Noninfiltrating Adenocarcinoma of the Lung Causing ST-Segment Elevation

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ST-segment-elevation myocardial infarction is a leading cause of cardiovascular morbidity and death. We describe the case of a 51-year-old woman with advanced adenocarcinoma of the lung who presented with ST-segment elevation in the presence of an extracardiac lung mass but no objective evidence of myocardial ischemia or pericardial involvement. After the patient died of hypoxic respiratory failure, autopsy findings confirmed normal-appearing pericardium and myocardium, and mild-to-moderate atherosclerosis in the coronary arteries. A 4.5 × 4-cm extracardiac left hilar lung mass was confirmed to be poorly differentiated adenocarcinoma of the lung adjacent to the myocardium. The persistent current of injury that had been detected electrocardiographically was thought to occur from direct myocardial compression. ST-segment elevations secondary to direct mass contact on the myocardium should be considered in patients who have a malignancy and ST-segment elevation. (Tex Heart Inst J 2015;42(4):381-4)

Key words: Arrhythmias, cardiac/diagnosis; diagnosis, differential; electrocardiography; lung neoplasms/complications/pathology; myocardial infarction/classification

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A leading cause of cardiovascular morbidity and death is ST-segment-elevation myocardial infarction (STEMI).¹ Myocardial ischemia occurs when diminished blood supply to the heart cannot meet the demand of the myocardium. Infarction can occur when this supply-and-demand mismatch leads to cell necrosis. Usually, STEMI is caused by an occlusive thrombus and is typically recognized on electrocardiography (ECG) from the presence of ST-segment elevation (STE). Clinically, the diagnosis of acute myocardial infarction is reached after the detection of a rise or fall of cardiac biomarker values with at least one of the following: symptoms of ischemia; new or presumed new STE at the J point in at least 2 contiguous leads of ≥ 2 mm (0.2 mV) in men or ≥ 1.5 mm (0.15 mV) in women in leads V_2 and V_3 , and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb lead; the development of pathologic Q waves; imaging evidence of new loss of viable myocardium or new regional wall-motion abnormality; or the identification of an intracoronary thrombus on angiograms or at autopsy.^{2,3} ST-segment elevation can also be seen in other clinical contexts, including normal variants, left ventricular hypertrophy, left bundle branch block, pulmonary embolism, and pericarditis.⁴ We describe the case of a woman with advanced adenocarcinoma of the lung who presented with STE in the presence of a lung mass close to the heart but with no objective evidence of myocardial ischemia.

Case Report

In December 2011, a 51-year-old woman presented at the emergency department with a 3-day history of worsening nausea, weakness, and upper abdominal pain. She reported no chest discomfort or dyspnea. Her medical history included hypertension and former tobacco use. She had recently been diagnosed with poorly differentiated adenocarcinoma of the lung with metastases to the brain, peritoneum, and gluteal and paraspinal muscles. The malignancy had been treated with left-upper-lobe wedge resection, gamma-knife radiation to the brain, and systemic chemotherapy with use of carboplatin and pemetrexed. On presentation, the patient's temperature was 37.2 °C; heart rate, 114 beats/min; blood pressure, 93/74 mmHg; respiratory rate, 15 breaths/min; and pulse oximetry, 99% on 2 L of oxygen. Results of physical examination included a jugular venous pressure of 7 cm H₂O, normal heart sounds, and no murmurs, rubs, or gallops. Auscultation revealed mild bilateral end-expiratory wheezes and

crackles at the bases. Laboratory studies were notable for a white blood cell count of $22.5 \times 10^9/L$, and levels of sodium, 129 mmol/L; creatinine, 1.51 mg/dL; and cardiac troponin I, <0.02 ng/mL. An ECG showed STE in leads II, III, aVF, V_5 , and V_6 , and no PR-segment changes (Fig. 1). A chest radiograph showed an extracardiac left hilar lung mass with bilateral airspace opacities (Fig. 2), and echocardiograms with Definity® contrast medium (Lantheus Medical Imaging, Inc.; N. Billerica, Mass) revealed a normal-sized heart, a left ventricular ejection fraction of 0.60 to 0.65, and no wall-motion abnormalities.

