

Relapsing Pulmonary Embolism in a Patient with a Spinal Cord-Injure

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Spinal cord injuries are associated with a high incidence of deep vein thrombosis (DVT), and, in consequence, associated also with a greater frequency of pulmonary emboli (PE), reported in the literature to occur in 70 to 100% of those cases¹. The extent of the pulmonary vascular obstruction is probably the most important determinant of the right ventricular dysfunction, which in turn leads to worse prognosis and higher risk of death and recurrence². These patients are therefore good candidates for thrombolytic therapy. After clinical suspicion of PE, if there are unequivocally visible thrombi and signs of right ventricular dysfunction at echocardiography, treatment, including thrombolysis, can be started³. We report the case of a paraplegic patient with unusual echocardiographic findings in whom two different therapeutic approaches for PE could be evaluated.

Case Report

A 52-year-old male had been paraplegic for 17 months due to spinal cord injury resulting from an automobile accident. He had undergone surgery for medullary decompression, without success 9 months prior. Approximately two months after surgery (February 97), he experienced sudden dyspnea and syncope, and was admitted to another hospital with the diagnosis of probable pulmonary embolism. Enzymatic evaluation was then performed and an increased LDH was observed. The electrocardiogram showed secondary abnormalities of ventricular repolarization in the anterior wall, probably due to right ventricular overload. These abnormalities were not present in his prior ECG performed six months earlier (fig. 1). Chest radiography revealed elevation of the left diaphragm with obliteration of the costophrenic angle due to pleural reaction. The transthoracic echocardiogram showed significant right ventricular and moderate right atrium enlargement, an important right ventricular dysfunction and dilatation of the inferior vena cava. The echocardiographic image suggested a large, multilobular, highly mobile right atrial thrombus with an apparent free pedicle, partially

protruding into the right ventricle during diastole (fig. 2). Doppler echocardiography disclosed a slight tricuspid reflux. The highest estimated systolic pressure at the pulmonary artery was 70mmHg. There was also a moderate left ventricular hypertrophy with slight ectasia of the descending aorta. The ejection fraction was 74%, and the shortening fraction was 36% (table I).

The patient had low blood pressure that regressed with the infusion of crystalloid fluids. Full heparinization was started, maintaining activated partial thromboplastin time (APTT) at 1.5 to 2.5 times the basal value. On the fifth day, oral anticoagulation treatment was started maintaining prothrombin time (PT) at 1.5 to 2.0 times the basal value. On the eighth day of treatment, echocardiography was repeated. The image of the thrombus could no longer be seen. The pulmonary flow showed a mesosystolic notch, suggesting pulmonary hypertension, which could not be estimated. A moderate right ventricular deficit was also

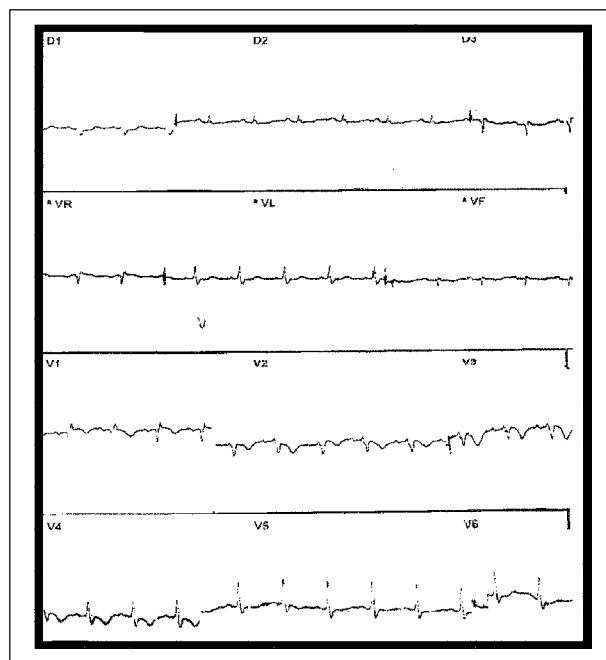


Fig. 1 – Electrocardiogram performed during the patient's first admission. NOTE: Q wave in D3 and aVF, S wave in D1 and aVL, inverted T wave in D3 and V1-V4, and low-voltage QRS complexes.

