

Transthoracic Echocardiography Assessment of Early Pulmonary Revascularization for a Sub-massive Pulmonary Embolism: A Case Report*

Évaluation par échocardiographie transthoracique d'une revascularisation pulmonaire précoce dans le cas d'une embolie pulmonaire submassive : étude de cas

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To the Editor,

A 28-year-old patient, with no medical history of pulmonary embolism (PE), was referred for syncope and chest pain with spontaneous resolution. His past medical history included a right bi-malleolar fracture surgically treated two months ago with antithrombotic prophylaxis [tinzaparin (Innohep®)]. His systolic blood pressure was 100 mmHg; no clinical signs of shock were present. However, jugular turgor was observed. Arterial blood gas analysis showed respiratory alkalosis with shunt effect (pH = 7.45, PaCO₂ = 23 mmHg, PaO₂ = 59 mmHg, HCO₃⁻ = 16 mmol/l, SaO₂ = 92%). Arterial lactate was 2.63 mmol/l and troponin 1.095 mg/l (N < 0.05), with B natriuretic peptide (BNP) at 14 ng/l (normal range < 100). Electrocardiogram (ECG) showed sinus tachycardia at 132/min with a complete right bundle branch block. Transthoracic echocardiography (TTE) revealed a significant dilatation of the right ventricle (RV) [RV diameter/left ventricle (LV) diameter ratio > 1] with severe impairment of RV systolic function [peak S-wave velocity = 8 cm/s, tricuspid annular plane systolic excursion (TAPSE) = 12 mm]. Tricuspid regurgitation (TR) could not be used to reliably assess systolic pulmonary artery pressures

(sPAP). No pulmonary regurgitation was observed to estimate the mean pulmonary artery pressure (PAPm). However, the triangular appearance, with meso-systolic notch of the pulmonary systolic ejection flow, and short pulmonary acceleration time (75 ms) were suggestive of pulmonary hypertension; and systolo-diastolic paradoxical septum motion (Fig. 1) confirmed RV volume and pressure overload associated with suprasystemic pulmonary pressures. The inferior vena cava (IVC) was dilated to 22 mm (RAP > 20 mmHg) with no IVC respiratory variation [(Max - Min)/Max × 100] of 9%. With short axis view, we showed a 1 cm occlusive thrombus with low mobility in front of the bifurcation of the pulmonary trunk (Fig. 1). Computed tomography (CT) scan confirmed the presence of bilateral proximal emboli with a RV/LV ratio greater than 1 (Fig. 2). Thrombolysis with rtPA was administered (10 mg over 10 min followed by a 2 h perfusion of 90 mg) after this confirmation. The clinical evolution was favorable without bleeding complications. A TTE was performed 12 h after the thrombolysis and showed an improvement of RV function. There remained a discrete flattening of the inter-ventricular septum in diastole. Systolic pulmonary pressures were sub-systemic, measured at 30 mmHg on TR flow. The right ventricular systolic function was almost normalized (wave S = 10.3 cm/s, TAPSE = 23 mm) and right cavities were not dilated (RV diameter/LV diameter < 0.6). Short axis view confirmed the regression of the thrombus (Fig. 1) as well as the CT scan findings (Fig. 2). The clinical course was uneventful without bleeding. The patient was discharged two weeks later under rivaroxaban.

Intermediate-risk or sub-massive PE is defined as an acute right ventricular dysfunction consecutive to a pulmonary arterial thrombosis without the sign of shock. The poor prognostic markers associated with this life-threatening condition are elevated troponin, and BNP and NT-proBNP levels [1,2]; RV/LV CT-scan ratio higher than one [3]; and the impairment of right ventricular systolic function as seen

