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Case Report

Acute myocardial infarction due to malignant neoplastic coronary embolus

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Summary A 54-year old man was diagnosed with right lung carcinoma (squamous cell carcinoma, SCC), stage IIIB (c-T2N3M0). Transthoracic echocardiography (TTE) showed a huge 8.9 cm × 1.3 cm tumor in the left atrium (LA) that was invaded by a pulmonary vein, and the tumor moved under the mitral valve at LA systole. After 3 months, he was diagnosed with acute myocardial infarction (AMI) and emergency coronary angiography (CAG) was performed. CAG showed that the distal segment of the right coronary artery was totally occluded. TTE showed that the shape of the mass tip became sharp. He was discharged on hospital day 15. He died 4 months after discharge because of right lung carcinoma with respiratory failure. An autopsy showed that the cause of AMI was tumor embolism. SCC clearly occupied a blood vessel lumen in the distal segment. This is a rare case of AMI due to embolism of lung carcinoma during progression of the disease with direct invasion to the LA. TTE is useful for assessing lung carcinoma invasion.

Introduction

Direct invasion of the left atrium (LA) by lung carcinoma is rare, and the area that is most often involved in the spread of carcinoma is the pericardium [1,2]. Lung carcinoma rarely induces tumor emboli in coronary or pulmonary arteries [3].

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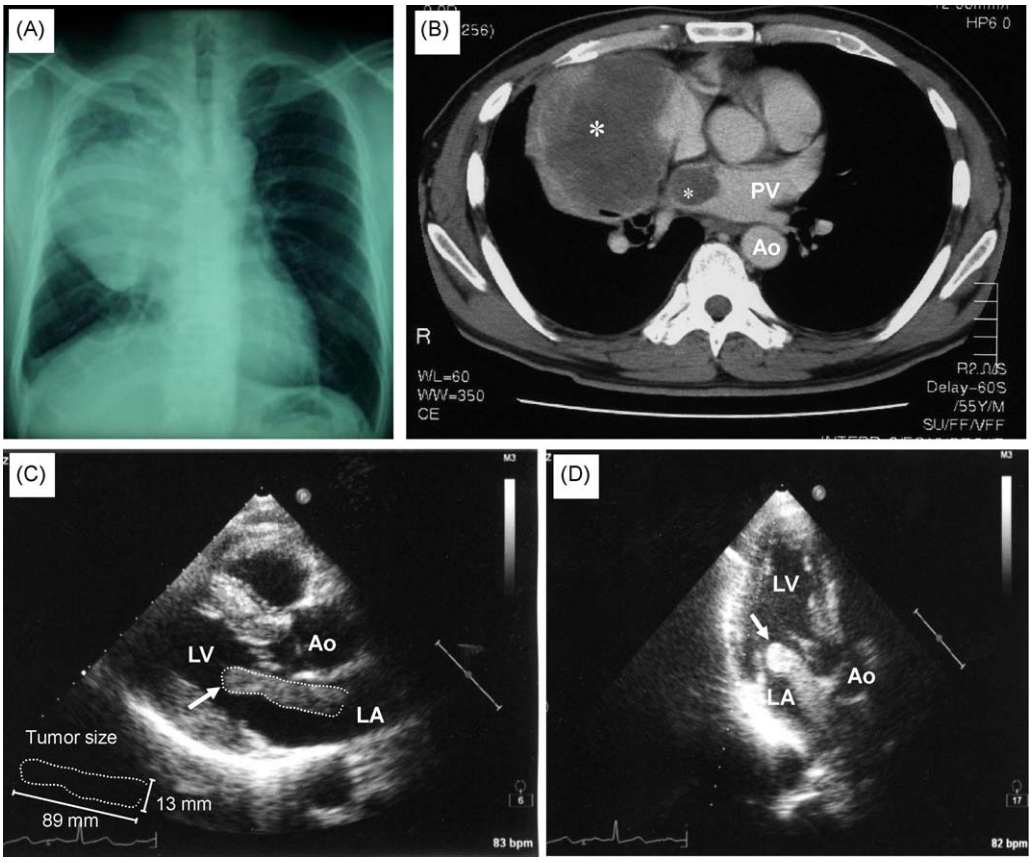

Figure 1 (A) Chest X-rays demonstrated a huge mass on the right side. (B) Chest computed tomography showed that the known lung carcinoma (*) invaded the right pulmonary vein (PV). (C and D) Transthoracic echocardiography showed a tumor (→) in the left atrium (LA) that moved under the mitral valve at LA systole. Ao, aorta.