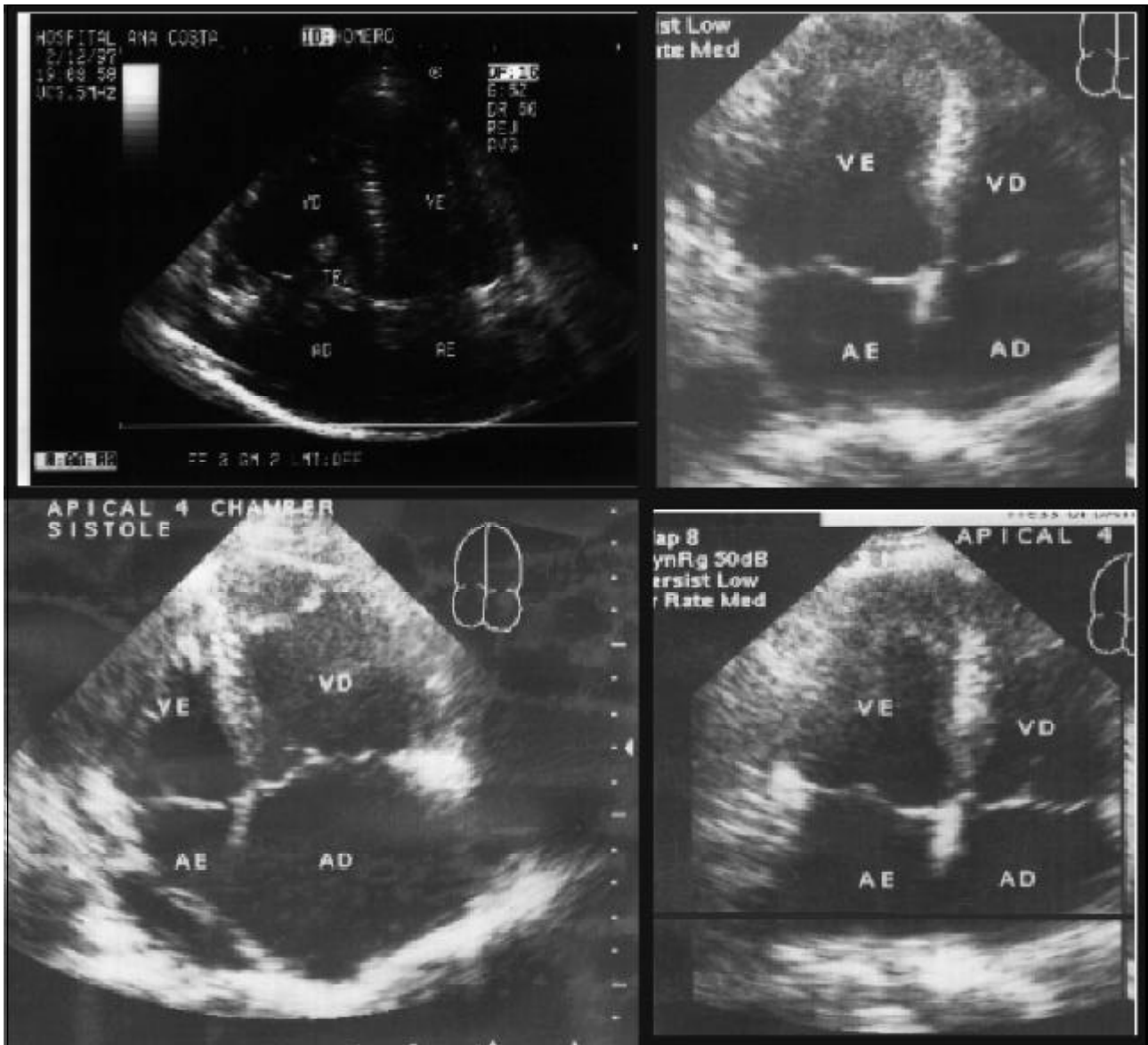


Fig. 2 – Echocardiogram performed at the patient's first admission (A), showing right atrial thrombus, and another normal two months later (B). Echocardiogram performed at the patient's second admission (C), showing the shift towards the left ventricle of the interventricular septum, and another two months later (D), normal.

present (table I). The patient was discharged, and the international normalization ratio (INR) was maintained at 2 to 2.5 times the basal value.

