The use of transthoracic echocardiography for diagnosis of pulmonary embolism: a case report and review literature

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Abstract

Introduction: Pulmonary embolism (PE) is a common condition but if it is diagnosed with delay, it can cause serious morbidity or mortality. Herein we evaluated the role of transthoracic echocardiography (TTE) as a real-time, non-invasive, portable, and cost-efficient modality in diagnosis of PE in critically ill patient.

Case: We report a successful application of bedside TTE in a 55-years-old man presenting with dyspnea and unstable hemodynamic. Bedside echocardiogram showed a free-flouting thrombus at the main pulmonary artery bifurcation.

Discussion: Although TTE can be used for rapid risk stratification in patients with PE, it provides a means for rapid, reproducible, portable, and cost-efficient diagnostic modality in the setting of high clinical suspicion of PE. The sensitivity and specificity of TTE for diagnosis of PE ranged between 29% and 52% and between 96% and 87%, respectively. Echocardiographic signs used in the diagnosis of acute PE include reduced RV size and function, direct visualization of thrombus, reduced LV cavity size, dilated pulmonary arteries, interventricular septal flattening, paradoxical septal motion, tricuspid regurgitation, increased velocity of tricuspid regurgitation jet, and dilatation of IVC.

Conclusion: Although TTE had a low sensitivity for diagnosis of PE, this modality is non-invasive, cost-efficient, portable, and helps us to differentiate between the etiologies of hypotension in the unstable patients.

Key words: Pulmonary embolism, Transthoracic echocardiography, right ventricle dysfunction, thrombus

Introduction

Pulmonary embolism (PE) is a frequent cardiovascular emergency and still remains one of the leading causes of cardiovascular morbidity and mortality. The PE is responsible for 100,000 to 300,000 deaths per year in the United States[1-3], and it is frequently not diagnosed until after the patient dies.[4,5] Prognosis of PE strongly depends on hemodynamically status and echocardiographic findings. A short-term mortality in normotensive patients that do not have evidence of right ventricular dysfunction (RVD) is 2%, but the mortality rate rapidly increases up to 30% in patients with shock and up to 65% in patients with cardiac arrest at presentation.[6] In the other hand, delay in presentation and diagnosis is very common and it can lead to increase the morbidity and mortality. Thus, rapid diagnosis and treatment of possible causes of hypotension or shock in the critically ill patient that highly suspicious to PE are very important. Transthoracic echocardiography (TTE) may be invaluable as a real-time, non-invasive, and cost-efficient modality which allows serial bedside examinations. Herein, we reported a case of unstable hemodynamic PE that was diagnosed by TTE, and then reviewed the literatures about it.

Case

A 55-years-old man (174 cm, 95 kg) presented with an atypical chest pain and sudden onset dyspnoea from one day ago. Also, he was complaining of left leg pain from 3 days ago, but did not refer to any medical center. Initial physical examination revealed an ill-appearing man in significant respiratory distress using accessory muscles of respiration. Vital signs were: blood pressure 78/45 mm Hg, heart rate 124 beats/min, respiratory rate 32 breaths/min, SpO_2 82% on non-rebreather mask, and temperature 37 ^{0}C. The cardiac examination was regular tachycardic, lungs were clear, and left lower limb had pitting edema, compatible with deep vein thrombosis. After examination, a bedside TTE examination was performed. Bedside echocardiogram showed normal RV size and function, mild tricuspid regurgitation, and a free-flouting thrombus at the main pulmonary artery bifurcation (Fig. 1). Unfortunately, the multidetector computed tomography was not available at same time. So, base on clinical condition and TTE finding, we decided to use the thrombolytic agent (streptokinase) for the patient. Then, the patient was treated by full anticoagulant therapy. After one day, the patient significantly feel better, and physical examination revealed stable hemodynamic; blood pressure 112/78 mm Hg, heart rate 86 beats/min, respiratory rate 18 breaths/min, SpO_2 94%. After two weeks admission, he was discharged home in good condition.

Discussion

Relationship between delay in diagnosis and prognosis

Identification of patients with PE who are at risk for mortality or severe morbidity in the early observation period is important. Because the non-specific signs and symptoms, patients may experience significant delays from the onset of symptoms to presentation and diagnosis. So that

may majority of cases diagnosed after died. Pineda et al reported only a 45 % rate of correct antemortem diagnosis of PE.[7] In previous studies, 16% to 30.4% of the patients with acute PE were diagnosed one week or longer after the symptom onset; while only 31.6% to 50% of the patients presented within 24 hours of symptom onset.[8-11] Same as delay in diagnosis, misdiagnosis of PE is frequent and occurred in 50% of patients. Higher age, more days of delay and the absence of syncope or sudden onset dyspnea were factors associated with misdiagnosis.[12] Kline et al. found that in-hospital adverse outcomes including death, cardiogenic shock, or endotracheal intubation for respiratory distress in patients with delayed diagnosis were worse than those of patients with PE diagnosed in the emergency department. They showed that adverse events were occurred in 30% of the patients who have delayed diagnosis compared to 8.5% in the patients with early diagnosis.[13] Also, same as early diagnosis, early treatment is very important. Smith et al showed that patients who received heparin and achieved a therapeutic activated partial thromboplastin time within 24 h from presentation had lower in-hospital and 30-day mortality rates as compared with patients who achieved a therapeutic activated partial thromboplastin time after 24 h (1.5% vs. 5.6% and 5.6% vs. 14.8%, respectively).[14] Because, hours or even minutes are important, especially high risk patients, guidelines recommend initiation of anticoagulation if clinical suspicion for PE is high, even prior to confirmatory testing.[15-17]