Although it has been reported that the incidence of coronary embolization in patients with myxomas is only 0.06% [4], acute myocardial infarction (AMI) due to lung carcinoma embolization to a coronary artery is even rarer. We report here a case of AMI due to embolism of lung carcinoma during the progression of carcinoma with direct invasion of the LA.

Case report

A 54-year old man visited the Department of Respiratory Medicine at Fukuoka University Hospital because of a sustained fever and left shoulder pain. He had no medical history, but had a history of smoking for 30 years and a laboratory examination showed diabetes mellitus (fasting serum glucose 148 mg/dL and hemoglobin A1c 8.6%). Chest X-ray and computed tomography (CT) of the thorax demonstrated a huge mass on the right side. Transbronchial lung biopsy proved that the mass was squamous cell carcinoma (SCC). He was diagnosed with right lung carcinoma, stage IIIB (c-T2N3M0). Since there was no indication for an operation, he was treated with chemotherapy (cisplatin + vinorelbine). However, he became resistant to the treatment.

Eight months after the first visit to our hospital, he had to change his chemotherapy. Chest X-rays showed a huge mass on the right side (Fig. 1A). Chest CT demonstrated that the known lung carcinoma had invaded the right pulmonary vein (Fig. 1B). Transthoracic echocardiography (TTE) showed a huge 8.9 cm × 1.3 cm tumor in the LA that was invaded by a pulmonary vein, and the tumor moved under the mitral valve at LA systole (Fig. 1C and D). Other parameters of left ventricular (LV) function were normal and there was no valvular heart disease. Therefore, the huge tumor in the LA was considered to be the lung carcinoma invasion. His attending physician judged that the prognosis was poor and chose not to perform extirpation with the patient's informed consent. The patient was given erlotinib as a second chemotherapy.

He visited the emergency center of our hospital because of sudden chest pain 3 months after beginning the second chemotherapy. Electrocardiography showed ST elevation on leads II, III, and aVF (Fig. 2A). Laboratory examination of a blood sample showed an increased white blood cell (WBC) count and serum creatine kinase (CK) (WBC: 12,100/μL, CK: 932 IU/L), and troponin T was positive. He was diagnosed with AMI and emergency coronary angiography (CAG) was performed. CAG showed that the distal segment of the right coronary artery was totally occluded (Fig. 2B), and other coronary arteries did not show any significant stenosis. Although ballooning was tried on the distal segment, there was no reperfusion in that region. The severity of MI was mild (CK max 1290 IU/L), and there were no complications. As depicted in Fig. 2C and D, TTE showed that the shape of the mass tip became sharp and smaller, and the long

