

Echocardiographic findings in pulmonary embolism: An important guide for the management of the patient

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ABSTRACT

Echocardiography can be used as an easy, inexpensive, devoid of complications and, for the most part, universally available tool for the risk stratification of patient with acute pulmonary embolism. Because of its low sensibility, an echocardiogram is best indicated in a patient in whom hypotension and or shock are suspected to be due to pulmonary embolism. Pertinent findings in the echocardiogram can justify the use of fibrinolytic therapy. Most common echocardiographic findings in acute pulmonary embolism are: dilatation of the right ventricle, right ventricular dysfunction in some cases with preservation of the motility of the apex, dilatation of the inferior vena cava with lack of collapse during inspiration flattening of the interventricular septum suggesting right ventricular pressure overload and pulmonary hypertension based on the jet of tricuspid regurgitation if available.

Keywords: Pulmonary Embolism; Echocardiography; Venous Thromboembolism; Deep Vein Thrombosis

1. INTRODUCTION

Pulmonary embolism (PE) is a serious medical condition, potentially fatal, caused by partial or total obstruction of the pulmonary arteries and or its branches usually by blood clots but also air, tissue and fat. The most common cause is secondary embolization from thrombosis of veins usually originating from lower extremities or pelvis. Risk factors for thrombosis include endothelial injury, hypercoagulability of blood and stasis [1]. PE is an important cause of patient morbidity and mortality, with estimated 200,000 patient deaths per year in the US. The mortality reaches 30% for untreated PE; however with treatment the mortality rate decreases to 2.5% - 10% [2]. It is estimated that more than half of all PE are probably undiagnosed and frequently not discovered until autopsy [2].

Symptoms of PE include chest pain, hemoptysis, and dyspnea. Because the clinical presentation tends to be very nonspecific, diagnostic testing plays a crucial role in the diagnosis. Evaluation should include history taking, chest x-ray, electrocardiogram, D-dimer assay (high sensitivity but poor specificity), computed tomography (sensitivity 53% - 100%, specificity of 83% - 100%), ventilation-perfusion scan (high sensitivity but poor specificity). Although pulmonary angiography is the “gold” standard of reference for confirming or refuting a diagnosis of PE [3], spiral CT with contrast is the most widely used modality for diagnosis of pulmonary embolism.

ECG findings are usually nonspecific. In fact, 33% of patients with pulmonary embolism have a normal ECG. The most common abnormal findings are sinus tachycardia and nonspecific ST-segment and T-wave changes. Changes that strongly suggest pulmonary embolism indicate strain on the right side of the heart; manifesting as T-wave inversion in precordial leads V₁ through V₄, transient right bundle branch block, new right or left deviation of the QRS axis, sudden onset of atrial fibrillation or other atrial arrhythmia, and right atrial enlargement may be seen [4].

The S₁Q₃T₃ pattern (prominence of S wave in lead I, Q wave in lead III, and T-wave inversion in lead III) also suggests pulmonary embolism [4].

Echocardiography may play a significant role in making therapeutic decisions in patients with pulmonary embolism. This paper discusses the utility of using echocardiogram in diagnosing and guiding treatment in patients with pulmonary embolism.

Echocardiography is most appropriately recommended for patients with hypotension and or shock and suspected pulmonary embolism.

2. ECHOCARDIOGRAPHIC FINDING IN ACUTE PULMONARY EMBOLISM INCLUDE

Right ventricular enlargement, right ventricular dysfunction, in some cases with preservation of the apical con-

tractility (McConnell's sign). When the pulmonary embolism is large, more than 90% of patients have right ventricular hypokinesis [5].

Signs of pulmonary hypertension: flattening of the interventricular septum during systole, tricuspid regurgitant flow velocity higher than 2.7 m/sec.

In patients with acute PE there is marked prolongation of the isovolumic contraction time and isovolumic relaxation time over the tricuspid annulus. During chronic pulmonary hypertension the isovolumetric contraction and relaxation time of the LV is also increased, hence both RVMPI and LVMPI are increased. During acute pulmonary hypertension, because the LV has had no time to adapt, the LVMPI is not increased so there is a discrepant increase of the RVMPI over the LVMPI helping differentiate chronic pulmonary hypertension from acute pulmonary hypertension of PE [6,7].

