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Case Report

Acute myocardial infarction with concomitant pulmonary embolism as a result of patent foramen ovale

Abstract

Acute myocardial infarction (MI) and pulmonary embolism can alone lead to life-threatening conditions such as sudden cardiac death and congestive heart failure. We discuss a case of a 74-year-old man presented to the emergency department with acute dyspnea and chest pain. Acute anterior MI and pulmonary embolism concomitantly were diagnosed. Primary percutaneous coronary intervention performed because of preliminary acute anterior MI diagnosis. Transthoracic echocardiography was performed to determine further complications caused by acute MI because patient had a continuous tachycardia and dyspnea although hemodynamically stable. Transthoracic echocardiography revealed a thrombus that was stuck into the patent foramen ovale with parts in right and left atria. Anticoagulation therapy was started; neither fibrinolytic therapy nor operation was performed because of low survey expectations of the patient's recently diagnosed primary disease stage IV lung cancer. Patient was discharged on his 20th day with oral anticoagulation and antiagregant therapy.

In routine clinical practice, acute myocardial infarction (MI) or pulmonary embolism (PE) are frequent entities solitarily. Myocardial infarction's most frequent reason is coronary plaque rupture, whereas deep vein thrombosis is the most frequent reason for PE. Although both diseases have different pathologic backgrounds, in rare cases, acute MI may be seen concomitantly with PE. In this situation, underlying pathology may be procoagulant state and/or patent foramen ovale (PFO).

We describe an unusual case of a patient with acute anterior MI and massive PE, which was presented with severe chest pain and dyspnea to the emergency department with preliminary diagnosis of acute anterior MI and subsequent diagnosis of concomitant PE.

A 74-year-old man with a recent diagnosis of lung cancer presented to emergency department with severe crushing chest pain and dyspnea. On physical examination, he was cachectic and tachypneic. His blood pressure was 125/86 mm Hg, and oxygen saturation was 87% with pulse oximetry. Auscultation of the patient revealed diminished S1 and loud S2; no additional sound was heard. Bilateral ronchi and basilar rales were heard on pulmonary auscultation. The electrocardiogram revealed sinus tachycardia in addition to ST-segment elevation in precordial derivations (V1-V6) with QS formation (Fig. 1).

The patient was transferred to the catheter laboratory for coronary angiography with acute anterior MI as preliminary diagnosis. Coronary angiography showed totally occluded proximal left anterior descending artery. Left main coronary artery, left circumflex artery, and right coronary artery were normal (Fig. 2A).

Left anterior descending artery was occluded with thrombus. After manual thrombus aspiration, percutaneous transluminal coronary angioplasty was performed (Fig. 2B). Left anterior descending artery showed bifid structure, one of the branches was proximally totally occluded, and the other branch was distally totally occluded. Multiple balloon dilatations were performed to the proximally totally occluded branch; unfortunately, no distal flow was obtained suggesting large thrombus burden occluding entire distal coronary lumen (Fig. 2C). Because there were no underlying obvious atherosclerotic plaques, no stent implantation has been performed.

The patient was transferred to the coronary care unit under tirofiban infusion. Overnight heart rate was between 115 and 130 beats per minute. Despite no anginal symptoms and effective treatment, persisting tachycardia and dyspnea raised a suspicion of complication because of MI. Bedside transthoracic echocardiography (TTE) showed right heart dilatation with basal right ventricular diameter of 48 mm. There was a mobile hyperechogenic mass 3.4 × 1.3 cm in the right atrium, which was consistent with a thrombus. Basal segment of this thrombus was sited interatrial septum, and there was a concomitant thrombus image in the left atrium (Fig. 3A and B).

Pulmonary computed tomographic angiography (CTA) was performed in suspicion of PE. Computed tomographic angiography revealed enlarged main pulmonary artery in addition to complete obstruction of right pulmonary artery and partial obstruction of left pulmonary artery and distal branches (Fig. 4). Patient was evaluated for fibrinolytic therapy, and before treatment, cranial computed tomography (CT) was performed to exclude a metastatic disease. Cranial CT revealed metastasis in the left parietotemporal region of the brain. Fibrinolytic therapy was not performed because of the metastatic brain lesion and biatrial thrombi. Low molecular weight heparin therapy was started on consultations of neurology and pulmonary disease departments. To investigate the deep vein thrombosis, we performed venous Doppler ultrasonography, and bilateral deep venous thrombosis was determined.

Transesophageal echocardiography (TEE) was performed for better characterization of both atrial masses and to display the nature of left atrial mass. Transesophageal echocardiography showed biatrial thrombus formation connected via PFO (Fig. 3C).

On detailed patient history, we have found that patient had experienced shortness of breath and hemoptysis about a month ago, and he was admitted to pulmonary department of another hospital, where stage IV lung cancer was diagnosed with positron emission tomography CT.