Relationship between Hemodynamic status and prognosis

Hemodynamic status at the time of presentation in patient with acute PE has the strongest prognostic implications for short-term mortality.[18] Secondary hemodynamic destabilization, usually within the first 24h to 48 h, may occur in hemodynamically stable patients with PE, as a result of recurrent emboli or deterioration of right ventricular function. Also, early PE recurrences commonly occur in patients with undiagnosed or inadequately anticoagulant treatment[19], and may deteriorate the hemodynamic status.[20] In previous studies, the in-hospital mortality of normotensive patients with PE has been reported to vary from 3 to 15%;[21,22] while this rate in patients with massive PE, defined on the basis of systemic hypotension or cardiogenic shock rapidly raise to ranges from 25 to more than 50%.[18,23,24]

Diagnostic utility of TTE in patients with suspected PE

Despite TTE can be used for rapid and accurate risk stratification in patients with PE, it provides a means for rapid, reproducible, portable, and cost-efficient diagnostic modality in clinical situations where PE is suspected. It may be most useful in the setting of high clinical suspicion of PE.[25,26] In previous studies an increasing interest in the use of TTE in the diagnostic investigation of patients with suspected PE were seen.[27-29] In spite of these benefits, only 30% to 40% of patients with acute PE will demonstrate echocardiographic abnormalities.[30-32] In many centers, TTE is not routinely used as a diagnostic modality in patients with suspected PE; and transesophageal echocardiography is still preferable in certain specific clinical situations in which TTE is likely to fail or be suboptimal.[33] But, although transesophageal echocardiography was once the principal diagnostic approach using ultrasound to evaluate critically ill patients,

advances in ultrasound imaging, including harmonic imaging, digital acquisition, and contrast for endocardial enhancement, has improved the diagnostic yield of TTE.[34] However in massive PE, when patients are hemodynamically unstable with unexplained dyspnea, syncope, or right heart failure, that is a relatively common occurrence in critically ill patients, prompt TTE is useful within minutes at the bedside to justify the use of thrombolytic therapy.[25,34-37] The sensitivity and specificity of TTE for diagnosis of PE ranged between 29% and 52% and between 96% and 87%, respectively.[27] Also, Adding TTE to the diagnostic strategy for PE would avoid about 12% to 28% of lung-scan angiography procedures.[27]

TTE can be used to detect acute RV dilatation and dysfunction in massive PE. Regional RVD has a sensitivity of 77% and a specificity of 94% for the diagnosis of acute PE; positive predictive value is 71% and negative predictive value is 96%. The presence of regional RVD that spares the apex (McConnel sign) should raise the level of clinical suspicion for the diagnosis of acute PE.[34,38] In 1993, Goldhaber et al suggested a correlation between echocardiographic RVD and poor outcome in patients with acute PE.[39] Since then, several studies have evaluated the importance of echocardiographic RVD as a predictor of mortality. According to previous studies, the short-term (in-hospital) and long-term mortality are 2% to 14% and 9% to 17%, whereas in patients with RVD these percentages raise to 4% to 33% and 18% to 21%, respectively.[18,36,39-43] Even, RVD is associated with an increased risk of mortality in patients with hemodynamically stable PE.[44,45] In a study, the sensitivity and specificity of RVD for 30-day mortality in normotensive patients were 52.4% and 62.7%, respectively.[31]

It may also be possible to visualize the thrombus directly in the main pulmonary artery, although Transesophageal echocardiography is more useful in visualizing the thrombi lodged in main and right pulmonary artery or the right sided chambers.[46] Central PE are present in half of patients with symptoms of PE and acute pulmonary hypertension on TTE.[47] Other echocardiographic signs used in the diagnosis of acute PE include reduced reduced LV cavity size, dilated pulmonary arteries, interventricular septal flattening, paradoxical septal motion resulting in a "D-shaped" LV in short axis view, significant (moderate to severe) tricuspid regurgitation, increased velocity of tricuspid regurgitation jet, and dilatation of IVC.[35,36,38,46,48,49]

Despite these advantages, some limitations have restricted the use of TTE in diagnosis of PE. This method is highly dependent on operator skills. Thrombus rarely visualize in the pulmonary artery and left pulmonary artery distal to left main is not good visualized by TTE due to interposition of the left main bronchus. Also, while RVD may portend a poor prognosis in acute PE, these findings alone without a consistent clinical picture are not specific and may also be seen in COPD, obstructive sleep apnea, pulmonary hypertension, right sided myocardial infarction, cardiomyopathy, or valvular heart disease.[25,26]

