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Pulmonary Air Embolism: An Infrequent Complication in the Radiology Suite

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Statistical Analysis C
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Conflict of interest: None declared

Patient: Female, 64
Final Diagnosis: Pulmonary air embolism
Symptoms: Shortness of breath
Medication: —
Clinical Procedure: —
Specialty: Critical Care Medicine

Objective: Unusual or unexpected effect of treatment
Background: Air embolism can occur in a number of medical-surgical situations. Venous air embolism is frequently lethal when a substantial amount enters the venous circulation rapidly and can lead to significant morbidity if cross-over to the systemic arterial circulation occurs. The diagnosis of massive air embolism is usually made on clinical grounds by the development of abrupt hemodynamic compromise. The true incidence, morbidity, and mortality of this event is unknown given the difficulties in diagnosis.
Case Report: An inadvertent antecubital venous injection of 150 mL of air using a contrast power injector during a computed tomography (CT) is reported. Immediate imaging (CT) showed a significant amount of air in the right atrium and right ventricular cavity, and air mixed with contrast in the main pulmonary artery and proximal divisions of the pulmonary circulation. Patient condition deteriorated requiring mechanical ventilation for 48 hours. Condition improved over the next few days and patient was successfully extubated and discharged home.
Conclusions: Air embolism is a rare complication, the potential for this to be life threatening makes prevention and early detection of this condition essential. This condition should be suspected when patients experience sudden onset respiratory distress and/or experience a neurological event in the setting of a known risk factor. Treatment options include Durant's maneuver; left-lateral decubitus, head-down positioning; to decrease air entry into the right ventricle outflow tract, hyperbaric therapy, 100% O₂ and supportive care.

MeSH Keywords: Anoxia • Embolism, Air • Hyperbaric Oxygenation

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/901098>

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Background

Venous air embolism can occur in a number of medical situations including placement, use, and removal of intravenous catheters and devices, trauma, surgery, and gynecological interventions but can also occur in the radiology suite during angiography, needle biopsy, or pneumoradiographic procedures. Contrast enhanced computerized tomography (CT) using power injectors are an essential diagnostic tool for the pulmonologist but also provide a particularly high-risk situation for the accidental rapid injection of air into the venous circulation and single fatal event has previously been reported [1].

We report a case of inadvertent rapid injection of a large amount of air during a non-invasive radiological procedure with imaging capture as it traveled through the pulmonary circulation. Our patient's survival after the complication – air embolism – in the presence of significant concurrent cardiac stress from acute pulmonary thromboembolism, was remarkable. This case report also contributes to the controversy of the lethal dose and rate-of-entry of venous air embolism and how we can prevent this from happening.

Case Report

A 64-year-old female was found lying down after 10–12 hours of binge drinking. She was taken to an outlying emergency department (ED), and subsequently transferred to our hospital with mental status changes and respiratory distress and hypoxia for further management. She had a past history of rheumatoid arthritis, congestive heart failure, controlled hypertension, and alcoholism. She smoked two packs a day for the past 50 years. On presentation, she had a blood pressure of 176/80 mm/Hg, heart rate of 80 bpm, respirations 24 bpm, temperature 98°F (36.7°C). Arterial blood gases on room air showed a pH of 7.30, PaCO₂ 60, PaO₂ 61 mm/Hg, H⁺-CO₃ 29 mEq/L, and saturation 92% which improved to 95% with oxygen by a high flow non-rebreather mask. On physical examination she was lethargic, disoriented, dysarthritic, but without neurological focalization. Scattered expiratory wheezes were noted bilaterally along with normal heart sounds. The remaining results of her physical examination and routine laboratory results were unremarkable with the exception of a leukocyte count of 14,000/dL with 88% neutrophils. Repeated vitals signs were consistent with a normal blood pressure but evidence of tachycardia in the monitor. Repeated ABG's on the non-rebreather mask showed: pH 7.22, PaCO₂ 78, PaO₂ 140 mm Hg, H⁺-CO₃ 30 mEq/L, and hemoglobin SaO₂ 97%. In the ED she was started on IV steroids and antibiotics for a COPD exacerbation. As part of the diagnostic workup, she underwent a chest x ray that did not show any infiltrates or any major abnormal findings. The ECG was only significant for sinus tachycardia. Given