Other signs: Short acceleration time of the pulmonary flow velocity in the presence of a tricuspid regurgitation gradient of less than 60 mmHg. RV to LV diameter ratio > 1 [8].

Thrombus seen within the right sided chambers [8].

However, echocardiography shows indirect evidence of pulmonary embolism in about 80% of patients with massive embolism (*i.e.*, $\geq 60\%$ perfusion defects) [9].

Echocardiography may directly visualize embolized thrombi (right heart chambers or central pulmonary arteries) or show right heart hemodynamic changes that indirectly suggest pulmonary embolism. For this a transesophageal echocardiogram holds a higher promise as a diagnostic tool [7,9].

3. ECHOCARDIOGRAM IN ESTIMATION OF PROGNOSIS IN PULMONARY EMBOLISM

The presence of right ventricular hypokinesis, among other factors, is a sign of adverse prognosis for the patient [10].

The finding of a patent foramen ovale in conjunction with pulmonary embolism indicates a relatively poor short-term prognosis and may encourage the use of more aggressive therapy for PE [10].

A study done by Stein *et al.* showed that the in hospital prognosis of patients with right ventricular enlargement following a pulmonary embolism is good in the absence of shock, a life threatening arrhythmia or ventilatory support [11].

4. USE OF ECHOCARDIOGRAPHY AS A TOOL FOR DIFFERENTIAL DIAGNOSIS

Echocardiography can help differentiate pulmonary embolism from other causes of hypotension: pneumothorax,

pericardial effusion, myocardial infarction and sepsis.

In conjunction with clinical assessment and the results of other noninvasive tests, echocardiography may enable PE to be diagnosed, or anticoagulants to be withheld in severely ill patients, at least until it becomes feasible to perform additional testing.

In addition, indirect parameters such as unexplained right ventricular dilatation/dysfunction and marked tricuspid regurgitation, which can be detected similarly by TTE and TEE, have a sensitivity of about 50% and a specificity of about 90% for pulmonary embolism.

5. TTE VS. TEE

With the transesophageal echocardiography it is possible to see the right-sided cardiac structures, the main pulmonary artery, a significant portion of the right pulmonary artery, and a portion of the left pulmonary artery. [12].

TTE visualizes intracardiac thrombi (usually right atrium) in about 5% of patients with acute pulmonary embolism and generally does not detect emboli in the pulmonary arteries. Central emboli can be seen by TEE in about 70% of the patients who have pulmonary embolism and right ventricular dysfunction. TEE can visualize thrombi in the central pulmonary arteries (main, right, proximal portion of left) with high specificity ($>90\%$), but its sensitivity has not been evaluated in unselected patients with pulmonary embolism (perhaps about 30%) [13-16].

Because of the limited specificity with the TTE, the invasiveness of the TEE and the low sensitivity with both approaches, echocardiography is not suitable as a routine diagnostic test for PE. Outside the ICU, as a stand-alone imaging technique for the diagnosis of PE, echocardiography should not be used. It is rather insensitive (reported range, 30% to 50%). Findings on echocardiography, even in patients with shock suspected to be due to a PE, are nonspecific. Although certain findings, such as free-floating thrombus or clot transiting through the right heart, are diagnostic for PE, these are rare findings. In the ICU, however, echocardiography is attractive since it is noninvasive and does not require the transport of an unstable patient. Moreover, 80% of patients with shock and PE show evidence of right-heart dysfunction on echocardiography. However, in the patient with unexplained shock, the differential diagnosis is broad and includes a number of processes that can cause both right ventricular dilation and dysfunction. Even if echocardiography is nondiagnostic for PE, it may still provide important information by helping to exclude other causes of shock, such as acute left ventricular dysfunction, tamponade, acute valvular disease, and aortic dissection, as noted above [14,15].

