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Acute Respiratory Distress and Cardiopulmonary Arrest in a 41-Year-Old Female with Massive Pulmonary Embolism: A Case Report

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Abstract | **Case Report**

We present the case of a 41-year-old female who developed acute dyspnea and rapidly progressed to cardiopulmonary arrest. She had a history of chronic tobacco and oral contraceptive use. Computed tomography pulmonary angiography (CTPA) revealed bilateral massive pulmonary embolism. During resuscitation, thrombolytic therapy with alteplase led to return of spontaneous circulation (ROSC). She was admitted to the intensive care unit (ICU), where she experienced progressive neurological deterioration and ultimately expired. This report underscores the importance of early recognition, prompt diagnosis, and the timely initiation of thrombolytic therapy in cases of high-risk pulmonary embolism.

Keywords: Pulmonary embolism (PE), Cardiopulmonary arrest, Thrombolysis, Alteplase, Oral contraceptive use.

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INTRODUCTION

Pulmonary embolism (PE) is a life-threatening cardiovascular emergency that can present with a spectrum ranging from mild dyspnea to cardiogenic shock and cardiac arrest. Its diagnosis remains challenging due to nonspecific presentations. The incidence of PE is increasing globally, in part due to heightened awareness and advanced diagnostic modalities. Risk factors include recent surgery, malignancy, immobility, thrombophilia, and hormonal therapy. Here, we describe a case of massive PE in a young female with cardiopulmonary arrest and discuss management strategies in the context of current evidence.

CASE PRESENTATION

A 41-year-old woman presented to the emergency department with complaints of sudden-onset dyspnea, fatigue, and asthenia. On arrival, she was tachypneic, hypotensive (BP 80/50 mmHg), and hypoxemic (SpO2 81%). She had a significant history of tobacco use and oral contraceptive therapy. Physical examination revealed no overt signs of deep vein thrombosis, though the patient reported right leg pain.

Initial stabilization included oxygen therapy, IV fluid resuscitation, and blood sampling. Given the high pre-test probability for PE, CT pulmonary angiography was ordered. On return from radiology, she suffered abrupt cardiopulmonary collapse.

Management and Outcome

Cardiopulmonary resuscitation (CPR) was initiated, and orotracheal intubation performed. CT angiography obtained during CPR confirmed bilateral massive PE. Systemic thrombolysis was immediately administered with 100 mg alteplase (50 mg bolus + 50 mg infusion). ROSC was achieved after approximately 70 minutes of CPR. Following ROSC, the patient was admitted to the ICU for post-resuscitation care. Despite vasopressor support and mechanical ventilation, she developed worsening neurological status with persistent coma (Glasgow Coma Scale score: 3), absent brainstem reflexes, and evidence of diffuse cerebral edema on cranial CT performed 24 hours later. Supportive care was continued, but she succumbed to multi-organ failure on day 3 of ICU admission.

DISCUSSION

Massive PE remains a diagnostic and therapeutic challenge, particularly when presenting with cardiac arrest. This case highlights several key aspects:

Recognition of risk factors such as hormonal therapy and smoking is essential in assessing PE likelihood.

Early imaging with CTPA, even in unstable patients, is critical when feasible.

Systemic thrombolysis during CPR is supported by current guidelines in massive PE with cardiac arrest and can result in ROSC, as seen in our patient.

However, prolonged low-perfusion states carry a high risk of post-arrest brain injury, as likely occurred in this case. The presence of refractory coma and radiological signs of diffuse cerebral edema indicated a poor neurological prognosis.

Alternative treatment strategies such as surgical embolectomy or catheter-directed thrombolysis may be considered in selected patients; however, they are often unfeasible in emergent cardiac arrest scenarios.

Conclusion

This case emphasizes the need for rapid recognition of PE in patients presenting with acute dyspnea and known risk factors. While thrombolytic therapy during CPR can achieve ROSC, it may not reverse the consequences of prolonged hypoperfusion. The case contributes to the ongoing discourse on optimizing post-resuscitation care and risk stratification in massive PE.

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