Fatal venous air embolism during lumbar spondylolisthesis surgery

Sir,

Perioperative venous air embolism (VAE) and cardiac arrest have been reported during spine surgery in the prone position; however, reports of VAE and cardiac arrest in knee-chest prone position for spondylolisthesis surgery are not very common. We report a case of fatal VAE in a 65-year-old woman, scheduled for spondylolisthesis surgery at L4-L5 level. She was on daily oral bisoprolol 2.5 mg and ramipril 5 mg for hypertension. Preoperative assessment revealed class II dyspnoea of New York Heart Association (NYHA), besides normal cardiac and neurological examinations. A 12-lead electrocardiogram (ECG) and transthoracic echocardiography (TTE) were unremarkable. Laboratory tests were normal. The patient had standard monitoring: non-invasive blood pressure (measured every 3 min), pulse oximetry, 5-lead ECG and capnography. Induction of anaesthesia was achieved with titrated intravenous doses of fentanyl (4 µg/kg), propofol (100 mg) and rocuronium (40 mg). Anaesthesia was maintained with rocuronium (10 mg/h), sevoflurane and fresh gas flow 4 l/min (air:oxygen = 1:1) using a semi-closed circle system. Then, the patient was positioned knee-chest prone. The surgical procedure consisted of transpedicular screws, bilateral rods and the placement of a cage following laminectomy with discectomy. It was going uneventful with the following spirometry and capnography parameters: baseline peak pressure 19 mmHg, positive end-expiratory pressure (PEEP) 5 mmHg, end-tidal carbondioxide (EtCO₂) 33 mmHg and peripheral oxygen saturation (SpO₂) 98%. Blood loss was about 300 ml and occurred predominantly during the release of spinal and vertebral gutters. During the placement of rods, EtCO₂ dropped suddenly to 20 mmHg, systolic blood pressure fell from 105 to 65 mmHg and heart rate (HR) remained unchanged at 80 beats/min. The procedure was interrupted immediately. Then, the fraction of inspired oxygen (FiO₂) was increased to 100% with intravenous bolus of isotonic saline and ephedrine. Given the cardiovascular history and the risk of poor tolerance of blood loss, the patient was transfused with packed red cells. After multiple doses of ephedrine, continuous infusion of norepinephrine (0.1 μg/kg/min) was promptly started via a peripheral venous catheter. Despite these measures, EtCO, continued to fall to 10 mmHg, ECG rythm remained constant with a widening of QRS complex and carotid pulse became non-palpable. The wound was covered with protective surgical drapes and the patient was turned supine and cardiopulmonary resuscitation (CPR) was started. The return of spontaneous circulation (ROSC) was achieved after 2 doses of intravenous epinephrine (1 mg each). Subsequently, a point-of-care TTE was performed and intracardiac air emboli were visualised [Figures 1 and 2]. Then, a right jugular central catheter was placed, but aspiration did not evacuate air bubbles. At the same time, a radial arterial line was secured. Arterial blood gas indicated: PaCO₂ of 52 mmHg, PaO₂ of 181 mmHg (FiO, 100%), PaCO, EtCO, gradient of 20 mmHg, haemoglobin level of 12.2 g/dL. Haemodynamic parameters were stabilised with norepinephrine via central line.

The patient was then put once more in the prone position uneventfully and surgical closure of the wound was done. She was then safely rolled back and transferred to intensive care unit, but ended up having cardiac arrest again with an unsuccessful resuscitation attempt. Autopsy was suggested, but the husband refused.

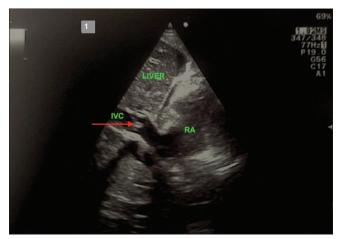


Figure 1: Subcostal echocardiographic view showing air bubble (red arrow) in the inferior vena cava (IVC)



Figure 2: Subcostal echocardiographic view showing air bubbles (red arrows) in the right atrium (RA)

Although blood loss is the common cause of acute haemodynamic instability in spine surgery, [1] in our case a normal haemoglobin level after blood transfusion does not support this diagnosis. Other probable differential diagnoses include dural traction leading to vasovagal response. The HR did not decrease and hence this cause is excluded. [2] Nevertheless VAE is frequently associated with tachycardia which was absent due to beta blocker usage. Additionally, pulmonary thromboembolism (PTE) can also be lethal in lumbar surgery. [3] Our patient had low risk scores of PTE and intraoperative intermittent pneumatic compression device was applied.

VAE might happen when there is a negative gravitational gradient between the surgical site and the right atrium with open blood vessels exposed to the atmosphere. Furthermore, abdominal pressures in the knee-chest prone position are low. [4] So the epidural veins are less engorged with blood and can become

relatively empty, especially in the hypovolaemic patient. Moreover, spontaneous respiration attempts can also increase the gravitational gradient and hence the risk of air entrainment. A sudden release of PEEP may also increase the rate of air entrainment.^[4]

Transesophageal echocardiography is the gold standard for VAE diagnosis; however, a point-of -care (POC) TTE coupled with ${\rm EtCO}_2$ and ECG changes seems to be useful in intraoperative cardio-circulatory failure to exclude other differential diagnoses. [5,6]

Hence, a careful preoperative cardiovascular assessment along with a high index of suspicion of VAE is required in patients undergoing lumbar surgery in knee-chest position for prompt diagnosis and better management.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. Consent is obtained from the husband for publishing the images and other clinical information in the journal. The husband understands that the patient name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

Youssef Elouardi, Youssef Zarrouki, Hasna Darouich, Mohamed Khallouki

Department of Anaesthesia and Intensive Care Medicine, Mohamed VI University Health Center, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco

Address for correspondence:

Dr. Youssef Elouardi.

Department of Anaesthesia and Intensive Care Medicine, Mohamed VI University Health Center, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco. E-mail: y.elouardi.rea@gmail.com

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