Case Report: Role of transesophageal echocardiography in a patient with an initially misdiagnosed acute pulmonary embolism [version 1; peer review: awaiting peer review]

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Abstract

Background: For the diagnosis of acute pulmonary embolism, chest computerized tomography (CT) angiography is considered as the gold standard. However, echocardiography could be useful especially in cases of high suspicion with normal CT scan or for patient with hemodynamic instability.

Case presentation: We present a case of a 47-year-old man admitted to the intensive care unit with a diagnosis of respiratory failure and hemodynamic instability 24 hours after closed fracture of the right leg. Before his admission, a thoracic CT angiography was done but did not show any sign of acute pulmonary embolism. During the first hours, he presented a bad evolution with a respiratory status which failed to respond to high dose of vasopressor, oxide nitric and ventilatory support. Therefore, due to the poor echocardiographic window, transesophageal echocardiography examination was done in emergency. It revealed high-probability diagnosis of massive pulmonary embolism based on right ventricular dysfunction and the presence of thrombus in the right pulmonary artery. Anticoagulant therapy (non-fractioned heparin) was administrated immediately achieving a favorable clinical outcome with rapid withdrawal of dobutamine, nitric oxide and norepinephrine.

Conclusions: This case illustrates the fundamental role of transesophageal echocardiography in a critically ill patient with shock due to high-probability pulmonary embolism. Echocardiography allows the initiation of adequate treatment without further delay.

Keywords

Acute pulmonary embolism, transesophageal echocardiography, transthoracic echocardiography, CT chest angiography
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Author roles: Daghmouri MA: Conceptualization, Funding Acquisition, Investigation, Project Administration, Resources, Supervision, Validation, Writing – Original Draft Preparation, Writing – Review & Editing; Oueslati M: Investigation, Validation, Writing – Original Draft Preparation; Tarhouni MA: Validation, Visualization; Faten O: Supervision, Validation; Zakhama S: Supervision, Validation; Rebai L: Writing – Review & Editing

Competing interests: No competing interests were disclosed.

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How to cite this article: Daghmouri MA, Oueslati M, Tarhouni MA et al. Case Report: Role of transesophageal echocardiography in a patient with an initially misdiagnosed acute pulmonary embolism [version 1; peer review: awaiting peer review] F1000Research 2022, 11:879 https://doi.org/10.12688/f1000research.74667.1

First published: 02 Aug 2022, 11:879 https://doi.org/10.12688/f1000research.74667.1
Introduction
Pulmonary embolism (PE) can be difficult to diagnose especially in critical ill patients who are hemodynamically unstable notably if the classic symptoms of PE are absent. However, many cases of PE are diagnosed in an emergency context. Echocardiography could be considered a useful technique at the bedside in critical care settings for the diagnosis of PE, especially as it is difficult to diagnose using other techniques. That is why, we present this case of massive pulmonary embolism diagnosed by the combined use of transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) due to the poor transthoracic window. TEE was useful in ruling out differential diagnoses and finding signs in favor of the diagnosis of PE, which allowed the initiation of adequate treatment without further delay. The aim of this case report was to highlight the pivotal role of TEE in the diagnosis of PE in a hemodynamically unstable patient especially when his mobilization to the radiology department was difficult to achieve.

Case presentation
A 47-year-old north-African man working as an engineer, with no relevant medical history, previous treatments or toxic habits (tobacco, alcohol) was admitted to hospital with a diagnosis of isolated closed fracture of his right leg due to a road accident (he was struck by a motor vehicle). At the time of admission, he was conscious, without any neurological alterations, or hemodynamic and respiratory disorders.

24 hours after admission, the patient suddenly presented a change in his level of consciousness (confusion with Glasgow comat scale of 12). He was tachypneic (30 breaths/min) with an oxygen saturation of 94% with a non-rebreather mask. Lung auscultation showed conserved vesicular murmur with bilateral basal crackles. He was tachycardic (heart rate 120 beats/min) and presented a hypotension (blood pressure was 80/40 mmHg). He was not febrile and did not present any cutaneous sign.

A 12-lead electrocardiogram showed only a sinusoidal tachycardia without other signs of acute coronary syndrome or right heart strain. The patient was immediately treated with crystalloid fluid infusion and bolus of epinephrine. After that, a brain scan was done which did not show signs of post traumatic abnormalities, in addition to thoracic CT angiography which did not show any sign of acute pulmonary embolism (Figure 1).

Therefore, he was transferred in emergency to the intensive care unit (ICU) and due to his bad evolution, he was intubated and required mechanical ventilation. Arterial acid-base balance at that time showed fraction of inspired oxygen 100%, pH 7.15, partial pressure of oxygen 86 mmHg, partial pressure of carbon dioxide 52 mmHg, bicarbonates 24 mmol/L, base excess -15, lactic acid 2.5 mmol/L and oxygen saturation 93%. Laboratory findings showed hemoglobin 10g/dl, leukocytes 6.10^3/mm^3, lymphopenia, creatinine 1.5 mg/dl, troponin T 34 µg/L, pro-BnP 400 pg/ml and procalcitonin < 0.05. His respiratory status failed to respond to high-dose of vasopressor and ventilatory

![Figure 1. Computerized tomography (CT) chest angiography (showing bilateral condensation with pleural effusion, without any sign of acute pulmonary embolism).](image-url)
support so nitric oxide was introduced in addition to continued infusion of cisatracurium. Chest radiography showed bilateral infiltrate (Figure 2).

