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Case Report

# Iatrogenic and fatal arterial air embolism during the CT scan

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**Abstract**

Systemic and fatal arterial air embolism during the computed tomography (CT) scan is rarely reported in English-based literature. Iatrogenic air embolism happening during the CT scan is often related to the injector, usually venous air embolism and asymptomatic. We report one fatal and extensive systemic arterial air embolism because of one error that happened during a brain CT scan. The mechanism is different from the reported cases in the literature. The possible mechanism and pathogenesis are well discussed to alert clinicians and prevent the recurrence of such complication.


*Keywords:* Air embolism; Iatrogenic error; Pneumocranium

## 1. Introduction

Cerebral air embolism may be caused by iatrogenic and traumatic introduction of air into the arterial or venous system.[1] The most common iatrogenic procedures that produce air embolism are lung biopsy and cardiovascular surgery.[2,3] Systemic arterial air embolism is caused by the entry of gas into the pulmonary veins or directly into the arteries of the systemic circulation.[2] Systemic and extensive arterial air embolism is very rare and is likely to result in serious complications, such as seizure, heart attack, hypoxemia, pneumothorax, apnea, circulatory collapse and even intra-cranial air formation. The purpose of this article is to introduce this serious complication, recognize the images and avoid the recurrence. If the complication is recognized earlier, it is more likely the patient will be resuscitated.

## 2. Case report

The 35-year-old female suffered from acute traffic accident with multiple abrasion wound and then was transferred to the emergency room. Initially, the gross inspective body images revealed no major injuries other than some abrasion wounds. However, a Glasgow Coma Scale (GCS) evaluation revealed poor consciousness, with a Glasgow Coma Scale score of E1M3VT (Eye: no response, Motor: flexion response to pain stimulation, Verbal: endo-tracheal tube insertion). The patient

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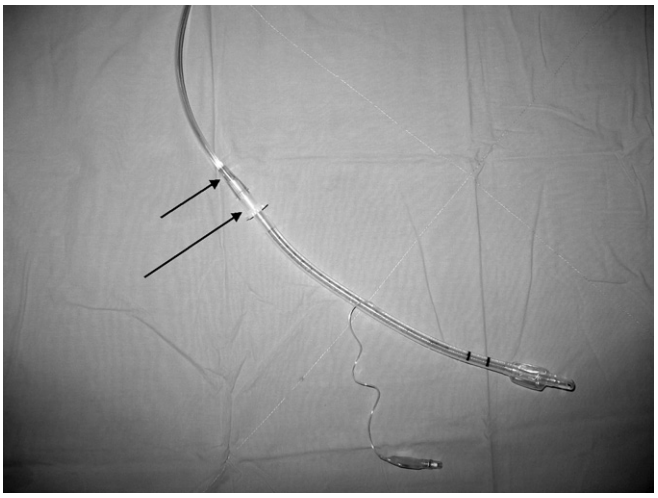

Fig. 1. Diagram of the wrong connection between the endo-tracheal tube (long arrow) and oxygen flow meter (short arrow). Such connection could result in one-way airflow pattern, only inflow without outflow from lung, increasing the intra-tracheal and intra-alveolar pressure markedly.

breathed spontaneously, with an oxygen saturation of 100% with 30% $FiO_2$ without any evidence of hypoxemia or apnea. An emergent cranial computed tomography (CT) scan was scheduled within 10 minutes after her arrival.

The iatrogenic error happened when the patient's oxygen flow meter was connected to the endo-tracheal tube incorrectly (Fig. 1). Extensive swelling of her face and trunk developed immediately after the CT scan. The oxygen saturation dropped to 35–40%, with no audible breathing sound. Pneumothorax was considered and the emergent chest tube insertion was done. Bilateral subcutaneous emphysema and pneumoperitoneum were demonstrated in chest film (Fig. 2). Apnea developed about 10 minutes later. CT images revealed extensive intra-vascular air content within the extra-cranial and intra-cranial vessels including the carotid, vertebral and cortical arteries (Fig. 3A and B) with pneumocranium (Fig. 3B) formation.

