on’t Take My Breath Away: A Case of High-Risk Pulmonary Embolism in the Setting of Patent Foramen Ovale Requiring Extracorporeal Membrane Oxygenation

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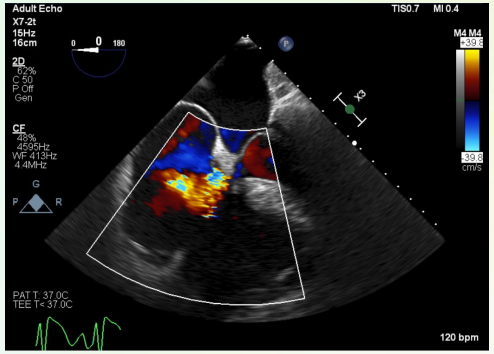
BACKGROUND

- Pulmonary emboli (PE) is the third most common cause of cardiovascular death, with 60,000-100,000 deaths per year.
- Approximately 35% of the general population have a patent foramen ovale (PFO) based on autopsy studies.
- We present a case of a patient with saddle pulmonary emboli who had persistent hypoxemia in the setting of a PFO.

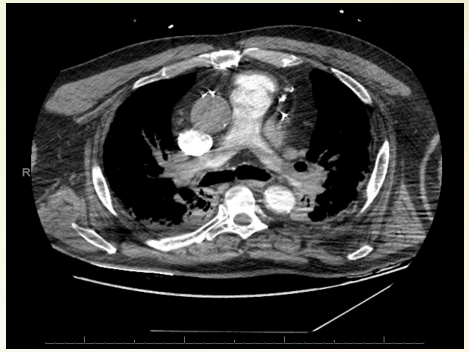
CASE DESCRIPTION

A 67-year-old man with a past medical history of coronary artery disease, alcohol abuse, hypertension, hyperlipidemia, and obstructive sleep apnea presented to the hospital with complaint of word-finding difficulty and behavioral changes. He was found to have acute occlusion of the bilateral middle cerebral arteries, requiring mechanical thrombectomy. He was recovering successfully when he suddenly developed dyspnea and lightheadedness with exertion, which rapidly progressed to mechanical ventilation. He remained hypoxic on optimal ventilator settings and inhaled nitric oxide, prompting suspicion for acute pulmonary embolism. Chest x-ray was unchanged. EKG showed inferior ST-segment elevations. Emergent transesophageal echocardiogram (TEE) revealed a severely dilated right ventricle, an underfilled left ventricle, McConnell’s sign, and right to left atrial septal bowing suggesting elevated right atrial pressures. TEE also noted an incidental PFO with right-to-left shunting and an atrial septal aneurysm. Computed tomography scan of the chest revealed a saddle pulmonary embolus. Veno-arterial extracorporeal membrane oxygenation (ECMO) was initiated as a bridge to mechanical thrombectomy. He was converted from veno-arterial ECMO to veno-venous ECMO with Protek Duo for right ventricular support. His hospital course was complicated by cardiogenic shock, septic shock, and progression of his initial ischemic stroke. He was able to be decannulated from ECMO and eventually extubated. He was discharged to a long-term care facility.

Refractory hypoxemia in pulmonary emboli should prompt high clinical suspicion for the presence of intracardiac shunts



Transesophageal echocardiogram demonstrated dilated right side of the heart and patent foramen ovale by color Doppler flow



Computed tomography scan of the chest revealed saddle pulmonary emboli

DISCUSSION

- Pulmonary emboli (PE) can precipitate cardiovascular collapse by mechanical obstruction of the right ventricular outflow tract and pulmonary artery vasoconstriction, leading to right ventricular overload. The presence of a patent foramen ovale (PFO) can subvert this typical physiology by creating a right-to-left shunt, permitting left ventricular filling and near-normal cardiac output. However, this also causes intracardiac mixing of oxygenated and deoxygenated blood, which can further worsen hypoxemia.
- Recent studies have recognized ECMO as a promising adjunctive therapy for pulmonary emboli for those who are too unstable for surgical thrombectomy or who have contraindications to systemic thrombolysis. Our patient was not a candidate for thrombolysis given his recent stroke.
- The patient’s PFO, while beneficial in preventing florid cardiogenic shock by offloading the right ventricle, caused hypoxemia refractory to mechanical ventilation alone. Thus, ECMO was a crucial temporizing measure for bridging to surgical thrombectomy.

CONCLUSION

- It is important to have high clinical suspicion for intracardiac shunts in cases of refractory hypoxemia in pulmonary emboli. This aid with early identification of patients who need advanced intervention such as ECMO.

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