American Journal of Medical Case Reports, 2020, Vol. 8, No. 5, 119-122
Available online at http://pubs.sciepub.com/ajmcr/8/5/3
Published by Science and Education Publishing
DOI:10.12691/ajmcr-8-5-3

Pulmonary Air embolism Associated with Pneumocephalus: A Case Report

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Received February 04, 2020; March 06, 2020; Accepted March 14, 2020

Abstract Iatrogenic air embolism is associated with significant morbidity and mortality. Retrograde cerebral venous air embolism is most frequently associated with manipulation of venous access most commonly from central venous catheters. The ascension of air to the cerebral circulation is possibly due to the low specific gravity of air compared to blood and the performance of procedures in the sitting position. Increased right ventricular pressures in the setting of pulmonary thromboembolism may also contribute to the retrograde flow of air. We present the case of a 61-year-old woman who developed a massive pulmonary embolism and pneumocephalus, which was evident during contrast enhanced CT pulmonary angiography. Neurological deficits were not apparent and air resorption occurred after 48 hours of high flow oxygen therapy.

Keywords: *air embolism, pneumocephalus, intravenous contrast, cerebral air embolism*

Cite This Article: Mohammed Al-Sadawi, Romy Rodriguez Ortega, Naseem Hossain, Yusra Qaiser, and Samy I. McFarlane, "Pulmonary Air embolism Associated with Pneumocephalus: A Case Report." *American Journal of Medical Case Reports*, vol. 8, no. 5 (2020): 119-122. doi: 10.12691/ajmcr-8-5-3.

1. Introduction

Air embolism is a complication of multiple medical scenarios involving line placement, and surgical procedures, including cardiac and vascular. Instrumentation of the human body and access to cavities or spaces with negative pressures leads to the possibility of air entering and remaining in closed systems. The consequences of these vascular-access related procedures can range from trivial to life threatening. Venous air embolism typically affects the right heart and lungs, but air can also gain access to the arterial circulation through intra-cardiac or intrapulmonary shunting. Cerebral arterial air embolism could be devastating with signs and symptoms indistinguishable from those of an ischemic stroke. On the contrary, cerebral venous air embolism typically occurs in the setting of traumatic skull fractures given that in most situations venous flow would take the air bubbles towards the right heart. Retrograde cerebral venous air embolism, where air ascends against forward venous flow, has been reported under manipulation of central venous catheters and other procedures that involve patients in the seated position.

In this report we present a patient who developed cerebral venous air embolism after inadvertent infusion of air while receiving a contrasted imaging study that revealed a significant arterial pulmonary embolus.

2. Case Presentation

A 61-year-old African American woman with a past medical history of peripheral vascular disease (PVD), chronic venous insufficiency and pre-diabetes mellitus presented to our Emergency Department (ED) with worsening left lower extremity pain for the past 3 days. The pain was continuous, progressive and the patient denied any history of trauma. Examination showed a clean, shallow ulcer 2x3 cm with surrounding hyperpigmentation from stasis dermatitis; without discharges or signs of infection. Upon assessment, the pain was deemed to be due to chronic venous insufficiency. It improved with non-steroidal anti-inflammatory drugs and she was subsequently discharged from the emergency department. Three days later, the patient returned to the ED with worsening leg pain unresponsive to the oral analgesics. At this admission, she also reported inability to bear weight on the affected extremity. She denied fevers, chills, nausea, vomiting, abdominal pain, or urinary symptoms. Physical examination revealed erythema around the ulcer with increased warmth and mild tenderness. CT of her left lower extremity with intravenous (IV) contrast revealed subcutaneous edema and fascial thickening most prominently at the medial aspect of the lower extremity, compatible with cellulitis in the absence of a drainable fluid collection (Figure 1). The patient was treated with IV ampicillin/sulbactam. There was evidence of symptomatic