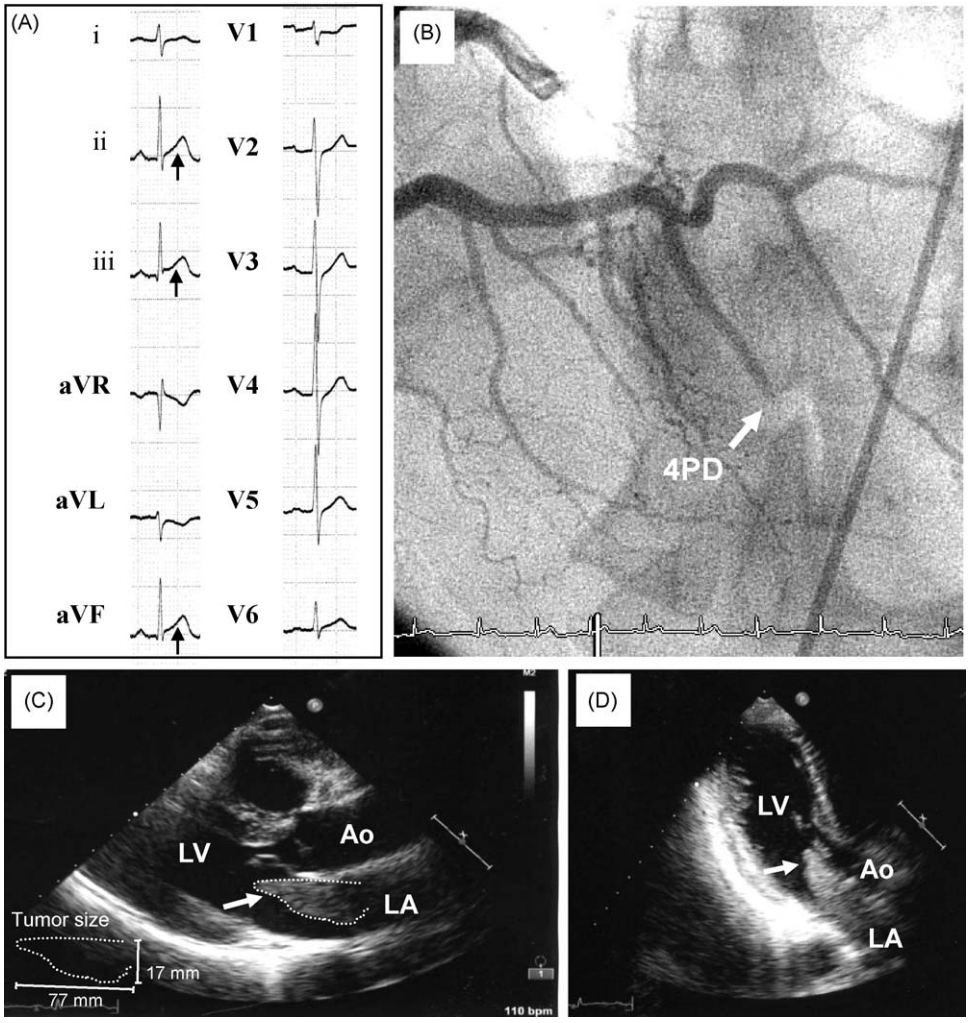

Figure 2 (A) Electrocardiography showed ST elevation in leads II, III, and aVF (↑). (B) Coronary angiography showed that the distal segment of the right coronary artery was totally occluded (→). (C and D) Transthoracic echocardiography showed that the shape of the mass tip became sharp and the size was smaller than before.

axis shortened, but the root enlarged (7.7 cm × 1.7 cm). CT was performed on the head, and brain infarction was not observed. He was discharged on hospital day 15.

Four months after discharge, his face became swollen because of superior vena cava (SVC) syndrome and he had dyspnea. He was hospitalized, and chest X-rays showed that the known right lung mass had grown and there was severe pneumonia in the right lung. Although he was given an antibiotic transfusion and oxygen, he developed disseminated intravascular coagulation and died 3 days after hospitalization.

An autopsy was performed after his family gave their consent and the results were as follows. The autopsy report concluded that the main cause of death was right lung carcinoma with respiratory failure and right thoracic cavity occupation, SVC syndrome, LA invasion, and multiple distant metastases. Tumor invaded superior vena cava and right pulmonary artery directly. In addition, tumor invaded right superior pulmonary vein directly and occupied a lumen, and subsequently invaded the LA. The cause of MI was tumor embolism. The heart weighed 370 g and the pericardial cavity contained 48 mL of effusion. Part of the LV posterior wall showed fibrinoid necrosis (Fig. 3A). There were moderate stenotic lesions of the right coronary artery (RCA) and left circumflex branch (LCX), and the distal segment was totally occluded. SCC clearly occupied a blood vessel lumen in the distal segment (Fig. 3B and C).

Discussion

We have reported a case of AMI due to embolism of lung carcinoma during progression of the disease with direct invasion to the LA. The direct invasion of the LA by lung carcinoma is rare [1,2]. Generally, tumors can spread to the heart through 4 main pathways: through the bloodstream; through the lymphatic system; by direct invasion; and by intracavitary diffusion through the inferior vena cava or pulmonary veins. The lymphatic system serves as a major pathway in cardiac metastases, and pericardial metastases are the most common type, followed by myocardial and epicardial metastases. Endocardial metastases comprise only 5% of all

