What Is the Best Management Strategy for Venous Air Embolism?

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Case

A tall, slender 24-year-old male sustained a single gunshot wound to the upper abdomen while attempting to wrestle his bicycle from a thief. On arrival to the emergency department, he was pale, unconscious, and flaccid with a heart rate on telemetry of 160 beats per minute and barely palpable carotid pulses. He was rushed to the operating room, moved to the operating table in a supine position, ventilated with 100 % oxygen, and tracheally intubated. Breath sounds were equal and clear. The patient was placed on volume-control mechanical ventilation with 600 ml tidal volumes and peak inspiratory pressures of 23 mm Hg. Scopolamine 0.6 mg and pancuronium 8 mg were the only drugs administered.

Despite the presence of a scaphoid abdomen, intraperitoneal and retroperitoneal bleeding was quickly ruled out during an emergency exploratory laparotomy. A left thoracotomy revealed tense pericardial tamponade. The pericardium was incised, and the pumping hole in the anteroinferior right ventricle was closed with a purse string suture. Circulatory function immediately returned permitting administration of normal doses of intravenous and inhalational anesthetic agents (fentanyl, isoflurane). An arterial line was inserted with the first blood gas showing normal oxygenation and a 12 mEq base deficit. The latter was treated with 200 mEq sodium bicarbonate.

The head surgeon breathed a sigh of relief and quickly moved on to the surgical exploration for bleeding vessels prior to chest closure. Less than 10 min later, the normal-appearing, vigorously contracting heart suddenly began to dilate and lose all efficacy at pumping blood. End-tidal carbon dioxide (CO2) dropped precipitously from 36 to 5 mm Hg. Inhalation anesthesia was discontinued. Equal bilateral breath sounds were auscultation, and normal inspiratory pressures were confirmed. No new surgical bleeding was identified.

Although the telemetry monitor showed normal sinus rhythm, the arterial pressure waveform was flat at 25 mm Hg and no carotid pulses could be palpated. Open chest cardiac massage was begun and epinephrine 1 mg administered intravenously. Despite vigorous manual squeezing of the heart, arterial systolic pressure remained below 40 mm Hg. Additional doses of epinephrine were not effective in elevating the arterial pressure, and carotid pulsations remained absent.

The anesthesiology resident said. "Let's use the Advanced Cardiovascular Life Support (ACLS) algorithm for causes of cardiac arrest, the '5 Hs and 4 Ts': Hypoxia, Hyperkalemia, Hypothermia, Hypoglycemia, H+ (acidosis), Trauma, Tension pneumothorax, and Thrombus (cardiac or air)' [1]. We haven't yet addressed the possibility of air embolism."

Saying "It's worth a try," the surgeon picked up a sterile 30-ml syringe with an 18-gauge needle attached. The needle was inserted into the right ventricle with subsequent evacuation of 60 cc of air, followed by aspiration of blood. The arterial pressure waveform immediately returned to normal. No source for air entrainment was identified.

Chest closure, chest tube placement, and patient transfer to the intensive care unit (ICU) were uneventful. The patient was stable overnight. The patient was later transferred to a neighboring private facility for continued rehabilitation and was thought to be lost to follow-up. However, 6 months later, he walked into the ICU to thank all those who had cared for him. He apologized for not coming sooner, his excuse being that he needed to first successfully pass all his examinations for engineering graduate school.

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Question

What is the best management strategy for venous air embolism?

PRO If you want to know the best management strategy for venous air embolism, it is imperative to first know what situations you can expect to see it in. VAE is classically associated with neurosurgical procedures performed in the sitting position, and occurs secondary to a gravitational gradient in 76 % [2].

CON That's great but just like anything in medicine, it is not always so cut and dry. Venous air embolism is not always associated with these common high-risk surgeries. Laparoscopic and minimally invasive procedures use gas for insufflation and exposure of the surgical field, leading to a risk for injection or entrainment of this gas into the venous system [1]. As an anesthesiologist, you have to be prepared for uncommon occurrences in common situations. This case is a prime example of that. Here, there was no clear source for introduction of air into the venous system. But, as is common in trauma cases such as this one, you may not have the time to fully identify the extent of the patient's injuries prior to the operating room. Especially in life-threatening hemorrhagic trauma, every second matters.

PRO One good thing about VAE is that it seldom results in clinically significant hemodynamic compromise; however, it naturally follows that many episodes occur without clinician awareness. While the majority of research has been done using animal models, the consensus is that clinically significant VAE in humans occurs between 3–5 ml/kg and 200–300 ml of air [2]. The rate of air accumulation is a key determinant in clinical significance [2]. Prompt identification of VAE is key. Currently, transesophageal echocardiography is the most sensitive monitoring device available with the ability to detect as little as 0.02 ml/kg of air [3]. Precordial Doppler has been shown to be the most sensitive noninvasive monitor for VAE detection [4].

CON The odds of you actually having the time or accessibility to use those methods in a situation in which venous air embolism is not suspected are slim to none. You can't lean on technologies that will waste time and delay proper treatment. The importance of the time from the onset of hemodynamic compromise to treatment can't be overstated. In this situation, the clinician successfully used clinical signs such as a drop in ETCO2 and loss of arterial waveform to successfully identify the problem. A drop of 2 mmHg of ETCO2 can signify VAE [2].

