**PO34   AIR EMBOLISM IN HEPATIC SURGERY, A RARE BUT DEADLY COMPLICATION**

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Background

Venous air embolism (VAE) is the entrapment of air into the venous vasculature.1 Significant VAE is a life-threatening event with mortality as high as 28%.2 Although previously thought of as a rare complication, recent studies show VAE is far more common, with incidences as high 73% in major liver surgery.3 It is a clinical diagnosis and should be considered in the anesthetized patient, particularly in the trendelenburg position, with sudden severe hypotension, increased central venous pressure (CVP) and decreased end-tidal carbon dioxide (EtCO2) and arterial oxygen saturation (SaO2).1 The correct use of monitorization coupled with an understanding of the pathophysiology of VAE will enable the successful management of this dangerous event.

Case report

A 62-year-old woman, ASA III, with liver metastasis from colorectal cancer, was admitted for elective metastasectomy. Because of venous thrombosis related to a central venous catheter (CVC) placed for chemotherapy, the patient was on anticoagulants, which were stopped according to guidelines. After monitorization with standard ASA monitoring, Bispectral Index (BIS) and Train-of-Four (TOF), the patient received total intravenous anesthesia. Due to thrombosis at the entrance of the superior vena cava, it was decided to place a CVC in the femoral vein, knowing it would not be possible to measure CVP. An arterial line and noninvasive bioreactance were used for hemodynamic monitoring.

During hepatic resection, the right supra-hepatic vein was injured and opened to atmosphere. An immediate drop in EtCO2 (35 mmHg to 11 mmHg), SpO2 (98% to 75%) and mean arterial blood pressure (81 mmHg to 52 mmHg) accompanied by an increase in heart rate (88 bpm to 110 bpm) and appearance of supraventricular extrasystoles was noted. Arterial blood gas analysis showed pCO2 45 mmHg. Embolism was immediately suspected, and vasopressor support and 100% FiO2 were implemented. The surgical team promptly repaired the injured vessel and, associated with the anesthetic measures, the patient stabilized. Given that the patient's condition stopped deteriorating after repairment of the injured vein, the suspicion of VAE as the culprit for this event increased. The patient lost 800 ml of blood during the procedure.

After surgery, the patient was admitted to intensive care. Extubation was possible after 12 hours and vasopressor support was stopped, with complete recovery.

Discussion

Although definite diagnosis of VAE requires visualization of air bubbles on echocardiography, we present a case of high suspicion for VAE. The execution of transthoracic echocardiography during the procedure was difficult due to positioning and type of surgery and a transesophageal approach was not possible. We lament that CVP was not monitored, but after careful consideration of risks and benefits, it was decided not to manipulate the thrombosed area. The small amount of blood loss made hemorrhagic shock an unlikely cause. Also, such major cardiovascular and respiratory changes could only be caused by a massive thrombus, which likely would not have come close to resolution by the end of surgery. Taking all of this into account, VAE becomes the most likely cause for this event.

Immediate recognition and action are extremely important for patient survival during major adverse events. This case serves as a reminder of a not so rare, but potentially deadly complication.

References

1. Anesthesiology 2007 106:164–177
2. Sci Rep 12 20487 2022
3. Eur J Anaesthesiol 2014 Feb;31(2):120-1

