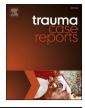


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#### Morbidity and Mortality Case Report

# Multiple systemic venous air emboli after fatal basilar skull fracture

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ARTICLE INFO	A B S T R A C T
<i>Keywords:</i> Venous air embolism Basal skull fracture Head trauma Traumatic air embolism	<ul> <li>This case is significant for extensive systemic vascular air emboli in the right ventricle (visually estimated at 30 ml), pulmonary artery, inferior vena cava, hepatic veins, and iliac vein, which is a rare and novel consequence of fatal basal skull fracture.</li> <li>Collections of air in the right ventricle and left external iliac vein were visible on imaging prior to contrast administration, making an iatrogenic etiology unlikely.</li> </ul>

#### Introduction

A venous air embolism (VAE) is a potentially life threatening collection of gas in the venous system that is well documented as an iatrogenic complication, such as from intravenous (IV) infusion or catheter insertion, but fewer reports explore traumatic etiologies [1]. Pneumocephalus, the presence of intracranial air, occurs in patients with severe head trauma, particularly basal skull fractures, and is a potential mechanism for the development of VAE [2]. This case describes air in the right ventricle and pulmonary artery, as well as extensive VAE present in the inferior vena cava (IVC), hepatic veins and left iliac vein from basal skull fracture extending into the mastoid air cells.

#### **Case summary**

The patient was a 25 year old male involved in a motor vehicle-related traumatic brain injury without reliable witnesses. He was unresponsive at the scene. Intubation was attempted prior to arrival, but uncontrolled bleeding obscured airway visualization. A laryngeal mask airway placement was successful, and he was airlifted to a level II trauma center. On arrival, the patient was immediately orally intubated. His blood pressure was 224/140 mmHg. Head examination showed apparent brain tissue exiting out the right ear and an open head wound with frank bleeding despite multiple attempts to dress the wound. Glasgow Coma Scale score was 3 with fixed, dilated pupils and no brainstem reflexes elicited.

Head computed tomography (CT) without contrast showed extensive subarachnoid hemorrhages in both hemispheres and the posterior fossa, with a 4 mm right to left midline shift and pneumocephalus consistent with open skull fracture (see Fig. 1a). Fracture of the right temporal skull extended into the mastoid, carotid canal, and sphenoid sinus (see Fig. 1b). There was also a right occipital bone

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https://doi.org/10.1016/j.tcr.2022.100608

Received 8 July 2021; Received in revised form 10 January 2022; Accepted 24 January 2022

Available online 31 January 2022

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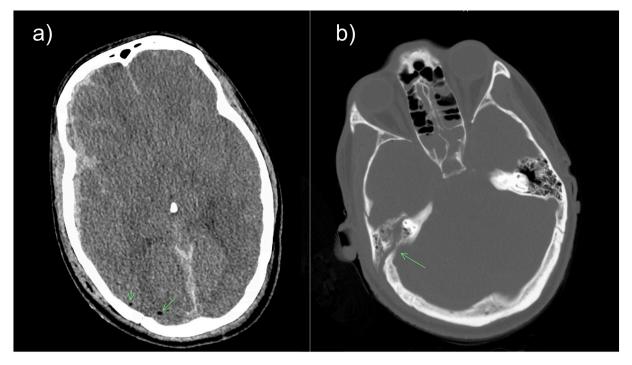


Fig. 1. CT of the head without contrast revealing (a) pneumocephalus and (b) basilar skull fracture involving mastoid air cells in bone view.

fracture extending to the left parietal bone, and a complete loss of grey-white junctions suggestive of a non-survivable anoxic injury. This was consistent with severe head trauma.

CT of the chest, abdomen, and pelvis revealed air in the right ventricle extending into the pulmonary artery, visually estimated at 30 ml (see Fig. 2), with additional air in the hepatic veins, IVC, and left external iliac vein (see Fig. 3). The emboli were initially believed to be from IV contrast injector. Notably, while being scanned, the patient's blood pressure had suddenly dropped to the 50's-60's systolic. Further investigation showed air in the heart and left iliac vein on the CT scout, prior to contrast (see Fig. 4).

The remainder of imaging showed no other evidence of acute trauma. The patient received resuscitation with blood products, and a left subclavian central line and arterial lines were placed. No air was able to be aspirated from the right atrium or ventricle. After initially stabilizing with resuscitation, the patient deteriorated and expired approximately 48 h after arrival despite maximum resuscitative efforts and additional hemodynamic support.

#### Discussion

Mortality of vascular air embolism ranges from 48 to 80% with variance based on volume of air, rate of accumulation, and patient position [1]. Large volumes of intracardiac air may cause hemodynamic instability and circulatory collapse due to interference with blood in the cardiac pump—known as "air lock" [1]. Treatment involves patient repositioning to prevent further accumulation of air, hyperbaric oxygen to eliminate nitrogen, and attempting to remove air via catheter insertion [1]. In this case, attempts to decrease the air already in the system were unsuccessful. Further attempts to decrease air entrance, such as repair of sinuses, seemed futile in this case with a fatal brain injury.

