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Pitfalls in the Diagnosis of Peri-Operative Acute Pulmonary Embolism: A Case Report

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Abstract **Case Report**

An 80 years-old man with hypertension, atrial fibrillation, and coronary artery occlusion disease presented for an elective right femur bipolar hip arthroplasty under general anesthesia. Preoperative transthoracic echocardiography (TTE) showed only preexisting regional wall motion abnormality. During the reversal of muscular relaxation following surgery, there was a sudden decrease in blood pressure associated with rapid ventricular response, which was successfully treated by cardioversion. No decrease in end-tidal carbon dioxide level was observed during the hypotensive episode. However, despite fluid and blood resuscitation and vasopressor treatment, there was a repeated hemodynamic collapse postoperatively. A portable TTE showed dilated right atrium and right ventricle (RV), D-shaped left ventricle, and septal wall motion abnormalities consistent with RV pressure overload (McConnell's sign). Computed tomography pulmonary angiography revealed multifocal PE (pulmonary embolism). Anticoagulation therapy was continued to avoid PE recurrence. The patient discharged uneventfully 30 days later. Perioperative PE is a rare and perhaps dangerous circumstance that can be challenging to diagnose when it happens under anesthesia. The likelihood of PE should always be suspected at the time of perioperative hemodynamic collapse.

Keywords: Pulmonary embolism, intra-operative, end-tidal carbon dioxide.

INTRODUCTION

Acute pulmonary embolism (PE) is a potentially fatal illness [1]. A better prognosis depends on early identification and treatment [1]. For patients who are under general anesthesia, early diagnosis is even more difficult because they lack common symptoms like dyspnea, chest discomfort, hemoptysis, and syncope [2]. PE in the operating room frequently begins with hemodynamic instability and has a greater morbidity and death rate [3,4].

A decrease in end-tidal carbon dioxide level ($ETCO_2$) has been postulated as a useful diagnostic for the early diagnosis of PE in the perioperative context, resulting in a better prognosis [5]. However, decrease in $ETCO_2$ is not a specific finding of PE, and the potential of PE cannot be fully ruled out even in the absence of dramatic change in $ETCO_2$ [6]. We provide a case of PE following hip fracture surgery under general anesthesia, where early diagnosis was challenging due to the lack of a significant change in $ETCO_2$.

CASE PRESENTATION

An 80 years-old man (172cm, 52kg) was presented for an elective right femur bipolar hip arthroplasty. His medical history included hypertension, atrial fibrillation (AF), coronary artery occlusion disease, chronic kidney disease, hypothyroidism, and dementia. The AF was diagnosed when he underwent left femur bipolar hip arthroplasty 1 year ago. His current medications included aspirin 100mg (which he stopped 3days before the surgery), rosuvastatin 10mg, and trimetazidine hydrochloride 35mg. A preoperative electrocardiogram (ECG) showed AF with heart rate of 68 bpm (Figure 1). A preoperative transthoracic echocardiography (TTE) examination revealed a decreased left atrium (LA) with reduced systolic dysfunction (ejection fraction: 48%) and preexisting regional wall motion abnormality (mainly on territory of left anterior descending and left circumflex artery). Laboratory investigations demonstrated normal creatine kinase-MB (CK-MB), troponin I, and elevated brain natriuretic peptide (NT-pro BNP) of 6158 pg/ml.