Two months after the onset of clinical findings (April 97), echocardiography was performed revealing only a slight left ventricular hypertrophy, normal contractile function in both ventricles, and no pulmonary hypertension nor thrombi (table I and fig. 2).

Six months after hospital admission (August 97), the oral anticoagulant treatment was suspended due to a new surgery at the lumbar level. Three weeks later, the patient presented again with chest pain, dyspnea, and signs and symptoms of low cardiac output. Electrocardiography showed the same pattern as before, and echocardiography revealed a thrombus in the main pulmonary artery with an estimated pulmonary hypertension of 75mmHg, and involvement of the right ventricle (table I and fig. 2).

Initially, streptokinase was administered at a dosage of 250,000 IU/h for 24 hours. It was then maintained at 100,000 IU/h for 24 hours. The oral anticoagulant treatment was restarted after thrombolytic infusion, which had an uneventful course. After seven days of hospitalization, the patient was discharged, and the INR was maintained at 2 to 2.5 times the basal value.

The echocardiography performed on the tenth day showed no thrombus, and the estimated pulmonary artery pressure was 37mmHg. One month later (October 97), the patient had mild right ventricular hypokinesia and no signs of pulmonary hypertension nor thrombi (table I and fig. 2).

Discussion

The majority of pulmonary emboli result from thrombi that originate in the deep veins of the upper and lower limbs

Table 1 – Patient's echocardiographic data during the acute phase of pulmonary thromboembolism and control after only anticoagulation or an association of thrombolysis and anticoagulation

Parameters	1	2	3	4	5	6
RV dd	36	36	22	30	21	22
LV dd	40	47	49	47	44	47
LV sd	26	29	27		29	32
Delta D	36	39	41		34	32
EF	74	77	72		64	60
PAP	70	--	normal	75	51	37

RV dd - right ventricle diastolic diameter; LV dd - left ventricle diastolic diameter; LV sd - left ventricle systolic diameter; Delta D - shortening fraction; EF - ejection fraction; PAP - pulmonary artery pressure.

and the pelvis. When a thrombus detaches from its original site, it moves through the venous system to the pulmonary circulation and most often obstructs a major branch of the pulmonary artery. The spinal cord lesion is responsible for a high occurrence of deep venous thrombi and, consequently, a higher frequency of pulmonary thromboembolism, with a reported incidence in 70% to 100% of cases¹.

The extension of the pulmonary vascular obstruction might be the main determining factor of the right ventricular dysfunction. The larger the obstruction, the higher the pulmonary artery pressure, the amount of vasoconstrictive substances released, and the hypoxemia. These in turn increase vascular resistance resulting in pulmonary hypertension. The sudden increase in pulmonary artery pressure causes increased afterload and tension in the right ventricular wall. These are followed by distension and dilatation of this chamber, leading to an interventricular septum shift towards the left ventricle, reducing chamber filling and, consequently cardiac output². These findings were documented by echocardiography and, after treatment, they disappeared on both occasions.

Pulmonary thromboembolism must be suspected in hypotensive patients when there is evidence of or predisposing factors to deep venous thrombosis and in the presence of signs of right ventricular failure. These signs include distension of the jugular veins, gallop rhythm, tachycardia, and tachypnea, especially when associated with electrocardiographic abnormalities. These electrocardiographic abnormalities include complete or incomplete block of the right branch, S wave in D1 and aVL > 1.5 mV, Qs in D3 and aVF but not in D2, QRS > 90° or undetermined, low voltage and inverted T wave in D3 and aVF or from V1 to V4³. In a series of 49 consecutive patients with pulmonary thromboembolism, at least three of these echocardiographic signs were present in 76% of the patients⁴. The electrocardiogram in this case showed QS in D3 and aVF, but not in D2, incomplete block of the right branch, and inverted T wave from V1 to V4.