Few reports have linked pneumocephalus and head trauma to VAE. Historical cases of fatal head trauma noted air limited to the right ventricle and pulmonary artery postmortem [3]. More recent trauma cases have linked air in the right ventricle and jugular vein specifically to basal skull fracture [4,5]. A common factor in these cases is the tearing of dural vessels, potentially granting air intracranial access due to the relative rigidity of those veins when compared to systemic veins [2].

This patient's findings were indicative of a large VAE in the right ventricle extending into the pulmonary artery, seen on scout films before injection of contrast. To our knowledge, our patient exhibited the largest reported amount of intracardiac air caused by these circumstances [3,4]. In addition, the patient's air embolism extended past the right ventricle and pulmonary arteries to include multiple systemic veins. The mechanism for the multiple VAE is unclear but likely was related to the basilar skull fracture. There were no signs of air in the arterial system, bilateral lungs were unaffected on imaging, and no other fractures seen. The patient's left antecubital IV received only 1 l of fluids. Any attempt to establish it would not have caused this much air to accumulate or explain the areas it accumulated in. The air embolism's presence prior to IV contrast administration or central line placement makes an iatrogenic cause unlikely. Patients with skull injury and resulting air emboli need to be recognized and treated quickly to prevent cardiovascular collapse.

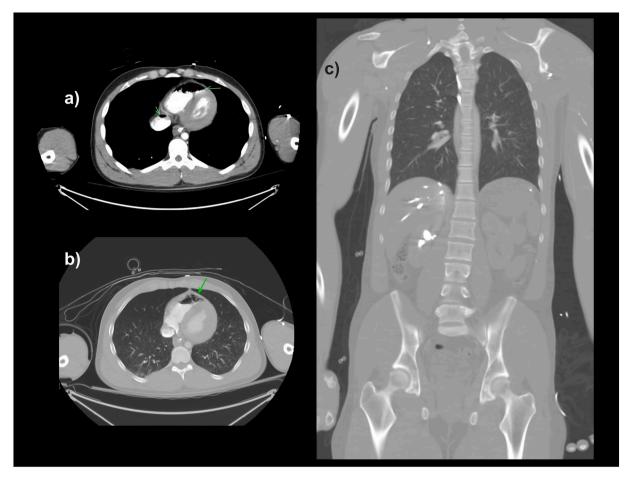


Fig. 2. Chest CT with contrast showing (a) air embolism in the right ventricle and pulmonary vein, and (b) axial and (b) sagittal view showing unaffected lung tissue.

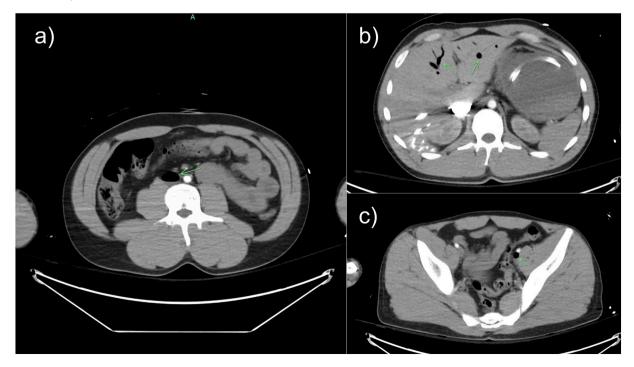


Fig. 3. CT abdomen and pelvis with contrast showing air within the (a) inferior vena cava, (b) hepatic venous system, and (c) left external iliac vein.

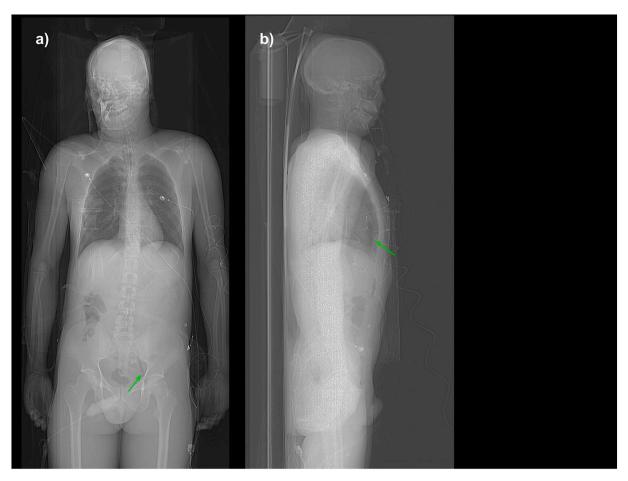


Fig. 4. Air emboli of (a) left external iliac vein on frontal view and (b) right ventricle of heart on sagittal view scout CT.

#### Statement of informed consent

No patient identifiers were used in this report.

#### Funding

No funds were received or are expected in support of this work.

#### Declaration of competing interest

The authors declare no competing interest(s).

#### Acknowledgements

We would like to acknowledge Gilman T. Wolsey, MD for his helpful discussion and technical contribution.

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