Successful Therapeutic Intervention for Massive Pulmonary Embolism Under General Anesthesia with Severe Hypercapnia: A Case Report

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Citation

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Abstract

Massive pulmonary embolism under general anesthesia is an acute fatal complication. Prompt diagnosis is important but difficult because some of clinical signs are masked by general anesthesia under controlled ventilation. The mortality rate can be as high as 70% if massive pulmonary embolism is left untreated. We report a case of successful therapeutic intervention, thrombolysis, for clinically suspected massive pulmonary embolism with severe hypercapnia under general anesthesia.

The annual incidence of venous thrombo embolism (PE) in a general Western population is approximately 0.1 %[1]. In spite of improved prevention and treatment of pulmonary embolism, the mortality is still estimated to be between 20 - 30%[2]. If the embolism is massive, patient's conditions deteriorate rapidly and the death rate in the first hour can reach as high as 70% [3]. Prompt diagnosis and treatment at the earliest possible opportunity is the most effective means for reducing the mortality from massive PE [4]. We report a case of successful therapeutic intervention, thrombolysis, for clinically suspected massive pulmonary embolism with severe hypercapnia under general anesthesia.

CASE REPORT

A 66-year-old man underwent a selective orthopedic surgery for his pelvic fracture caused by a fall from a high building 5 days earlier. His physical and lab exams were normal including KPTT 28.5s and PT 12.1s. His past medical history was clear except for chronic bronchitis. When he admitted into the operating room, his initial blood pressure was 134/82mmHg, HR 100bpm and his SpO_2 was 88% on air. The patient was routinely induced with propofol 1ml/kg and fentanil 4mcg/kg following preoxygenation. Endotracheal intubation was performed smoothly after the muscle-relaxant vecuronium 0.1mg/kg took effect. The tube was connected to an anesthesia ventilator with the parameters set as follows: IPPV, VT 8ml/kg, RR10bpm and Isoflurane 1% with pure oxygen flow 1L/min. The vital signs after induction were 102/60mmHg, HR101bpm SpO_2 99% and PetCO2 30mmHg. The patient was put into a lateral position with the left side on and his vital signs kept within normal range until several minutes after the operation started. His blood pressure and SpO_2 were unobtainable but HR climbed to 120bpm regular. After an intravenous infusion of ephedrine 30mg failed to raise BP, dopamine 50ug/kg/min were administered intravenously along with fast fluid infusion. Although the blood pressure reached 60/34mmHg and SpO_2 was 96%, the PetCO2 fell to 23mmHg from 35mmHg. Blood-gas analysis showed PH7.06, $PaO_2$334mmHg, $PaCO_2$99.5mmHg, BE-3. The operation stopped, the incision was sutured and the patient was turned to supine position.. The situation was improving as the fluid infusion rate kept high enough to maintain BP around 130/80mmHg, HR110bpm and SpO_2 99%. The patient was prepared for transfer to the ICU for further treatment. As soon as he was put onto a stretcher, his blood pressure dropped again to 50/25mmHg, HR70bpm, SpO_2 64% and the patient's color was ashen. Then after 3 times of epinephrine 0.1mg iv, the BP was 85/40mmHg HR100bpm SpO_2 96%. Chest auscultation found rochi on the both sides, blood-gas analysis revealed PH7.05, PaO_2 449mmHg, $PaCO_2$ 103.2mmHg, BE-1. The $PaCO_2$ increased steadily to 170mmHg in the next hour while the PaO_2 declined slightly. The patient had no urine output for half an hour despite 1L of fluid administering. ECG showed sinus heart rate with RBBB, S1Q2T3 and the jugular vein was elevated. D-dimer was 2.6mg/L (normal range 0-0.3mg/L), KPTT 49.5s and PT 19.2s. There were no other tests to perform due to the cardiovascular collapse and the limited availability of

devices. Massive pulmonary embolism was highly suspected and thrombolysis was discussed and started with continuous peripheral intravenous infusion of 100mg rTPA along with an injection of low molecular weight heparin (LMWH) 1mg/kg subcutaneously when the patient arrived to ICU. One hour later the infusion was stopped due to severe bleeding from the incision. At that time PT was 14.5 and KPTT was 40.9. Fortunately the patient started to have urine output and the $PaCO_2$ dropped down to 70.2mmHg. Bed-side ultrasound revealed thrombolus in right deep thigh vein. LMWH was administrated for 5 days followed by warfarine taken orally for further anticoagulation treatment. The $PaCO_2$ and vital signs returned to normal, EKG showed normal sinus heart rate. The patient was checked out of the ICU 6 days later and followed up for one year without recurrence. No further operation was done. The patient is now totally recovered and he can walk with the help of stick.

