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Importance of the Study of the Right Heart Chambers in Symptomatic Acute Pulmonary Embolism

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ABSTRACT

We present the case of a 42-year-old woman on oral contraceptives that presented to the emergency department with pain and swelling in the left lower limb. Diagnosis of extensive deep vein thrombosis was established. A few minutes later, she exhibited signs of shock and hemodynamic instability, thus raising suspicion of high-risk acute pulmonary thromboembolism. Prior to the administration of fibrinolytic treatment, a bedside transthoracic echocardiography was performed that excluded right ventricular dilatation. Then, the study was complemented with a thoraco-abdominal computed tomography scan that demonstrated a large retroperitoneal hematoma as the cause of the shock. In conclusion, a transthoracic echocardiography should be performed before initiating thrombolytic therapy in hemodynamically instable patients with strong suspicion of high-risk pulmonary embolism.

1 | Introduction

Thrombosis is the most common underlying pathology of the three major cardiovascular disorders: ischemic heart disease, stroke and venous thromboembolic disease (VTE), with pulmonary embolism (PE) being the most severe clinical presentation of VTE [1]. Hemodynamic instability is the main prognostic variable in patients with PE, in which case mortality may exceed 15%, as a consequence of right ventricular failure due to acute pressure overload [2].

Early diagnosis and treatment are crucial in high-risk PE. In this setting, assessing right ventricular (RV) function by a transthoracic echocardiography (TTE) is essential. Assuming that hemodynamic stability is due to the presence of a PE alone entails a risk that may have fatal consequences.

We present a case of deep vein thrombosis and acute PE with shock in a patient with normal RV function. If it had not been taken into account that RV function was normal and thrombolytic therapy had been initiated to manage hemodynamic instability, the patient would most likely have died.

2 | Case Report

We present the case of a 42-year-old woman on oral contraceptives that presented to the emergency department with acute pain in

the left inguinal region and swelling of the ipsilateral lower limb. In the previous days, the patient had had pain in the left lumbar region that radiated to the ipsilateral buttock, with no history of previous trauma. On admission, the patient was afebrile (36.7°C), HR was 88 bpm; BP was 120/75 mmHg; and O_2Sat was 96%. ECG demonstrated sinus rhythm at 92 bpm, without any relevant findings. An echo-Doppler confirmed the presence of deep vein thrombosis at the level of the left iliofemoral vein. A few minutes later, the patient complained of dizziness and pain in the left iliac fossa. She was pale, sweaty, hypotensive (65/40 mmHg) and tachycardic (145 bpm). On cardiac auscultation, heart sounds were rhythmic without murmurs. Breath sounds were normal on pulmonary auscultation. Palpation of the left iliac fossa was painful with guarding. The left lower limb was edematous and swollen, with cyanotic coloration and weak distal pulses. In the blood test, D-dimer was 14,684 ng/mL and NT-pro-BNP was 153 pg/mL (troponin determination was not requested). A decrease in haemoglobin (from 12.6 to 7.9 g/dL) was observed. The arterial blood gas test (with FiO_2 of 0.5) showed pH 7.18; pCO_2 37.7 mmHg; pO_2 185.7 mmHg; HCO_3 13.9 mmol/L; and lactate 3.38 mmol/L. The Geneva scale score was 12 (high probability for PE). A thoraco-abdominal computed tomography (CT) scan confirmed the diagnosis of bilateral PE (Figure 1A), extensive left ileo-femoral deep vein thrombosis (Figure 1B) and a large retroperitoneal hematoma (Figure 1C). Due to the presence of the hematoma, we decided not to administer anticoagulation. A blood transfusion was performed. Then, an arteriography of the right lumbar arteries was performed for any evidence of vascular lesion or active bleeding susceptible to embolization. Finally, an iliocavography was performed to locate the renal veins prior to the insertion of a temporary vena cava filter through the internal jugular vein (Option Elite) (Figure 1D). A bedside TTE impressed hypercontractility but excluded right ventricular dilatation. Biventricular function was preserved.

