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A Saddle Pulmonary Embolism at Transthoracic Echocardiography: A Case Report

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Abstract **Case Report**

Acute Pulmonary embolism (PE) is a life-threatening disease and is considered the third major cause of cardiovascular death with higher incidence and mortality rates. Identifying a saddle PE between the left and the right pulmonary trunk in transthoracic echocardiography (TTE) is rare, and helps to make a rapid diagnosis to avoid complications. Clinical presentation, electrocardiogram, and X-ray guide the diagnosis but are not specific. TTE is helpful and confirms the diagnosis especially when the CT is not available or in other situations when it is not realizable. Visualizing a pulmonary artery thrombus in TTE is unusual. It can be identified in the right cardiac chambers in less than 5% of patients with PE. The severity of acute PE is determined by its hemodynamics, the sudden pulmonary hypertension, and the development of obstructive shock. Anticoagulation therapy is the cornerstone of PE management and should be initiated as soon as possible.

Keywords: Pulmonary embolism, pulmonary artery thrombus, saddle pulmonary embolism.

INTRODUCTION

Pulmonary embolism (PE) is a common acute cardiovascular disorder and can result in acute right ventricular (RV) failure. It is a life-threatening condition due to the pulmonary bed obstruction.

Saddle PE is the presence of a straddling thrombus in the bifurcation of the main pulmonary artery trunk. Identifying a thrombus in the pulmonary artery is rare, and helps to make a rapid diagnosis to avoid complications. In this case, we report a saddle pulmonary thrombus in TTE, which is an uncommon finding.

CASE REPORT

A 34-year-old female patient with only a history of hormonal contraception was admitted to the Emergency Room for acute dyspnea Class IV of New York Heart Association, associated with orthopnea. Physical examination showed a heart rate of 125 beats per minute (bpm), blood pressure of 110/66 mm Hg, and oxygen saturation of 94% without any hypoperfusion signs. Cardiovascular examination did not reveal any right or left heart failure signs or any additional heart murmur.

The electrocardiogram (EKG) showed sinus tachycardia at 125 bpm, and ST depression in the precordial leads. D-dimer was significantly positive. Because of the unavailability of CT-pulmonary angiography (CT-PA), transthoracic echocardiography (TTE) was realized and showed a dilated right ventricle, with preserved function and segmental kinetic (Figure 1). The pulmonary artery (PA) was dilated with the presence of a saddle thrombus in the PA bifurcation trunk, (Figures 2 and 3). The systolic PA pressure was at 65mmhg. The left ventricle had a preserved function and a normal cardiac output.

CT-PA confirmed the diagnosis. Risk stratification of acute PE was intermediate-low. Auto-immune and Assessment of the thrombophilia, blood clotting tests blood tests, and lower limb echocardiography were without anomalies.

Anticoagulation was started immediately using low-molecular-weight heparin. The hormonal contraception was stopped and switched to a mechanical contraception.

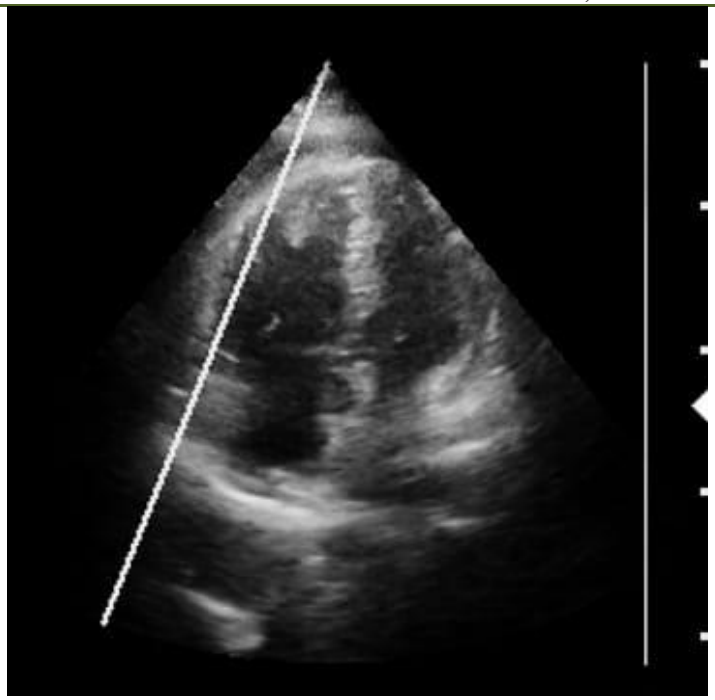

Figure 1: Apical 4 cardiac chamber TTE view showing an enlarged right cardiac chamber

