Case Report

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A Case of Massive Cerebral Arterial Air Embolism Induced by Artificial Pneumothorax and Its Analysis

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Significance of the Study

- Artificial pneumothorax may induce cerebral arterial air embolism, requiring a high degree of vigilance. If cerebral arterial air embolism occurs, the patient should be placed in the left lateral or supine position to eradicate gas in the thoracic cavity and be treated with high-flow oxygen until hyperbaric oxygen therapy is available.

Keywords
Artificial pneumothorax · Cerebral artery · Air embolism · Paradoxical embolism · Analysis

Abstract
Objective: This study aimed to investigate the characteristics of cerebral arterial air embolism. ***Clinical Presentation and Intervention:*** The clinical data of a patient with cerebral arterial air embolism induced during artificial pneumothorax were retrospectively analyzed. The patient needed the induction of artificial pneumothorax for medical thoracoscopy but developed hemiplegia and disturbance of consciousness during the induction. Cerebral arterial air embolism was detected by head computed tomography. ***Conclusion:*** Artificial pneumothorax may induce cerebral arterial air embolism.

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Introduction

Air embolism is mostly iatrogenic and is mostly correlated with invasive examination and treatment [1]. Cerebral arterial air embolism is a rare type of air embolism with serious outcomes. Some patients may have no clinical symptoms [2], and it is sometimes misdiagnosed as thromboembolia. Here, we present a case of cerebral arterial air embolism that occurred during the induction of artificial pneumothorax.

Case Presentation

The patient was a 55-year-old woman with dyspnea after activity which had started 1 month prior to presentation. Chest computed tomography (CT) revealed effusion in the right pleura. Highly suspicious malignant pleural effusion, but no pathological diagnosis was obtained. Thoracoscopy was scheduled. Before the

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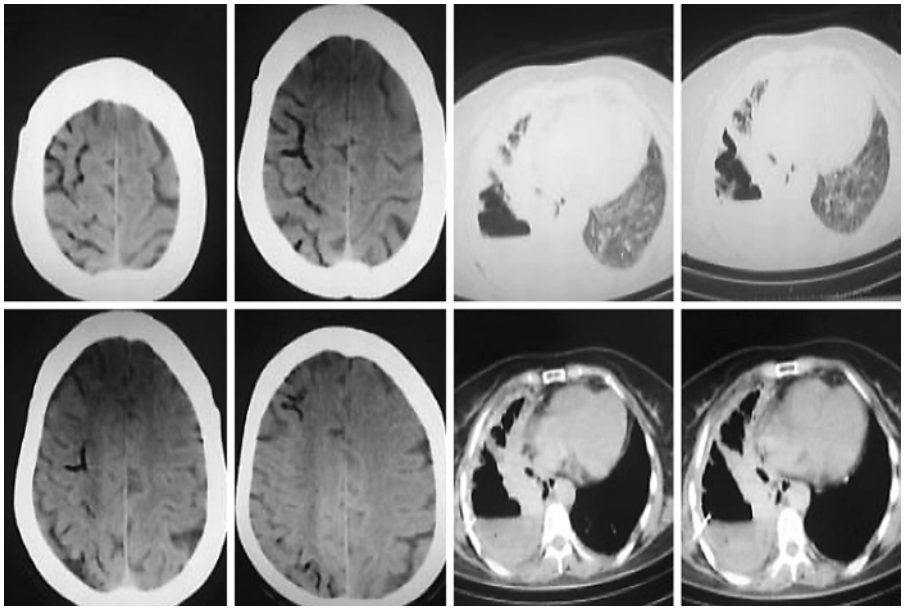

Fig. 1. CT of the head and chest at the time of onset.

operation, air was injected into the thoracic cavity through a central venous cannula to prepare the artificial pneumothorax. When 260 mL of air was injected, slurred speech and decreased left limb muscle strength occurred. The injection of air was immediately stopped. Head CT revealed an air shadow in the right cerebral vessels (Fig. 1, left). Chest CT showed right pleural adhesions; air shadows and pleural effusion were observed in the pleural cavity (Fig. 1, right). Approximately 150 mL of air and 50 mL of pleural effusion were withdrawn from the right thoracic cavity. The patient was treated with hyperbaric oxygen therapy once a day. Inhalation of pure oxygen lasted for 60 min at a pressure of 2.5 ATM (atmospheres) and the pressure was set at 2.5 ATA (atmosphere absolute). The step depressurization method was adopted. A total of 5 courses of treatment were given. After 5 days, the patient was re-examined. Head CT revealed cerebral infarcts in the right frontal, temporal, and parietal lobes of the patient (Fig. 2, left). Chest CT revealed patchy shadows in the 2 lungs, right lung consolidation, pleural effusion, and small air shadows in the right thoracic cavity (Fig. 2, right).