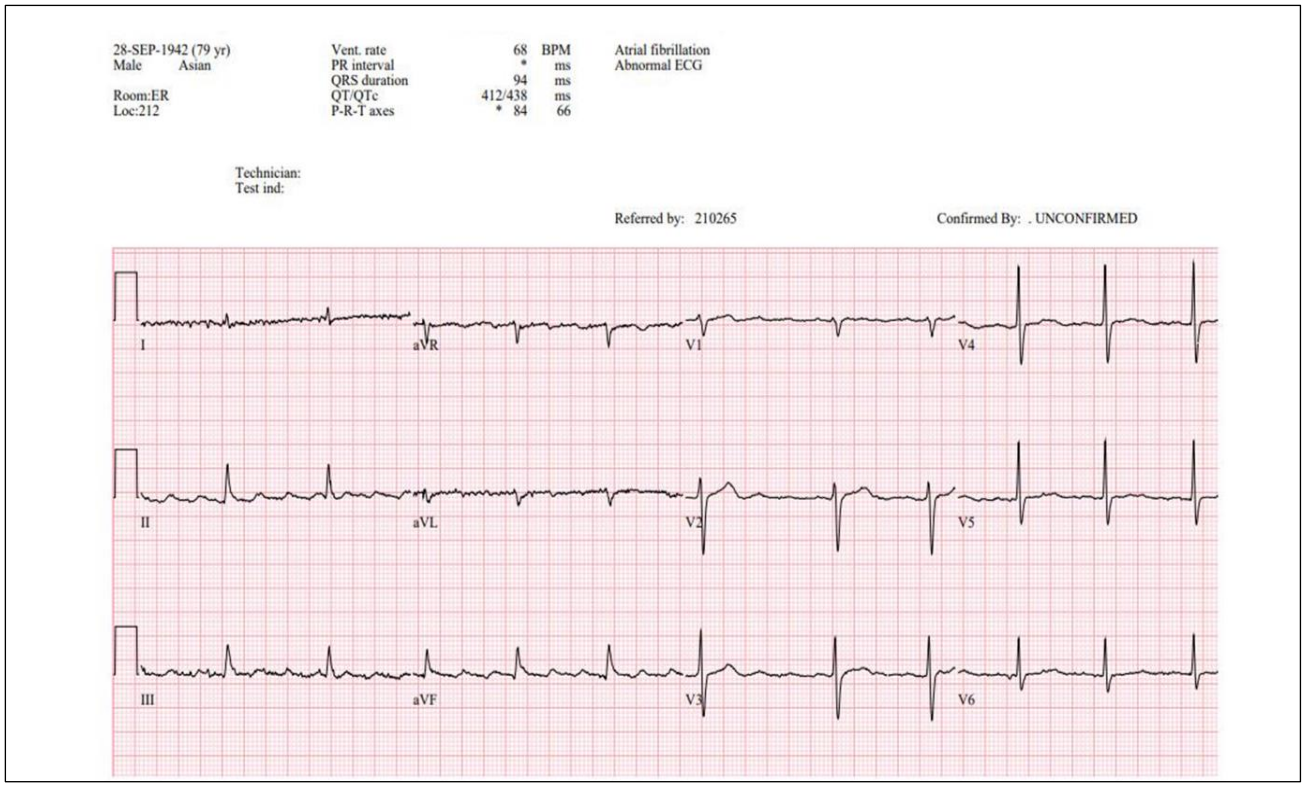

Figure 1: A preoperative electrocardiogram

After the patient arrived in the operating room, standard monitor including pulse oximetry, end-tidal carbon dioxide, ECG, and non-invasive blood pressure was applied. During examination in the operation room, the patient had no symptoms concerning the cardiorespiratory system. His noninvasive blood pressure was 119/60 mmHg, his pulse rate was 117 bpm, his respiratory rate was 16 breaths per minute, and his oxygen saturation (SpO_2) was 97% on room air. ECG showed AF with heart rate of 100 – 120 bpm. After anesthesia induction, an arterial catheter was cannulated into the radial artery for arterial blood pressure (ABP) monitoring and arterial blood gas analysis (ABGA) showed pH 7.412, PaO_2 407.7 mmHg, and $PaCO_2$ 38.5 mmHg with a base deficit of 1.1 mmol/L on FiO_2 1.0. About 25 minutes after the start of the operation, the patient's ABP dropped to 70/35 mm Hg with AF, heart rate of 78 bpm.

After a phenylephrine 50 μg bolus injection, systolic blood pressure (SBP) was maintained above 90 mmHg until the end of the operation. End-tidal carbon dioxide level ($ETCO_2$) was kept within the range of 30 to 35 mmHg. The operation lasted for 45 minutes and the estimated blood loss was about 150ml. After pyridostigmine 20 mg and glycopyrrolate 0.4 mg were given to reverse neuromuscular block, there was a sudden decrease in SpO_2 to 39% and ABP to 59/38 mmHg, associated with rapid ventricular response (heat rate up to 220 bpm). The patient was tilted to a head-down position, ventilated with 100% oxygen and hydrated with crystalloid solution. As profound hypotension persisted (ABP 55/33 mmHg, heart rate of 199 bpm, and SpO_2 80%) despite two bolus injections of phenylephrine 50 μg, a single synchronized cardioversion of 50 J was applied and restored relatively stable hemodynamics (ABP 128/69 mm Hg with AF, heart rate of 154 bpm, and SpO_2 94%). No decrease in $ETCO_2$ was observed during the hypotensive episode. ABGA showed pH 7.373, PaO2 573.1 mmHg, and PaCO2 43.7 mmHg with a base deficit of 0.2 mmol/L on FiO_2 1.0. As ABP was normal without any hemodynamic support, amiodarone 150mg was infused after securing central venous line in the right internal jugular vein (IJV) for ventricular rate control.

