

# Complete Cognitive Recovery and Survival From Massive Pulmonary Embolism During General Anesthesia after Administration of Alteplase: A Case Report

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Presented herein is the case of a 37-year-old male who was scheduled for an anterior decompressive laminectomy after suffering trauma to the cervical area (C6–C7). An intraoperative acute pulmonary embolism (APE) was suspected after persistent hypoxemia and a decreased end-tidal CO<sub>2</sub> that was refractory to proper management. After 6 intraoperative episodes of cardiac arrest that followed, intravenous alteplase (thrombolytic therapy) was administered, and the patient was stabilized without major complications. Eventually, APE was successfully diagnosed and treated. The experience served as reference for the diagnosis and management of APE under general anesthesia. [*PR Health Sci J* 2020;39:62-63]

*Key words: Intra-operation, Pulmonary embolism, General anesthesia, Cardiac arrest*

An acute pulmonary embolism (APE) is a life-threatening event for which early identification and management are key to improving the sufferer's prognosis and survival. Deep vein thrombosis (DVT) is the main cause of an APE (1). In the United States, more than 100,000 patients die each year due to this complication (1). Although it is more commonly seen in the preoperative or postoperative period, cases have been reported to have occurred intraoperatively. Due to the effects of general anesthesia on the physiologic responses of the afflicted patient, the common signs and symptoms of an APE are blunted, which makes the prompt identification and treatment of this phenomenon even more difficult (2). Advances in biomedical technology, such as echocardiography and computed tomography angiography (CTA), have made the diagnosis and proper treatment of this disease more efficient, nowadays.

## Case Description

Herein, we describe the case of a 37-year-old male patient who had been scheduled for an anterior decompressive laminectomy at C6–C7 5 days after his admission to our hospital posterior to having sustained a 10-story fall; he had no other trauma-related injuries. Previous to the traumatic event, the patient was in good health, with no past medical history of thrombotic disease. The patient arrived at the operating room alert, awake, and oriented but presented symptomatic bilateral upper-extremity paresthesia and weakness in the C6–C7 distribution. To avoid further injury, the patient was intubated, nasotracheally, via awake fiber optic bronchoscopy, without any complications. A left radial arterial line and 2 large-bore (18G) intravenous (IV) access lines were placed. Initial monitoring showed that the patient had a BP of 140/90 mmHg, an HR of 55 bpm, and an SpO<sub>2</sub> of 98%. To sustain an end-tidal CO<sub>2</sub> (EtCO<sub>2</sub>) of 35 to 45 mmHg, the ventilator was used in the volume-control mode,

with the following parameters: tidal volume (V<sub>t</sub>) was 600 mL; the respiratory rate (RR) was 10 breaths per minute, with an inhalation to exhalation ratio of 1:2; the fraction of inspired oxygen (FiO<sub>2</sub>) was 100%. Immediately after positioning, the patient had a sudden onset of hypoxemia (peripheral capillary oxygen saturation [SpO<sub>2</sub>] at 89%) and experienced an acute decrease of EtCO<sub>2</sub> (falling from 35 to 18 mmHg), followed by cardiac arrest, consistent with pulseless electrical activity. A total of 6 intraoperative cardiac arrests occurred, each one lasting approximately 5 minutes and with an average interval of 15 minutes between each arrest. All were successfully managed, per the advanced cardiac life support (ACLS) protocol. Although the return of cardiac hemodynamics was evident, the patient continued to suffer from hypoxemia, which was apparent by his SpO<sub>2</sub> and persistent low EtCO<sub>2</sub>. Arterial blood gases (ABGs) were taken and revealed relatively marked hypoxemia (pH, 7.13; partial pressure of oxygen [PaO<sub>2</sub>], 72.9 mm Hg; partial pressure of carbon dioxide [PaCO<sub>2</sub>], 69.1 mm Hg; bicarbonate [HCO<sub>3</sub>], 22.6; and base excess [BE], -7.9) with primary respiratory acidosis. These results revealed an increase in dead space secondary to a ventilation/perfusion (V/Q) mismatch based on an increased Alveolar–arterial (A–a) gradient where, in spite of the patient's having received an FiO<sub>2</sub> of 100%, PaO<sub>2</sub> was markedly decreased. The fact that a V/Q mismatch was present along with a decreased PaO<sub>2</sub>, despite the patient's having been optimally ventilated, suggested decreased alveolar perfusion

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(thus, the increased V/Q mismatch) (2). A limited number of causes for high V/Q mismatch exist, the most common being chronic obstructive pulmonary disease (COPD—emphysema) and APE (2). With this in mind, and taking into consideration the patient's noncontributory medical history, an APE secondary to DVT was highly suspected. After the 5th cardiac arrest and a multidisciplinary discussion, therapeutic anticoagulation with 90 mg of enoxaparin was administered.

