Case Report

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TANAFFOS

Pulmonary Thromboembolism Following Spine Surgery: Clinical Suspicion is the Key

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Pulmonary thromboembolism following spine surgery, although rare, could end into devastating outcome. Gold standard for it diagnosis is pulmonary CT angiography but in operating theatre, clinical suspicion is the key to diagnose. Here we report a case of pulmonary embolism with classic clinical findings which approved using pulmonary CT angiography and echocardiography.

Key words: Pulmonary embolism, Venous thromboembolism, Spine surgery, Pulmonary CT angiography, Qanadli score

INTRODUCTION

Although perioperative pulmonary embolism (PE) is an uncommon event, it can challenge medical team and patient. It can manifest as an asymptomatic incidental finding or a fatal acute catastrophe. Immediate postoperative PE is an uncommon event and can be devastating.

CASE SUMMARIES

A 58 year old male known case of ankylosing spondylitis (AS) since 5 years ago was candidate for cervical spinal laminectomy and fixation due to post-traumatic fraction of the fourth cervical vertebrate in prone position. All preoperative laboratory data including ECG and chest x-ray were acceptable. Cardiologist consult predicted low risk of cardiac events. Fiberoptic bronchoscopy was performed as the patient was awake and in a semi sitting position. Nasal phenylephrine drops, nebulizing lidocaine through nasopharyngeal cavity and recurrent laryngeal nerve block with lidocaine were used to prepare the patient for the procedure. Following successful intubation, intravenous anesthetics were injected. Monitoring including ECG, pulse oximetry (SpO_2), capnometry, invasive/noninvasive blood pressure, airway pressure, and FiO_2 were attached. The surgery took place in prone position without any hemodynamic instability throughout the procedure and lasted for 10 hours. Estimated blood loss volume was near 2000 milliliters (Hb=16 g/dL, 2 units of packed RBCs were transfused). Serial Arterial blood gas analysis reports were all in acceptable range.

At the end of surgery, patient started spontaneous breathing, reversed accordingly with neostigmine and atropine. While monitored by the pulse oximeter, the position of the patient was slowly changed into lateral decubitus and kept there for one minute to avoid severe cardiovascular changes. Then, he was turned into supine position successfully. Relating the type of surgery and the preoperative neck range of motion we planned for awake extubation. Five minutes in supine position, he was alert

and obeying with acceptable leak test and the nasotracheal tube was discarded. Concerned about airway patency, the patient was monitored in the operating theatre for 10 more minutes. However, he suddenly became unconscious, desaturated and end-tidal CO_2 ($ETCO_2$) dropped significantly despite of present deep spontaneous breathing. Due to concomitant bradycardia, 0.5 mg atropine was injected, followed by 10 micrograms of epinephrine. Infusion of crystalloid was started and the patient became conscious. Three minutes later the former scenario repeated and this time we decided to intubate the patient using video laryngoscope and also administered one more of the same dose of intravenous epinephrine. After intubation, arterial blood sample obtained for analysis showed metabolic and respiratory mixed acidosis; so 50 meq of sodium bicarbonate was infused for compensation. Regarding acute changes of capnograph, while the lungs were clear in auscultation and effective spontaneous breathing and increased arterial CO_2, we planned to transfer the patient for emergent CT angiography, suspecting pulmonary thromboemboli. Simultaneous 12 lead ECG was recorded (Figure 1), and showed bradycardia at first and after intervention, heart rate was in normal range but in both traces, S1Q3T3 pattern was obvious.

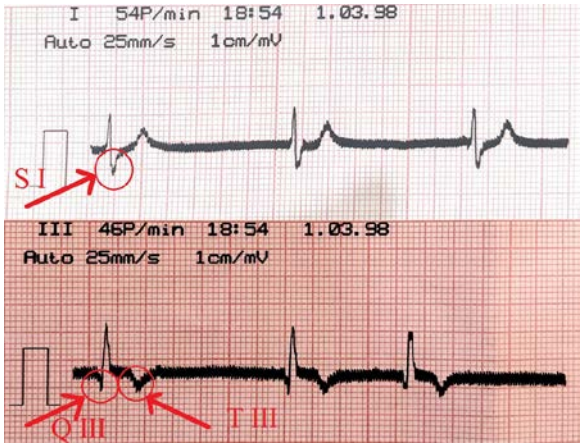

Figure 1. Electrocardiogram during the phase of bradycardia showing S1Q3T3 pattern, indicating right ventricular raised pressure and wall strain

After infusing 1000 ml of normal saline and 500 ml of ringer, with stable vital signs patient was transferred to CT angiography which confirmed the diagnosis (Figure 2-4). Bed side echocardiography in ICU again approved the diagnosis and anticoagulant therapy and norepinephrine infusion was started. Six hours later, he was extubated with stable hemodynamic without any neurologic sequels.

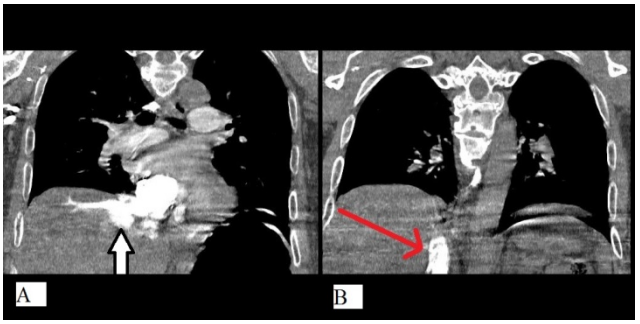

Figure 2. Coronal views of pulmonary CT angiography (with PTE protocol)
A. Reflux of contrast material into the hepatic veins representing right ventricle pressure elevation which can be a sign of pulmonary embolism.
B. Reflux of contrast material into the lower parts of inferior vena cava, indicating right ventricular strain.

