

Case Report

Role of Echocardiography in Suspected Acute Pulmonary Embolism in a Patient with Normal Hemodynamics: A Case Report with Literature Review

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ABSTRACT

Pulmonary embolism is a disease with high mortality if left untreated, but early diagnosis and treatment are compromised on account of its variable and non-specific presentation. Although ventilation/perfusion scan is the widely used diagnostic modality of choice, in our opinion echocardiography, being an easily available and

rapid bedside modality, can be used more frequently and effectively in the diagnosis and management of a suspected case of acute pulmonary embolism in a patient with normal hemodynamics as was evident in our case report.

KEYWORDS: acute pulmonary embolism, right ventricular dysfunction, thrombolysis

INTRODUCTION

Echocardiography can play a very important role in the management of a suspected case of acute pulmonary embolism (PE) in a subset of patients with strong clinical suspicion of acute pulmonary embolism but normal hemodynamics^[1-2].

Nearly 40% of patients with acute PE have normal blood pressure (BP) but some echocardiographic evidence of right ventricular (RV) dysfunction^[2]. The correct strategy of treatment in this subgroup of patients is an important but contentious issue^[3]. RV dysfunction in these patients may be a marker of a large thrombus load, incipient hemodynamic instability, or a poor outcome^[4-6]. Here we present a case of echocardiographic evidence of RV dysfunction with pulmonary hypertension which showed significant improvement following thrombolysis.

CASE REPORT

A 41-year-old gentleman with nephrotic syndrome was admitted to the ward for renal biopsy. Next day early morning he developed sudden chest discomfort coupled with dyspnea and mild dizziness. Physical examination revealed a mildly dyspneic gentleman with pedal edema and a prominent a-wave in the jugular venous pulse but without cyanosis. Blood pressure was 100/70 mmHg with mild tachypnea and tachycardia. There was no clinical evidence of deep vein thrombosis (DVT). Chest was clinically clear.

Cardiovascular examination showed sinus tachycardia, normal first heart sound but mildly accentuated pulmonary component of second heart sound. A soft systolic murmur I-II / VI was audible at tricuspid area, which was increasing with inspiration. Right ventricular (RV) atrial gallop was also audible. ECG showed sinus tachycardia but no evidence of right axis deviation or significant ST-T changes. Arterial blood gas (ABG) analysis showed mild hypoxia coupled with carbon dioxide (CO₂) wash out.

Bedside echocardiogram showed mildly dilated and hypokinetic RV with mild to moderate tricuspid regurgitation (TR) and a pulmonary artery pressure of 50 mm Hg.

On the basis of clinical presentation, ABG and echocardiographic findings a diagnosis of PE was made. Although decision to give thrombolytic therapy was already taken, on the echocardiographic evidence of RV hypokinesis and TR with pulmonary hypertension, since the facility of V/Q scans was immediately available in house, a V/Q scan was done which was reported later as highly suggestive of PE. Thrombolysis with 100 mg of rt-PA was started even before the result of V/Q scan was available.

Patient showed clinical improvement and ABG normalized. Follow-up echocardiogram later showed only mild TR with normal RV size and kinesis with pulmonary artery systolic pressure of 30 mm Hg. On discharge patient was totally asymptomatic and he was put on warfarin therapy.

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DISCUSSION

Recently, a number of studies have underscored the importance of echocardiographic evidence of RV in the management of patients with acute pulmonary embolism in a patient with normal hemodynamics^[1,7].

PE is a disease with high mortality if left untreated. Most deaths occur because of delay in establishing the diagnosis and starting appropriate therapy because clinical symptoms are variable and non-specific^[1].

Pulmonary angiogram is considered the gold standard in the diagnosis of PE but it is invasive and is often not available in smaller centers.

Ventilation/ perfusion scan has also been found to be quite useful in the diagnosis but this modality is also not so easy and urgently available in smaller centers to establish the diagnosis^[1].

The role of echocardiography is increasingly being recognized in the prompt diagnosis of PE^[1]. This is an easily available modality in most centers and can be arranged quickly at the bedside. Although it has its own limitations, in a proper clinical setting it can be a very useful tool for early diagnosis and prompt treatment.

Echocardiography is useful not only in establishing a prompt diagnosis but it can play an important role in deciding the mode of therapy as well^[8]. There is consensus regarding thrombolysis in PE in patients who are hemodynamically unstable particularly in the presence of systemic hypotension but still there is controversy regarding its use in those with echocardiographic evidence of RV dysfunction but no hypotension^[9].

Many authors recommend treating stable patients with thrombolytic therapy if there is echocardiographic evidence of RV dysfunction with a view to prevent further deterioration^[4,10,11].

Konstantinides *et al*^[10] reported a mortality of 4.1% in patients with stable hemodynamics and no echocardiographic evidence of RV dysfunction compared to 10% in patient with RV dysfunction at 30 days.

The 30-day mortality in the MAPPET registry was 4.7% in patients with echocardiographic evidence of RV dysfunction treated with thrombolytic therapy compared to 11.1% in heparin treated patients^[11].

Echocardiographic parameters of RV dysfunction used are:

- The presence of dilated RV more than 30 mm end-diastolic size, (RV/LV ratio > one, four chamber view)

- Septal fluttering or paradoxical septal motion

- The presence of pulmonary arterial hypertension by tricuspid regurgitation (jet > 2.8 m/sec)

- Regional hypokinesia with sparing of the RV apex (Mc Connell's sign)^[1]

CONCLUSION

Echocardiography can play a crucial role in the management of a subset of patients with strong clinical suspicion of acute PE but with normal hemodynamics^[1-2]. It can help us in deciding the modality of treatment that a particular patient should receive. If there is a strong clinical suspicion of PE but no hemodynamic compromise, echocardiography should be employed to identify the subset of patients showing echocardiographic evidence of RV dysfunction. Probably this subset will accrue benefit with thrombolysis, while the group showing no hemodynamic compromise and normal echocardiogram can be treated with intravenous heparin only.

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