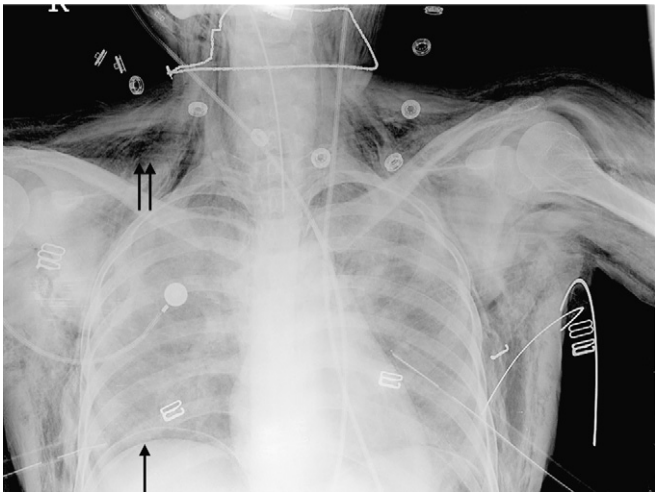

Fig. 2. Chest film revealed both-side pneumothorax with chest tube insertion. Intra-peritoneal free air collection (single arrow) and extensive subcutaneous emphysematous change (double arrows) were demonstrated.

Reconstructed 3D CT images revealed extensive negative contrast intra-cranial vessel images (Fig. 4A and B). Pneumocranium, intra-parenchyma air formation (Fig. 4B) and some air passage into the superior sagittal sinus were noted. Despite the usage of 100% oxygen, huge intravenous fluid supplement and both-side chest tube insertion, the patient suffered from sudden cardiac arrest 2–3 hours later. She responded poorly to cardiopulmonary resuscitation and expired finally.

## 3. Discussion

Systemic arterial air embolism is caused by the entry of gas into the pulmonary veins or directly into the arteries of the systemic circulation.[2] Air can enter the systemic arteries as a result of overexpansion of the lung or by decompressive barotraumas or as a result of paradoxical embolism,[3] then into the pulmonary vein and systemic circulation. Most iatrogenic arterial air embolism occurred during the lung biopsy procedure[4] or during cardiovascular surgery.

Air that enters the pulmonary vein can then enter the systemic artery by three possible mechanisms. First, ambient air may be directly introduced if a biopsy needle tip enters a pulmonary vein. Second, air introduced into the pulmonary arterial circulation may reach the venous side by traversing the pulmonary microvasculature, even in the absence of artery-venous fistulas.[5] Third, air may enter the pulmonary vein by bronchus-venous fistula or other communication between airway trees and vein finally.[5]

Iatrogenic venous air embolism after the CT scan is usually related to the CT-injector and causes air within the right ventricle and pulmonary artery.[6] According to a previous report, its incidence could be up to 23% and it is usually asymptomatic.[7]

Our patient suffered from a systemic, diffuse arterial air embolism immediately after the CT scan, by a mechanism that may be different from previous described. The oxygen flow meter could not connect to the endo-tracheal tube directly, so there must be some media between the two devices such as Ambu bag and so forth. Because of the direct connection between the endo-tracheal tube and oxygen flow meter (Fig. 1), the oxygen flow could be delivered into the tracheabronchial tree system continuously with one-way flow pattern, only inflow without outflow. The intra-bronchial, intra-alveolar pressure could have increased markedly, and eventually, air could diffuse into the pulmonary venous system through the alveolar-capillary membrane, then reach the pulmonary venules, pulmonary vein, left atrium, left ventricle and finally extensive, systemic air embolism occurred. CT scan revealed extensive and large amount of air content within the carotid, vertebral artery and intra-cranial vessels.

Some air content was delivered to the patient's superior sagittal sinus and intra-cranial cavity. The most likely reason is that air could penetrate through the blood-brain barrier and even rupture it focally because of large volume of intra-vascular air. Furthermore, the intra-alveolar pressure could

