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A case of double cancers with myocardial metastasis mimicking acute myocardial infarction both on an electrocardiogram and on Tc-99m-MIBI myocardial SPECT

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We report a rare case of double cancers with myocardial metastasis presenting acute myocardial infarction (AMI)-like findings both on an electrocardiogram (ECG) and on Tc-99m-MIBI myocardial SPECT. The ECG showed abnormal Q-waves and ST-segment elevation in leads V_1–V_4, and Tc-99m-MIBI SPECT showed a photon deficient area in the anteroseptum. These findings were suggestive of AMI, but the patient had been simultaneously suffering from two adenocarcinomas, which were lung cancer and gastric cancer, and consecutive ultrasonic cardiography (UCG) demonstrated a growing mass lesion in the septal aspect of the left ventricle. After a month he died of severe heart failure. The histological diagnosis of a specimen of the cardiac mass lesion was invasive adenocarcinoma infiltrating to the heart, which revealed that the myocardial metastasis had mimicked AMI. This case shows that it is difficult to distinguish between myocardial infarction and myocardial metastasis with myocardial perfusion SPECT. It is necessary to consider the possibility of myocardial metastasis when a patient with malignancy presents AMI-like findings.

Key words: myocardial metastasis, AMI-like findings, ECG, Tc-99m-MIBI

INTRODUCTION

CARDIAC METASTASES OCCASIONALLY ARISE from some malignant tumors, such as lung cancer, leukemia and breast cancer. The incidence of cardiac metastasis is estimated to be about 1.5–20.6% of malignant tumors.[1] In most cases, cardiac metastases spread to the pericardium[2] and are first noticed at autopsy. In this report we present a rare case of double cancers that metastasized to the myocardium with acute myocardial infarction (AMI)-like findings both on an electrocardiogram (ECG) and on myocardial perfusion imaging with Tc-99m-MIBI.

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CASE REPORT

A 69-year-old man had transient pains in the anterior chest and the left shoulder when he was riding a bicycle in December 1997. After two weeks, he visited Fukui Prefectural Hospital because of a persistent cough, and then ECG abnormalities and an abnormal lung shadow on a chest radiograph were detected. The ECG showed atrial fibrillation and abnormal Q-waves and ST-segment elevation in leads V_1–V_4 (Fig. 1) even though the serous cardiac enzymes were within normal limits. Because the ECG findings were suggestive of anteroseptal AMI, urgent cardioangiography (CAG) was performed, but no stenosis of the main coronary arteries was seen. Meanwhile, a chest X-ray CT showed a mass lesion in the lower lobe of the right lung and pericardial effusion (Fig. 2A and 2B).

Myocardial scintigraphy was performed 50 min after intravenous injection of 740 MBq (20 mCi) Tc-99m-MIBI with a triple headed gamma camera (GCA-9300A/

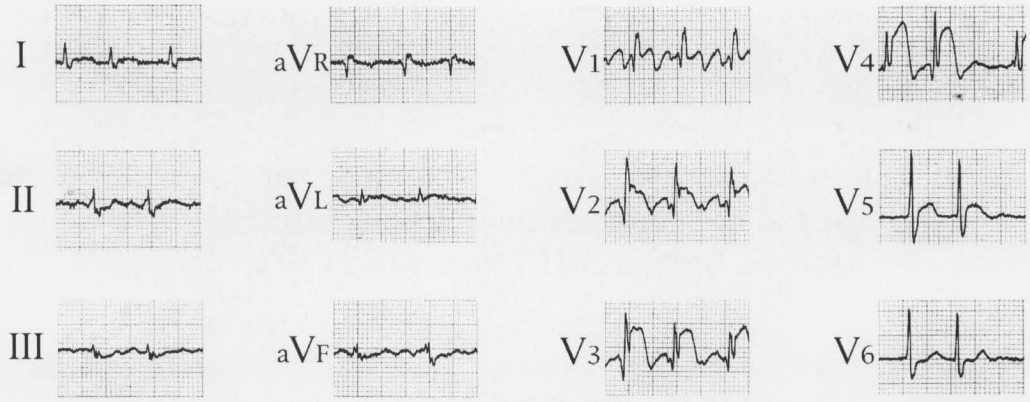

Fig. 1 ECG showed abnormal Q-waves and ST-segment elevation in leads V_1–V_4 besides atrial fibrillation.

