Pseudo-Infarction Pattern Secondary to Lung Cancer Tumor Mass

Rajdeep S. Gaitonde, DO
Naveen Sharma, MD
Irmina Gradus-Pizlo, MD

The 3 most common causes of ST-segment elevation as seen on electrocardiogram (ECG) are acute myocardial infarction (MI), pericarditis, and Prinzmetal's angina.[1] Other causes for ST-segment elevation include early repolarization phenomenon and ventricular aneurysm. ST-segment elevation also is occasionally observed in acute cor pulmonale, hyperkalemia, cerebrovascular accidents, left ventricular hypertrophy, left-bundle branch block, hypertrophic cardiomyopathy, hypothermia, and invasion of the heart by neoplastic tissue.[1,2] A few case reports of ST-segment elevation secondary to metastatic carcinoma invasion have been published.[3–9] We present a case in which neoplastic tissue invasion of the heart led to ST-segment elevations. This case emphasizes the importance of a wide differential diagnosis when encountering chest pain and focal ST-segment elevation in patients with cancer.

CASE PRESENTATION

Initial Presentation

A 72-year-old white man presented to the emergency department with a chief complaint of continuous, nonpositional, nonradiating, pressure-like, left-sided chest pain that worsened with exertion and was associated with shortness of breath and diaphoresis. He stated that he had been having similar intermittent chest pain episodes for the past few months prior to presentation. Over the past few days, the chest pain had increased in intensity and had recently become continuous in nature. He denied dizziness, fevers, or palpitations, although he did complain of the recent onset of worsening fatigue.

The patient had a history of atrial fibrillation, an MI 12 years prior that was treated medically, gastroesophageal reflux, and poorly differentiated squamous cell lung cancer. As part of his lung cancer treatment, he underwent left pneumonectomy 8 months prior to admission. His admission medications were remarkable for amiodarone and rabeprazole.

Examination revealed an elderly man in mild-to-moderate distress. He was afebrile with a stable blood pressure of 142/92 mm Hg and a heart rate of 70 bpm. Oxygen saturation levels were within normal limits. No elevated jugular venous distension, carotid bruits, or body lymphadenopathy were noted on examination. Cardiac auscultation revealed a regular rhythm with no rub, murmur, or gallop and with a nondisplaced point of maximal impulse. No air movement was noted in the left lung. The rest of the physical examination was unremarkable. An ECG obtained on presentation revealed normal sinus rhythm with a left anterior fascicular block as well as evidence of an inferior MI of undetermined age. Strikingly present were ST-segment elevations in leads I and aVL with reciprocal ST-segment depression in leads II, III, and aVF (**Figure 1**). Laboratory studies revealed normal electrolyte levels and blood counts. His initial cardiac enzyme levels were creatine kinase (CK), 35 U/L (normal, 0–232 U/L); CK-MB, 3.5 ng/μL (normal, 0–5.9 ng/mL); and troponin I, 0.8 ng/mL (normal, 0–0.5 ng/mL).

Hospital Course

Based on clinical presentation, ECG, and troponin elevation, the patient was diagnosed with an acute high lateral MI. The patient underwent emergent coronary angiography, which revealed the following findings:

- Serial proximal 80% and 70% distal left anterior descending (LAD) lesions
- Luminal irregularities in the circumflex coronary artery
- A completely occluded distal right coronary artery

Dr. Gaitonde is a cardiology fellow, Dr. Sharma is a recently graduated cardiology fellow now in private practice, and Dr. Gradus-Pizlo is an assistant professor of medicine, Krannert Institute of Cardiology, Indiana University School of Medicine, Indianapolis, IN.

