

CAZURI CLINICE

NONATHEROSCLEROTIC MYOCARDIAL INFARCTION WITHOUT ST-ELEVATION

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Summary

We present a clinical case (a 44-years-old man) of bronchioloalveolar carcinoma with massive pulmonary embolism and nonatherosclerotic ischemia. The patient has been examined by noninvasive methods. The presence of myocardial infarction without ST-elevation (non STEMI) was established by electrocardiography and elevated activity of CK-MB isoenzyme. The nonatherosclerotic etiology of non STEMI was confirmed by the presence of metastasis of bronchioloalveolar carcinoma that compresses the coronary artery at morphopathological examination. Massive pulmonary embolism was another complication of bronchioloalveolar carcinoma and facilitated by the presence of deep venous thrombosis. Evolution of disease was poor and prognosis is unfavorable because of association of many risk factors that lead to patient's death.

Introduction

The primary malignant heart tumors account for 0.5-10% of all heart tumors. The metastatic heart tumors represent more than 21% of all cardiac tumors.

The most frequently causes of cardiac metastasis are [4]:

- Lung cancer - 28%
- Melanoma - 37%
- Thyroid cancer - 30%

Metastatic spread of tumoral cells in heart is realized by lymphatics, bloodstream and by invasion. Systemic symptoms occur in 30% and include weight loss, anorexia, fever, rised erythrocyte rate sedimentation (ERS) [1,2,3].

Increased level of clotting factors in the blood in some types of cancer, especially pancreatic, lung and ovarian cancers, predispose to thrombosis. Patient with pulmonary embolism and cancer have a higher risk of dying than other patients with pulmonary embolism, and malignant tumors represent the most frequent cause of death in those patients (in 34.7%) [4,5]

Diagnosis of heart tumors is achieved by ECG, ECHO-CG, coronary angiography, myocardial scintigraphy.

Treatment usually is combined (surgical, chemo~ and radio - therapy).

Notice

A 44-year-old man was brought by ambulance to the Cardiology Division. He experienced intermittent retrosternal chest pain, dyspnoea during exertion, dry caught, low-degree fever (37.6°C), weakness and calfache associated to trombophlebitic nodes.

History. He reports a 2-weeks history of retrosternal discomfort at minimal exertion, last about 15 minutes, not fully relieved by nitroglycerin or rest, calfache, ankleache, local swelling, redness, warmth, temperature about 37-38°C and then appear shortness of breath and cough.

The patient did not currently take any medication, he is non-smoker, drank alcohol occasionally. There was no family history of sudden death or coronary artery disease.

Physical examination revealed severs general state, calfoedema, tenderness and cords deep in the calf, local swelling and warmth, body temperature was 37.6°C. The result of lymph nodes examination was normal, without evidence of lymphadenopathy. Bilateral breathing sounds were faint, with coarse crackles at the basis of each lung. The blood pressure was 110/70 mmHg, heart rate was 11 b/min with regular rhythm. There was a loud pulmonary second sound.

Laboratory data

Hematological examination shows a slight leukocytosis ($L-10.4 \times 10^9$) with left deviation (segmented neutrophils - 81%, bands - 9%), ERS -20 mm/h.

Biochemical constituents of blood: glucose and blood lipids were normal. CK-MB isoenzyme activity was elevated (CK-MB - 32 U/L), and liver and kidney biochemistry were normal.



Fig.1. Chest X-ray

Electrocardiography denotes sinus tachycardia, normal QRS axis and signs of subendocardial anteroseptal and apical ischemia (T-wave inversion in V_1 - V_4 leads). The next ECG presents changes in electrical axis S_1Q_3 pattern and incomplete right bundle branch block.

Chest X-ray detects oligoemic peripheral lung fields, hilar engorgement of pulmonary artery, proemination of pulmonary artery in the left border of the heart (Fig. 1).

Lung perfusion scans (Fig.2) demonstrates underperfused areas in the apex and base of right lung and middle area of the left lung. In conclusion there are modifications suggestive for pulmonary embolism in the respective vascular areas.