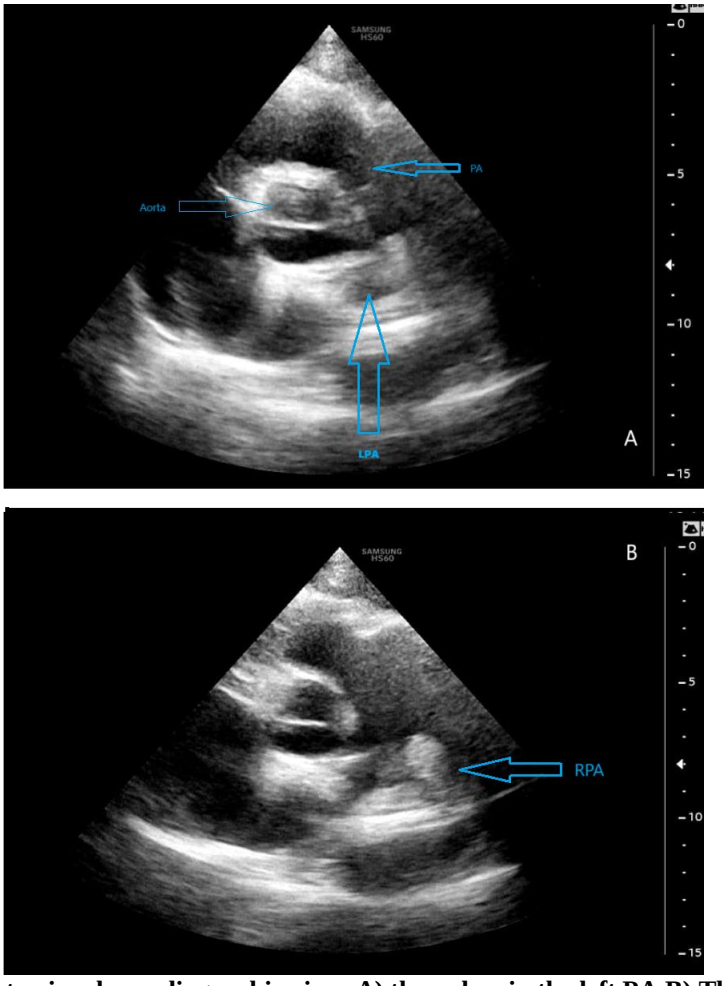

Figure 2: Parasternal short axis echocardiographic view. A) thrombus in the left PA B) Thrombus in the right PA. PA: pulmonary artery, LPA: Left pulmonary artery, RPA: Right pulmonary artery

Figure 3: Parasternal short axis echocardiographic view, showing a straddling thrombus in the bifurcation of the main pulmonary artery trunk

DISCUSSION

Acute PE is a life-threatening disease and is considered the third major cause of cardiovascular death with higher incidence and mortality rates [1]. It is estimated that more than half of all PE are probably undiagnosed and frequently not discovered until autopsy and among them 29% of saddle PE. Identifying a saddle PE between the left and the right pulmonary trunk is unusual and had an estimated prevalence between 2.6 and 5.2% in all patients with PE with a mortality risk at 2 weeks at 5.8% [2].

PE can present with varying degrees of severity; its hemodynamic alteration had a relation with the RV overload and dysfunction in the development of additional changes which results in low cardiac output. These consequences result in a vicious cycle, that must be broken rapidly.

Clinical symptoms can be various and nonspecific, including chest pain, hemoptysis, palpitations, and dyspnea. It is important to search for the thromboembolic factors. The clinical examination helps to determine the pretest probability, and hypoperfusion signs and to stratify the prognosis. Evaluation should include also chest X-ray and D-dimer assay. EKG can be normal, and its findings are usually nonspecific. The common findings are sinus tachycardia, inversion of T waves in precordial leads V1 to V4, Right bundle branch block, new right axis deviation, sudden onset of atrial fibrillation or atrial arrhythmia, and the S1Q3 pattern.

PE is usually determined by a pulmonary CT-PA. TTE is helpful and confirms the diagnosis especially when the CT is not available or in other situations when it is not realizable like pregnancy, or in critical patients who can't be transported.

TTE is useful in guiding the management by evaluating hemodynamics, prognosis, and risk stratification. It is a highly specific exam but lacks sensitivity and excludes other differential diagnoses. It provides also an assessment of RV and LV size, systolic function, regional wall motion, and valvular abnormalities [4].

Visualizing a thrombus in the pulmonary artery is rare. It can be identified in the right cardiac chambers in less than 5% of patients with acute PE. However, in transesophageal echocardiography (TEE), central thrombus can be seen in 70% of patients. TEE is helpful in critical patients in the intensive care unit who have poor transthoracic windows, with a higher sensibility [5].

The optimized view to detect a thrombus in the pulmonary trunk, and to assess the pulmonary valve and the proximal and distal RV outflow tract segments is the parasternal short-axis view. Its detection can be challenging due to poor acoustic view [6].

Indirect signs in TTE of increased pressure on the right cardiac chambers, an interventricular septum flattening, and the short acceleration time of the pulmonary flow velocity help the diagnosis. If these findings are absent PE is uncertain.

RV overload signs are frequent in saddle PE. It is associated with mild-to-moderate RV enlargement in 90% and mild-to-severe one in 80%. The presence of an RV dysfunction is related to an increased relative risk of death [7]. The severity of acute PE is determined by its hemodynamics, the sudden pulmonary hypertension, and the development of obstructive shock.

The presence of a Saddle PE doesn't influence the management. Anticoagulation therapy is the cornerstone of PE management and should be initiated as soon as possible in all patients, especially with a high pre-test probability. Fibrinolytic treatment is indicated in patients with hemodynamic instability. Invasive or surgical procedures can be proposed in other situations.

CONCLUSION

Saddle PE is a rare type of acute PE that can lead to sudden hemodynamic collapse and death. Identifying a thrombus in PA at TTE is very rare, and it allows to start treatment rapidly.

This case illustrates the importance of TTE in PE when CT-PA is not available, it highlights an unusual TTE finding of a patient with a pulmonary embolism.

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