The patient was sent to surgical intensive care unit (ICU) under sedation, intubated and had been consulted to cardiologist. He commenced subcutaneous enoxaparin 40 mg for routine thromboprophylaxis after hip fracture surgery. Three hours after arrival in the ICU, he developed profound hypotension again (ABP 47/36 mmHg and heart rate of 73 bpm) and norepinephrine infusion of 0.4mcg/kg/min norepinephrine was started and the rate of the infusion was adjusted to maintain the arterial SBP >80 mm Hg. Laboratory tests revealed a normal cardiac troponin I levels but mild anemia (hemoglobin level of 12 g/dL on preoperative, which dropped to 9.2 g/dL on postoperative.). ECG showed sinus rhythm with heart rate of 85 bpm. Under clinical suspicion of hypotension due to hypovolemia, blood products and fluids were actively administrated along with norepinephrine infusion.

On postoperative day (POD) 1, despite fluid and blood resuscitation and vasopressors, hemodynamic fluctuation was persisted (the patient's arterial SBP sometimes dropped to between 60 and 70 mmHg). Laboratory tests demonstrated elevated CK-MB of 8.21 ng/mL, troponin I of 709.6 pg/mL, and pro-BNP of 6854 pg/mL while chest X-ray revealed no signs of heart failure. A portable TTE to determine the cause of hypotension showed dilated right atrium (RA) and right ventricle (RV), D-shaped left ventricle (LV), and septal wall motion abnormalities consistent with RV pressure overload (McConnell's sign) (Figure 2). The estimated RV systolic pressure increased from 32 to 41 mmHg.

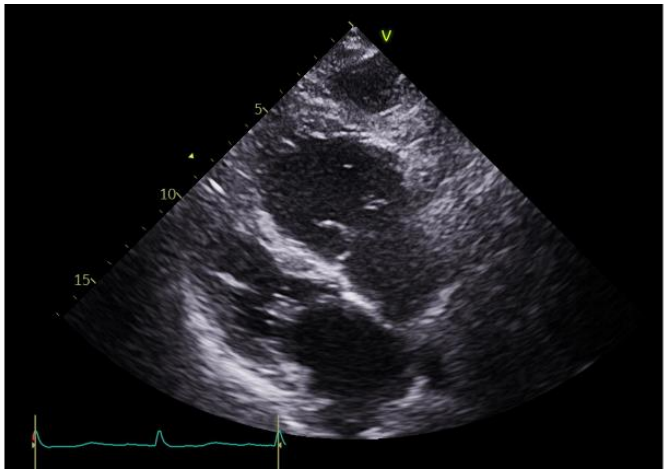

Figure 2: A portable transthoracic echocardiography on postoperative day 1

On POD 2, it was able to maintain the patient's SBP above 90mmHg with supportive care. With the suspicion of acute pulmonary embolism (PE), computed tomography (CT) pulmonary angiography was performed. The CT showed multifocal pulmonary thromboembolism in both main pulmonary artery (PA), lobar, segmental or subsegmental PA (Figure 3). Immediately, anticoagulant treatment (enoxaparin 0.6 mg/kg subcutaneous every 12 hours) was subsequently started. On POD 4, stable hemodynamics were achieved without need of vasopressor. The dose of enoxaparin was increased to 60 mg per day. On POD 5, the patient extubated without complications, high flow oxygen therapy was applied in the following days because of a mild pneumonia that gradually resolved. On POD7 follow-up TTE showed decrease RA, RV, and decreased RV systolic pressure (27 mmHg) and D-shaped LV was no longer shown. On POD 24, the anticoagulant medication was changed to edoxaban according to clinical decision of cardiology. The patient was transferred to the general ward on POD 17, the subsequent course was uneventful and he was discharged from the hospital on POD 30.

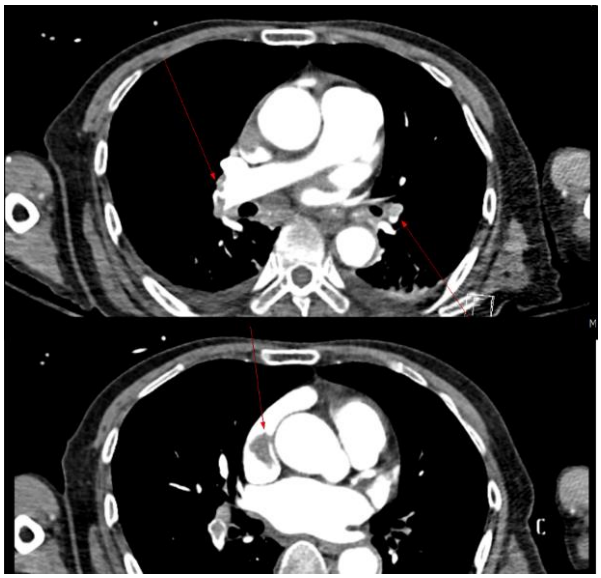

Figure 3: computed tomography pulmonary angiography on postoperative day 2

DISCUSSION

Abrupt hemodynamic collapse such as hypotension, tachycardia, and hypoxemia are the most