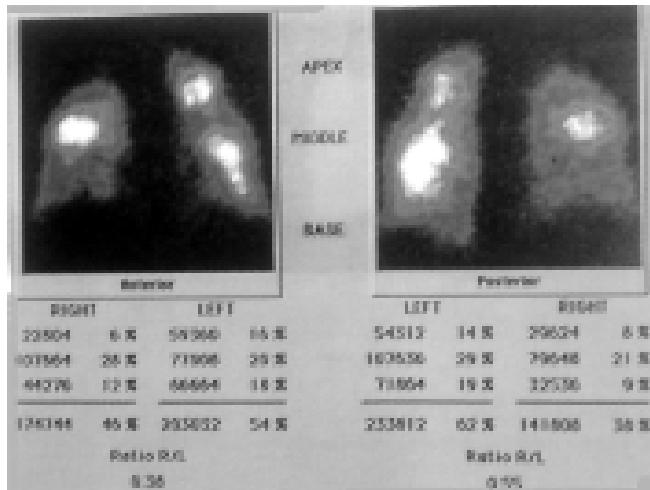


Fig.2. Lung perfusion scans

Ultrasound examination (Fig.3) detects an adrenal mass in the right gland.



Fig.3. Ultrasound examination

Echocardiography shows a slight dilatation of right ventricle (RV – 28 mm). There were no signs of regional wall motion abnormalities with LV function preserved. The peak pulmonary artery pressure was 45 mmHg.

On the basis of clinical and paraclinical dates it was suspected the diagnosis of acute coronary syndrome without ST-segment elevation (possible non STEMI). Non massive pulmonary embolism. Deep venous thrombosis.

Administered treatment consist of anticoagulant therapy (Clexan 1mg/kg bid s/c), antiplatelet agents (Aspirin 75 mg/day), beta-blockers (Metoprolol 25 mg bid.), nitrates (isosorbid mononitrate 20 mg bid) and antibiotics (Cefoperasone 2 g/day)

In 4 hours after hospitalization the patient was getting worse; the chief complaint was major dyspnoea, persistent hypotension

and patient died despite all emergency measures performed. It was suspected the massive pulmonary embolism.

Pathological discussion

Morphopathological examination revealed massive thrombus (Fig.4) in the pulmonary artery, pulmonary infarction and multiple metastases in heart, liver, kidney, bronchopulmonary lymph nodes, and right adrenal gland.

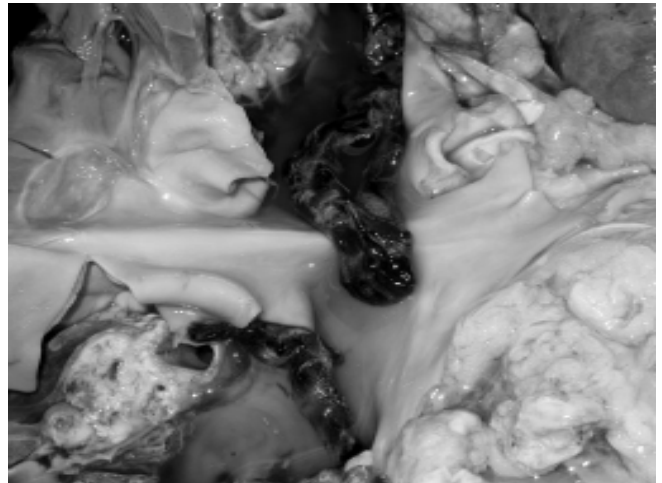


Fig.4. Massive thrombus in the pulmonary artery

Histopathologically was detected bronchioloalveolar carcinoma (Fig.5) with primary location in the right lung.