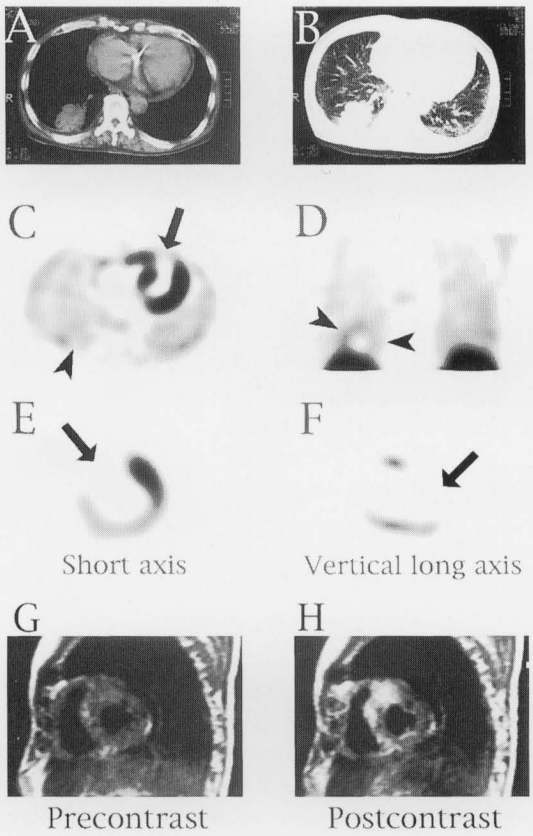

Fig. 2 (A and B) Thoracic CT showed a mass lesion in the lower lobe of the right lung and pericardial effusion. (C, D, E and F) Tc-99m-MIBI SPECT showed a photon deficient area in the anteroseptum (arrows) and an abnormal accumulation corresponding to the periphery of the right lung mass lesion (arrowheads). (G and H) T1-weighted MR images showed an enhancement with contrast medium in the anteroseptum.

HG; Toshiba Medical, Japan) equipped with low-energy, high-resolution parallel-hole collimators to assess the myocardial status. Acquisition parameters were a matrix 128 by 128 pixels, a step angle of 6 degrees, acquisition time of 30 sec and 60 acquisition directions. The energy window was set at 140 keV ± 20%. The reconstructed images were obtained with a Butterworth filter (cut-off frequency of 0.15 cycles/cm, order 8) and Ramp filter. Tc-99m-MIBI SPECT showed a photon deficient area in the anteroseptum and increased tracer uptake in association with the periphery of the mass lesion of the right lung (Fig. 2C, 2D, 2E and 2F). Ultrasonic cardiography (UCG) showed a small flap in the septal aspect of the left ventricle (Fig. 3A). MRI showed an enhancement with contrast medium in the anteroseptal wall but failed to delineate the strange flap (Fig. 2G and 2H). Transbronchial lung biopsy revealed infiltrating adenocarcinoma in the S_{10} of the right lung. Based on these findings, it seemed as if this patient with lung cancer had accidentally suffered from AMI caused by vasospasm. But the true character of the flap in the left ventricle remained unclear. Subsequent UCG showed that the flap had expanded rapidly, which suggested that it was a metastatic cardiac tumor (Fig. 3B). Furthermore, gastroscopy detected an advanced gastric cancer (Borr. 3) in the cardiac part of the stomach, and pathological examination revealed moderately differentiated tubular adenocarcinoma.