improvement and the patient was discharged with an oral course of antibiotics and zinc oxide compression bandages for local wound care. Her discharge diagnosis was lower extremity cellulitis with associated chronic venous insufficiency ulcer. Immediately upon leaving the hospital, the patient had a syncopal episode and collapsed; which prompted her to be rushed to the ED for further evaluation. In the ED, the patient presented with tachypnea and confusion and reported feeling short of breath. Physical examination revealed a blood pressure of 85/61 mmHg, heart rate of 131 beats per minute and respiratory rate of 32 breaths per minute with an oxygen saturation of 92% on non-rebreather mask 15 litres per minute. The patient was sedated and intubated due to dyspnea, increased work of breathing and hemodynamic instability. A central venous catheter was placed, along with initiation of vasopressors. CT scan of the head showed pneumocephalus within the cavernous sinus and clival venous plexus in the absence of skull base fracture, acute hemorrhage, mass effect or evidence of an acute ischemic infarct (Figure 2). CT with IV contrast of pulmonary artery revealed saddle pulmonary embolus with pulmonary emboli extending into the right and left main pulmonary arteries, bilateral lobar and segmental arteries. The Figures also showed marked right heart strain with bowing of the interventricular septum and reflux of contrast within the IVC and hepatic veins, and pooling of contrast within the right heart. It also demonstrated air within the right ventricle as well as subcutaneous emphysema involving anterior chest wall and right supraclavicular fossa. Foci of air are also seen tracking into the hepatic vasculature likely iatrogenic from contrast administration (Figure 3). Due to her clinical condition and imaging findings, the patient received thrombolytic therapy (recombinant Tissue Plasminogen Activator) for pulmonary embolism. Subsequently she was admitted to the critical care unit. Her initial laboratory results revealed lactate of 3 mmol/L (reference range 0.5-2.2), troponin I <0.01 ng/mL (reference range <0.01) and N-terminal pro B-type natriuretic peptide (NT-pro BNP) at 61 pg/mL (reference range <300 pg/mL). She was started on therapeutic anticoagulation with heparin drip afterwards. The patient was extubated next day and put on high flow 100% O2 as a trial to improve resorption of air in the vasculature. Once the patient was hemodynamically stable, there were no apparent neurological deficits noted as a result of cerebral air embolism. Two days after extubation, repeat CT head revealed resolution of air in cavernous system (Figure 4). Venous duplex of her lower extremities revealed no evidence of thrombosis bilaterally (Figure 5). Patient was transitioned to oral anticoagulation and discharged after two days of hemodynamic and clinical recovery.

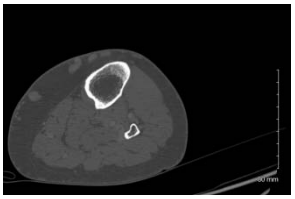

Figure 1. CT of patient's left lower extremity with IV contrast revealed subcutaneous edema and fascial thickening most prominently at the medial aspect of the lower extremity (blue arrow), compatible with cellulitis without visualization of drainable fluid collections.

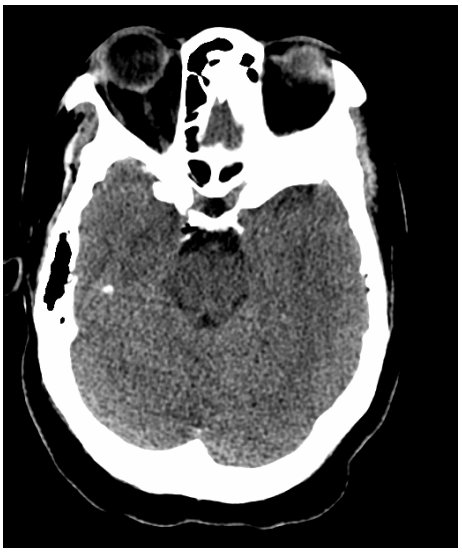

Figure 2. CT head showed pneumocephalus (white arrows) within the cavernous sinus and clival venous plexus in the absence of skull base fracture, acute hemorrhage, mass effect or evidence of an acute ischemic infarct.