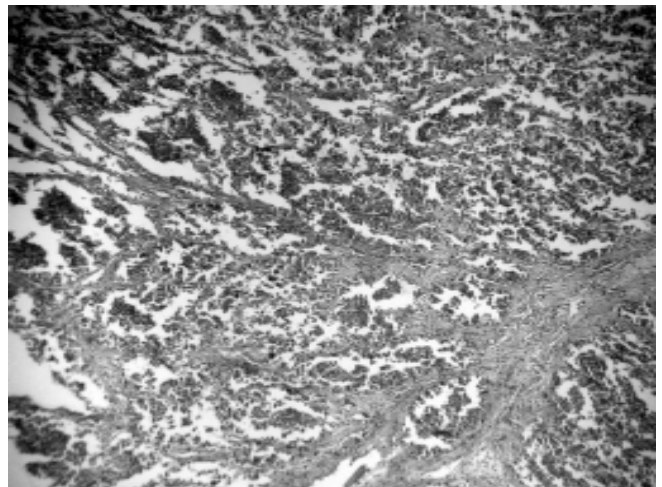


Fig.5. Bronchioloalveolar carcinoma

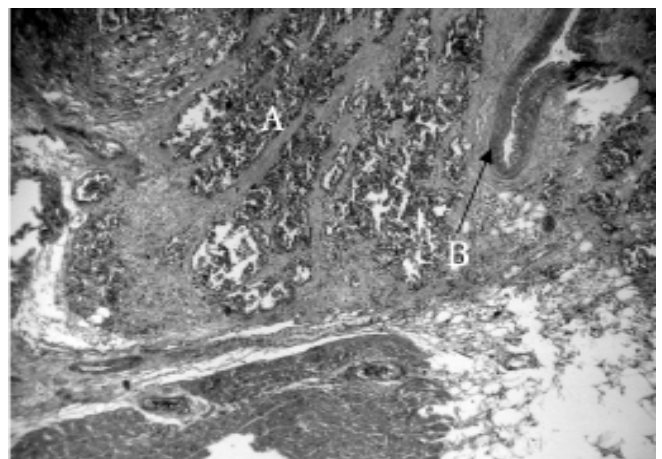


Fig.6. Myocardium with metastasis (A) of bronchioloalveolar carcinoma that compress the coronary artery (B)

In the figure 6 it is possible to see myocardium with metastasis of bronchioloalveolar carcinoma that compress the coronary artery and determine nonatherosclerotic ischemia, so probably that was the cause of ECG modification and elevated activity of CK-MB isoenzyme.

The cancerigenic intoxication was complicated by pulmonary embolism and pulmonary infarction that caused the patient's death.

Discussion

This case presents a peculiar interest because of the fact that ischemia unassociated with atherosclerosis has never been reported in malignant tumors. The existence of ischemia was proved by ECG changes and increased CK-MB activity. The explanation of pathophysiological mechanism may be the compression of coronary artery by the metastasis of bronchioloalveolar carcinoma in myocardium, as it was illustrated at hystopathological examination (Fig. 6)

Metastatic tumors in the heart represent about 21% of all heart tumors [4,5,6]. The most frequently causes of cardiac metastasis are: lung cancer - 28%, melanoma - 37%, thyroid cancer - 30%.

Metastatic spread of tumoral cells in heart is realized by lymphatics, bloodstream and by invasion. Systemic symptoms occur in 30% and include weight loss, anorexia, fever, rised erythrocyte rate sedimentation (ERS) [3].

Several small studies have indicated an association between deep venous thrombosis (DVT) or pulmonary embolism (PE) and a subsequent diagnosis of cancer, but the subject is controversial [7,8].

According to the data of nationwide study of a cohort of patients with DVT/PE drawn from Danish National Registry for the years 1977-1992, 45% of the patients given a diagnosis of cancer within one year after hospitalization for thrombembolism, had distant metastases at the time of the diagnosis of cancer. There was strong association of PE with several cancers, most pronounced for those of the pancreas, ovary, liver and brain. This fact can be explained by increased blood level of procoagulants.

An association between venous thromboembolic disease is well documented and recent studies suggest that patients with so-called idiopathic PE develop subsequent malignant neoplasms in 10% of cases [1], but various risk factors for venous thromboembolism obviously act together. According to a recent French multicentre registry [2,9] almost one in two cases of PE and DVT occurred in the absence of a predisposing factors.

Natural history and prognosis: The prognosis of treated non-massive PE is mainly dependent on co-existing illnesses such as malignancy or cardiovascular diseases. Factors, associated with higher mortality are advanced age, cancer, stroke and cardiovascular diseases.

In conclusion, an aggressive search for a hidden cancer in patients with a primary DVT or PE is not warranted but there was strong evidence that proves the higher risk of death in patients with PE and cancer.

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