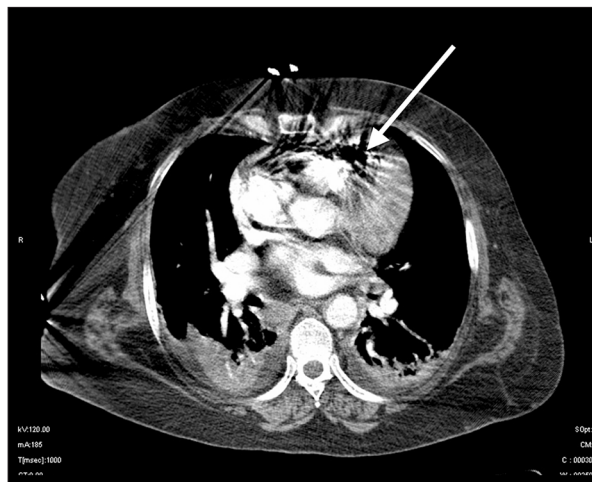


Figure 1. Air embolus in the right atrium and ventricle.

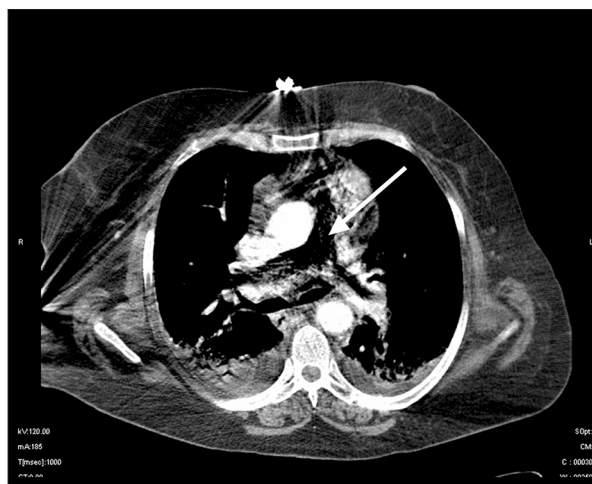


Figure 2. Air and contrast in the main pulmonary artery and major divisions.

the negative findings on chest x ray, sinus tachycardia on ECG, and an increased A-a gradient, the patient was sent for a spiral chest CT with contrast to rule out a pulmonary embolism.

During CT about 100–150 mL of air was inadvertently injected through the right antecubital vein using a power contrast injector (estimated by the technician and approximation of volumes on available imaging). Concurrent imaging (CT) showed a significant amount of air in the right atrium and right ventricular cavity (Figure 1), and air mixed with contrast in the main pulmonary artery and its proximal branches divisions of the pulmonary circulation (Figure 2). Concurrently, a filling defect was noted in the right lower lobe artery consistent with pulmonary thromboembolism (Figures 3, 4). The patient maintained hemodynamic stability with Trendelenburg, and left lateral decubitus positioning (Durant's maneuver), and supportive care alone and she was transferred to the intensive care unit (ICU) for observation. Her respiratory distress worsened,

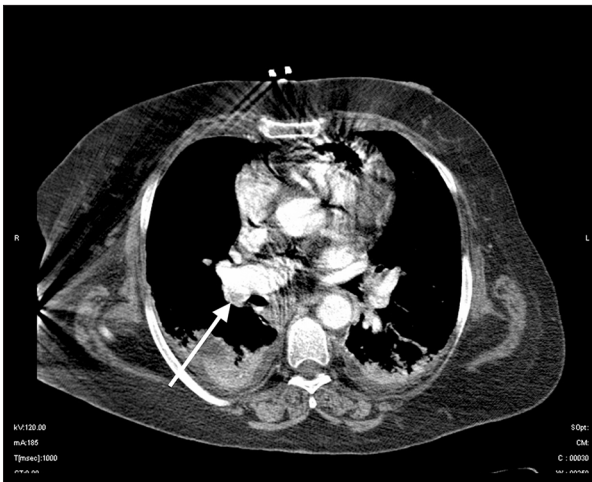


Figure 3. Filling defect in the right lower lobe branch.

