Atypical Electromechanical Dissociation in a Patient With Recurrent Pulmonary Embolism*

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A 47-year-old man experienced recurrent pulmonary embolism resistant to aggressive medical and surgical prophylaxis. Although paraneoplastic hypereosinophilia was suspected, no endoscopic or radiologic signs of malignancy were detected. Death was the result of electromechanical dissociation, which was attributed to right ventricular outflow obstruction. At autopsy, anaplastic lung carcinoma was found in the left basal segment with superimposed pulmonary infarction. A huge pedunculated thrombus was attached to the left ventricular apex and extended into the ascending aorta, obstructing the left ventricular outflow. To our knowledge, this is the first case of electromechanical dissociation due to left ventricular thrombus in a patient with pulmonary embolism. Radiologic and echocardiographic evaluation of such patients should take into account possible masking of the underlying neoplasm by embolic or hemorrhagic phenomena, or both, and the presence of left-sided cardiac thrombi, which may cause catastrophic events. (CHEST 1996; 109:562-63)

Key words: cardiac arrest; left ventricular thrombus; paraneoplastic syndromes; pulmonary embolism; pulmonary infarction

Electromechanical dissociation represents the most frequent mechanism of death in a patient with a massive pulmonary embolism. It is generally the result of acute right ventricular failure secondary to obstruction of the main pulmonary arteries. The patient reported herein had recurrent pulmonary embolism notwithstanding intensive medical and surgical prophylactic treatment and died from electromechanical dissociation independent of right ventricular outflow obstruction.

CASE REPORT

A 47-year-old man developed bilateral femoral and saphenous deep venous thrombosis without any evident predisposing condition. His family history did not consist of any thromboembolic phenomena; cigarette smoking (20/d) was the only cardiovascular risk factor. Treatment with subcutaneously administered heparin (5,000 U/d) and acetaminophen (Paracetamol) (400 mg/d) was initiated. Laboratory investigations did not demonstrate any deficiency in protein C, S, and antithrombin III; antiphospholipid antibodies and lupus anticoagulant were absent. Assessment of tumor markers showed a significant increase in CA-125 antigen (286 U/mL; normal range, 0 to 35 U/mL), whereas other markers (CA 19-9 antigen, carcinoembryonic antigen, tissue polypeptide antigen, α-fetoprotein) were within normal ranges.

Because of progressing thrombophlebitis, thromboticolytic treatment was initiated on the third day of admission (urokinase, 175,000 U/h) and was suspended on the eighth day because of intestinal bleeding. A regimen of heparin drip at 2,000 U/h was then begun. Notwithstanding such high dosage, activated partial thromboplastin time did not show any significant prolongation.

On the 20th day, the patient developed sudden dyspnea, tachycardia with hypoxia, and appearance of a right bundle-branch block evidenced on the ECG. Emergency cardiac catheterization revealed pulmonary hypertension (54/28 mm Hg; mean, 38 mm Hg); angiography showed massive bilateral pulmonary embolism and a floating thrombus in the inferior vena cava. A permanent Greenfield caval filter was inserted just below the renal veins. Gastroscopy, colonoscopy, and bronchoscopy were negative with regard to hemorrhagic and neoplastic lesions. The patient began receiving treatment with high-dose (4,000 U/d) heparin; the activated partial thromboplastin time reached but never passed the ratio of 2.5. A total body CT scan did not show any evidence of malignant neoplasms. In the basal segment of the left lung, a parenchymal density with pleural base and convex borders was noted; linear strands extended from the apex of the lesion toward the hilum, and low attenuation areas were present within the lesion. This lesion was interpreted as a pulmonary infarction. Five days later, the patient had a clinically evident recurrence of pulmonary embolism that was treated with front-loaded (15·50±35 mg) recombinant tissue plasminogen activator. Emergency pulmonary angiography revealed increased pulmonary hypertension (75/22 mm Hg; mean, 41 mm Hg) with worsened pulmonary obstruction and thrombosis of the inferior vena cava extending proximally beyond the filter. A temporary filter was placed just below the hepatic veins, and surgical removal of the Greenfield filter and ligation of the inferior vena cava were performed. Two days later, there was mild improvement in the pulmonary arterial obstruction on angiography, without evident thrombus in the vena cava. Improvement in clinical findings and in oximetry values was observed during the following week while the patient was receiving intravenously administered heparin and warfarin sodium.

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The first day after leaving the ICU the patient developed sudden electromechanical dissociation. A two-dimensional echocardiogram during resuscitative maneuvers showed a massive enlargement of the left ventricle with minimal systolic excursion. A huge echoluent mass was seen within the left ventricular cavity extending toward the outflow tract (Fig 1).

At autopsy, there were multiple thrombi in different stages of organization within the pulmonary arteries and multiple pulmonary infarctions. Histologic findings of a specimen taken from the infarcted left basal segment revealed the presence of anaplastic lung carcinoma with invasions of the lymphatics. At the opening of the heart, a huge (10 cm) pedunculated thrombus was found within the left ventricle (Fig 2). It was attached to the left ventricular apex and then extended through the left ventricular outflow tract into the ascending aorta which looked completely occluded by the thrombus. The foramen ovale was closed, and no myocardial metastaises were found. Coronary arteries presented extensive calcific atherosclerosis without evidence of acute thrombosis or plaque ulceration in the proximal segments.

**Discussion**

Thromboembolic events rank second only to infections as the cause of death in patients with solid tumors. The treatment of thromboembolic complications in patients with cancer is difficult. Such patients are resistant to anticoagulation, but their prognosis seems to be favorably affected by the placement of permanent caval filters. In our patient, however, the hypercoagulable state was so intense that even the most aggressive therapy failed. In most cancer patients, the hemostatic abnormalities are corrected by successful treatment of the underlying malignancy. In this patient, a paraneoplastic hypercoagulable state was highly suspected. Unfortunately, the lung carcinoma was localized in the left basal segment where a massive pulmonary infarction had occurred. The CT scan showed features consistent with pulmonary infarction, which masked the underlying neoplasm and hindered diagnosis.

Electromechanic dissociation was presumably the result of acute obstruction of the left ventricular outflow tract by the left ventricular thrombus. Left ventricular thrombi occur generally in three clinical conditions: acute myocardial infarction, left ventricular aneurysm, and dilated cardiomyopathy. Fatal left ventricular outflow tract obstruction was observed in a heart transplant patient after embolization from the left atrium. In this case, the thrombus originated presumably from the left ventricular apex, since no remnants of atrial thrombi, patent foramen ovale, or metastatic lesion were found within the heart. The presumed mechanism of thrombus formation might have been slow flow in the left ventricular apex due to hypoxia, concomitant coronary artery disease, and hypercoagulable state.

To our knowledge, this is the first case of electromechanical dissociation due to left ventricular outflow obstruction in a patient with pulmonary embolism. In patients with hypercoagulable states and concomitant pulmonary embolism, echocardiographic assessment should focus not only on the right side of the heart and the venae cavae but also should scan attentively the left heart chambers for possible presence of thrombi, which may cause catastrophic events.

**References**