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

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Abstract

We describe a case of systemic inflammatory response syndrome associated with air embolism following the removal of a central line catheter, coupled with a deep inspiratory maneuver. The presence of a patent foramen ovale allowed the passage of a clinically significant amount of air from the venous circulation to the systemic circulation. The interaction of air with the systemic arterial endothelium may have triggered the release of endothelium-derived cytokines, resulting in the physiologic response of systemic inflammatory response syndrome.

Keywords foramen ovale, hypotension, paradoxical air embolism, sepsis

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/cm^5. 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 transthoracic echocardiogram.

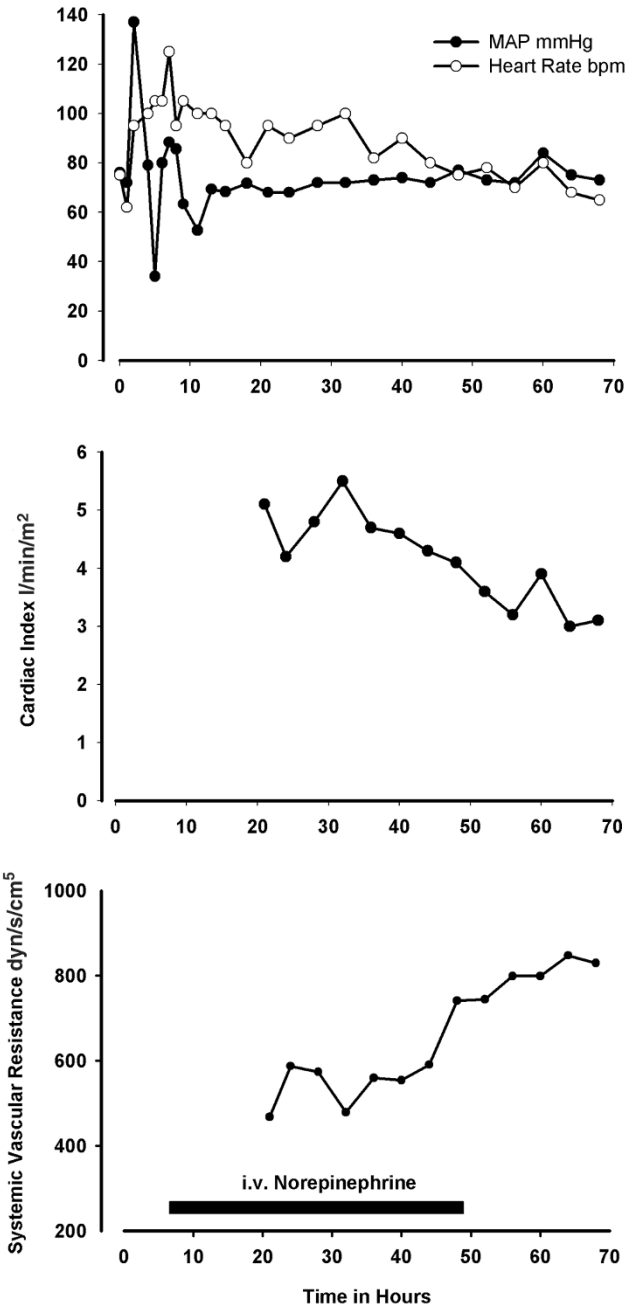
Bleeding had stopped and volitional movement had returned to all extremities by the next morning. Over the next 24 hours, the patient's hemodynamic parameters normalized and vasopressor support was discontinued. Forty-eight hours later the patient was weaned off the ventilator without difficulty and 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 'paradoxical' 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 decompensation, 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

Figure 1



Changes in hemodynamic parameters following the removal of the right internal jugular vein introducer (time = 0). There was an initial rise in blood pressure and in heart rate at the time of air entry into the circulation. This was followed by severe hypotension, which was treated with intravenous (i.v.) norepinephrine. At the time of insertion of a pulmonary artery catheter, approximately 20 hours after the removal of the introducer, the patient's cardiac index was elevated and the systemic vascular resistance was low. These parameters normalized during the next 2 days. MAP, mean arterial pressure.

air into the patient's circulation, but a few hours after the episode of air embolism the patient's hemodynamic and clinical condition (elevated cardiac output, decreased systemic vascular resistance, tachypnea, fever and diffuse intravascular coagulation) was compatible with the diagnosis of SIRS.

The rapidity of the patient's recovery, as well as the lack of positive cultures, suggest that sepsis was not the cause of his hemodynamic decompensation, although this possibility cannot be totally ruled out. Instead, we hypothesize that entry of air into the systemic circulation through the PFO, a condition present in approximately 30% of the population [11], must have triggered the release of inflammatory mediators that resulted in SIRS. Perhaps owing to the episode of air embolism being a single, self-limited event, the systemic response in this patient was relatively short lived and resulted in no permanent organ dysfunction.

Inflammatory states associated with systemic air embolism have been described in animal models [10,12]. Air can form microscopic bubbles that disrupt microvascular flow, resulting in platelet aggregation and the release of plasminogen-activator inhibitor [13]. This mechanism has been implicated as a trigger to the cascade of cytokines thought to be causative agents of SIRS, among them interleukin-1 and tumor necrosis factor [14]. The host response to these cytokines may include diffuse endovascular injury, microvascular thrombosis, organ ischemia, multiorgan dysfunction, and death. Animal studies suggest that agents such as heparin [10] and lidocaine [15] attenuate the thrombo-inflammatory response of the endothelium to luminal air.

In conclusion, the removal of an internal jugular vein sheath introducer in this patient, coupled with a deep inspiratory maneuver, allowed the passage of a clinically significant amount of air from the venous circulation to the systemic circulation through a PFO. The interaction of air with systemic arterial endothelium may have triggered the release of endothelium-derived cytokines, resulting in the physiologic response of SIRS. This complication of air embolism has not been previously documented in the clinical literature.

Key messages

- 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.

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