A rare interesting case of fatal air embolism after intravenous contrast during computed tomography

Abstract. We are reporting a fatal case of air embolism. Although minor cases of air embolism may go unnoticed, this is a case of fatal air embolism after intravenous entry of air, which presented with sudden onset of pulseless electrical activity during a computed tomography scan in the radiology department, requiring cardiopulmonary resuscitation for 15 min. Subsequently, after admission to the intensive care unit, we achieved return of spontaneous circulation. The patient was intubated and ventilated in a shock state. He remained in refractory shock despite of supportive care. Cardiac arrest was registered again in the catheterization lab and the patient could not be revived after 4 h from the initial cardiac arrest. A computed tomography scan was reported to reveal a significant amount of intra-cardiac air, which was the likely cause patient’s death. The case is a rare condition, which highlights the importance of early diagnosis and delivers a message to the medical staff to have a high index of suspicion in patients who have risk factors, and who develop sudden shock with hypoxemia, in order to treat this potentially life-threatening condition effectively in a timely manner.

Keywords: air embolism; pulseless electrical activity; cardiopulmonary resuscitation; shock; mortality

Introduction
Fatal air embolism is a rare condition, because of entry of air into the venous or arterial vasculature, producing hemodynamic and systemic effects. This occurs during several operative or non-operative procedures, which involve communication with external air. Most of the episodes are preventable, if all precautions are taken, and managed properly. A high clinical index of suspicion is required with an increase in physician awareness [1].

Case Summary
A 58-year-old Saudi male. He was an ex-smoker.

He was a known case of non-insulin-dependent diabetes mellitus, hypertension, and cholecystectomy.

He presented with a 2-week history of passing black colored stools, and feeling dizzy.

He was admitted to the ward for further workup, including endo/colonoscopy and CT scan.

He had undergone upper GI endoscopy 6 months ago which showed mild duodenitis and gastritis.

At the time of admission to the ward, he was hemodynamically stable, oxygenating well on room air.

A CT abdomen with IV contrast was done for further evaluation of his anemia.

At the end of CT scan, the patient developed PEA, which required CPR for 10–15 minutes.

He was revived, intubated, ventilated, and transferred to the Intensive Care Unit.

On Examination, the vital signs include pulse 110–140 per min and regular.

The patient was severely hypotensive with blood pressure 80/60 mm Hg, oxygen saturation was 98 % on FiO2 100 %, on mechanical ventilator.

Lungs were clear with bilateral equal air entry. Secretions were minimal. Heart sounds were audible. No S3 or S4 was heard. No murmurs was detected. Abdomen was soft and non-tender. Bowel sounds were audible. Neurologically, patient was mildly agitated, otherwise grossly intact. Sedation...
was optimized to prevent any asynchrony with the ventilator. He was also started on muscle relaxant with atracurium.

Ventilator settings were high with ARDS protocol on controlled mode, tidal volume (V, 450 ml), Rate 14 per min, positive end-expiratory pressure (PEEP) 12 cm, FiO₂ was 100 %.

Plateau pressure was maintained below 30. Plan was to titrate FiO₂ as tolerated to keep oxygen saturation 88—92 %.

The patient received 2 liters of IV fluids, followed by high doses of norepinephrine and dopamine, but remained in refractory shock as shown by the rising lactate level in the blood gases.

The patient appeared to be in cardiogenic shock, and was urgently transferred to the Cardiac Cath Lab. An IABP (Intra-aortic balloon pump) was inserted. He again developed cardiac arrest in the Cath Lab, and could not be revived. After the patient died, a CT scan final report confirmed the cause of his arrests.

Laboratory results of blood analysis are shown in the table 1. Arterial Blood Gas: PH: 7.09, PaCO₂: 39 mmHg, PaO₂: 86 mmHg, lactate: 8.0 mmol/L – On FiO₂ 100. After 2 h: PH: 7.03, PaCO₂: 74 mmHg, PaO₂: 70 mmHg, lactate: 10 mmol/L – On FiO₂ 90. The above ABG shows: Worsening severe hypoxemia, a very high alveolar-arterial gradient and a severe lactic acidosis due to shock. 2D Echocardiogram (fig. 1): sinus tachycardia and right bundle branch block (RBBB); LV systolic function was severely reduced with ejection fraction 20—30 %; RV systolic function was mildly reduced; RVSP 30—40 %. Doppler was not performed. Chest X-ray (fig. 2): bilateral air space disease; endotracheal tube and central venous catheter in place; the cardiac shadow within normal limits. CT scan Chest images (fig. 3) show air in the right ventricle and few specks of air in coronary vessels, consistent with air embolism.

