Pulmonary Thromboembolism – A Clinical Report A Combination of Different Therapeutic Strategies

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1. Abstract

1.1. Background: Pulmonary embolism (PE) is common and can be fatal. For rapid diagnosis and adequate treatment, it is essential to have a high index of suspicion, essential elements for a favorable outcome and low mortality.

1.2. Case Presentation: A 79-year-old man presented with syncope and was diagnosed with pulmonary embolism. The initial therapeutic strategy was low molecular weight heparin (LMWH). Given the worsening with hemodynamic instability, systemic thrombolysis was performed, and clinical improvement was observed. As the patient remained in shock with the need for vasoactive amines, and due to the maintenance of proximal pulmonary thrombi, catheter-directed percutaneous thrombectomy was performed, which when fragmenting and aspirating the thrombi, reversed the right heart failure with rapid hemodynamic improvement.

1.3. Conclusion: This case included the combination of different strategies, allowing the patient's survival and, probably, a faster subsequent recovery of the optimized cardio-respiratory function.

2. Introduction

Pulmonary embolism (PE) is common and can be fatal. According to European Society of Cardiology, the annual incidence rate of PE varies between 39-115 per 100,000 inhabitants [3]. The clinical presentation of a patient with PE is variable and nonspecific, representing a clinical challenge. Therefore, it is essential to have a high index of suspicion.

3. Case Report

A 79-year-old man, previously under hypocoagulation with rivaroxaban for suspected Deep Venous Thrombosis (DVT) of the right lower limb 3 years earlier. Admitted to the emergency department due to a syncope with Traumatic Brain Injury (TBI). No dyspnea or chest pain.

On admission, Glasgow Coma Scale 15, without focal deficits, presenting a hematoma in the left occipital region, blood pressure (BP) 117/82mmHg, heart rate 99bpm, peripheral oxygen saturation 94% under oxygen therapy at 2L/min, tachypnoea, slight asymmetry of the lower limbs with pre-tibial edema on the left, although without tightness in the calf.

Arterial blood gas analysis in room air revealed respiratory alkalemia and hypoxemia (pH 7.51, pCO2 28mmHg, pO2 76mmHg), normal lactate and glucose. D-dimers 14.03mg/dL, troponin 1.01ng/dL. Chest radiography was normal, electrocardiogram in sinus rhythm with the S1Q3T3 pattern (Figure1). Head computed tomography (CT), showed an epicranial hematoma.

A chest Computed Tomography Pulmonary Angiography (CTPA) revealed endoluminal repletion defects in both the right and left pulmonary arteries (Figure2A) and its lobar and segmental branches, in relation to central and segmental Pulmonary Thromboembolism (PTE) and dilation of the right cardiac chambers (RCs) with contralateral bulging of the interventricular septum. Transthoracic Echocardiography (TTE) showed dilation of the RCs.
Given the diagnosis of PE without hemodynamic instability, despite signs of right ventricular dysfunction and elevation of troponin, it was decided to start Low Molecular Weight Heparin (LMWH) 5 hours later, he had an episode of seizure, progressing to shock: hy potension without response to fluid therapy (BP 50/30mmHg) and generalised signs of poor peripheral perfusion (lactate 5.6mmol/L). Noradrenaline was started, titrated up to a maximum of 2.7mcg/Kg/min and orotracheal intubation for invasive mechanical ventilation was performed. The TTE was repeated, revealing a worsening of the dilation of the RCs, with pressure overload and a significant reduction in the left ventricular (LV) cavity, and an image suggestive of a tricuspid transvalvular thrombus. A head-CT was repeated excluding intracranial hemorrhage. Due to the clinical severity and risk of fibrinolysis due to the TBI, thrombectomy was considered. However, none of the reference centres contacted had the capacity to respond at that time.

Concomitantly, the patient developed electrical instability with wide complex tachydysrhythmia and the need for synchronized electrical cardioversion. It was decided to start thrombolysis (alteplase 100mg), as the only life-saving solution, with immediate improvement of clinical and echocardiographic signs. He was admitted to the ICU. There were improved signs of poor peripheral perfusion and reduction of noradrenaline up to 0.5mcg/Kg/min in the first hours. The re-evaluation by TTE showed a significant improvement in the dilation of the RCs and the diastolic involvement of the LV.

On the 1st day of admission to the ICU, there was a new clinical worsening. He repeated a CT angiography, which revealed the persistence of proximal pulmonary thrombi (Figure2B) and excluded thrombi of the inferior vena cava or venous proximal lower limbs. Therefore, an interventional cardiology center was contacted and the patient was transferred to perform thrombectomy. In the pre-procedure hemodynamic evaluation: pressure in the right atrium 18mmHg, pressure in the right ventricle (RV) 54/7/17mmHg, Systolic Pressure In The Pulmonary Artery (SPPA) 58mmHg, mean pressure in the pulmonary artery 34mmHg, telediastolic pressure of the LV of 11mmHg and a cardiac index of 1.95, pulmonary vascular resistance 6.3 Wood Units.