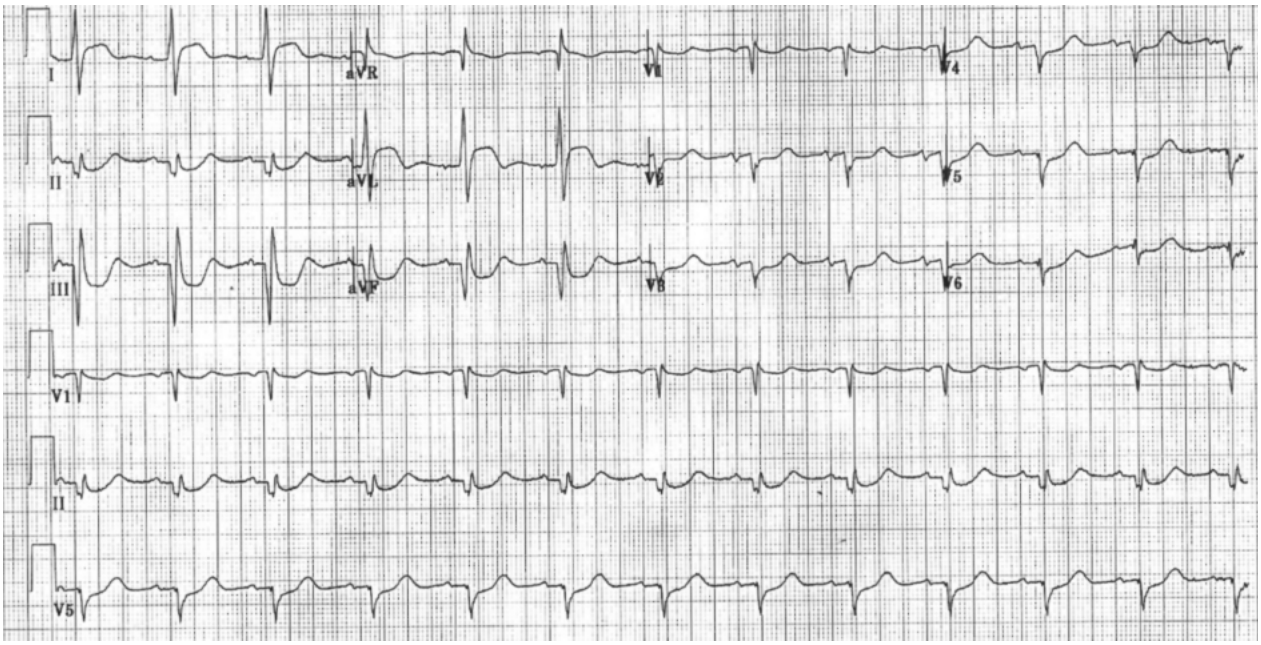

Figure 1. Electrocardiogram of the case patient obtained at the initial presentation.

- A left ventricular ejection fraction of 45% to 50%, with mild anterolateral and apical hypokinesis
- Left-to-right collateral flow

A 3.0 mm × 9 mm stent was placed in the proximal LAD. The patient tolerated the procedure well and left the catheterization laboratory free of chest pain. However, ECGs obtained during and after the catheterization revealed continued ST-segment elevations in leads I and aVL (**Figure 2**). Follow-up evaluation of cardiac markers showed CK levels of 22 and 21 U/L, CK-MB levels of 3.4 and 3.8 ng/uL, and troponin levels of 0.9 and 0.4 ng/mL, respectively. A few hours after the cardiac percutaneous intervention, despite optimal medical therapy, the patient again began to experience chest pain with associated diaphoresis. His appearance was gray and ashen on examination. ECG revealed no change, showing the same degree of ST-segment elevations in the high lateral leads and ST-segment depressions in the inferior leads. Although sublingual nitroglycerin initially alleviated his chest pain, subsequent episodes were responsive only to morphine. Vital signs, ECG, and cardiac enzyme levels remained unchanged throughout the chest pain episodes. The patient returned to the cardiac catheterization laboratory, and the LAD coronary artery was confirmed to be patent.

In an attempt to determine a cause for the chest pain, computed tomography was performed (**Figure 3**), revealing a mass eroding into the posterior portion of the left sixth rib and invading the myocardium. The heart was deviated into the left pleural cavity occupying the empty space vacated by the removal of the left lung (post-pneumonectomy). After being placed on an appropriate pain medication regimen, the patient was discharged chest-pain free with oncology follow-up.

We hypothesized that the changes suggesting an acute current of injury evident on this patient's ECG were in fact due to myocardial invasion by the lung mass.

DISCUSSION

Myocardial tissue metastasis from neoplastic disease often remains clinically inapparent and thus is very difficult to diagnose. Of 151 consecutive autopsies of lung cancer patients, 67 demonstrated cardiac metastases (44.4%), but myocardial tissue metastases were found in only 8 patients (11.9%).[10]

Supraventricular arrhythmias, such as atrial fibrillation, are the rhythm disturbances usually found in patients with metastasis to the heart. Ventricular tachycardia is rare.[11] Heart metastases manifest themselves on ECG as (1) diffuse T-wave inversion (10%), (2) focal T-wave inversion specific for a coronary distribution (80%), and (3) ST-segment elevation (10%).[1,12,13] Lestuzzi et al[14] reported the sensitivity and specificity of electrocardiographic ST-T changes as markers of neoplastic myocardial invasion by comparing the echocardiographic results to computed tomography, nuclear