6. CURRENT THERAPIES FOR PE

Accurate diagnosis followed by effective therapy with anticoagulants decreases mortality. Conventional therapy for acute PE is the administration of intravenous heparin or Low Molecular Weight Heparin (LMWH). Heparin prevents clot formation and extension. Because the risk of death from pulmonary embolism is greatest in the first few hours of development of a clot and because diagnostic test results often are not available for 8 to 12 hours, heparin should be given to patients with a moderate to high clinical probability of pulmonary embolism or deep vein thrombosis until all diagnostic results are available. LMWH is preferred to unfractionated heparin. LMWH can be given subcutaneously once or twice a day, and laboratory monitoring may not be necessary. Long-term anticoagulation is continued after discharge, usually with warfarin.

Thrombolytic therapy should be considered for patients with deep vein thrombosis involving the iliofemoral system. Thrombolytic therapy accelerates the resolution rate of pulmonary occlusion and improves right ventricular function. These effects are seen within 24 hours, as opposed to two weeks with heparin or LMWH; therefore thrombolytic agents are used for patients with massive PE and hemodynamic instability or with echocardiographic findings of right ventricular failure. It is also useful for patients with massive pulmonary embolism who have significant pulmonary hypertension, obstruction of multiple segments of the pulmonary circulation, right ventricular dysfunction, or systemic hypotension [17].

Clot lysis may reduce the incidence of recurrent thrombi and postphlebotic syndrome and returns pulmonary arterial pressure to normal faster than heparin. Thrombolytic therapy also relieves strain on the right side of the heart faster than heparin does. However, thrombolytic therapy does not improve survival. Risks include hemorrhage, including an approximate 1% to 2% risk of intracranial bleeding. The hemodynamic response and the rate of bleeding are similar across age groups. Contraindications to thrombolytic therapy include eye or central nervous system surgery within the preceding 2 weeks, intracranial neoplasms or vascular abnormalities, stroke within the preceding 2 months, active bleeding, severe hypertension, and allergy to thrombolytic agents. Patient's age should not be a consideration when deciding on whether to use thrombolytics [16].

Interruption of the inferior vena cava, usually with a Greenfield filter, may be required in patients who have a contraindication to anticoagulation, have recurrent emboli while on anticoagulation, have pulmonary emboli from septic thrombophlebitis or those with massive embolization from a clot in the legs. Endarterectomy may be helpful in patients who have chronic pulmonary hy-

pertension due to a clot occluding the main pulmonary artery or a lobar pulmonary artery [16].

7. PREVENTION OF PE

Prophylaxis decreases the incidence of fatal pulmonary emboli by two thirds in hospitalized patients at risk of developing venous clots. LMWH (e.g., enoxaparin 40 mg sc once daily) is as effective and safe as prophylaxis with subcutaneous heparin (5000 IU sc bid or tid) and may reduce drug-induced adverse effects. Postoperative prophylaxis with LMWH (e.g., enoxaparin 30 mg sc q 12 h for up to 14 days) also dramatically reduces the incidence of venous thrombosis after knee or hip replacement. For total hip replacement, some investigators find that 4 to 6 weeks of LMWH postoperatively may be more effective [16].

Pulmonary embolism is believed to recur in 5 to 10% of patients despite ongoing heparin therapy. The likelihood of recurrent emboli is greatest in patients who have massive pulmonary embolization or for whom anticoagulant therapy is inadequate. If recurrence develops in the first few days of heparin or thrombolytic therapy, this treatment is usually continued. Recurrent pulmonary embolism leading to chronic pulmonary hypertension and cor pulmonale is uncommon [15].

8. CONCLUSION

Echocardiography, included in this term bedside transthoracic and transesophageal, is a very helpful tool for the clinician that faces acute ill patients in whom the diagnosis of pulmonary embolism is in doubt. By demonstrating signs of right ventricular strain and pulmonary hypertension, echocardiogram is able to help in the treatment of these patients. Echocardiography is also able to determine which patient is at highest risk of an adverse outcome by means of evaluating the right ventricular function.

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ABBREVIATION

PE: pulmonary embolism.

RV: right ventricle.

LV: left ventricle.

RVMPI: right ventricular myocardial performance index.

LVMPI: left ventricular myocardial performance index.

TEE: transesophageal echocardiography.

TTE: transthoracic echocardiography.

LMWH: low molecular weight heparin.

APPENDIX