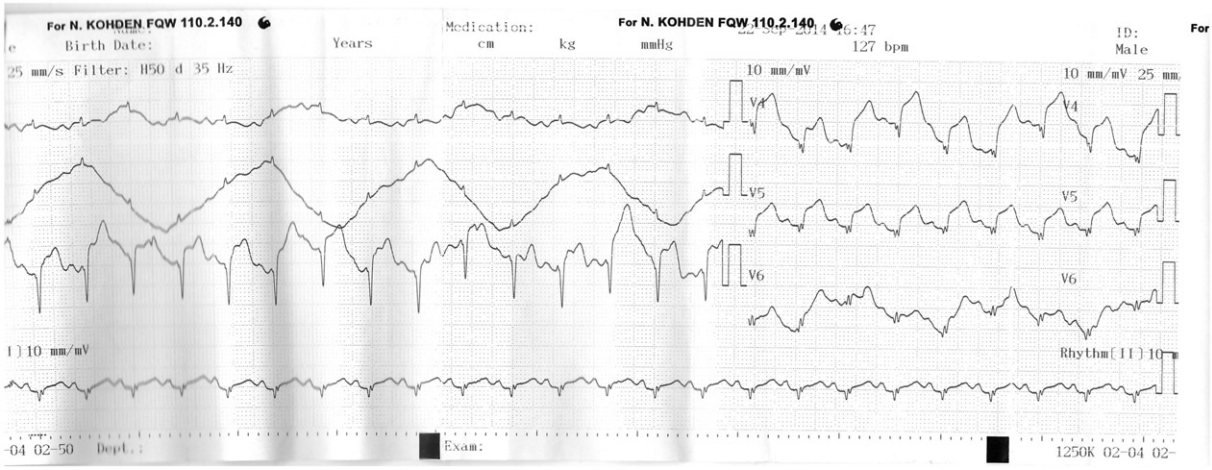

Fig. 1. Figures of the case report

Patient has been consulted with cardiovascular surgeons because of biatrial and pulmonary thrombus for the intention of thrombecthomy. However, medical treatment was decided because the patient was hemodynamically stable and has low survival expectation because of stage IV lung cancer and brain metastasis.

He was discharged uneventfully on his 20th day of hospital stay with oral anticoagulant and antiagregant therapy.

This case is an important example of initial presentation of acute MI that can be a complication of underlying PE. This case is also important to show us that bedside echocardiography has primary importance in the coronary care unit to reveal the underlying physiopathology, especially when patient clinical findings do not match the clinical problem.

Tachycardia and dyspnea should be a cautionary signal in patients with acute MI suggesting a complication such as pump failure or acute mitral insufficiency [1]. In our patient, failure of the percutaneous coronary intervention because of massive coronary thrombus burden primarily suggested a left ventricular failure secondary to the anterior MI. However, physical findings of acute pulmonary edema were not detected. Mechanical complications of acute MI could be another possible cause, which generally appear on 3 to 4 days after MI. Transthoracic echocardiography was performed to evaluate left ventricular functions, which incidentally showed right ventricular dilatation, and subsequent TEE excluded mechanical complications besides revealing biatrial thrombus via PFO [2].

We suspected from PE on the strength of the patient's clinical state and TTE findings. Pulmonary CTA confirmed acute PE.

Concomitantly acute MI and PE suggested a transition between the atria and/or a procoagulant state. It has been known that cancers predispose patients to procoagulant state. Cancer-associated procoagulant state often lacks a clear etiology. Many studies have been trying to understand the metabolic alteration; one of the latest studies proposed that cancers predispose neutrophils to release DNA traps that contribute to thrombosis [3,4]. In our patient, bilateral deep vein thrombosis in lower extremity may be caused by lung cancer causing procoagulation. The thrombus in right atrium may occur separately and passed through the PFO to the left atrium, or it could be detached from the deep veins of the lower extremities.

In this case, TEE showed us the PFO that causes paradoxical embolism [5]. The PFO found in about 25% to 30% of the population is an asymptomatic condition, whereas it may be responsible for the 5% to 10% of paradoxical embolism [6].

The criteria for diagnosis of paradoxical embolism are (1) evidence of arterial embolism in the absence of a source in the left heart, (2) evidence of a source of embolism in the venous system, and (3) evidence of communication between venous and arterial circulation [7]. Paradoxical embolism is a rare cause of coronary embolism and may be underdiagnosed in patients with PFO.

Silent PE has been reported in 40% to 60% of patients with deep venous thrombosis regarding lung scintigraphic and angiographic abnormalities. Systematic lung scans reveal a high frequency of silent PE in patients with proximal deep venous thrombosis [8]. In our case, PE was also silent and is not clinically recognized until paradoxical embolism.

