T-wave deflections render the ECG ostensibly more "normal" in appearance, they are indicative of post-infarct pericarditis.

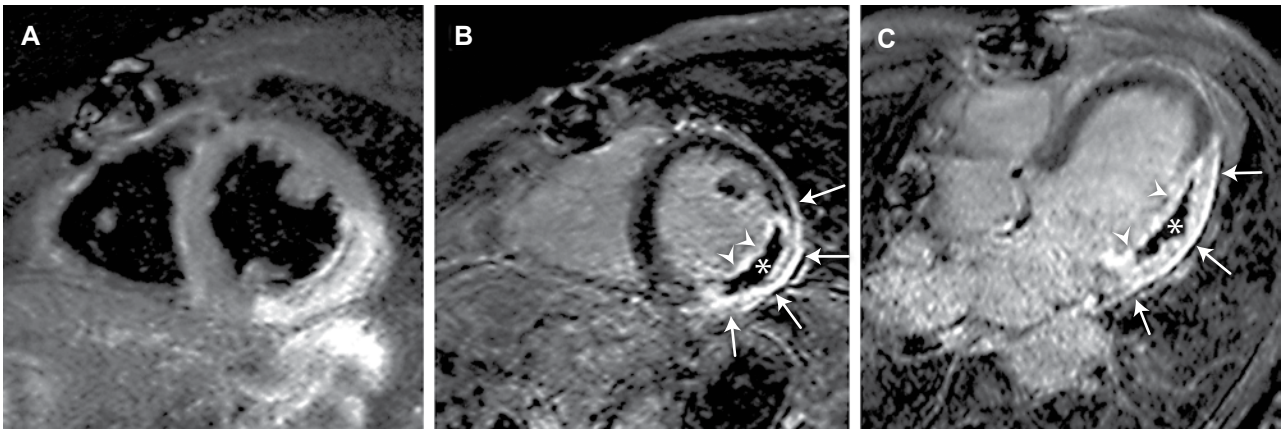

Figure 2. Cardiac magnetic resonance (CMR) findings. Comprehensive CMR performed 8 days after the acute event, including T2-weighted short-τ inversion recovery (T2W-STIR) imaging (A), with late gadolinium imaging in the short-axis (B) and horizontal long-axis (C) planes. The extend of edema can be well appreciated on T2W imaging of the inferior and inferolateral wall (A). Transmural enhancement on late gadolinium imaging at the inferolateral wall (arrowheads, B and C) is present, corresponding to the area of edema. The hypointense area within the infarcted myocardium is suggestive of an extensive no-reflow zone (asterisk, B and C). Strong pericardial enhancement along the inferolateral wall (arrows, B and C) confirms the presence of regional pericardial inflammation.

with current diagnostic tools: pericardial rub is not always present or has a fleeting nature, positional chest pain might be mistaken for recurrent ischemia, and ECG patterns suggestive of post-infarct pericarditis are not always present. There is growing evidence that CMR may be of use for depicting pericardial inflammation *in vivo*.[1,2] According to a recent CMR study, post-infarct pericardial damage can be accurately detected between days 2 and 5 after the infarction. In the same study, a follow up CMR exam after 4 months showed that pericardial abnormalities had disappeared in 80% of patients.[3] The current case suggests a role for CMR in depicting early pericardial damage. Particularly in cases where the clinical presentation is unclear, CMR may be of use for depicting concomitant pericardial injury.

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