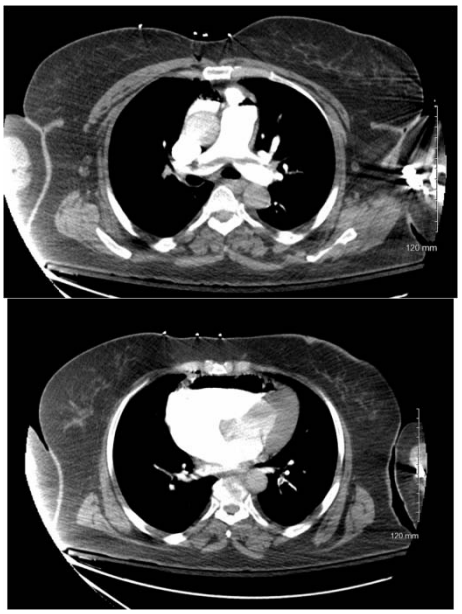

Figure 3. CT with IV contrast of pulmonary artery revealed saddle pulmonary embolus extending into the right and left main pulmonary arteries (red arrow), bilateral lobar and segmental arteries. The Figures also showed marked right heart strain with air seen within the right ventricle (blue arrow).

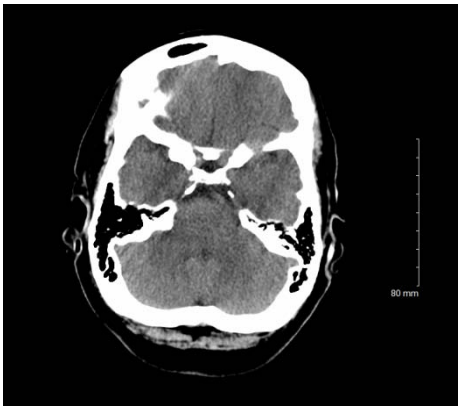

Figure 4. CT head revealed resolution of air in cavernous system.

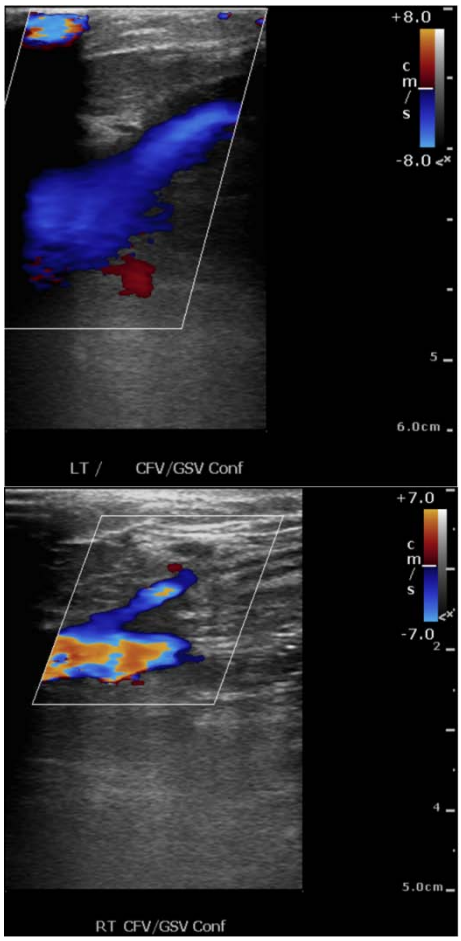

Figure 5. Venous duplex of patient's lower extremities revealed no evidence of thrombosis bilaterally (Figure 5).

