Malignant ST segment elevation

Alfonso Ielasi\textsuperscript{1}, Azeem Latib\textsuperscript{1,2}, Andrzej Okreglicki\textsuperscript{2}, and Antonio Colombo\textsuperscript{1,3*}

\textsuperscript{1}Interventional Cardiology Unit, San Raffaele Scientific Institute, Milan, Italy; \textsuperscript{2}Division of Cardiology, Department of Medicine, University of Cape Town, South Africa; and \textsuperscript{3}Interventional Cardiology Unit, EMO Centro Cuore Columbus, 48 Via M. Buonarroti, 20145 Milan, Italy

* Corresponding author. Tel: +39 02 481 2920, Fax: +39 02 481 93 433, Email info@emocolumbus.it

A 62-year-old male with an unremarkable medical history presented to an emergency department with heart failure, paroxysmal atrial fibrillation, and ST segment elevation in leads V\textsubscript{2}–V\textsubscript{6}, I, aVL, in the absence of Q waves. The patient did not complain of chest pain at the moment electrocardiographic alterations were identified, and serial cardiac enzymes were within normal limits. Echocardiogram showed a mass in the right atrium and a small pericardial effusion. The heart failure was treated and the patient was transferred 1 week later to our institution for evaluation.

On admission, the ECG showed persistence of the ST segment changes as did serial ECGs during hospitalization. On the basis of the pronounced and persistent ST elevation we were concerned about ventricular myocardial involvement. This was confirmed on CT thorax and cardiac MRI which demonstrated the presence of a mass in the posterior right ventricular wall infiltrating the right atrial wall and the pericardium which also had a small effusion (Panel B, arrow). There were also secondary masses noted in the right ventricular anterior wall and left ventricular apex (Panel C, arrows). Histopathology demonstrated an undifferentiated pericardial mesothelioma.

Tumour invasion of the myocardium should be considered high in the differential diagnosis in patients presenting with persistent and marked ST elevation. Although previously described with cardiac metastases, this is not so for primary cardiac tumours. ST elevation due to a cardiac tumour may occur secondary to tumour invasion of the myocardium, tumour emboli to the coronary artery, or compression of a coronary artery.

Panel A. ECG showing persistent ST elevation in leads V\textsubscript{2}–V\textsubscript{6} up to 5 mm in V\textsubscript{2}–V\textsubscript{3}; and 1.5 mm in I and in aVL, in the absence of Q waves.

Panel B. CT thorax demonstrating large mass infiltrating right atrium (arrow), right ventricle, and pericardium (arrowhead).

Panel C. Cardiac MRI demonstrating large secondary in left ventricular apex (arrow).

Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2008. For permissions please email: journals.permissions@oxfordjournals.org