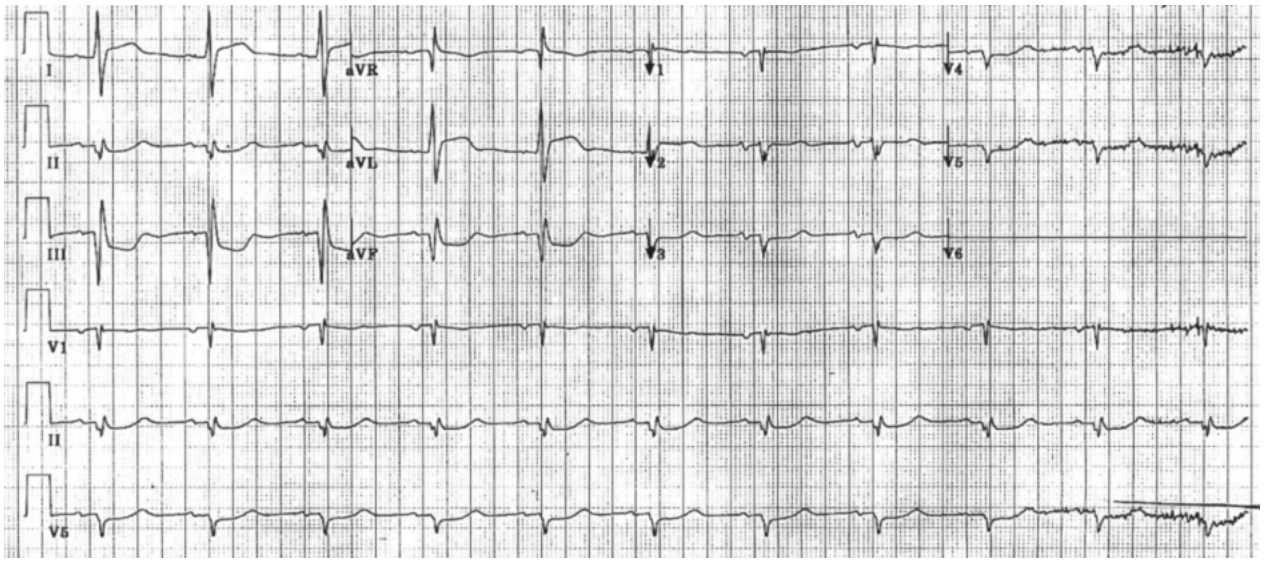

Figure 2. Post-catheterization electrocardiogram of the case patient.

magnetic resonance, surgery, or autopsy data in 49 patients. They found that significant ST-T changes were present in 77.7% of the patients with myocardial infiltration at echocardiography. The false negatives in their study (ie, normal ECG, nonspecific changes) were mainly related to infiltration limited to the right side of the heart. The false positives (ie, ST-T changes without echocardiographic signs of infiltration) were observed in older patients as well as those with pericardial effusion or other heart diseases. They concluded that ST-segment elevation was a more specific sign of myocardial infiltration as compared to negative T waves (86% versus 47%, respectively).

In asymptomatic, clinically stable lung cancer patients, ST-segment elevation with a QS pattern has been reported to be highly suggestive of myocardial injury due to myocardial metastasis.[15] Others have suggested that in lung cancer sufferers without Q waves on ECG, persistent ST-segment elevation is pathognomonic for tumor invasion of the heart.[7,16] It has even been suggested that in patients with lung cancer, an ECG representative of acute MI rarely can be induced by myocardial involvement itself.[17]

Little has been written regarding the possible etiology of ST-segment elevations in the presence of neoplastic tissue invasion. The prevailing thought is that ST-segment elevation results from myocardial irritation, as occurs in pericarditis.[1] Myocardial contusions frequently result in electrocardiographic changes similar to those of MI. In effect, any irritation to cardiac myocytes can result in electrocardiographic changes. Unlike what is typically seen in pericarditis, however, this patient did not demonstrate PR depression or concave ST elevation. Furthermore, his chest pain symptoms were not typical for pericarditis. Indeed, neoplastic myocardial invasion rarely results in an ECG pattern typical for pericarditis. It is highly likely that another process, whether simple pressure irritation from the tumor or cell membrane changes from tumor-myocyte interaction, is taking place.

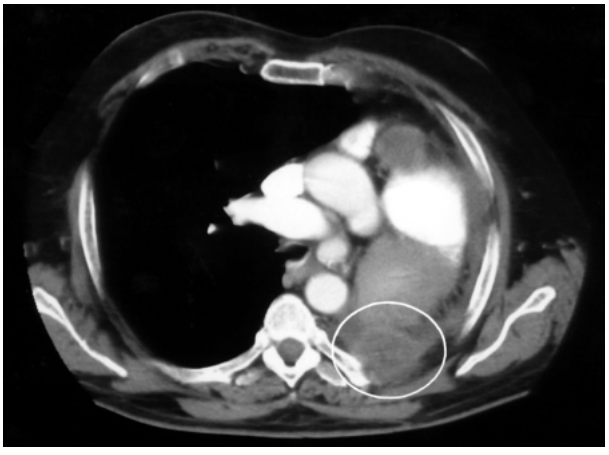

Figure 3. Thoracic computed tomographic scan of the case patient. Lung tumor mass is seen eroding into the rib and infiltrating into the cardiac myocardium (*circle*).

