Research

Air embolism as a cause of the systemic inflammatory response syndrome: a case report

Tarun Kapoor1 and Guillermo Gutierrez2

1Chief Medical Resident, Department of Internal Medicine, The George Washington University, Washington, DC, USA
2Professor of Medicine and Director, Pulmonary and Critical Care Medicine Division, Department of Internal Medicine, The George Washington University, Washington, DC, USA

Correspondence: Guillermo Gutierrez, Ggutierrez@mfa.gwu.edu

Introduction

Tachycardia, tachypnea, fever or hypothermia and leukocytosis or leukopenia are the hallmarks of systemic inflammatory response syndrome (SIRS) [1]. Although SIRS is commonly associated with infectious etiologies, it also occurs in patients with noninfectious conditions, including trauma, burns, pancreatitis, anaphylaxis, adrenal insufficiency, pulmonary embolism, myocardial infarction, massive hemorrhage, and following cardiopulmonary bypass [2–4]. We describe a case of SIRS associated with air embolism following the removal of a central line catheter.

Case presentation

A 65-year-old male with adult immune deficiency syndrome (CD4+ cell count, 90), chronic obstructive pulmonary disease and hepatitis-related cirrhosis was admitted for a transjugular intrahepatic porto-systemic shunt procedure for recurrent bleeding from esophageal varices. The procedure was performed without complications.

The following afternoon, an internal jugular sheath used to gain access to the vena cava during the procedure was pulled in anticipation of discharge. Approximately 20 min later, and against medical advice, the patient went to the bathroom, where he collapsed while attempting to defecate. He was found on the floor, incoherent and not moving his extremities. Vital signs showed tachycardia (heart rate, 96 beats/min), tachypnea (28 breaths/min) and arterial blood pressure of 170/100 mmHg. A physical examination revealed an unconscious man with a weak gag reflex, with sluggishly reactive, 3 mm pupils and who was able to withdraw to pain. Chest auscultation revealed diffuse wheezes and crackles over both lung fields. The cardiac rhythm was regular and no murmurs were heard. The patient was orally intubated and placed on mechanical ventilation. A diagnosis of venous air embolism was made and the patient was taken to a hyperbaric chamber for treatment with 100% oxygen at 2.5 atm for 90 min [5,6]. Upon removal from the hyperbaric chamber, the patient’s blood pressure was 58/40 mmHg with a heart rate

PFO = patent foramen ovale; SIRS = systemic inflammatory response syndrome.
of 105 beats/min, and aggressive volume and vasopressor (norepinephrine) resuscitation was initiated.

A pulmonary artery was inserted approximately 20 hours after the air embolism episode. As shown in Fig. 1, initial hemodynamic measurements showed an elevated cardiac output at 8.2 l/min and a low systemic peripheral resistance of 460 dynes/s/cm². At that time the patient developed hematemesis and profuse bleeding both from a tongue laceration and from the internal jugular puncture site. Laboratory results confirmed a clinical diagnosis of diffuse intravascular coagulation. The patient was transfused several units of packed red blood cells, fresh frozen plasma, platelets and cryoprecipitate. Intravenous antibiotic therapy with vancomycin and imipenem was initiated for suspected sepsis. An interval physical examination revealed a right-sided hemiplegia. A patent foramen ovale (PFO) was noted by trans-thoracic echocardiogram.

Bleeding had stopped and volitional movement had returned to all extremities by the next morning. Over the next 24 hours, the patient’s hemodynamic parameters normalized and vasopressor support was discontinued. Forty-eight hours later the patient was weaned off the ventilator without difficulty and the antibiotics were discontinued. Several days later, he was discharged from the hospital with no neurological sequelae. Blood, urine and sputum cultures taken during hospitalization failed to grow pathogenic organisms.

Discussion

Air embolism is defined as the entry of air into the vasculature, and it can occur during the insertion or removal of central venous catheters [7]. For air to enter the venous circulation, there must be both a direct communication between the atmosphere and a noncollapsed vein and a pressure gradient favoring the passage of air into the circulation [8]. The patient described in the present report met these criteria by having a patent lumen from skin to the central vein formed by the internal jugular sheath and by developing a pressure gradient while taking a deep inspiration immediately before or after a Valsalva maneuver. Moreover, this patient exhibited symptoms compatible with arterial air embolism, implying that a large portion of air sucked into the central veins found its way into the left ventricle and the systemic circulation. The PFO provided the mechanism by which venous air passed into the systemic circulation, a condition defined as ‘paradoxic’ air embolism [9].