3. Discussion

Cerebral venous air embolism is rarely encountered in clinical practice. Although the specific incidence is not available in the literature, a review article on air embolism by *Brull et al.* reported at least 700 instances of air embolism on a search from an FDA mandatory device reporting database from 2011 to 2016 [1]. Central venous access has been frequently reported as a source of cerebral venous air embolism [2,3,4]. The proposed mechanism is that once air gains access to the venous system; ascension against blood flow is possible given the low specific gravity relative to blood [5]. In clinical scenarios involving contrast enhanced studies with power injectors, the risk for retrograde cerebral venous air embolism is likely increased [6]. A prospective study from India examined the risks of air embolism in 200 patients that underwent contrast enhanced CT and found that iatrogenic air embolism occurred in 7% of all studies [7].

The etiology of air embolism in this patient was iatrogenic, but pin-pointing the exact moment at which it occurred is difficult. She received IV contrast injections on two different occasions during a short period of time and had a central venous catheter placed before undergoing the imaging that ultimately diagnosed the air embolism. All these interventions are known to be associated with the development of this complication. Although it has been established that ascension to the cerebral veins is feasible, we believe than in this specific scenario the increased right sided pressures as a result of the massive pulmonary embolism were likely an additional contributor for the retrograde migration of air. This was evidenced by the pooling of contrast material and air in the right ventricle, the reflux of contrast material into the hepatic circulation and the absence of air in the pulmonary circulation.

Various neurological manifestations have been described in the setting of cerebral venous embolism such as stroke, seizures, loss of consciousness, altered mental status and paralysis. Most cases report non-specific signs and symptoms ranging from syncopal episodes to confusion and coma [2,4,8]. These possible neurologic sequelae prompt an inquiry of whether the syncopal episode that preceded the clinical deterioration in this patient may have been caused by cerebral air embolism. This hypothesis poses a problem as the patients' symptoms developed 24 hours after the initial contrasted study. The syncopal episode can also be better explained by an alternative diagnosis; i.e. the saddle pulmonary embolus and hypotension. After hemodynamic improvement, there was no evidence of neurologic deficits. There were also no deviations from standard practice of central venous catheter placement. Given the significant amount of contrast material injected, the infusion rates commonly used during angiography protocols and the temporal relationship with the CT pulmonary angiography, we believe that infusion of IV contrast was likely the moment at which the passage of air occurred.

The first step in managing air embolism is attempting to interrupt the intervention responsible for the event. Multiple case reports and reviews have suggested positioning (Trendelenburg and left lateral decubitus), high flow 100% oxygen and hyperbaric oxygen therapy as specific treatments along with supportive measures [1,4,9]. No randomized controlled trials to date have proven the effectiveness of hyperbaric oxygen therapy for cerebral air embolism. An animal study failed to demonstrate improvements in cerebral function in a model of arterial air embolism [10]. Use of IV catheters for direct removal of air is currently not recommended due to lack of evidence for efficacy [11]. For our patient, a repeat CT head after 48 hours revealed resorption of air following a trial of high flow oxygen therapy through nasal cannula.

Reported morbidity and mortality associated with cerebral air embolism is notable as exemplified by a case series where among 23 patients that suffered an acute neurological change due to air embolism, 4 patients died and 10 had residual deficits [12]. In our patient no other neurological complications arose, thus no further interventions were required.

4. Conclusion

Cerebral venous air embolism is a complication of contrast enhanced studies and is associated with significant morbidity and mortality. In the setting of significant pulmonary embolism, the elevated right sided ventricular pressures could increase the risk of air, if inadvertently administered, flowing retrogradely to the cerebral venous system. Neurological deterioration after

an intravenous contrast administration should raise the suspicion for cerebral air embolism. Hyperbaric oxygen therapy and high flow oxygen therapy are proposed treatments for cerebral air embolism but evidence for its' effectiveness is lacking.

Acknowledgements

This work is supported, in part, by the efforts of Dr. Moro O. Salifu M.D., M.P.H., M.B.A., M.A.C.P., Professor and Chairman of Medicine through NIH Grant number S21MD012474.

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