A PRIMARY LUNG CARCINOMA CASE WITH HEART INVASION GIVING THE PSEUDOIMPRESSION OF MYOCARDIAL INFARCTION

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SUMMARY: One of the rare reasons of ST segment elevation in electrocardiogram is the malign invasion of the heart. In this paper, a patient with a diagnosis of epidermoid lung carcinoma who had undergone pericardiectomy due to pericardial invasion is presented. On the patient's reexamination six months later, a newly ST segment elevation was found on the ECG. Fix perfusion defect was present in myocardial thallium scintigraphy suggesting basal, anterolateral, lateral and inferolateral myocardial infarction, although there was no related history or myocardial enzyme elevation. The coronary angiography was entirely normal. The two dimensional echocardiography and thoracic computerized tomography revealed the presence of a mass narrowing the left ventricle cavity. The recent ECG findings which were not present six months ago pre or postoperatively, although there was pericardial invasion at that time, can be attributed to a more intense pericardial invasion or to the involvement of myocardium which could not be differentiated our case.

Key Words: Lung Carcinoma, Heart invasion, Acute Myocardial Infarction Image In Electrocardiogram.

INTRODUCTION

Although the incidence of primary tumors of the heart in autopsy series is % 0.0017, metastatic tumors are encountered more often (2). The patients who died from malignancy were shown to have metastasis in the heart at a rate of % 1.5 - 20.6 (5). Metastasis is generally observed in the pericardium and myocardium, the valves and endocardium are affected less frequently. His bundle involvement of the tumor was observed in a patient with atrioventricular block and lung carcinoma (5). The invasion can be seen by direct invasion, hematogenic spread or via lymphatic ducts.

In this report, a case with lung carcinoma, and metastasis to the heart possibly through direct invasion has been presented.

CASE REPORT

A 65 - year - old male patient who was planned to operate for left renal tumor was consulted by Cardiology Department before the operation because of the changes in his ECG.

The patient had no cardiac complaints. According to New York Heart Association (NYHA) classification, his functional capacity was rated as group 2. Of the risk factors for atherosclerotic heart disease, he had a cigarette smoking history only. It was clear from his background information that he
had undergone a left lung lingual lobectomy and partial pericardiectomy six months ago. It was established that there was no pathologic features in his ECG both pre and post operatively during this first hospitalization. The histopathologic diagnosis of the material obtained during the operation was reported to be modestly differentiated epidermoid carcinoma and reactive lymph nodes, as well as epidermoid carcinoma infiltrated to the pericardium. The physical examination revealed that the patient had no pathologic findings other than a trace of operation under the left scapula with decreased respiratory sounds in the region. He did not come to his control examinations after the operation unless he was hospitalized in Urology Clinic due to left flank pain and hematuria. The patient was transferred to the Cardiology Clinic so that the ECG findings could be cleared up.

LABORATORY FINDINGS

Hemoglobin: 10.5 g/dl. Leukocyte: 11.600/mm³. Erythrocyte sedimentation rate: 120 mm/h. No pathologic features were found in the blood biochemistry. Complete urine examination revealed 4-5 erythrocytes. Posterior anterior lung radiography: There was elevation in the left diaphragm, the left costophrenic sinus was obliterated. The left ventricle appeared large (Fig 1). Electrocardiography: 0.5-2 mm ST segment elevation was established in inferoposterior and anterolateral region (V1, V2, V3). (Fig 2 a, b, c). Colored Doppler echocardiography: The inferior, inferoposterior, basal, anterolateral and lateral walls of the left ventricle are akinetic: there was an ecogenic mass related to those walls, narrowing the left ventricular cavity.

Fig - 1: Posterior anterior lung radiography.

Fig - 2 a, b, c: The electrocardiograms showing ST segment elevation in inferoposterior and anterolateral derivatives.
with the size of 76x44 mm and with about the same
density to the soft tissue, which was hardly to diffe-
rentiate from the myocardium (Fig 3). Thoracic
computed tomography: It revealed the presence of
a mass narrowing the left ventricle cavity. (Fig 4).
Thallium-201 myocardial perfusion SPECT: Lack
of perfusion was established in the basal anterolate-
ral, lateral, inferolateral walls during stress myo-
cardial perfusion which persisted at rest (Fig 5).
Left Ventriculography: RAO position: inferobasal
akinesia was established. LAO position: There was
an irregular appearance on the left section of vent-
ricle facing the left atrium (Fig 6). Coronary angi-
ography: It was found normal (Fig 7, 8).