DISCUSSION

PE is the implantation of material into branches of the pulmonary arterial bed. This material usually consists of clots dislocated from peripheral veins but can also be neoplastic cells, fat emboli, amniotic fluid, air, pieces of catheters and other diagnostic devices, or exogenic material when delivered in the veins. Most commonly the clots migrate from deep veins of the pelvis and the lower extremities. The classical triad of Virchow is the main cause of such clots (endothelial injury, stasis, hypercoagulability). The incidence is highest in those undergoing emergency surgery following trauma (for hip fractures, for example) and pelvic surgery [5]. In this case, the patient was immobilized for 5 days following a pelvic fracture, so the possibility of DVT was extremely high. The bed-side ultrasound confirmed it.

Clinical signs and symptoms of PE depend on the size of the embolus, the location of the embolus, the patient's underlying cardiopulmonary status, and compensatory neurohumoral adaptations. Post mortem findings have shown that 73% of pulmonary embolisms are not clinically diagnosed [6]. Attempts to diagnose massive pulmonary embolism under general anesthesia are further complicated since the use of controlled ventilation precludes the onset of a potentially valuable warning sign, i.e., rapid shallow breathing followed by apnoea. In this case, the initial symptoms were unstable haemodynamics with both BP and SpO_2 dropping to unobtainable levels. This could be suspected as just hypovolmia but the patient's PetCO2 decreased remarkably and the $PaCO_2$ soared to 170mmHg. Hypercapnia has previously been described in clinical and experimental cases of overwhelming pulmonary thrombo embolism [7]. To our knowledge, this is the most severe hypercapnia that has ever been reported. In acute PE, hypercapnia reflects massive embolism accompanied by marked increases in both anatomic and physiological dead space. The alveolar volume of each tidal breath is severely reduced, and the ventilatory muscles are unable to sustain the marked increase of minute ventilation needed to maintain normal $PaCO_2$. [8] and massive pulmonary embolism with hypercarbia were associated with severe hemodynamic instability [7].

Inspite of minimally invasive diagnostic strategies for pulmonary embolism are in constant evolution, early diagnosis of acute massive pulmonary embolism depends on clinical signs and symptoms and D-dimer. Transesopheal echocardiography (TEE), spiral computed tomorgraphy of the chest, alveolar dead-space measurement and other diagnostic tools have been widely validated and reviewed[9,10]. In this case, the situation came suddenly and deteriorated quickly so that it was almost impossible to move the patient for further investigation. TEE is not a routine tool in our operation theatre and the case happened later after 6pm, staff who perform TEE in our hospital had left. But TEE should have been performed later in ICU for further observation.

Thrombolytics are plasminogen activators, converting plasminogen to plasmin, which then degrades clot bound fibrinogen, resulting in clot lysis. Thrombolysis may result in faster and more complete clot lysis, which could lower morbidity and mortality. The causal clot may also be more efficiently lysed and so recurrence reduced. The use of thrombolysis in patients with PE and systemic hypotension, cardiopulmonary arrest, or RV dysfunction is widely advocated, because the mortality rate is high and case reports of its effectiveness are encouraging [11]. This case again indicated that rTPA could lyse the clot within an hour and therefore reduce the mortality rate. However, thrombolysis risks haemorrhaging. In this case, the patient was on the blinck of death and the thrombolysis was the last resort that might be able to save him. We administered rTRA while closely monitoring the incision and other signs about bleeding. Time of using thrombolysis in acute PE has been reviewed [11], our case showed that early thrombolitic intervention could save life and reduce mordality.

There is a suspicion that the clot can migrate within certain area. For the first episode, the patient suffered severe hypotension and no pulsation, a situation similar to PEA. After the administration of epinephrine and turning the position, the patient's situation improved quickly. But another movement caused even more severe blockage leading to a catastrophic condition. Again, we should have performed more noninvasive diagnostic tools, such as TEE, to further check the emboli.

In conclusion, massive pulmonary embolism under general anesthesia is an acute fatal complication. Prompt diagnosis is of the utmost importance but difficult in practice, not only because some of the signs and symptoms are masked by general anesthesia with controlled ventilation, but also because an unstable haemodynamic condition may not allow for further exmainations. Clot migration may fool the clinicians and cause more severe blockage. The lesson to be learnt from the case is that it is important to be aware of the possibility of PE and to start therapeutic intervention as early as possible to confirm the diagnosis and more importantly to save lives. If possible, non-invasive diagnotic tools, such as TEE and spiral CT of the chest should be performed to observe the progress.

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