In order to determine the real cause of this instability, TTE was performed however we obtained poor quality images, so it was necessary to do a TEE which was performed by an experiment anesthesiologist. TEE demonstrated a dilated and dysfunctional right ventricle (RV) with a hypertrophic dysfunctional left ventricle (LV). The right atrium (RA) was also severely dilated with a patent foramen oval and septum bowing (Figure 3). The RV end-diastolic diameter to LV end-diastolic diameter ratio was 1.2 suggesting RV pressure overload. RV dilatation led to functional tricuspid regurgitation as the tricuspid annulus enlarged. There was a pulmonary arterial hypertension with a pulmonary artery systolic pressure of 70–80 mmHg. Initially, there was no evidence of a thrombus either in the pulmonary arteries or on the right side of the heart. Due to global heart failure and the low-cardiac-output state, dobutamine was used with the doses of 3–5 µg/kg/min. However, after 24 hours, a control TEE showed an evident thrombus in the right pulmonary artery which was dilated (Figure 4). Massive pulmonary embolism was suspected but we could not confirm it by other complementary test because the unfavorable hemodynamic situation of the patient prevented his transfer. Anticoagulant therapy (non-fractioned heparin) was administrated immediately achieving a favorable clinical outcome with rapid withdrawal of dobutamine, nitric oxide and cisatracurium. No follow up information about the patient was available.

**Discussion**

This case highlights the crucial role of echocardiography in ICU for patients with severe shock due to massive pulmonary embolism associated to an unfavorable hemodynamic situation. In addition, like another similar case published in literature, it illustrates the value of TEE over TTE for those who have poor transthoracic window secondary to some clinical situation (supine position or mechanical ventilation). Pulmonary embolism can be difficult to diagnose particularly for patients in ICU who are sedated or on mechanical ventilation because key symptoms are absent (dyspnea, chest pain and syncope). For the diagnosis of PE, pulmonary angiography and spiral CT is the gold standard with a sensitivity of 83% and a specificity of 96% according to the PIOPED II trial. However, in our scenario, the CT angiography performed initially did not show any sign of acute pulmonary embolism despite the high probability of PE and this could be explained by the occurrence of artifacts or secondary migration of subsegmental thrombosis. So echocardiography was useful in order to rule out some differential diagnoses which caused this hemodynamic instability (tamponade, aortic dissection, hypovolemia...) according to the guidelines of European Society of Cardiology.

Vignon et al. showed that TEE helped in 98% of clinical decisions in a critical care population so it has higher impact on patient care than TTE which provided adequate images in only 38% of cases. Concerning the confirmation of PE, TEE has 70% sensitivity and 81% specificity. In the context of PE, TEE usually shows indirect signs like RV dilatation (RV end-diastolic diameter/LV end-diastolic diameter ratio > 0.9) and exclude other causes. In addition, serial assessment of RV size, determination of RV systolic pressure and inferior vena cava
assessment could be performed in patients with massive PE. Although, thrombus may be seen in some cases. According to Pruszczyszk et al., the central pulmonary arteries including the proximal lobar branches on both sides could be precisely visualized by biplane TEE. Only the proximal left pulmonary artery is difficult to assess because it is shielded by the left main bronchus. But a perimural artifact may be potentially misinterpreted as thrombus especially when it is present in the right pulmonary artery.

Significant hemodynamic instability is present in 8% of patient with acute pulmonary embolism. The main cause is acute right ventricular failure which increases mortality from 15% to 42%. That is why, TEE could be useful for analyzing response to medical interventions such as fluid and drug therapy. It could also be helpful for monitoring RV function and pulmonary artery systolic pressure especially if thrombolytics or anticoagulant were administrated.

**Conclusion**

We reported this case in order to show the fundamental role of TEE in ICU especially when the transthoracic window is poor. TEE allows the initiation of adequate treatment without further delay, by avoiding unnecessary mobilization of an unstable patient to perform CT chest angiography and can lead to a better clinical outcome. Although, TEE has some limitations like the cost of the equipment or the inability to place a probe (esophagectomy, esophageal diverticula or varices), complication rates from TEE use are fairly low at 0.2%. In addition, it has been demonstrated to have a steep learning curve and that physicians could successfully perform focused TEE assessments with a high retention rate after 6 weeks of 4-hour simulation workshop.

**Data availability**

All data underlying the results are available as part of the article and no additional source data are required.

**Consent**

Written informed consent for publication of their clinical details and clinical images was obtained from the patient.

**Acknowledgements**

An earlier version of this article can be found on Authorea (doi: 10.22541/au.161467284.43760460/v1).

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