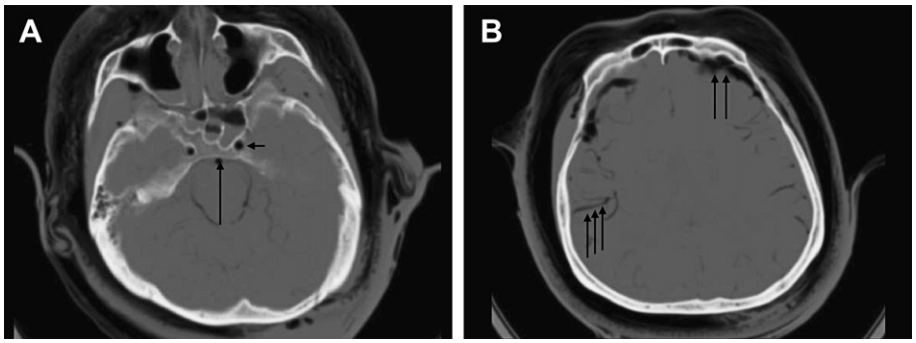


Fig. 3. Axial section CT scan (window: 3,200, level: 700): (A) Diffuse intra-vascular air content, included both carotid (short arrow), vertebral, basilar (long arrow) and (B) cortical arteries (triple arrows), was seen. Additionally, intra-cranial pneumocranium formation (double arrows) was demonstrated, indicating that some air had penetrated through the blood-brain barrier and ruptured it focally. Marked subcutaneous emphysematous change was also noted.

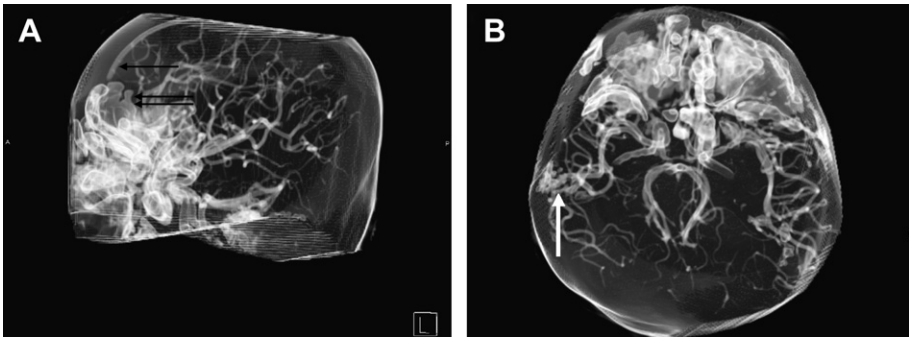


Fig. 4. (A, B) Reconstructed air-contrasted 3D images from the original CT scan: Some air content was noted within the parenchyma (single thick arrow), superior sagittal sinus (single arrow) and confluent pneumocranium (double arrows).

have increased markedly and the alveolar sacs ruptured with pneumothorax formation eventually.

Emboli cause pathologic changes by two mechanisms: a reduction in perfusion distal to the obstruction and an inflammatory response to the air bubbles.[8] The effect of arterial embolism is much more critical, as compared with venous embolism, dependent on the volume of air introduced and the ischemia of vital organs, especially the heart and brain. Studies in dogs suggest that as little as 1.0 mL of air injected into a pulmonary vein inevitably leads to death from coronary air embolism. In humans, 0.5 mL can be sufficient to induce fatal arrhythmia from focal coronary ischemia.[9] Cerebral air embolism may occur concomitantly; intra-cranial ischemia is typically reflected by generalized seizures, circulatory collapse, or multiple neurological defects corresponding to the affected vascular territories.[5,9]

The treatment strategy in arterial gas embolism is the protection and maintenance of vital function, including the usage of 100% oxygen, marked IV fluid resuscitation and hyperbaric oxygen therapy.[10] Cerebral gas embolism often causes generalized seizure,[11] which may not respond to benzodiazepine. In such condition, the usage of barbiturates coma is recommended.[12]

Unfortunately, because of poor response to extensive medical care, our patient suffered from fatal downhill sequences. The error could have been prevented. A simple connection error can result in complicated and fatal outcome. Double-checking of the tube connection is mandatory, especially in changing tube system.

We have to monitor the patient all the time and stop scanning when there are any gross body images changing, such as facial swelling or emphysema appearing. Alertness to the fatal complication and familiarity with CT images could also help to resuscitate such patients. The treatment goal is the protection of vital organ perfusion such as heart and brain. The strategy includes the hyperbaric oxygen therapy, infusion therapy, and steroid therapy and seizure control.

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