Detecting right heart thrombi by TTE

Right heart thrombi can be diagnosed by TTE and are observed almost exclusively in patients with massive PE.[50,51] There is infrequent data available about thrombi in right heart in patients with PE. According to previous studies, the prevalence of thrombi in right heart among patients with PE is infrequent, approximately 4% to 18%.[48,52-56] Patients with right heart thrombi are

more hemodynamically unstable at the time of diagnosis than other patients. It may secondary to higher prevalence of hypotension, higher heart rate, and frequent RVD on echocardiography in patient with thrombi.[35,48] Patients with right heart thrombi had a worse short and long-term survival when compared to those without thrombi. In the ICOPER study, mortality rate in acute PE patients with free floating right heart thrombi was 21% compared with those without right heart thrombi 11%.[48] Prognosis is mostly depend on conventional clinical and hemodynamic status rather than the size and location of the thrombus.[57] Also, when thrombus is visualized, especially accomplished with other echocardiographic criteria, thrombolytic therapy is recommended.[34-36]

Conclusion

Accurate and prompt diagnosis of PE is crucial in the emergency department and the easiest and least invasive method of imaging cardiac structures is TTE. This modality is non-invasive, cost-effevtive, portable, and helps us to differentiate between the etiologies of hypotension in the unstable patients.

Conflict of Interest: None declared.

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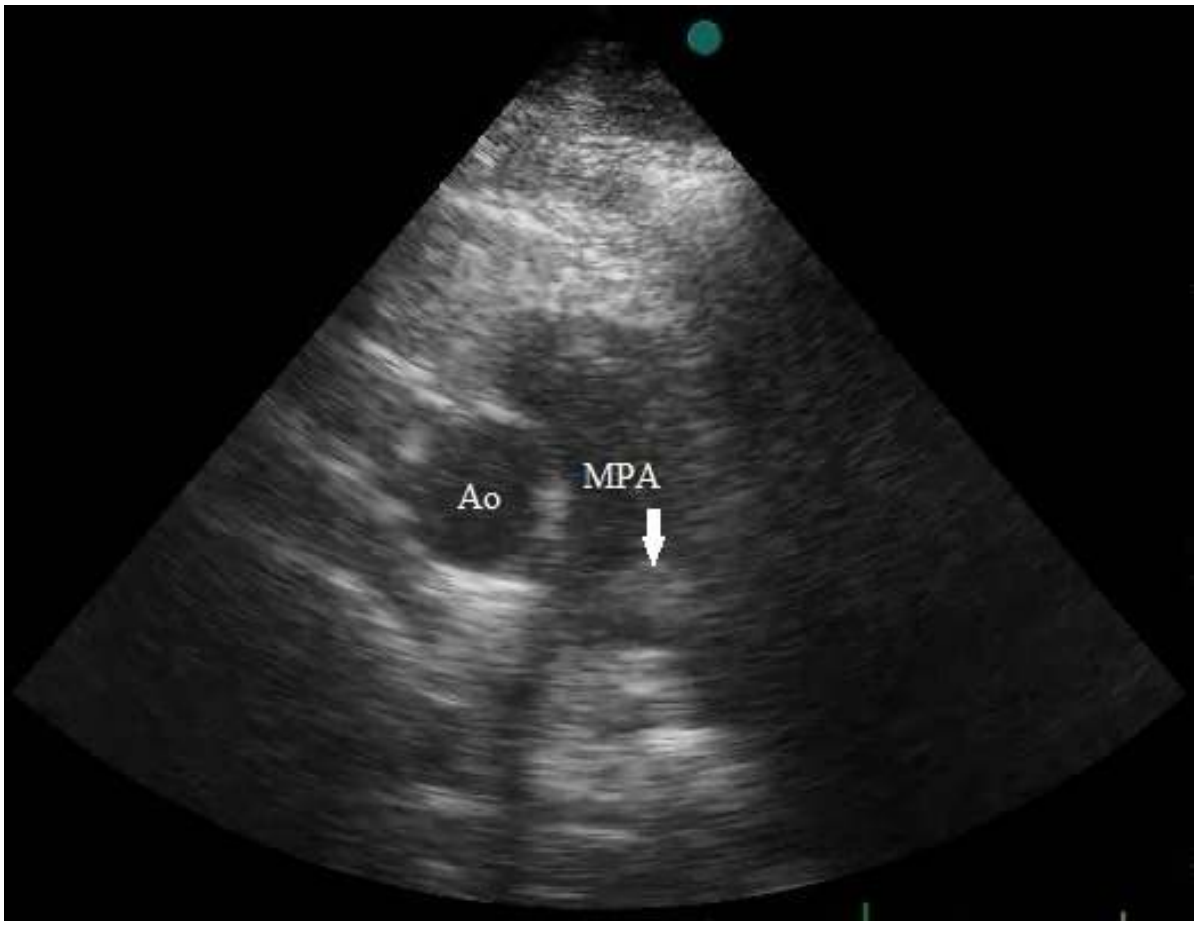

Figure 1: Parasternal short axis echocardiographic view: Arrow indicates a free-flouting thrombus at the pulmonary artery bifurcation. MPA: Main Pulmonary Artery; Ao: Aorta.