Anticoagulation with low molecular weight heparin was only started once the patient had been stabilised, both from a

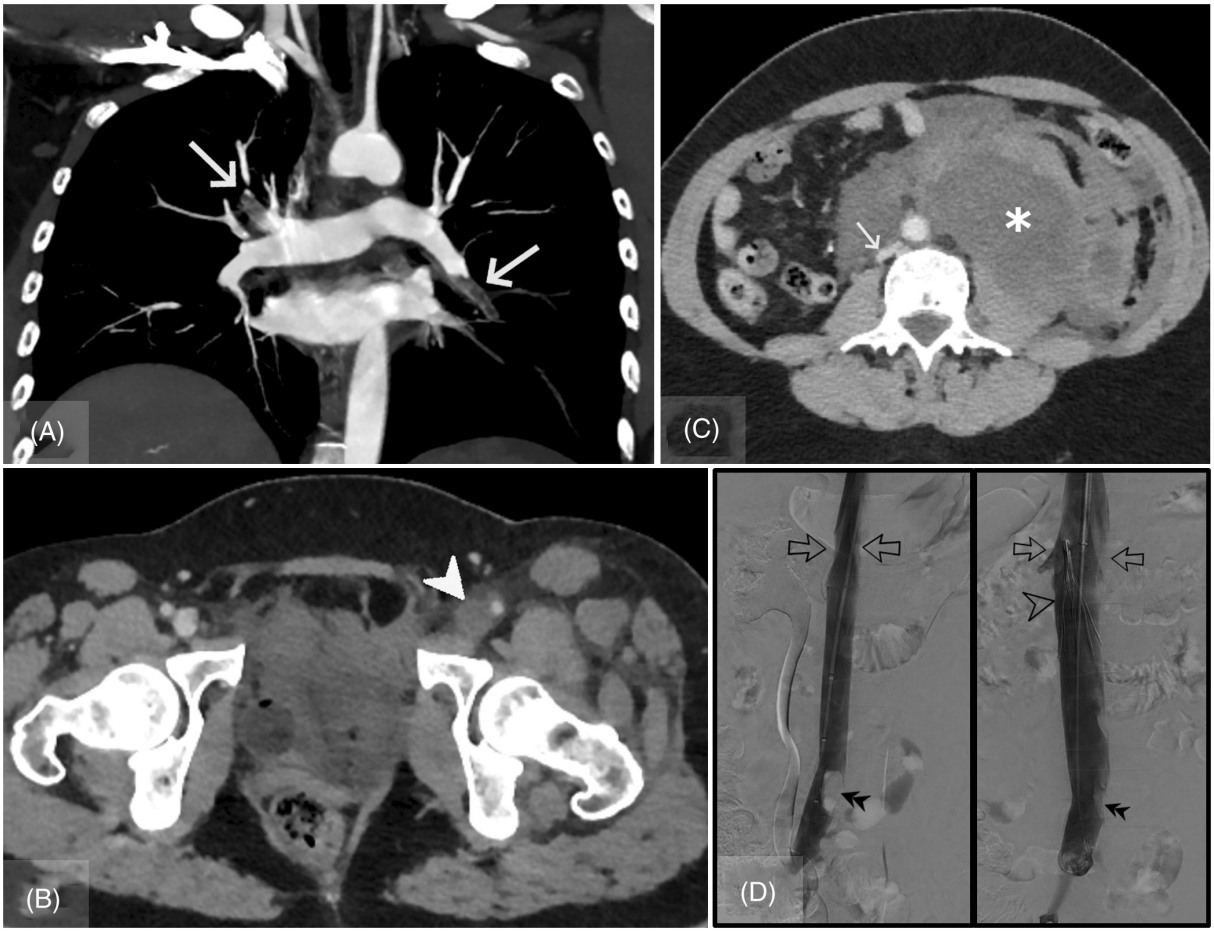

FIGURE 1 | (A) Coronal reconstruction of pulmonary angio-CT. Repletion defects in left inferior lobar and right superior lobar arteries (arrows), with extension to all its segmental branches. It also had involvement of several segmental branches of the right lower lobe (not visible in this projection). (B) Axial abdominal-pelvic CT image in venous phase. Deep venous thrombosis in left common femoral vein (arrowhead). The vein is visualised with increased calibre, with the lumen completely occupied and with absence of contrast filling. Edema of the interfascial planes at the level of the left thigh root (see difference with the contralateral side). (C) Axial abdomino-pelvic CT image in venous phase. Extensive left retroperitoneal hematoma (*), with no active bleeding point identified, extending towards the pelvis and exceeding the midline towards the right side. Collapse of the inferior vena cava (small arrow) in probable relation to hypovolemic shock. (D) Digital angiography image with subtraction. Iliocavography from jugular access, prior to placement of the temporary filter (left) and prior to its removal (right). Patent inferior vena cava. The contrast washout of the renal vein flow is located (hollow arrows) for filter placement immediately below them (hollow arrow head). Floating thrombus is seen protruding from the origin of the left common iliac vein into the lumen of the inferior vena cava (double arrowhead). Patent right common iliac vein.

respiratory and hemodynamic point of view, haemoglobin values had been restored to normal and reduction of the retroperitoneal hematoma had been confirmed. After a few days, the temporary vena cava filter was removed. The patient was discharged 20 days after admission. Final diagnosis was iliac-femoral deep vein thrombosis of the left lower limb, hemorrhagic shock secondary to retroperitoneal hematoma, and bilateral acute segmental PE.

3 | Discussion

Due to RV geometry, RV function and size cannot be assessed in a rapid and reliable way by a single echocardiographic parameter. In patients with hemodynamic deterioration and suspected PE, the unequivocal signs of RV pressure overload are, in addition to RV dilatation or dysfunction and an elevation of the estimated systolic blood pressure, akinesia of the medial right ventricular free wall and apical hypercontractility, the so-called McConnel's sign [3]. These findings, together with a high clinical probability of PE and the absence of other obvious causes of RV pressure overload, justify emergency thrombolytic therapy for PE [4]. In contrast, isolated evidence of RV dysfunction on TTE is not sufficient to initiate thrombolytic therapy (it can be found in 25% of patients with PE) [5]. For thrombolytic therapy to be initiated, there must be a high clinical suspicion of PE (as in this Case), and other obvious causes of RV overload must have been excluded. The echocardiographic parameters most frequently associated with an unfavourable prognosis include a RV/LV diameter ratio ≥ 1.0 and a TAPSE < 16 mm [6]. However, these are not exclusive to acute PE, but they can also be observed in other cardiac or respiratory diseases (pericardial tamponade, acute valvular dysfunction, severe general or regional LV dysfunction, aortic dissection or hypovolemia, among others) [4]. Patients with these conditions may present with RV overload/dysfunction in the absence of acute PE. In the Case reported, none of these echocardiographic signs were observed. However, the absence of echocardiographic data of RV overload/dysfunction does not exclude PE, as this study has a negative predictive value of 40%–50% [7]. What is important is that, in the presence of suspected high-risk PE, the absence of echocardiographic signs of right ventricular overload/dysfunction virtually excludes it as the cause of hemodynamic instability [2]. For this reason, systemic fibrinolysis was not administered, which would have been fatal, given the presence of abdominal bleeding with hypovolemic shock.

In summary, hemodynamic instability cannot be assessed by the presence of arterial hypotension or cardiogenic shock but, as recommended by clinical practice guidelines, should be based on demonstrating RV dysfunction by TTE. In this case, deep vein thrombosis, PE, and arterial hypotension were confirmed and could have justified initiating thrombolytic therapy. Failure to do so, based on not finding signs of right ventricular overload/dysfunction on TTE, ruled out PE as the cause of hemodynamic instability and helped save the life of this patient. A TTE should be performed in patients with hemodynamic stability and strong suspicion of high-risk PE before initiating thrombolytic therapy.

Ethics Statement

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable—no new data generated, or the article describes entirely theoretical research.

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