**Discussion**

Vascular air embolism is a known entity since the early nineteenth century. All critical care physicians must know this catastrophic medical emergency, as mortality remains high [1].

The patient described above went into a sudden right ventricular failure and cardiac arrest in the radiology department during a procedure, with no warning signs. The other possibility to consider was anaphylactic reaction. However, anaphylaxis usually present with hemodynamic instability only, at least early on, and does not cause refractory hypoxemia related to severe ventilation perfusion mismatch and shunt physiology as evident by arterial blood gases.

One interesting finding which was not detected in our case was the absence of classical murmur described as “mill wheel murmur”. This finding although has clinically significance but has been described to be temporary and a late finding. Therefore, it may not be detected all the time. The
The presence of tachycardia may also limit its detection. It may require a focused auscultation in highly suspected cases to hear any faint added heart sounds which was not the situation in this patient. The CPR has also been reported to disperse the intra-cardiac air out of the cardiac chambers, which may ultimately limit the auscultatory findings. Severity of air embolism is reflected by refractory shock and hypoxemia. The severity of air embolism mainly depends on 3 factors. The first factor is the amount of air infused. Even a small of air can cause severe morbidity and even mortality. Presence of mill wheel murmur usually requires a sizeable amount of air up to 200 ml. The second factor is the speed at which the air is infused. Air infused under high pressure carries a much higher risk of severity even if the amount of air is less. The third factor which determines the severity is the position of the patient at the time of occurrence, especially Fowler and sitting position.

The presence of RBBB is a recognized finding in air embolism due to RV strain.

The Chest X-ray showed bilateral air space disease consistent with pulmonary edema, which could be due to capillary leak, which is well known to be caused by air in the pulmonary circulation. The air induces an inflammatory cascade leading to the damage of endothelial cells and ARDS like picture.

Trans esophageal echocardiography (TEE) could have been diagnostic but it’s an invasive procedure and requires a stable patient. The 2D echocardiogram was done as a focused and limited study and showed severely reduced EF and pulmonary hypertension. No Doppler/ultrasound was performed.

Finally, significant amount of air in the cardiac chambers and a few specks of air in the coronary vessels on CT scan, which was completed just before the commencement of CPR, clinch the final diagnosis. However, these CT findings were only known retrospectively, after the patient was expired. Nowadays, intravenous contrast agents are infused by pumps and must be given at a rapid speed. So, the risk of air embolism is real, if a significant amount of air goes in along with the contrast at a rapid speed. It is highly imperative, that all intravenous contrasts should be given by a protocol and all the infusion pumps should be periodically checked, for any dysfunctionality, to minimize the risks. The exact incidence is difficult to know as the sub-clinical cases will be undetected.

Etiology. Surgery, if the operative site is above the level of the heart, especially during neurosurgery, obstetric-gynecological and laparoscopic procedures. Central venous catheters, insertion and removal [2, 3]. Several studies have shown the risk. Trauma, especially penetrating chest injuries requires a focused approach to diagnose it as it may not be easy to confirm it by routine tests as patient already have air leaks in the mediastinum.

Scuba diving — 7/100,000 dives [4, 5]. This can particularly occur if they ascent rapidly towards the surface. Pilots and astronauts with bronchogenic cysts or cystic air-filled lesions in the lung are also at risk. Positive pressure ventilation barotrauma has also been shown to cause air embolism. Positive pressure infusion pumps as in our case is a well-known risk factor.
Diagnosis is based on a high clinical suspicion. An echocardiogram especially TEE is highly sensitive to show intracardiac air; contrast-enhanced CT scan Chest may also show it [4]. Doppler/Ultrasound is also useful.

Differential diagnosis includes anaphylactic reaction to contrast, acute coronary syndrome with cardiogenic shock, and acute pulmonary embolism.

Treatment [4–6]. Put the patient on 100 % oxygen to improve oxygenation and remove nitrogen. Optimal fluid support and inotropic agents are required to keep mean arterial pressure above the mean pulmonary artery pressure.

Left lateral decubitus head down position, and aspiration of air from the right atrium, preferably using Buneigin-Albin multi-orifice catheter with up to 60 % success.

Hyperbaric Oxygen, by creating a high diffusion gradient [6, 7]. However, its availability is limited to certain centers only.

Conclusions

This case of massive air embolism reflects the importance of identifying an air embolism as early as possible. Urgent measures such as high oxygen therapy/ hyperbaric oxygen, patient positioning with aspiration, and close hemodynamic monitoring should be started in cases of high clinical suspicion to reduce mortality. A strict intravenous contrast protocol to avoid air entry along with the contrast, must be instituted. Equipment, including automated pumps must be checked regularly, and staff competency must be maintained by all radiology departments.

References