During the 15-day follow up of the patient, it
was observed that the initial ECG findings re-
mained the same and there was no change in the myo-
cardial enzymes. Urology, Oncology, Radiation
Oncology, Thoracic Diseases and Thoracic Sur-
gery consultations were made. It was considered
that due to the clinical condition of the patient, he
could not benefit from surgery, chemotherapy and
radiotherapy and he was discharged.

**DISCUSSION**

The leading causes of ST segment elevation of
organic origin are acute myocardial infarction, pe-
ricarditis and Prinzmetal's angina pectoris. Acute
pulmonary emboli, hyperkalemia, cerebrovascular
incidents, left ventricular hypertrophy, right bundle

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**Fig 3:** Two-dimensional echocardiogram, apical two-
chamber view shows a mass narrowing the left ventricular cavity.

**Fig 4:** Thoracic computed tomography.

**Fig 5:** Tomographic ²⁰¹Thallium image shown in the horizon-
tal long axis.

**Fig 6:** Left ventriculography in left anterior oblique projection.
branch block, hypertrophic cardiomyopathy, hypothermy and invasion of the heart by a neoplastic tissue can be mentioned among other conditions which causes ST segment elevation (3). In a normal heart one of the reason for the widely diffused ST segment elevation is early repolarization which is a normal variant.

Although in the history and physical examination, there was any symptoms and findings in favor of atherosclerotic coronary heart disease in our patient, ECG and myocardial thallium scintigraphy suggested coronary ischemia. Coronary angiography revealed normal coronary arteries. It was considered that the findings in ECG and thallium myocardial scintigraphy in this case were caused by the tumor created cardiac metastasis or showing cardiac invasion.

We could not elucidate which part of the heart (i.e., the pericardium or the myocardium) was invaded by the tumor in our case. The ECG findings of the patient didn’t suggest anything on this point, for both pericardial and myocardial invasion may lead to changes in ST-T wave (1). Although pericardial invasion was established during the operation six months ago, ECG findings were not present at that time. The echocardiogram and cineangiogram show akinetic areas in left ventricle, but we can not say firmly that there is a myocardial invasion, because a pericardial mass adhered to myocardium can lead to the same kinesia disorder. However, under exercise and resting conditions, stable perfusion defects observed in the thallium myocardial scintigraphy, made us think that there was a myocardial invasion which disturbed at least the microcirculation. The fact that the clinical condition of the patient was very stable and cardiac failure or angina pectoris, which should be expected in the case of myocardial invasion, was absent led us to hesitate on myocardial invasion. It is possible that the coronary arteries can be spared although myocardial invasion is present. In the literature, in 23 cases of cardiac metastasis, only one myocardial infarction related to compression of the coronary artery by tumor invasion, was established (7). It was considered that an endomyocardial biopsy could assist in establishing the exact location of the patient so it was abandoned from ethical point of view. Pericardial invasion was present at a rate of %15.7 in a series of 402 patients with lung carcinoma and cardiac invasion in the other sections of the heart was reported to be %10.2, and the invasion of both %5.4 (2). Due to both direct and lymphatic invasion, in patients with lung carcinoma, the cardiac sections involved most frequently are the pericardium and epicardium (4). Pericardial effusion which could lead to pericardial tamponade is an important finding (6). The development of such a condition in our case may have been prevented due to the partial pericardiotomy performed earlier. Based on the available data we cannot remark whether the tumorous invasion in our patient is only on the pericardium or the myocardium is also affected. For a better idea on this point, Nuclear Magnetic Resonance (NMR) examination was planned, but since the equipment was under maintenance, this examination could not be performed. As a conclusion, we presented this case in
order to emphasize that when myocardial infarction is suspected in ECG, neoplastic cardiac invasion should also be considered, and in such a case, ECG, and thallium-201 myocardial perfusion SPECT findings could be false positive for coronary artery disease.

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REFERENCES