In the patient here reported, besides the electrocardiographic abnormalities, dyspnea, arterial hypotension, and syncope were observed, suggesting a massive thromboem-

bolism, probably with an obstruction superior to 50% of the pulmonary arterial tree. Generally, the presence of dyspnea, syncope, and cyanosis reflects a more threatening episode of pulmonary embolism.

The echocardiographic findings of diagnostic usefulness include thrombus direct visualization (rare), right ventricle dilatation, anomalous septal movement, tricuspid valve regurgitation, pulmonary artery dilatation, and reduction of the inspiratory collapse of the inferior vena cava⁵. In the present case, all these echocardiographic signs could be observed, including the thrombus visualization at both hospital admissions. Patients with right ventricular failure due to pulmonary embolism, as in the present case, have a poorer prognosis and might be at a greater risk of relapse and death, when compared with those with normal right ventricular function⁶.

Even though perfusion studies and angiography were not performed, this might have been a case of massive pulmonary embolism, according to clinical and laboratory findings of right ventricular failure, low cardiac output, and a quick and positive hemodynamic answer to volume infusion without the necessity of vasoactive drugs. As these patients seem more predisposed to relapsing and fatal pulmonary embolisms, in spite of anticoagulation, the current practice is the primary treatment of pulmonary embolism with thrombolytics or mechanical intervention⁶. The primary therapy reduces the thrombus and the hemodynamic impact of embolism, preventing the continuous release of serotonin and other neurohumoral factors that aggravate the pulmonary hypertension. It may also dissolve the embolic sources located in the pelvis and in the deep veins, and when combined with anticoagulation helps in preventing relapse⁷.

There is a tendency to use thrombolysis in cases diagnosed using noninvasive techniques, without angiography, which reduces the incidence of bleeding complications, particularly hematomas at the catheterization sites. In the two services where this patient was hospitalized, ventilation and perfusion scans were not available. It would have been necessary to remove him to a radiological clinic in a mobile intensive care unit to perform these tests. In case pulmonary embolism is clinically suspected and if there is unequivocal visualization of the thrombus in the atrium, in the right ventricle, or inside the pulmonary artery, and also echocardiographic signs of right ventricle overload, then the treatment should be started. It should include thrombolysis or surgery, especially if other tests are not available or not advisable³. As there was no diagnostic doubt and the onset of appropriate therapy was imminent, full heparinization was chosen during the first occasion of treatment of this patient. Due to a prior extensive surgery, to the described hypothetical risk of thrombus fragmentation, and the occurrence of hemodynamic stabilization with only volume infusion, the classic treatment was chosen using no thrombolytic agent.

At the second admission, due to the smaller extension of the last surgery, as well as to the risk of cor pulmonale, the

thrombolytic agent was used, without performing ventilation and perfusion scans or angiography, for the reasons already explained.

Comparing the use of heparin alone to its association with thrombolysis, Goldhaber et al⁷, in a study of 101 patients, concluded that in the second group there was an improvement of right ventricular function, a decrease of the final diastolic diameter of the right ventricle and an improvement in pulmonary perfusion. All these parameters were statistically significant. The authors stressed that the subgroup of patients with right ventricular hypokinesia, due to the high risk of adverse events with the isolated use of heparin, seemed to be excellent candidates for thrombolytic therapy. Contrary to thrombolysis for acute myocardial

infarction which is limited to the first 12 hours, in pulmonary embolism the patients within 6-14 days of symptom onset benefited equally from this treatment.

Despite of the current recommendation of thrombolysis in cases of moderate or massive pulmonary embolism and considering the contraindications, such as cerebral vascular disease, recent surgery or trauma, we had the opportunity to evaluate two different therapeutic approaches for the same clinical presentation, in the same patient, on different occasions. Although the results were similar, it is not possible to compare the two treatments because our patient was an isolated case. In conclusion, with the available data and in the absence of contraindications, thrombolysis should be, whenever possible, the treatment of choice.

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