Letter to the Editor

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Fatal pulmonary thromboembolism during total hip replacement under spinal anesthesia

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The development of pulmonary thromboembolism (PTE) in high risk patients during surgery is not uncommon. Although it is difficult to make a prompt diagnosis due to its nonspecific symptoms, it is imperative to make an early diagnosis and provide proper treatment for a good prognosis. In our case, PTE developed in a high risk patient even after preventive measures of venous thromboembolism (VTE) during total hip replacement (THR) under spinal anesthesia.

An 86-year-old male patient, weighing 79 kg, with a height of 165 cm was admitted for the treatment of subluxation of the right hip for which he had undergone revision THR 5 years ago. He was on hypertensive medication. After reduction and 12 days of absolute bed rest, we decided to carry out re-revision THR of the right hip because the pain did not subside. Considering the patient's old age, the 11 days of immobilization after the reduction, and the several risk factors for VTE (obesity, THR, etc.) that were present, graduated compression stockings were applied 4 days prior to the surgery and 40 mg of enoxaparin was administered 12 hours before the operation. Preoperative laboratory findings and examinations were normal except for mild aortic regurgitation on echocardiography and mild fatty liver on abdominal ultrasonography. Before initiating spinal anesthesia, the initial blood pressure (BP) was 160/90 mmHg, heart rate (HR) at 70 beats/min, and pulse oxygen saturation (SpO_2) was 99% with an O_2 2 L/min via nasal prongs. After achieving adequate spinal anesthesia, major surgical procedures were carried out without significant events and the estimated blood loss was 900 ml, and 2 pints of packed red blood cells were transfused. During skin closure, the BP decreased to 60/40 mmHg and the HR increased to 120 beats/min. The SpO_2 abruptly decreased from 99 to 78%. The patient was tachypneic and complained of chest pain. Soon after, his mental status quickly deteriorated. Ten L/min of 100% oxygen via facial mask was applied and fluid resuscitation was initiated. The patient's position was changed to supine as soon as the skin closure was completed. Meanwhile, ephedrine (total of 25 mg) and phenylephrine (100 ug) were injected and invasive arterial BP measurement was started through the radial artery. The BP increased to 80/50 mmHg and HR was 110 beats/min. Arterial blood gas analysis (ABGA) results were pH 7.29, $PaCO_2$ 69.8 mmHg, PaO_2 61 mmHg, and SaO_2 81%. After intubating the patient, dopamine (15 μg/kg/min) and epinephrine (0.1 μg/kg/min) infusions were initiated. BP, HR, and end-tidal CO_2 ($ETCO_2$) were checked 15 minutes after intubation and they were 140/90 mmHg, 120 beats/min and 28 mmHg, respectively. ABGA results were pH 7.36, $PaCO_2$ 41 mmHg, PaO_2 138.6 mmHg, and SaO_2 98.2%. Under suspicion of PTE, trans-esophageal echocardiography (TEE) was carried out and emboli in the right atrium with bulging of the right atrial wall intothe left atrium were noted. After transferring the patient to the intensive care unit (ICU), the ventilator was set to the following: FiO_2 1.0, synchronized intermittent mandatory ventilation mode 12 breaths/min, and tidal volume 600 ml. ABGA done at the ICU did not improve; pH was 7.26, $PaCO_2$ 58 mmHg, PaO_2 68 mmHg, and SaO_2 86.2%. Thus, heparin therapy was initiated 2 hours after the operation (5000 IU bolus injection followed by 800 IU/hr to maintain 1.5–2 times the normal activated partial thromboplastin time level). This resulted in an improvement of the ABGA results: pH was 7.37, $PaCO_2$ 46

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mmHg, PaO_2 110 mmHg, and SaO_2 97%. 5 days later, the endotracheal tube was removed and 5 L/min oxygen via a face mask was initiated. Fourteen days later, no thrombosis was found on spiral computer tomography (CT) and the heparin infusion was stopped. Seventeen days later, the patient's vital signs were stable and thus, he was transferred to the general ward.

PTE is a relatively common cardiovascular emergency. VTE is a broad concept which includes PTE and deep vein thrombosis (DVT). One investigation done in the US claims that 5 million cases of DVT develop every year and 10% of these cases causes PTE and 10% of the patients die from it [1]. VTE prevention can be divided into non-pharmacologic (stockings and pneumatic compression devices) and pharmacologic treatment (low-dose subcutaneous unfractionated heparin,low molecular weight heparin, and fondaparinux). Clinical findings of PTE include dyspnea, chest pain, cough, syncope, hemoptysis, high fever, tachycardia, hypotension, and tachypnea. ABGA results may show respiratory alkalosis, hypoxemia, increased arterial to $ETCO_2$ gradient, etc. Righini et al. [2] reported that only 20% of clinically suspected patients are finally diagnosed as having PTE. When PTE is clinically suspected and the patient is hemodynamically stable, with anincreased D-dimer, the diagnosis can be made through multidetector CT scanning or ventilation-perfusion scanning. If the patient is hemodynamically unstable, immediate multidetector CT scanning is performed for the PTE diagnosis. Multidetector CT has 97% sensitivity in finding main pulmonary artery emboli [3]. As in our case, if the CT is not a viable option due to the patient's condition, bedside TEE can be used to make the diagnosis. Pruszczyk et al. [4] reported that diagnosing PTE with TEE compared to diagnosis with angiography results in 100% sensitivity and 80% specificity, and compared to CT, results in 90% sensitivity and 100% specificity. If a patient is hemodynamically unstable and has clinical and laboratory findings that are suspicious for PTE, appropriate and timely management must be performed even if emboli are not found and only indirect signs such as wall dysfunction, tricuspid valve regurgitation, and right to left interseptal bowing are noted. When PTE develops, it causes acute RV failure which may result in death due to systemic output loss. Thus, hemodynamic and respiratory support in a PTE patient is very important. The mainstay of PTE treatment includes anticoagulation, pharmacological thrombolysis, and mechanical thrombolysis. In hemodynamically unstable patients, aggressive treatment such as pharmacologic or mechanical thrombolysis must be performed. Intracranial disease, uncontrolled hypertension, recent major surgery (within 3 weeks) and trauma are contraindications of thrombolytic therapy. Since the early diagnosis and treatment of PTE is essential for a good prognosis, we must always take into consideration the possibility of PTE when non-specific clinical symptoms (dyspnea, hypotension, chest pain, etc) develop, and adequate diagnostic tools must be utilized, followed by proper and prompt management.

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