Arial fibrillation and tachycardia later became worse with enlargement of the cardiac tumor, and he died of severe heart failure. ST-segment elevation on the ECG lasted until his death. The histological diagnosis of the cardiac tumor through needle necropsy was invasive adenocarcinoma infiltrating into the heart (Fig. 4). The extent of fibrotic changes in the myocardial tissue was completely in agreement with that of tumor invasion. Unfortunately it has remained unclear which of the two adenocarcinomas, lung cancer or gastric cancer, had metastasized to the heart.

DISCUSSION

Cardiac metastases of malignancies are unexpectedly not very rare. Several investigators have estimated the inci-

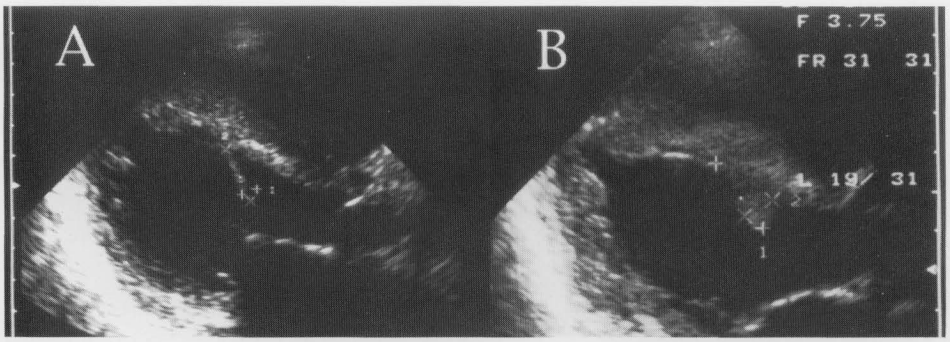

Fig. 3 UCG showed the rapid growth of the flap in the septal aspect of the left ventricle. (A) Initial UCG at the admission. (B) Following UCG three weeks after the admission.

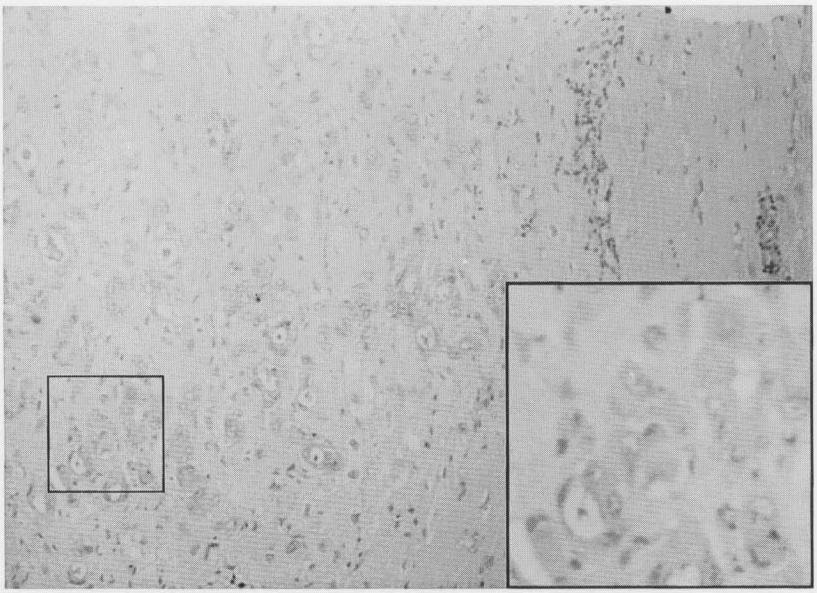

Fig. 4 Adenocarcinoma infiltrating into the heart with desmoplastic change. Normal myocardium is at right.

dence of cardiac metastases to be about 1.5–20.6% of patients with malignancies.[1] It is said that cardiac metastases in particular occur in 25–40% of patients with lung cancer,[3–6] but because it is very hard to detect cardiac metastases in life while the patient is alive, most of them were first noticed at autopsy.[7]

When the heart is divided into the following three parts, pericardium, myocardium and endocardium, the most common site that metastatic tumors involve is the pericardium.[8] Myocardial involvement is considerably rare, and mostly results from direct invasion of pericardial metastases.[9]

The metastatic pathways to the heart are generally lymphatic or hematogenous metastases. Tamura et al. reported that the major metastatic pathway from lung cancer to the heart is lymphatic, and patients with lymphatic metastasis had a higher incidence of malignant pericardial effusion than those with hematogenous metastasis.[6] Therefore, it may have been that in our patient lung cancer had lymphogenously metastasized to the heart.