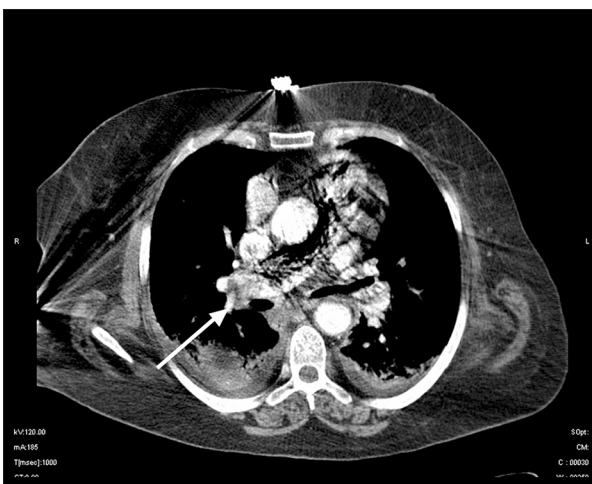


Figure 4. Filling defect in the right lower lobe branch along with "air-contrast".

and she was placed temporarily on non-invasive positive pressure ventilation (NIPPV) without improvement and a few hours later she was intubated and placed on mechanical ventilation. Intravenous full dose heparin infusion (initial bolus, 80 units/kg, followed by 18 units/kg/hour) was initiated for treatment of concurrent thromboembolism. Echocardiography did not show any evidence of right or left ventricular failure. Subsequent echocardiography done 24 hours later did not show any evidence of intracardiac air and complete resolution of the air embolism.

During the next 48 hours, she remained hemodynamically stable with no requirements of vasoactive agents. She was finally extubated 48 hours after the initial presentation. She was later discharged home on warfarin with subsequent outpatient follow-up.

Table 1. Causes of pulmonary (venous) air embolism.

Intravenous devices
Central venous catheters (placement, use, and removal)
Pulmonary artery catheter (swan ganz)
Hemodialysis
Cardiac pacemakers
Pressurized infusion (contrast, blood, fluids)
Trauma and surgery
Head, neck & thorax
Coronary artery bypass
Neurosurgery, dentistry
Genitourinary and pelvic procedures
Childbirth, caesarean section
Vaginal insufflation, curettage
Prostatectomy
Percutaneous nephrostomy
Oral-genital sexual relations
High altitude & diving accidents
Pulmonary disease
Asthma, penetrating injury
Positive pressure ventilation
Air injection
Laparoscopy, arthroscopy
Wound irrigation, hydrogen peroxide
Pneumoradiology
Radiology suite
Arterial or venous angiography
Needle biopsy
Contrast procedures

Discussion

Air embolism occurs when gas enters the circulatory system. This may occur in a number of medical-surgical situations. When this happens, secondary to the placement of an IV access or an IV infusion, it is termed intravenous air embolism (Table 1). The diagnosis of massive air embolism is usually made on clinical grounds by the development of abrupt hemodynamic compromise. Air embolism should always be suspected when patients experience sudden onset respiratory distress (venous air embolism) and/or experience a neurological event (arterial embolism) in the setting of a known risk factor (e.g., intravenous catheter insertion). The spectrum of findings depends upon the degree of severity of the embolism and the end-organs affected. Minor cases are usually asymptomatic or have non specific symptoms including dyspnea,

chest pain, lightheadedness, and a gasp or cough when the bolus of air enters the pulmonary circulation. Occasionally, a systolic “mill-wheel murmur” is described on cardiac auscultation, or an abrupt drop in end tidal CO₂ may be noted during monitoring in the operating room [2]. Often the diagnosis is overlooked entirely until neurological complications are evident or it is made from retrospective reconstruction of the embolic event.