CONCLUSION

Electrocardiographic ST-segment elevation, although frequently caused by obstructed coronary flow, has many other etiologies. In most cases, a cause can

be identified based on an adequate history, physical examination, and subtleties in the ECG. Unfortunately, this is not always the case and as a result, millions of health care dollars are spent to "rule-out" coronary artery disease.

Myocardial tissue metastasis masquerading with a MI-like pattern on ECG occurs rarely. Regardless of a patient's cancer status, acute MI should remain on the top of the differential diagnosis in all patients presenting with ST-segment elevation, especially given the hypercoagulable state of patients with cancer. However, in cancer patients presenting with evidence of an acute current of injury by ECG, it is necessary to consider that cardiac metastases may be responsible for the ECG changes. Furthermore, the appearance of ST-T changes or of conduction disturbances in patients with neoplasms should suggest the need for further work-up with 2-dimensional echocardiography in order to better define the diagnosis.[14] These patients may benefit from early cardiac catheterization rather than thrombolytic therapy.[13,18] **HP**

REFERENCES

1. Mirvis DM, Goldberger. Electrocardiography. In: Braunwald E, Zipes DP, Libby P, editors. Heart disease: a textbook of cardiovascular medicine. 6th ed. Philadelphia: WB Saunders; 2001:82–128.
2. Murphy JG, editor. Mayo Clinic cardiology review. 2nd ed. Philadelphia: Lippincott, Williams & Wilkins; 2000: 574–8.
3. Houghton JL, Sinden JR, Gross CM. Case report: acute presentation of pseudo myocardial infarction secondary to metastatic cancer. Am J Med Sci 1992;303:170–3.
4. Guy JM, Lamaud M, Guichenez P, et al. [Elevation of the ST segment caused by cardiac metastasis {letter}.] [Article in French.] Presse Med 1989;18:2067.
5. Vallot F, Berghmans T, Delhaye F, et al. Electrocardiographic manifestations of heart metastasis from a primary lung cancer. Support Care Cancer 2001;9:275–7.
6. Nishikawa Y, Akaishi M, Handa S, et al. A case of malignant lymphoma simulating acute myocardial infarction. Cardiology 1991;78:357–62.
7. Hartman RB, Clark PI, Schulman P. Pronounced and prolonged ST segment elevation: a pathognomonic sign of tumor invasion of the heart. Arch Intern Med 1982; 142:1917–9.
8. Zatuchni J, Burris A, Vejviboonsom P, Voci G. Metastatic epidermoid cardiac tumor manifested by persistent ST segment elevation. Am Heart J 1981;101:674–5.
9. Watanabe Y, Tanaka H, Kanayama H, Katou, K. [A case of lung cancer with myocardial metastasis with ECG suggestive of myocardial infarction.] [Article in Japanese.] Nihon Kyobu Shikkan Gakkai Zasshi 1993;31:619–23.
10. Abe S, Sukoh N, Ogura S, et al. Nucleolar organiser regions as a marker of growth rate in squamous cell carcinoma of the lung. Thorax 1992;47:778–80.
11. Tamura A, Matsubara O, Yoshimura N, et al. Cardiac metastasis of lung cancer. A study of metastatic pathways and clinical manifestations. Cancer 1992;70:437–42.
12. Berge T, Sievers J. Myocardial metastases. A pathological and electrocardiographic study. Br Heart J 1968;30: 383–90.
13. Cates CU, Virmani R, Vaughn WK, Robertson RM. Electrocardiographic markers of cardiac metastasis. Am Heart J 1986;112:1297–303.
14. Lestuzzi C, Nicolosi GL, Biasi S, et al. Sensitivity and specificity of electrocardiographic ST-T changes as markers of neoplastic myocardial infiltration. Echocardiographic correlation. Chest 1989;95:980–5.
15. Abe S, Watanabe N, Ogura S, et al. Myocardial metastasis from primary lung cancer: myocardial infarction-like ECG changes and pathologic findings. Jpn J Med 1991; 30:213–8.
16. Astorri E, Fiorina P, Pattoneri P, Paganelli C. Persistent ST segment elevation in a patient with metastatic involvement of the heart. Minerva Cardioangiol 2001;49:81–5.
17. Yao NS, Hsu YM, Liu JM, et al. Lung cancer mimicking acute myocardial infarction on electrocardiogram. Am J Emerg Med 1999;17:86–8.
18. Konishi S, Kojima T, Ichiyanagi K, et al. A case of double cancers with myocardial metastasis mimicking acute myocardial infarction both on an electrocardiogram and on Tc–99m-MIBI myocardial SPECT. Ann Nucl Med 2001;15:381–5.