Inferolateral STEMI was considered as a diagnosis; however, in view of the brain metastases and relative contraindications to antithrombotic medications, coronary angiography was deferred. The patient's subsequent clinical course was significant for 3 serial troponin I measurements of <0.02 ng/mL and no evolutionary ECG changes. She was treated for pneumonia and a partial small-bowel obstruction that was seen on abdominal computed tomography (CT). Prior CT and positron emission tomographic-CT (PET-CT) scans of the chest showed an enlarging mass in the medial and posterior aspect of the left upper lobe, abutting the myocardium (Figs. 3 and 4). Given the patient's rapidly progressing malignancy, her family decided not to pursue further aggressive measures, and she died of hypoxic respiratory failure after a 2-day hospitalization. Autopsy findings included normal pericardium, normal myocardium, and mild-to-moderate atherosclerotic lesions

in the coronary arteries. A 4.5×4 -cm extracardiac left hilar lung mass was confirmed to be poorly differentiated adenocarcinoma of the lung. This mass was not noted to infiltrate the pericardium or myocardium.

Discussion

Acute myocardial infarction is not the only cause of STE: other conditions can mimic an infarction pattern on ECG. The authors of several case studies have reported persistent STE in the presence of malignancies with cardiac metastases.⁵⁻⁹ One case report describes a tumor that caused extrinsic compression of an epicardial vessel, which led to myocardial damage.¹⁰ Here, we

Fig. 1 Patient's electrocardiograms **A**) en route to the hospital and **B**) upon arrival at the emergency department show ST-segment elevations in leads II, III, aVF, V_5 , and V_6 .

Fig. 2 Chest radiograph shows an extracardiac left hilar lung mass with bilateral airspace opacities.

Fig. 3 Chest computed tomogram with contrast shows a 2.2×1.9 -cm extracardiac left hilar lung mass, 5 months before presentation.

Fig. 4 Positron-emission tomogram shows a 2.9×2.5 -cm mass in the left hilar lung with increased fluorodeoxyglucose uptake, 2 months before presentation. Also seen is disease metastatic to the abdomen with multiple peritoneal, liver, left gluteal muscle, and right paraspinal muscle implants.

report an apparently unique case of malignancy that caused STE—by direct apposition of a lung mass on the myocardium without any evidence of cardiac metastases, coronary artery invasion or compression, or pericardial involvement. The persistent current of injury detected on ECG is thought to have occurred from direct myocardial compression. During the 2-month period between the PET scan and the above clinical presentation, the lung mass had grown from 2.9×2.5 cm to 4.5×4 cm—large enough to impinge on the epicardial surface.

ST-segment elevation has been described as a normal variant in healthy populations. In one study, over 91% of 6,014 males between the ages of 16 and 58 years had STEs of 1 to 3 mm in one or more precordial leads. Females might also have a normal variation of STE; however, the elevation is typically less than 1 mm.¹¹ ST-segment changes in left ventricular hypertrophy and left bundle branch block are typically seen discordant to the QRS complex. In acute pericarditis, diffuse STE and PR-segment depression without reciprocal ST depression are seen in all leads except aVR and V₁; however,

focal ECG changes have been seen in localized pericarditis.¹² Severe hyperkalemia has classic ECG findings: a characteristic pseudo-right bundle branch block and persistent coved STE in the precordial leads, peaked T waves, widened QRS complexes, and low-amplitude P waves.¹³ Similarly, STE can be seen in the presence of a pulmonary embolus¹⁴ and transiently after transthoracic cardioversion.¹⁵

Evolutionary changes occur in each pathologic condition. In myocardial infarction, initially hyperacute T waves are present, followed by J-point elevation and then STE. The ST segment will eventually become normal and Q waves will form, as will loss of R-wave amplitude and T-wave inversion.² In pericarditis, 4 stages are seen on ECG: STE, descent of the J points, T-wave inversions, and return to baseline.¹² In direct myocardial invasion, the STE is noted to be persistent without the evolutionary changes associated with infarction, as in our patient.¹⁶

Our report describes an occurrence of STE on ECG outside the above clinical situations. Efficiently and effectively scrutinizing ECG findings of STE in patients on the basis of their clinical presentations is paramount in providing timely therapy. Withholding appropriate reperfusion therapy in a patient with STEMI can be devastating; however, using antithrombotic drugs to treat a patient who has pericarditis or metastatic cancer can be injurious. ST-segment elevations secondary to direct contact on the pericardium and myocardium should also be considered in patients who have a malignancy and STE on ECG.

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