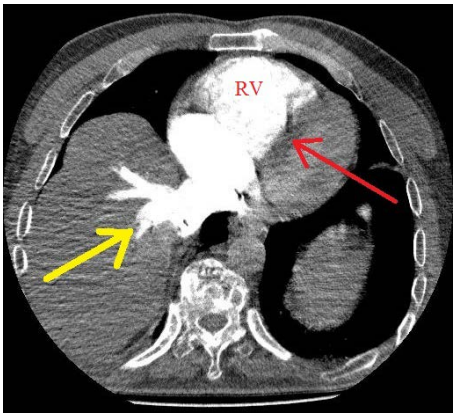

Figure 3. Red arrow points to straitened ventricular septum due to right ventricular (RV) strain which was confirmed later using echocardiography. Yellow arrow points to a back flow of contrast material into the hepatic veins.

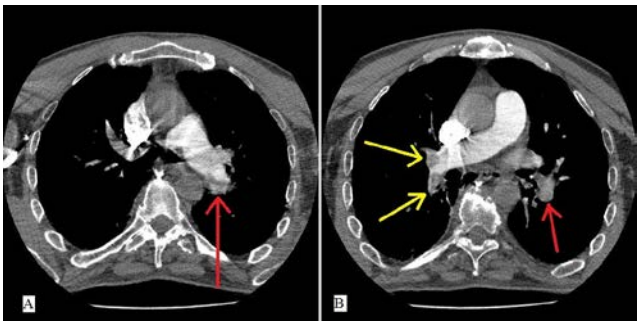

Figure 4. Horizontal views of pulmonary CT angiography (with PTE protocol). Left main pulmonary artery (A, red arrow) and right pulmonary artery (B, yellow arrow) are partially obstructed by thrombosis and distal branches of left pulmonary artery (B, red arrow) are completely blocked. All these findings reflect Qanadli score of more than 20 out of 40. Qanadli index of greater than 40% provides an accurate method for diagnosis of massive pulmonary embolism.

DISCUSSION

Asymptomatic venous thromboembolism (VTE) after spine surgery has been reported in about 1% of cases (1), while symptomatic VTE are rare (2). Venous thromboembolic events comprise two distinct entities: deep vein thrombosis (DVT) and pulmonary embolism (PE). These two entities are not necessarily concomitant. Predisposing factors are length of surgery, concurrent diseases, complexity of surgery, and obesity (3-6). Rheumatoid arthritis (RA) has been shown to be a predisposing factor itself (7). Our patient was not a known case of RA but suffered severe AS, an inflammatory disease which may be related to increased propensity of thromboembolism. Steroids have been reported as an independent risk factor for VTE among patients undergoing spinal surgery (8). This could be another underlying component in our patient's susceptibility to VTE.

Length of surgery, as a distinctive predisposing factor (9), may be related to different variables such as surgeon's expertise, complexity of spinal deformity and inappropriate devices. In our case the supplier of devices could not timely secure appropriate instruments for the surgery resulting in unusual prolonged surgery.

Confronting massive pulmonary embolism, physicians expect a tumultuous clinical presentation. However, in our case, despite the relatively vast emboli which led to rise in pulmonary artery pressure and interventricular septal straightening (Figure 3) and even backflow of the dye into inferior vena cava (Figure 2), the patient responded quite well to minimal cardiovascular support. We reported a similar case with massive PE after lumbar spine instrumentation who did not respond well to anticoagulant therapy and unfortunately passed away following paradoxical emboli to the brain (10). Of importance is early suspicion and detection of the emboli.

Not only there is no consensus on risk factors of VTE following spine surgery (1, 3, 4, 11), there is no common approach towards VTE prophylaxis among different authors. While Cox believed in decreased rate of VTE by chemical prophylaxis, McLynn and Senker did not report any difference in incidence of VTE (3, 12, 13). Even when decreasing incidence of deep vein thrombosis (DVT), prophylaxis may not change the mortality rate (14).

The only proposed anesthesia related factor which may decrease the incidence of VTE is epidural anesthesia/analgesia if it does not interfere with implementation of prophylaxis; on the other hand, general anesthesia, especially in orthopedic procedures, is considered as a predisposing factor for VTE (15).

Most of the literature is focused on post-operative chemical prophylaxis, whilst in cases like ours, PE manifests right after the surgery inside the operating theatre. The available data do not provide concrete material about such condition. Starting VTE prophylaxis would not be applicable in this situation. Concerning the nature of this procedure and risk of bleeding, preoperative chemical prophylaxis is not an option and the only available tool is intermittent pneumatic compression (IPC) device. Meanwhile, IPC did not prevent massive PE in any of our reported patients.

Medical team must be vigilant and suspect PE in spine surgery as the cause of acute hemodynamic changes. In operating theatre with no access to diagnostic tools such as CT angiography, clinical findings such as discrepancy between suddenly decreasing $ETCO_2$ and arterial CO_2, decreased SpO2 despite efficient ventilation and high FiO2 and inexplicable tachycardia, could be valuable clues for early clinical diagnosis of PE.

We recommend complex spine surgeries to be performed in centers which are equipped with efficient diagnostic instruments (e.g. CT angiography) and skilled specialists.

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