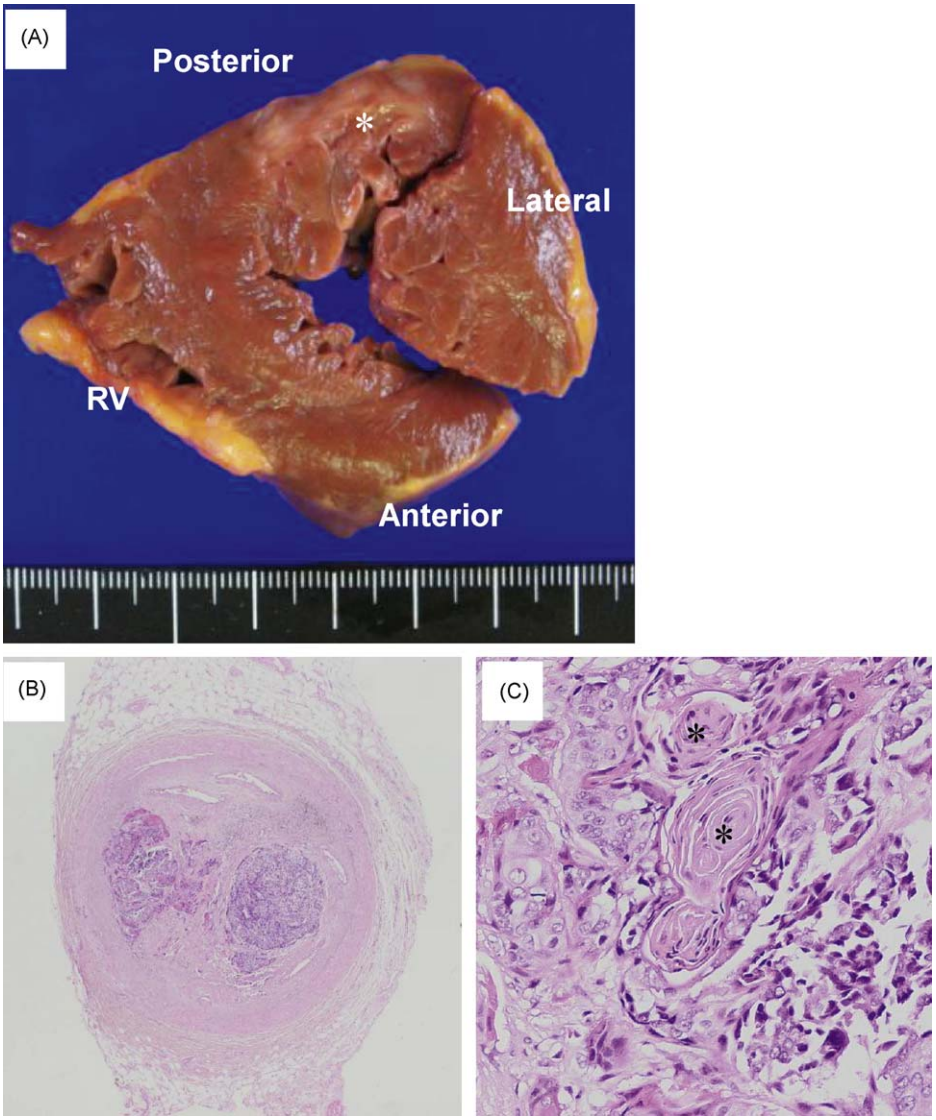

Figure 3 (A) Part of the LV posterior wall showed fibrinoid necrosis. RV, right ventricle. (B and C) Histological sections of a coronary segment (distal segment of the right coronary artery). Total occlusion of the distal segment was clearly observed (B), and squamous cell carcinoma (*) clearly occupied a blood vessel lumen in the distal segment (C).

cardiac metastases. These metastases are generally localized to the right heart and are usually associated with tumors with endovascular growth [2]. The direct invasion of lung carcinoma to the LA, as in the present case, is very rare.

Embolic obstruction of the coronary arteries due to tumor is also rare. Wenger and Bauer reported that embolic obstruction of coronary arteries was present in the left main or left anterior descending artery in 67% of patients, and 60% of them showed sudden death [5]. It has been reported that systemic embolization occurs in 30—40% of patients with myxomas. Although left atrial myxomas are the most common source due to neoplastic embolism [6], the incidence of coronary embolization in patients with myxomas is only 0.06% [4]. Since coronary embolism due to malignant non-cardiac tumor is rarer than embolization with myxomas, the present case is also very rare in this regard.

To our knowledge, only 16 cases of malignant coronary emboli have been reported [7—10]. Lung carcinoma was the most common source and accounted for 11 of the 17 cases including our case. Neoplastic invasion of the pulmonary veins may be the most frequent route for access to the systemic arterial circulation. This process was seen in the present and 8 previous cases. Moreover, carcinoma in 8 cases invaded to the LA chamber in addition to the pulmonary veins. Ten patients had coronary obstruction due to embolism in epicardial vessels, and 4 patients had embolism in 2 coronary arteries. According to these data, the left anterior descending artery was obstructed in 8 patients, the LCX in 5, the RCA in 3, including our patient, and the left main coronary artery in 2. Therefore, the left coronary artery was the main site of coronary embolism, and the RCA was the site of coronary embolism in only 17% of cases. In the present case, TTE after AMI showed that the shape of the mass tip became sharper and smaller than before, and SCC occupied the distal segment according to the autopsy report. Therefore, we confirmed that the lung carcinoma directly

invaded the LA, and neoplastic emboli occurred in the distal segment.

Echocardiography is a diagnostic method for assessing cardiac metastases. Since tumor in the LA usually comes from the inferior vena cava or PA, TTE is more useful for assessing carcinoma invasion in the heart.

In conclusion, the direct invasion of lung carcinoma to the LA with AMI due to neoplastic emboli in the RCA is very rare. In addition, TTE is useful for assessing the invasion of lung carcinoma.

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