Pulmonary angiography documented a proximal thrombus in the right pulmonary artery, with complete obstruction of the middle lobe perfusion (Figure3A) and partial obstruction of segmental branches of the right and left upper lobes. Thus, mechanical thrombectomy of the right lung was performed with an Indigo CAT 8 Penumbra device (Penumbra Inc., Alameda, CA, United States), which is a vacuum aspiration system through a flexible 8Fr catheter. It was achieved reperfusion of the middle lobe and improvement of hemodynamics at the level of the pulmonary vasculature in the immediate post-procedure phase, with a decrease of 10mmHg in the mean pulmonary artery pressure (Figure3B). 5000IU of Unfractionated Heparin (UFH) was started in bolus followed by perfusion at 18IU/kg, in accordance with the activated partial thromboplastin time value. After the procedure, there was immediate improve of the pressures in the RCs, with a reduction of the SPPA to 40-45mmHg and the ratio between arterial oxygen pressure and the fraction of inspired oxygen from 90 to 125mmHg, allowing decreased aminergic support.

The patient returned to ICU on the same day, with a favorable evolution, allowing for ventilatory weaning and suspension of amines 24h after thrombectomy. UFN perfusion was also changed to enoxaparin 80 mg/day (dose adjusted to creatinine clearance). During the remaining stay in the ICU, no complications were observed. After 4 days he was transferred to the ward waiting for a doppler of the lower limbs. At the time of discharge, the TTE revealed a LV with preserved global systolic function, an enlarged RV with normal systolic function, a 100ms acceleration time in the RV outlet chamber, without tricuspid regurgitation that would allow estimation of SPPA.

Figure 1: S1Q3T3 pattern on the electrocardiogram.
### Figure 2: Bilateral endoluminal repletion defects on chest CTPA (A). Persistence of proximal thrombus after fibrinolysis (B).

### Figure 3: Selective pulmonary angiography showing a large proximal obstructive thrombus in the right pulmonary artery that compromises the perfusion of the middle lobe (A). After mechanical thrombectomy with the Indigo CAT 8 PENUMBRA® device, reperfusion of the right middle lobe was possible, with a 10mmHg drop in mean pulmonary arterial pressure (B). PAP- pulmonary artery pressure

## 4. Discussion and Conclusion

PE has a high mortality rate. Syncope occurs in approximately 11% of patients with PE [1], and is generally considered to be a sign of hemodynamic instability and poor prognosis1. It is the result of reduced cardiac output with subsequent cerebral hypoperfusion. In PE with hemodynamic instability, there is obstruction of a main pulmonary vessel, with a sudden increase in peripheral vascular resistance and pulmonary arterial pressure. This pressure causes dilation of the RV and bulging of the interventricular septum to the LV, causing impaired its filling and contraction [2].

Since the patient was clinically stable at admission, the initial therapeutic strategy was LMWH, whose efficacy and safety is demonstrated, and whose administration is indicated in these cases [3]. Treatment with fibrinolytic agents in high-risk patients with hemodynamic instability and RV dysfunction is well established. These cause a rapid decrease in pulmonary arterial pressure, with a greater benefit when they are administered in the first 48 hours after the event3. Rapid intravenous administration of the recombinant tissue plasminogen activator (rtPA 100mg for 2 hours) is indicated in the first line3. However, thrombolysis can precipitate intracranial hemorrhage3,5. In the presence of a contraindication for thrombolysis or when hemodynamic instability persists after thrombolysis, thrombectomy should be performed5. The knowledge about catheter-directed percutaneous thrombectomy comes from clinical reports, which demonstrate an 87% success rate3, with a lower risk of bleeding compared to thrombolysis.

In the case described, given the worsening with hemodynamic instability, systemic thrombolysis was performed, and clinical improvement was observed. Subsequently, because the patient remained in shock, and due to the maintenance of proximal pulmonary thrombus, catheter-directed percutaneous thrombectomy was performed, with rapid hemodynamic improvement. From the pathophysiological point of view, it is widely recognized that
RV failure due to pressure overload is the main cause of death in severe PE. The abrupt bulging of pulmonary vascular resistance after PE leads to mechanical, hemodynamic and neurohormonal compensatory mechanisms that are perpetuated and, ultimately, cause right heart failure, cardiogenic shock and death. The early cessation of this vicious cycle is essential for the patient's survival.

Stratification of the risk of death is important for a correct therapeutic approach to PE. We must change our therapeutic approach according to the patient clinical condition.

References


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