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Background:
Acute pulmonary embolism (APE) is a life-threatening event on which early identification and management are the most important variables for improving patient’s prognosis and survival. Approximately more than 100,000 patients die each year due to this complication in the United States.(1-4). Intraoperative pulmonary embolism under general anesthesia is a rare but life threatening condition, where prompt recognition and treatment is necessary for patient survival. General anesthesia blunts the common signs and symptoms of an APE, making harder the prompt identification and treatment of this phenomenon. Despite advances in technical modalities for diagnosis, such as echocardiography this measure is invasive and not always available in rural setting. In this study we present a case of a massive APE during the anesthesia that was clinically diagnosed solely on hemodynamics and clinical presentation.

Case description:
Case of an 37-year-old male patient with a BMI of 33 kg/m2, past medical history of hypertension, scoliosis surgery without any complication, and American Society of Anesthesiologists (ASA) physical status of III, who was scheduled for anterior C6-C7 decompressive laminectomy 5 days after admission due to sustaining a fall from 10 feet, without any other trauma related injuries. Patient arrived at the OR alert, awake, and oriented but presented symptomatic bilateral upper extremity paresthesias and weakness in the C6-C7 distribution (ASAI Level C). Patient was intubated nasotracheally via awake fiberoptic bronchoscopy without any complications. A left radial arterial line was placed for invasive hemodynamic monitoring after induction. Initial monitoring showed a BP 140/90 mmHg, HR 55 bpm, and SpO2 98%. Immediately after positioning, the patient had sudden onset of hypoxemia (SpO2 at 89%) and acute decrease of EtCO2 (from 35 to 18 mmHg), followed by cardiac arrest consistent with pulseless electrical activity (PEA). A total number of 6 intraoperative cardiac arrests occurred, lasting approximately 5 minutes and with an average interval of 15 minutes between each arrest. All were successfully treated as per ACLS protocol. Although return of cardiac hemodynamics was evident, patient continued with hypoxemia noticeable by the SpO2 and persistent low ETCO2. ABG’s taken revealed marked hypoxemia (pH 7.13, PaO2 72.9 mm Hg, PaCO2 69.1 mm Hg, HCO3 22.6, and BE -7.9) and significant increase in dead space with a primary respiratory acidosis. After ruling out other causes (like hypoglycemia, pneumothorax, hypothermia, and electrolyte disturbances), pulmonary embolism was highly suspected as a possible etiology. After the 5th cardiac arrest and thorough discussion with neurosurgery team,
therapeutic anticoagulation with 90 mg of enoxaparin SC was administered. Simultaneously, the patient was placed on continuous epinephrine drip. Approximately 45 minutes later, the patient had a 6th cardiac arrest, alteplase IV was administered; acknowledging all the risks and detrimental outcomes this can ensue (since diagnosis was not confirmed due to lack of echocardiogram availability). A loading of 10 mg IV over 2 minutes were administered followed by 90 mg drip administered over 2 hours. Patient remained with a high dose epinephrine drip at 15 mcg/kg/min and after stabilization, he underwent a chest CTAngiogram, which confirmed a massive saddle pulmonary embolism with severe right heart dilated cardiomyopathy. Patient was then sent to the cardiac cath lab for an emergent endovascular left pulmonary artery thrombectomy and IVC filter placement. Patient was then put 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. Patient was closely followed for the next days, observing a satisfactory improvement on his condition. On day 10, patient was able to follow commands and was successfully extubated two days later. Cognitive assessment was evaluated through a Mini Mental Test, in which the patient scored a 26/30, without evidence of cognitive dysfunction. Patient was discharged to an inpatient rehabilitation center on Day 12, with out cognitive dysfunction.

This case demonstrates that despite limitations in invasive measurements for diagnosing intraoperative pulmonary embolism, prompt clinical diagnosis and multidisciplinary team efforts all contributed to markedly improved outcome in this patient.