A noteworthy aspect of this case was the patient’s physiological response to the acute embolic event. Embolization of large quantities of air into the right ventricle usually results in pulmonary hypertension, a phenomenon that appears to be related to the release of endothelin-1 from the pulmonary vascular endothelium [10]. The rapid increase in pulmonary artery pressure leads to right ventricular decompression, to decreased left ventricular preload, and to a rapid decline in cardiac output with profound hypotension. These mechanisms may have been present immediately after the entry of
Air embolization can occur following the removal of a central venous catheter

A patent foramen ovale (PFO) allows air to pass from the venous to the arterial circulation (paradoxical air embolism)

Air may trigger the release of cytokines by the arterial endothelium resulting in the development of the systemic inflammatory response syndrome (SIRS)

Competing interests
None declared.

References
1. Muckart DJ, Bhagwanjee S: American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference: definition of the systemic inflammatory response syndrome and allied disorders in relation to critically injured patients. Crit Care Med 1997, 25:1789-1795.
2. Pittet D, Rangel-Frausto S, Li N, Tarara D, Costigan M, Rempe L, Jabotin P, Wenzel RP: Systemic inflammatory response syndrome, sepsis, severe sepsis and septic shock: incidence, morbidities and outcomes in surgical ICU patients. Intensive Care Med 1995, 21:302-309.
3. Abraham E, Matthay MA, D’Acquisto CA, Vincent JL, Cohen J, Opal SM, Glauser M, Parsons P, Fisher CJ Jr, Repine JE: Consensus conference definitions for sepsis, septic shock, acute lung injury, and acute respiratory distress syndrome: time for a reevaluation. Crit Care Med 2000, 28:232-235.
4. Rangel-Frausto MS, Pittet D, Costigan M, Hwang T, Davis CS, Wenzel RP: The natural history of the systemic inflammatory response syndrome (SIRS). A prospective study. JAMA, 1995, 273:117-123.
5. Dexter F, Hindman BJ: Recommendations for hyperbaric oxygen therapy of cerebral air embolism based on a mathematical model of bubble absorption. Anesth Analg 1997, 84:1203-1207.
6. Muth CM, Shank ES: Gas embolism. N Engl J Med 2000, 342:476-482.
7. McGee DC, Gould MK: Preventing complications of central venous catheterization. N Engl J Med 2003, 348:1123-1133.
8. Duran TM, Long J, Oppenheimer MJ: Pulmonary (venous) air embolism. Am Heart J 1947, 33:269-281.
9. Gronert GA, Messick JM Jr, Cocchiara RF, Michenfelder JD: Paradoxic air embolism from a patent foramen ovale. Anesthesiology 1979, 50:548-549.
10. Tanus-Santos JE, Gordo WM, Udelson A, Ciddorino MH, Moreno H Jr: Nonselective endothelin-receptor antagonism attenuates hemodynamic changes after massive pulmonary air embolism in dogs. Chest 2000, 118:175-179.
11. Lynch JJ, Schuchard GH, Gross CM, Wann LS: Prevalence of right-to-left atrial shunting in a healthy population: detection by Valsalva maneuver contrast echocardiography. Am J Cardiol 1984, 53:1479-1480.
12. Ryu KH, Hindman BJ, Reasoner DK, Dexter F: Heparin reduces neurological impairment after cerebral arterial air embolism in the rabbit. Stroke 1996, 27:303-310.
13. Matthay MA: Severe sepsis – a new treatment with both anti-coagulant and anti-inflammatory properties. N Engl J Med 2001, 344:759-762.
14. Hotchkiss RS, Karl IE: The pathophysiology and treatment of sepsis. N Engl J Med 2003, 348:138-150.
15. Evans DE, Catron PW, McDermott JJ, Thomas LB, Kobine AI, Flynn ET: Effect of lidocaine after experimental cerebral ischemia induced by air embolism. J Neurosurg 1989, 70:97-102.