PRO Treatment of VAE is best accomplished by lowering the operative site below the level of the heart, flooding the operative field to prevent further air entry, and positioning the patient left side down and in Trendelenburg [5]. This helps to prevent further air entrainment into the venous system. The patient should also be placed on 100 % oxygen. Other rescue maneuvers include the initiation of cardiopulmonary resuscitation and chest compressions.

CON Actually, recent studies have refuted this conventional method of thinking [6]. Animal studies failed to show that repositioning to the left lateral position improved hemodynamic performance [6]. Keeping the patient in the supine position may actually be better for the patient because it allows for better access to perform chest compressions and other rescue maneuvers [6]. This case shows that the life-saving treatment is not always the one that is written about or studied the most in textbooks and papers. The quick actions of the anesthesiologist and surgeon allowed for rapid evacuation of the air embolus from the right ventricle and saved the young man's life.

Summary

Venous air embolism is an under-recognized and often insignificant event. However, when VAE leads to hemodynamic compromise, the clinician often only has seconds to identify the source (Table 112.1) and formulate a plan for treatment. More research needs to be done in order to

Table 112.1 Gremlids: anesthetic catastrophes, often assumed to be simple accidents, are actually the work of gremlids (anesthesia goblins)

Gremlids
Oxygen insufficiency (hypoxic mixture of inhaled gases)
Absolute versus relative to an individual patient's needs
Obstruction of the breathing system or the patient's airway
Anesthesia machine or circuit (peep valve, soda lime, HME, exhaust tubing, draeger muffler)
Upper airway (teeth, tongue, nasal turbinates, redundant pharyngeal tissue)
Larynx: epiglottis pushed down by oral airway or LMA tip; laryngospasm
Tracheal tube or LMA (mucus, blood, clot)
Trachea or major bronchus (mucus, blood, clot, tumor of airway or anterior mediastinum)
Distal airways: bronchospasm, air trapping
Overdosage or other drug administration error
Wrong inhalational or intravenous agent; exaggerated drug reaction; more concentrated drug (ketamine, lidocaine); look-alike drug
Intolerance (including allergic reaction) to drugs given by anesthesia, surgeon, perfusionist (including irrigation, methyl methacrylate, etc.)
Hypoventilation
Ventilator turned off
Major disconnection or breathing circuit leak
Tracheal extubation
Disconnection or major leak at corrugated circuit connection to machine or tracheal tube
Pop-off (APL) valve wide open during mechanical ventilation (older machines)
Soda lime canister seals not correctly mated
Gastric tube (Salem sump) in trachea, especially if connected to suction
Shock
Hypovolemic
Dehydration
Hemorrhage (especially concealed hemorrhage)
Cardiogenic
Myocardial infarction
Cardiac tamponade
Pharmacologic/toxicologic (local or general anesthetic overdose, beta blocker, methyl methacrylate, acidosis)
Arrhythmia (V-Tach—sux, ischemia, $\updownarrow K^+$, SVT or rapid atrial fib, bradycardia, heart block)
Obstructive (physical obstruction to circulation)
Cardiac tamponade (blood, effusion, air; e.g., from jet ventilation in presence of upper airway closure)
Inferior vena cava compression (pregnant uterus) or portal compression (surgeon)
Tension pneumothorax or air trapping from inadequate exhalation time
Embolism [air/other gas, (venous) thromboembolism]
Distributive/cytotoxic
Septic
Anaphylactic (drugs, blood product, latex)
Neurogenic (high spinal/epidural anesthesia, spinal cord injury, brainstem herniation)
Endocrine/metabolic (Addisonian crisis, thyroid storm, hypothyroidism/myxedema, carcinoid), ↓ ionized [Ca ++]—e.g., due to blood product administration, especially in patients with liver hypofunction: cirrhosis/thoracic aortic crossclamp)
Amniotic fluid embolism
Pharmacologic/toxicologic (IV or inhaled anesthetic agent, vasodilator, methyl methacrylate, acidosis)
Hyperthermia/hypothermia
Malignant hyperthermia

(continued)

Table 112.1 (continued)

Passive hyperthermia/hypothermia (e.g., massive transfusion with cold blood, especially with blood warmer off)
Hyperthyroid/hypothyroid
Intubation catastrophe
Esophageal intubation
Submucosal placement of nasal tube
Mediastinal intubation via perforation of pyriform sinus or airway tumor
Pre-tracheal misplacement of tracheostomy tube
Tension pneumothorax or other pulmonary catastrophe
Pneumothorax from disease, surgery, airway instrumentation, ventilator barotrauma/jet ventilation, subclavian puncture
Atelectasis (secretions, bronchial intubation, microatelectasis)
Aspiration (e.g., of gastric contents)
Asthma/bronchospasm/air trapping
Pulmonary edema (fluid overload, cardiogenic, non-cardiogenic; e.g., negative pressure)

The presence of a gremlid in the operating room can be deduced when, during the administration of a general anesthetic, the patient suddenly and unexpectedly becomes cyanotic, hypotensive, or difficult to ventilate
HME heat and moisture exchanger, *LMA* laryngeal mask airway, *APL* adjustable pressure limiting, *V-Tach* ventricular tachycardia, *K* potassium, *SVT* supraventricular tachycardia, *Ca* calcium, *IV* intravenous

validate a definitive treatment for clinically significant venous air embolism.

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