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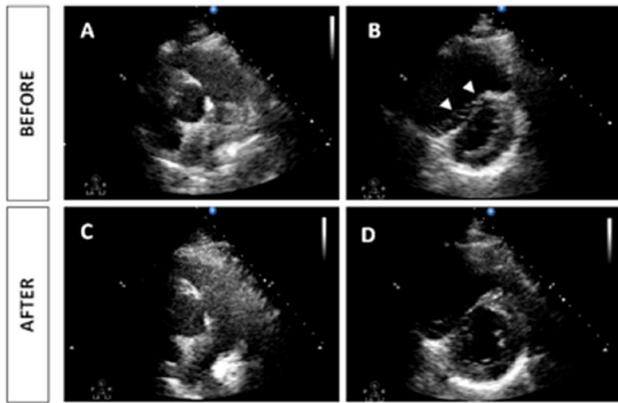


Fig. 1 Bedside transthoracic echocardiography in the ICU before and after thrombolysis (parasternal short axis view). A and C: Large and mobile thrombus next to the pulmonary artery bifurcation (A) with dramatic regression after thrombolysis (C). B and D: Systolo-diastolic paradoxical septum motion (white arrows) suggesting a volume and pressure overload of the right ventricle (B). Normalization of the interventricular septum motion after thrombolysis (D)

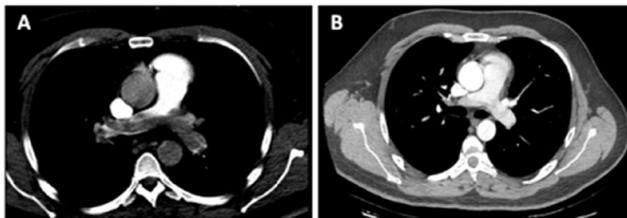


Fig. 2 Multi-detector computed tomography pulmonary angiogram revealing a proximal and bilateral artery pulmonary embolism (A) with a dramatic regression after thrombolysis (B)

on TTE [4]. High-risk PE care is well-codified. However, intermediate-risk PE treatment is still debated. Indications of early revascularization for sub-massive PE are even more controversial in view of increased bleeding risk [5] and lack of outcome benefit reported so far [6]. The Pulmonary Embolism Thrombolysis (PEITHO) study compared thrombolysis associated with heparin versus heparin alone in intermediate PE defined by the association of RV dysfunction and myocardial injury. In this study, thrombolysis prevents hemodynamic decompensation but increases the risk of major hemorrhage and stroke without significant effect on mortality [7]. In contrast, Kline et al. find less adverse outcome in the group treated with thrombolysis [8] in a study of 88 normotensive patients treated for PE associated with RV strain (by echocardiography or biomarkers). The latest recommendations of the European Society of Cardiology (ESC) suggest the use of thrombolysis case by case, depending on the risk/benefit ratio [9]. The main difficulty remains

to balance the risk of bleeding with the potentially lethal risk of PE. However, reports of low risk of bleeding after thrombolysis are encouraging, particularly in case of difficult decision-making [6,10].

In this case report, we endorsed the indication of fibrinolysis because of the severe echocardiographic condition including an occlusive thrombus in the pulmonary trunk and a severe impairment of the RV function with high risk of LV off-loading. In PE with circulatory failure (systolic blood pressure < 90 mmHg) requiring inotropic support, the presence of metabolic acidosis is associated with a higher risk of mortality [11]. In our case, even if we never required inotropic support, we observed metabolic acidosis with hyperlactatemia, sign of an intermediate PE with high risk of decompensation. As recommended by the ESC, we decided to use rtPA to faster hemodynamic improvement after its administration (2 h) [8,9]. We administrated 0.6 mg/kg of rtPA in a short infusion (over 15 min). Bedside TTE is interesting that it decides thrombolysis in sub-massive PE. We can define RV strain and even see the thrombus in some cases [12]. The ESC has highlighted the relationship between the presence of right heart thrombi in a patient with PE and the increased mortality [9].

While the ESC advocated that systematic use of echocardiography is not recommended in non-high-risk PE (III C) [9], we suggested that bedside TTE is an easy-access imaging modality that could provide reliable evidence for the decision of thrombolysis in sub-massive PE.

Link of interest: J. Carvelli, A. Theron, J. Tobarias, V. Ho, G. Perrin and M. Gainnier have no link of interest to declare.

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