There are three criteria for the diagnosis of acute myocardial infarction: ECG change (ST-segment elevation or Q wave present), elevation of serum cardiac enzymes (creatine phosphokinase, creatine kinase-myocardial band, and troponin I), and clinical symptoms such as chest pain, referred pain, cold sweating, and shock. There are other less frequent reasons for such symptoms and signs, including cardiac metastasis of lung cancer. We present the case of a patient with lung cancer and myocardial metastasis. Clinical symptoms and ECG change were compatible with acute myocardial infarction, but the laboratory data were not. Chest CT and Echo were key in the diagnosis. Myocardial metastasis of lung cancer is often elusive. It may be revealed in 30% of patients with fatal lung cancer at autopsy. Thus, careful observations of Echo and ECG changes are of primary importance for an antemortem diagnosis of myocardial metastasis.

Like breast cancer, malignant melanoma, and/or malignant lymphoma, metastases of the lung can spread to the heart, and metastases in the pericardium or myocardium is present in 20%–30% of autopsied patients with lung cancer.1–4 Metastatic cancer in the heart is difficult to diagnose before a patient’s death because there are few typical signs that lead to the diagnosis.5 Before death, the diagnosis is usually made by the discovery of ST-segment modifications after excluding the much more frequent causes such as acute MI, pericarditis, and cardiomyopathy.

Two-dimensional Echo may help in the differential diagnosis. Our lung cancer patient presented with chest tightness, ECG changes, and elevation of cardiac enzymes. Acute MI was the initial diagnosis considered. The final diagnosis of cardiac metastasis of lung cancer was confirmed by Echo and chest CT scan. No thrombolytic therapy or coronary intervention was administered.

CASE REPORT

The patient was a 68-year-old woman. She had lung cancer 2 years previously and was being monitored at a local medical center. On December 31, 2002, she was admitted to the hospital because of a productive cough and intermittent chest tightness. The chest films showed an abnormal shadow in the left upper lung field (Figure 1). Chest CT showed a lung tumor in the left upper lobe and possible invasions to the pericardium and left ventricular wall (Figure 2). ECG showed normal sinus rhythm, low voltage of the QRS complex, and ST elevation in leads I, II, III, aVF, V4, V5, and V6, with reciprocal ST depression of V1 and V2 (Figure 3A). The creatine phosphokinase and creatine kinase-myocardial band levels were nearly normal (153/21 U/L), and troponin I was mildly elevated (1.10 ng/mL). Acute inferior lateral wall MI was considered as the diagnosis. The patient was admitted to the intensive care unit.

On the admitting physical examination, the patient showed general weakness and respiratory embarrassment. Her vital signs were stable. Her systolic blood pressure was 118 mm Hg and her diastolic pressure was 80 mm Hg. Her pulse rate was 62 bpm and her respiratory rate was 20 BPM. Her heart beat was regular, with normal first and second heart sounds.

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Echocardiographic and Electrocardiographic Manifestations of Heart Metastasis From Primary Lung Cancer

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second heart sounds and without third and fourth heart sounds. Her breath sounds were clear. There was mild engorgement of the jugular vein. No heart murmur was detected. Her abdomen was soft and flat, without a palpable mass, tenderness, hepatomegaly, or splenomegaly. Her lower legs showed grade I pitting edema. There was no significant abnormal neurologic reflex.

Echo showed a hyperechoic and thickened left ventricular posterolateral wall with hypokinesis. Two small masses with high motility were seen in the left ventricle (Figure 4). The serum levels of creatine phosphokinase and creatine kinase-myocardial band were consecutively rechecked in 6-hour intervals for 24 hours. There was no significant difference among the four results (Table). A repeat 12-lead ECG 4 days later (Figure 3B) showed no significant change compared with the previous ECG. The patient had poor response to conservative treatment and expired on the sixth day after admission due to respiratory and heart failure; the family refused an autopsy.