In cases of myocardial metastases, the ECG shows various findings, which are ST-T changes, conduction disturbances, arrhythmia or absolutely no abnormal findings, but AMI-like ECG findings are rare. In our case, obvious abnormal Q-waves and ST-segment elevation were observed in leads V_1–V_4. There are several reports of such cases mimicking AMI.[2,9–15] In those cases, some patients were first treated as AMI because of typical AMI-like ECG findings, which indicates the difficulty in diagnosing myocardial metastases by ECG. Berge et al. concluded that it is almost impossible to diagnose cardiac metastases in life only by ECG.[16] And Biran et al. emphasized the necessity for continuous observation of

ECG changes.[17]

The mechanisms of AMI-like ECG on myocardial metastases are still unclear. Rosenbaum et al. presumed that the ST-segment elevation is due to continuous myocardial injury caused by pressure or by physicochemical action, or by interference with the blood supply as the malignant tissue invades the heart.[10] Harris et al. mentioned the association with necrosis and extensive tumor infiltration of the myocardium.[11] Some researchers have presumed that the interference with coronary flow by the tumor causes secondary myocardial infarction,[18] but in most cases, including our patient, the values for the serous cardiac enzymes were within the normal range and/or ECG showed persistent ST-segment elevation.[2,9–15] Furthermore, Tamura et al. reported that AMI was detected in only 1 case among 23 autopsy cases of cardiac metastasis, which demonstrated that AMI is a rare complication in a patient with cardiac metastasis.[6] And in our case, although the histological specimens of the metastatic cardiac tumor were obtained only by the percutaneous needle necropsy, the extent of fibrotic changes in the myocardial tissue was completely in agreement with that of tumor invasion, which indicates that the fibrotic changes had resulted from tumor desmoplasia rather than AMI. These findings show that secondary myocardial infarction is not a principal factor in AMI-like ECG findings on cardiac metastasis.

Recent development of various imaging devices has made it easy to diagnose myocardial metastases in life. In our case, UCG, MRI and Tc-99m-MIBI SPECT were performed to assess the condition of the myocardium, and each of them detected myocardial abnormality. MRI is a promising imaging method and could detect the enhancement of the tumor tissue in the anteroseptal wall. Nevertheless, it was difficult to declare the myocardial lesion to be a cardiac metastasis at that time because myocardial enhancement itself is also seen in the case of AMI.[19] UCG, which is easy to repeat and successfully showed growth of the flap in the septum, was most useful in confirming the myocardial metastasis.

Even though myocardial SPECT may not be essential, Mikasa et al. reported that a combination study with Ga-67 and Tl-201 may be useful in distinguishing between AMI and myocardial metastasis.[9] According to those authors, both myocardial metastasis and AMI are described as cold lesions in Tl-201 studies, so that Tl-201 is suitable for the screening. On the other hand Ga-67 study shows temporary abnormal uptake for the first week in case of AMI, but abnormal uptake increases as time goes on in the case of myocardial metastasis. In our case, the myocardial scan with Tc-99m-MIBI showed a cold area at the site of the myocardial metastasis. It was presumed that intense uptake of the surrounding normal myocardium masked the tumor uptake, so that it may be impossible to differentiate myocardial metastasis from infarction with myocardial perfusion SPECT alone.

In conclusion, we reported a case of double cancers with myocardial metastasis, with AMI-like findings both on ECG and on myocardial perfusion SPECT. It is necessary to consider the possibility of myocardial metastasis when a patient with malignancy presents AMI-like findings.

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