Persistent ST-segment elevation due to cardiac metastasis

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DESCRIPTION

A man aged 68 years with a known history of squamous cell carcinoma of the lung presented to the emergency room complaining of an acute episode of dyspnoea. His symptoms had resolved on presentation to the hospital. An EKG was obtained, which showed marked ST-segment elevation (STE) in the anteroseptal leads (figure 1A). No prior EKG was available for comparison. The patient denied any chest pain or pressure, and had no evidence of myocardial necrosis by cardiac biomarkers. Nevertheless, he was taken emergently to the cardiac catheterisation laboratory for a presumed diagnosis of STE myocardial infarction (STEMI). He was found to have severe two-vessel coronary artery disease with possible plaque rupture in the mid-left anterior descending artery (LAD) (figure 2). Overlapping bare metal stents were deployed to the LAD. Concerningly, the patient’s right ventricle was noted to be in a fixed motion suggestive of tumour infiltration.

A transthoracic echocardiogram was subsequently performed, which demonstrated a large right ventricular mass with involvement of the interventricular septum and pericardium (figure 3). A CT of his thorax showed an anterior mediastinal mass invading the pericardial space, right ventricular free wall and the right ventricular outflow tract (figure 4). Despite the percutaneous coronary intervention, the STE persisted while the serial cardiac biomarkers remained negative (figure 1B). Additionally, the patient also continued to deny any cardiac symptoms. It was later believed that the STE was likely due to tumour infiltration into the right ventricle, and unlikely to be the classic STEMI due to acute coronary syndrome with resultant occlusive thrombus.

The differential diagnosis of STE is broad and extends beyond acute myocardial infarction (AMI). Once AMI is ruled out, other relatively more common considerations include acute pericarditis, Prinzmetal’s angina, hyperkalaemia, left ventricular...
hypertrophy, left bundle-branch block, Brugada syndrome and
pulmonary embolism.1 2 Although relatively rare, STE due to
metastatic tumour involving the myocardium had been described
in published literature dating five decades back. Although the
mechanism of this phenomenon remains unclear, one hypothesis
centers around the idea of an injurious current resulting from the
inflammation surrounding the tumour, ionic transfer of potassium
from necrotic tissue with resultant electropotential difference and
neoplastic stretch of adjacent muscle fibres.2 3 In this article, we
presented a case of a patient who was initially presumed to have
a STEMI based on EKG findings, but was later found to have
myocardium compression and infiltration from lung metastasis.
He did not have evidence of direct tumour invasion to the LAD
on his coronary angiogram. His persistent STE and negative serial
biomarkers despite coronary stent placement (as well as absence
of angina symptoms) supports this change in diagnosis. Our
patient had previously tried palliative carboplatin and paclitaxel
with progression of disease. He had been subsequently enrolled
in the Lung Cancer Master Protocol (Lung-MAP) trial and had
been treated with nivolumab and ipilimumab prior to this admis-
sion. Unfortunately, he had continued to demonstrate tumour
growth and had been ultimately switched to gemicitabine for a
brief moment without improvement. Given his overall disease
progression and poor prognosis, the patient declined additional
chemotherapy during this admission and wished to pursue hospice
care. He passed away 2 weeks later.

Competing interests None declared.

Patient consent Consent obtained from next of kin.

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