**DISCUSSION**

The most common neoplasms associated with cardiac metastasis are lung cancer, lymphoma, breast cancer, leukemia, stomach cancer, and melanoma. The incidence of cardiac metastasis is estimated to be about 1.5%–20.6% of malignant tumors at autopsy, according to a study by Nakayama and Kimura in 1966. Then Strauss et al., Adenle and Edwards, Skhvatshabaja, and Tamura et al. reported in succession that cardiac metastases occurred in 25%–40% of patients with lung cancer. However, heart metastases are usually not diagnosed before a patient’s death due to a lack of specific signs. Thus, most of them are found only at autopsy.

Myocardial involvement is considerably rare and results from direct invasion of pericardial metastasis. The incidence of myocardial metastasis from lung cancer is 5.3%–10.6% in autopsy studies. The signs and symptoms of myocardial metastasis are not specific enough to permit recognizing them in lung cancer patients. Nonetheless, ECG changes such as arrhythmia, low-voltage complexes, and nonspecific ST-T changes may provide evidence of cardiac involvement by a metastatic tumor. However, the ST-segment and T-wave changes are not specific signs of myocardial metastasis. The differential diagnosis has to be performed against much more frequent etiologies, such as myocardial ischemia, pericarditis, hypertrophic cardiomyopathy, and/or the result of drug treatments. Cerebrovascular accidents, especially subarachnoid hemorrhage, may also cause ST-T elevation on the ECG, which results from ventricular repolarization abnormalities.
The concurrent MI, a rare complication of malignancy, has occurred as a result of tumor embolization in the coronary arteries, tumor-induced compression of the coronary arteries,\textsuperscript{13,14} or tumor-induced alternation of cardiac oxygen consumption. Acute MI-like ECG changes due to metastatic involvement in the myocardium are rarely found. The mechanisms are still unclear. Rosenbaum et al.\textsuperscript{15} presumed that the ST-segment elevation was due to persistent myocardial injury caused by pressure or by physicochemical action or by interference with the blood supply as the malignant tissue invaded the heart. Harris et al.\textsuperscript{16} mentioned the association of ECG change with necrosis and extensive tumor infiltration of the myocardium. Some researchers have presumed that the interference with coronary flow by the tumor causes secondary MI.\textsuperscript{17} But, in most cases,
the values for the series cardiac enzymes are within normal ranges and/or ECG shows persistent ST-segment elevation.\textsuperscript{15,16,18}

In our patient, the diagnosis of lung cancer was confirmed at another medical center 2 years previously. CT showed that the heart was involved by the tumor. ECG showed ST-segment elevation in leads of inferior and lateral walls. Acute MI was originally suspected on the bases of chest tightness, ECG change, and slight elevation of serum cardiac enzymes. The Echo excluded the presence of massive pericardial effusion but demonstrated the presence of hyperechoic and thickening of the posterolateral wall of the left ventricle. There was no severe chest pain, referred pain, cold sweating, shock, or vital sign change. Thus, coronary angiography was not performed immediately. The diagnosis was revised due to the similarity of the serial cardiac enzyme values (Table) (Figure 3 and Figure 4) and ECG findings. If the patient had an acute MI, it might have been caused by the mechanism of compression of an epicardial artery by the tumor or by the tumor mass invading a coronary artery and/or branch that impeded the coronary blood flow. The patient received conservative treatment, and no histology report was made. Echo provided better evidence of cardiac metastasis in our patient. Recent development of various imaging devices has made it easy to diagnose myocardial metastases in vivo, such as 2-dimensional Echo, consecutive ultrasonic cardiography,\textsuperscript{19} CT, technetium Tc-99m sestamibi single-photon emission CT, and MRI.

In conclusion, we report a case of lung cancer with myocardial metastasis, with clinical findings similar to acute MI including clinical symptoms, elevation of cardiac enzymes, and ST elevation on ECG. It is necessary to consider the possibility of myocardial metastasis when a patient with malignancy presents with acute MI-like clinical findings. Two-dimensional Echo could be a noninvasive and sensitive supplementary tool to help detect pericardial effusion and metastatic cardiac mural tumors. Given careful consideration of acute MI-like findings, these diagnostic tools could be used to make an earlier diagnosis of cardiac metastasis in some patients.

\textbf{REFERENCES}