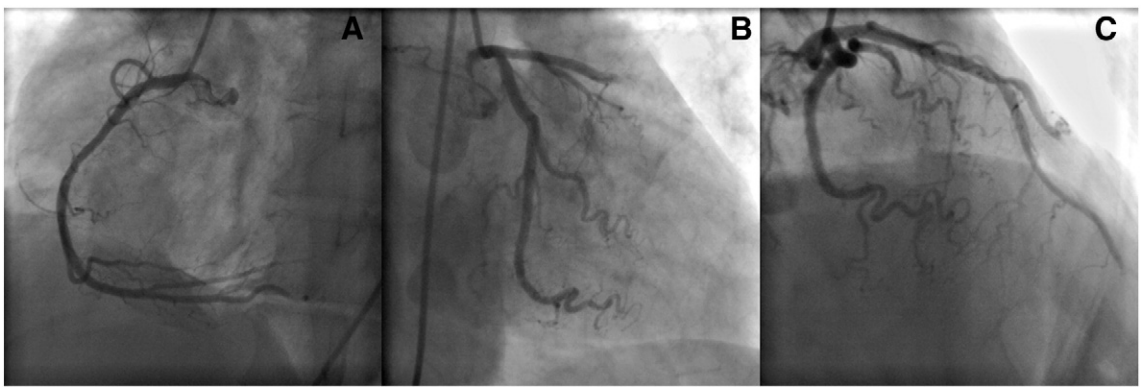

Fig. 2. Coronary angiography

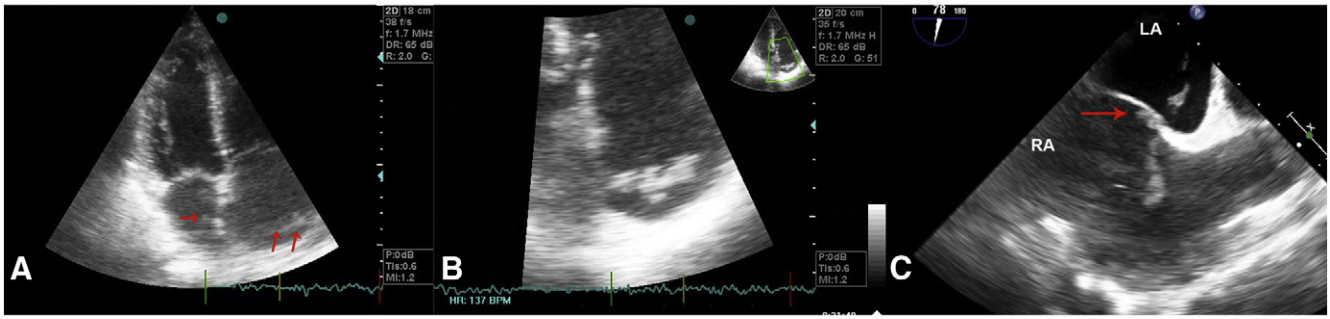

Fig. 3. Echocardiographic ımages

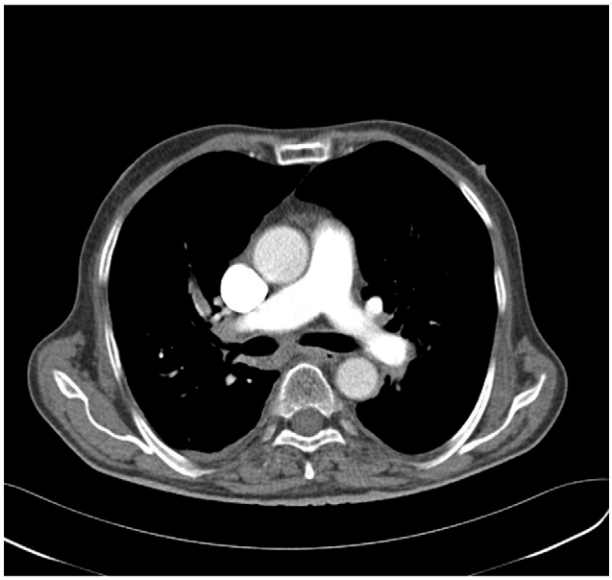

Fig. 4. Pulmonary computerized tomographic angiography

Pulmonary embolism is the most common cause of acutely elevated right atrial pressure and right to left in patients with PFO and atrial septal defect and occurs in at least 60% of cases with paradoxical embolism [9]. Data suggest that, in patients without preexisting pulmonary vascular disease, acute PE may result in paradoxical embolism if mean positive airway pressure increases to at least 30 mm Hg and there is a 35% to 40% pulmonary vascular obstruction and an intracardiac defect such as an atrial septal defect or PFO [10].

Our case illustrates a difficult clinical management related to suboptimal primary percutaneous coronary intervention, acute PE with a pulmonary severity index of 134, and metastatic lung cancer. Despite high pulmonary severity index in which fibrinolytic therapy is indicated, patient was hemodynamically stable and had a metastatic brain lesion, which is an absolute contraindication for fibrinolytic therapy [11-13].

Simultaneous acute anterior MI and massive PE is a rare fatal condition. It should be remembered that, when there are concomitant diseases such as malignancy, physiopathologic mechanisms may be different than expected. The second most important aspect in this case is to affirm the importance of bedside echocardiography in coronary care unit.

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