Minor venous air embolisms are fairly common and clearly evident on contrast enhanced CT imaging. A study of 100 patients after manual contrast injection demonstrated minimal embolisms in 20 and moderate (air fluid levels within major vessels) in another three patients. In a larger series of 677 [3] patients undergoing contrast enhanced electron beam CT, minor air emboli were noted in 79 (12%) with the commonest sites being the main pulmonary artery (8%), superior vena cava (1.8%), right ventricle (1.5%) subclavian or brachiocephalic vein (0.9%), and right atrium (0.7%). “Clinical reports” are rare, as events go under-reported. A few nonfatal cases of moderate air embolism have been reported [4,5] along with one fatal event in this setting [4]. The introduction of electron beam CT scanning has made it possible to show air-in-transit in the minor circulation. Air embolism can also be visualized by non-contrast non-invasive studies (echocardiography) [6] and plain radiographs [7].

The physiologic effects of venous air embolism depend on the amount, rate of injection, and underlying host cardio-pulmonary reserve. Though animal studies suggests that 6–8 mL/kg is a lethal dose in dogs [8,9]; 200–300 mL was thought to be fatal in adult humans [10,11]. Others estimates extend up to 500 mL [12], moreover, a rate-of-entry of at least 100 mL/second estimated for 2 seconds was found to be fatal during central venous line placement placement [13] and direct intracardiac injection of 200 mL was fatal in another report [14]. However, the volume in a similar although fatal power injector mishap was estimated at 100 mL [4]. Accumulation of air in the right ventricle (RV) results in “air lock”, ineffective right heart contraction and emptying, leading to obstructive shock, acute RV failure or asystole. Passage of air into the pulmonary circulation is associated with hypoxia from V/Q mismatching and acute pulmonary hypertension with/without right heart failure from direct vascular obstruction by air bubbles, reflex vasoconstriction, or formation of fibrin emboli in the ventricles. Frequently pulmonary edema ensues although the pathogenesis is still unclear.

Emergent treatment includes Durant’s maneuver (left-lateral decubitus, head-down positioning) to decrease air entry into the right ventricle outflow tract. Aspiration of air from the right atrium/ventricle through a multiport central catheter or pulmonary artery catheter (Swan Ganz) may be attempted but is

often of limited value. Alternatively, external cardiac compression may facilitate fragmentation, and dispersion of a large right ventricular air embolus [15]. Supportive care includes volume resuscitation for shock, vasopressors or inotropes as needed and 100% O₂ to enhance re-absorption or hyperbaric therapy to force dissolution of nitrogen.

Paradoxical embolism – air crossover into systemic circulation – can occur through a patent foramen ovale (present in 30% of the population) [16], or physiological intrapulmonary A-V shunts (60%) [17]. Complications result from cerebral (acute-focal neurologic deficits or generalized/global encephalopathy) or coronary embolism (less commonly). Cerebral circulation air-bubbles can be seen immediately on head CT scans or focal infarcts may be evident later. Treatment with hyperbaric oxygen immediately if available, is generally recommended though delayed treatment benefits also have been reported [17].

In our patient, although a fairly large amount of air was injected rapidly, no cardiovascular compromise was noted – catheter aspiration was not attempted – as most of the air was seen to transit through the proximal pulmonary circulation. Some neurologic changes preceded the event, but were clinically and temporally more likely related to her previous history of alcohol intake. Hyperbaric oxygen therapy was not used as the patient did not develop focal deficits and head imaging did not show any findings. Although this is a rare complication, the potential for this to be life threatening makes prevention and early detection of this condition essential. The incidence during non-invasive radiologic imaging needs to be kept in mind for better training of medical personnel, in particular to improve education of power injector technicians to prevent this avoidable hazard. In addition, early recognition of this condition by the medical personnel and technicians in charge of the power injectors can lead to better outcomes. Critical care physicians must be familiar with symptoms indicating air embolism and be ready to implement possible therapeutic maneuvers.

Conclusions

Air embolism is a rare complication, the potential for this to be life threatening makes prevention and early detection of this condition essential. This condition should always be suspected when patients experience sudden onset respiratory distress and/or experience a neurological event in the setting of a known risk factor. Treatment includes Durant’s maneuver; left-lateral decubitus, and head-down positioning to decrease air entry into the right ventricle outflow tract. Other treatment options include volume resuscitation, vasopressors or inotropes, and 100% O₂ to enhance re-absorption or hyperbaric therapy to force dissolution of nitrogen.

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