Massive Pulmonary Embolism in Recovery Period of General Anesthesia: Rapid Diagnosis and Successful Rescue by the Guidance of Transthoracic Echocardiography

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Massive pulmonary embolism (PE), an uncommon event in the perioperative period, is a major challenge to anesthesia management. Massive PE is often associated with an unexpected hemodynamic collapse and severe desaturation without other characteristic changes. It is difficult to make a rapid diagnosis during the perioperative period, especially under general anesthesia. We present a rare case of massive PE diagnosed with transthoracic echocardiography (TTE) in the recovery period of general anesthesia.

We obtained written informed consent from the patient and approval of the use of the data from the local Institutional Review Board. A 66-year-old male patient was admitted and scheduled for video-assisted thoracoscopic bronchial sleeve lobectomy. The medical history included hypertension and smoking. Ultrasonography of the lower extremities showed negative findings. TTE showed normal results of the right ventricle (RV), right atrium (RA), and pulmonary artery systolic pressure (PASP). Lung function test and other results were negative. The surgery was uneventful and lasted 571 min due to the severe adhesion of tissue, much longer than expected.

The patient was moved to the transport trolley and extubated after a full recovery with stable vital signs. His blood pressure (BP) sharply decreased to 40/20 mmHg, heart rate increased to 110 beats/min, and saturation of pulse oxygen (SpO₂) fell to 74%. Mask ventilation and vasopressors (phenylephrine and ephedrine) could not improve the situation. The patient was then reintubated (end-tidal CO₂, 30 mmHg). BProse to 90/60 mmHg with vasopressors (epinephrine 1 mg, norepinephrine 1 µg·kg⁻¹·min⁻¹, and dopamine 10 µg·kg⁻¹·min⁻¹). Central

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venous pressure was 12 mmHg. There were no abnormal findings in auscultation and chest drainage tube (airway pressure, 15 cmH₂O). Electrocardiography showed a sinus tachycardia with complete right bundle branch block. Subsequent arterial blood gas (ABG) analysis showed PaCO, of 73 mmHg, which led to the suspicion of PE. Emergency bedside TTE (duration 3 min) showed an enlarged RA (23 cm²) and RV (27.8 mm), moderate pulmonary hypertension (PASP 50 mmHg), and hypokinesia of the free RV wall compared with normokinesia of RV apex [Figure 1a]. The patient was diagnosed with massive PE and transferred to the catheter laboratory for catheter-directed thrombolysis. Pulmonary angiography revealed multiple filling defects in the left lung [Figure 1b and 1c]. After thrombus fragmentation and thrombus aspiration, urokinase (250,000 U) was sprayed transcatheter to the main pulmonary trunk. After completion of the intervention therapy, PASP soon decreased to 40 mmHg and the patient was transferred to the intensive care unit. After 22 days in the hospital, the patient was discharged home without any sequela of PE.

PE is a major clinical challenge and carries significant mortality in the perioperative period. PE occurs in about 1.3% of patients undergoing lung resection, despite routine

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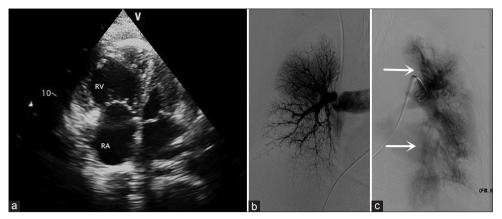


Figure 1: TTE image before the thrombolysis (a) and pulmonary angiography images (b and c). (a) Bedside TTE demonstrated remarkably enlarged RA and RV. (b) Right lung. (c) Left lung. Pulmonary angiography revealed multiple filling defects in pulmonary artery in the left upper lobe and inferior lingular segment (arrow in c). TTE: Transthoracic echocardiography; RA: Right atrium; RV: Right ventricle.

use of prophylaxis.^[1] However, rapid diagnosis may be difficult because of atypical clinical manifestations, especially during general anesthesia.

The first sign of PE in this patient was the unstable hemodynamics, followed by dyspnea and a decrease in SpO₂. Tension pneumothorax and massive hemorrhage were initially suspected and soon excluded; there may have been other causes, such as acute coronary syndrome or allergic reaction. The subsequent ABG led us to the suspicion of PE. Unfortunately, computed tomography, the recommended procedure for reaching a diagnosis of PE, was not available immediately.[1] However, indirect signs of RV overload (enlarged RA and RV and increased PASP) and depressed contractility of the RV free wall in conjunction with preserved apical contractility (McConnell sign) on bedside TTE showed a high probability of massive PE. The McConnell sign was first described in 1996 and has 77% sensitivity and 94% specificity for the diagnosis of acute PE. It also has the highest specificity of all findings of echocardiography in PE diagnosis, with a positive predictive value of 86%. [2] The McConnell sign indicates the regional RV dysfunction and is associated with an increased clot burden and systolic dysfunction of RV. It can also aid the differential diagnosis although the diagnostic power for the detection of PE is limited.[3] Echocardiography is not recommended as part of the workup in hemodynamically stable patients with suspected PE (not at high risk); it is strongly related to PE with moderate to severe RV dysfunction.^[4] In a severely hemodynamically compromised patient with suspected PE, unequivocal echocardiographic evidence of the McConnell sign justifies emergency reperfusion therapy without any further examination.^[1] From the onset of circulation collapse, it took us less than 10 min to make the diagnosis of massive PE. Undoubtedly, point-of-care echocardiography is the most useful initial test and is the preferred option for anesthesiologists conducting rapid diagnosis of massive PE.

The blood stasis of the dependent leg, prolonged major surgery, venous thrombus embolism (VTE) susceptibility,

and shortage of preventive measures during surgery perhaps contributed to the PE. Catheter-assisted treatment is an attractive therapeutic option for massive PE in situations with a high risk of bleeding. In our case, thrombus fragmentation and aspiration combined with catheter-directed thrombolysis were performed with no involvement of bleeding complications. Nevertheless, some adverse aspects of this case are worthy of further consideration. The risk of VTE should be evaluated periodically. More attention should be paid to the prevention of PE, including anticoagulation and mechanical preventive measures during the procedure.

Knowledge of prompt diagnostic strategies and assessment of clinical probability regarding PE are essential. Furthermore, in suspected massive PE with shock, bedside TTE can be used to identify the situation and justify reperfusion treatment, especially under general anesthesia.

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Conflicts of interest

There are no conflicts of interest.

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