common findings in patients receiving general anesthesia [2]. However, peri-operative setting, the most important clinical manifestation for PE is an abrupt decrease in $ETCO_2$ values [7]. Many case reports of intraoperative PE have also described sudden changes in $ETCO_2$ as a presenting symptom [7-10]. ETtCO2 and its gradient with $PaCO_2$ is a known measure of alveolar dead-space [11]. In PE, emboli totally impede blood flow to pulmonary capillaries, resulting in the creation of a lung compartment that is ventilated but not perfused, causing an acute increase in alveolar dead space and an abrupt decrease in the $ETCO_2$ value [12,13]. Compared with hemodynamic collapse, decrease in the $ETCO_2$ value as the presenting sign of massive peri-operative PE may be associated with a better prognosis because it may represent earlier detection and allow for more time to intervention [5].

However, even in the absence of a sudden change in $ETCO_2$, as in our case, the possibility of PE cannot be completely excluded. A recent meta-analysis investigates the utility of $ETCO_2$ values as a screening tool of PE, revealing much lower accuracy with a high number of false positives and false negatives, showing that PE cannot be excluded purely based on $ETCO_2$ values [6]. Previous research also has demonstrated that that $ETCO_2$ is a reliable screening technique in excluding PE only when used in conjunction with bedside prediction (when combined with low pretest probability risk of PE in the Wells score) [14]. Meanwhile, patients who undergo surgery are at a higher risk of developing PE [2]. Surgical patients have various patient-specific risk factors for PE, in addition to risk factors unique to the perioperative experience, including as acute inflammatory reaction produced by tissue trauma, activation of the clotting cascade, and immobilization/venous stasi [15,16]. Furthermore, it has been found that patients undergoing hip fracture surgery are particularly vulnerable to PE [2]. Therefore, even if $ETCO_2$ does not alter in patients with intra-operative hemodynamic collapse, such as our patient, PE should not be ruled out.

Transesophageal echocardiography (TEE) is another perioperative monitoring tool that has been recommended for quickly verifying the diagnosis of intra-operative PE [5]. We were unable to employ TEE to determine the cause of the hemodynamic collapse because there was no TEE in the operating room. The use of TEE enables the diagnosis of PE through the direct observation of pulmonary emboli or secondary indications of acute PA obstruction, such as acute RV dysfunction or interatrial septum bowing to the left [17,18]. TEE is also helpful in making alternative diagnoses, including those for aortic dissection, pericardial disease, hypovolemia, myocardial dysfunction/infarction, and valvular insufficiency, [19,20] as well as guiding therapy in the resuscitation of hemodynamically unstable patients [21].

Meanwhile, Rosenberger *et al.* found an embolus on TEE in just 26% of patients with severe PE, [18] implying that failure of TEE to directly demonstrate embolus within the PA circulation should not exclude a PE. Because indirect echocardiographic evidence of PA obstruction was detected in 98% of tests in Rosenberger *et al*.'s study, indirect echocardiographic data may support the diagnosis of PE. However, these secondary echography signs are not unique for PE and do not allow for a trustworthy separation from other potential causes of RV dysfunction. Therefore, intraoperative TEE can be a useful tool in the diagnosis of PE only when there is clinical suspicion.

Another reason we didn't suspect PE early on is distraction from focusing on AF with rapid ventricular response. Both the cause and the effect of PE may be the existence of AF in patients with PE [22]. The elevated RV afterload observed in some PE patients may result in AF. On the other hand, PE and atrial appendage thrombus development might result from AF. Risk factors for PE and AF overlap, such as advanced age, obesity, high blood pressure, and heart failure. It is not uncommon for them to coexist. Clinicians should be highly suspicious of this coexistence. Many clinical symptoms, such as palpitations, shortness of breath, syncope, hypoxia, and chest discomfort, can occur in both AF and PE, and treating simply AF might hide the PE, as it did in our case, leading to a delay in diagnosis. As a result, it is crucial for clinicians to be aware of the relationship between AF and PE because it has consequences for both clinical and prognostic factors.

PE can happen in many different peri-operative situations and is frequently linked to high rates of morbidity and mortality. Prior to the implementation of specific treatment options, a reliable and prompt diagnosis of PE is essential to ensure minimal morbidity and death. Clinical suspicion is crucial because no perioperative monitoring can accurately diagnose severe intraoperative PE. In the event of hemodynamic collapse, suspicion of PE should be maintained over the perioperative period.

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