Simultaneously, the patient was placed on a continuous epinephrine drip. Approximately 45 minutes later, the patient had a 6th cardiac arrest. Afterwards, it was decided to start thrombolytic therapy with alteplase IV, even given all the risks and the possible adverse effects that this treatment could bring about. The patient remained on a high-dose epinephrine drip (15 mcg/kg/min), and after stabilization, he underwent a chest CTA, which confirmed the presence of a massive saddle pulmonary embolism with severe right ventricular dilated cardiomyopathy. The patient was then sent to the catheterization unit for an emergent and selective endovascular left pulmonary artery embolectomy and inferior vena cava filter placement, which were performed without any complications. The patient was then started on a heparin infusion and transferred to the ICU, where he remained critically ill and developed acute renal failure (requiring hemodialysis) and shock liver secondary to cardiac failure. The patient was closely followed for the next few days, showed satisfactory improvement in his condition, and was successfully extubated 2 days later. Cognitive assessment was evaluated through a Mini Mental Test (scoring 26/30) without evidence of cognitive dysfunction.

## Discussion

An APE is an obstruction of the pulmonary artery or one of its branches by material (eg, a thrombus, a tumor, air, or fat) that originated elsewhere in the body (1). It consists most commonly of a thrombus originating in the proximal veins of the lower extremities, as was the case with this patient, and can result from immobility, trauma, or a hypercoagulable state. Various pathophysiologic responses can occur, such as pulmonary infarction, abnormal gas exchange, and sustained hypotension due to diminished stroke volume and cardiac output. Early detection under general anesthesia is even more difficult, as the common symptoms (chest pain, dyspnea, and cough) are not observed when the patient is anesthetized. The pathophysiologic course of intra-operative APE is influenced by ongoing surgical and anesthesia management procedures (3). APEs can be classified as massive, sub-massive, or low risk, according to hemodynamic changes. A massive APE, such as occurred with this case, presents with sustained hypotension (systolic blood pressure less than 90 mm Hg for at least 15 minutes) and pulselessness or profound bradycardia.

CTA is the current gold standard for the diagnosis of acute PE, because of its high accuracy, availability, and rapid turnaround time (4). But in the case of a sustained hemodynamic

instability, a transesophageal echocardiogram (TEE) remains the diagnostic study of choice for a quick bedside evaluation of PE in high risk patients with right ventricular dysfunction (4).

The treatment of a massive PE in the perioperative period typically involves a multidisciplinary discussion during which a risk-to-benefit ratio analysis is performed (5). Thrombolysis and systemic anticoagulation have been used successfully for surgical patients, but the risk of hemorrhage is greater in these patients. Despite the major risk of bleeding, thrombolytic therapy has been shown to result in significant reduction in hemodynamic collapse and the restoration of pulmonary perfusion, accomplishing these improvements more rapidly than do anticoagulants in a massive PE, which was what took place with this particular case.

It is worth mentioning that if diagnostic tests are not promptly available and the patient continues to be hemodynamically unstable (with a high arterial CO<sub>2</sub> and low EtCO<sub>2</sub> on ABGs) despite aggressive resuscitation efforts, as came to pass in this case, an APE should be suspected. ABG results consisting of high CO<sub>2</sub> and low EtCO<sub>2</sub> occurring under general anesthesia are known as the separation phenomenon (3).

## Resumen

Un caso de un varón de 37 años de edad que estaba programado para una laminectomía descompresiva anterior de C6-C7 después de sufrir un trauma en el área cervical. Luego de una hipoxemia sostenida y una disminución del EtCO<sub>2</sub> refractarios a manejo adecuado, se sospechó una embolia pulmonar intraoperatoria. El paciente sufrió de 6 episodios de paro cardíaco en la sala de operaciones. Por lo tanto, se administró Alteplase por vía intravenosa y el paciente se estabilizó sin mayores complicaciones. Inmediatamente después, el diagnóstico y el tratamiento de la embolia pulmonar se completaron con éxito. Esta experiencia sirve como referencia para el diagnóstico y tratamiento de la embolia pulmonar aguda bajo anestesia general.

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