Discussion

Manifestations of patients with air embolism are often atypical, making clinical diagnosis difficult [3]. The incidence of iatrogenic air embolism is 2.65/100,000 of admitted patients. Such cases have been reported in almost all surgical fields [4]. Air emboli entering into the cerebral artery or coronary artery may cause serious adverse consequences. The fatality rate in cerebral arterial air embolism is as high as 21%. A total of 53.8% of patients die of cardiac arrest.

The consequences of air embolism depend on the path through which the air enters the blood, air volume, and velocity, as well as on the location at which the air enters into the blood vessels. The air emboli in the systemic circulation can also enter the left heart artery system through the foramen ovale or septal defects, leading to embolism of the arteries of the brain, extremities, and viscera; this is also called paradoxical embolism. Furthermore, 77% of air emboli enter into the left heart artery system through the foramen ovale or septal defect [5]. The condition may be that the emboli enter the right heart system. This could be due to increased pulmonary artery pressure leading to increased right ventricular and right atrial pressure. In addition, the decrease in pulmonary blood flow leads to a decrease in left heart volume and a decrease in left atrial pressure. Then, the foramen ovale is opened under the action of the left and right atrial pressure gradient, causing a shunt from the right to the left. In addition, some abnormal pathways between the left and right heart, such as atrial ventricular septal defects and nonoccluded arterial ducts, can also cause paradoxical embolisms.

In organs with relatively poor collateral circulation, air embolism may induce ischemia or infarction, while surgery and trauma are the main causes of cerebral arterial air embolism. Air emboli can directly affect brain circulation, and patients are often accompanied by circulatory failure and secondary reduction of cardiac output; thus, such patients will develop focal neurological deficits,

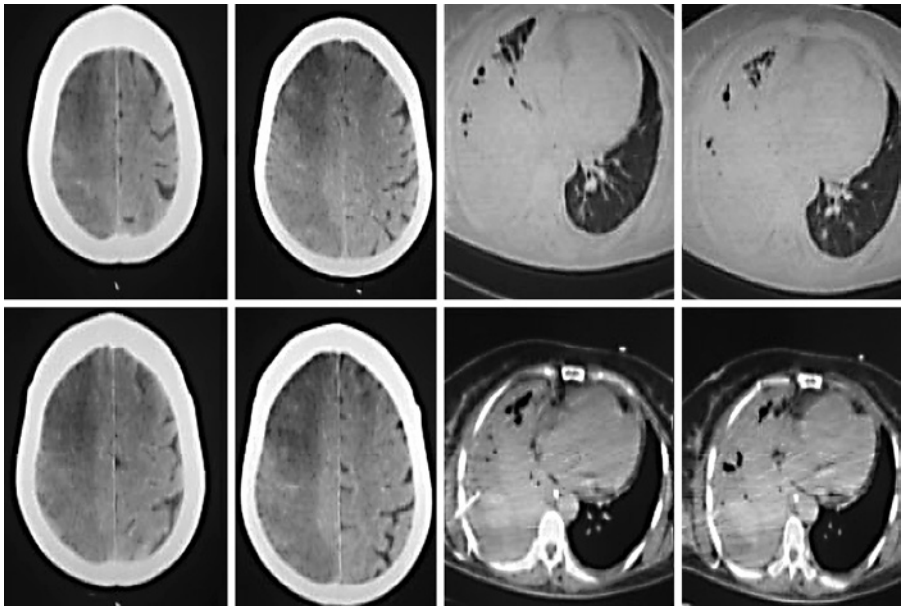

Fig. 2. CT of the head and chest after 5 days.

hemiplegia, and changes in mental state. Air embolism can also stimulate endothelial cells to release inflammatory factors, thereby causing secondary vasospasm and capillary exudation.

When arterial air embolism occurs, it is recommended that the patient is placed in the supine position, because the Trendelenburg position may aggravate cerebral edema. It is recommended that the patient should receive high-flow oxygen until hyperbaric oxygen is available, to promote the re-absorption of nitrogen from the air emboli into the blood and reduce the volume of air emboli. Thereby improving the ischemia of the diseased organs and reducing the fatality rate. It would be best for the patient to receive hyperbaric oxygen therapy within 4–6 h after the occurrence of air embolism. Even if the patient misses the best time of treatment, hyperbaric oxygen therapy within 30 h after the occurrence of embolism would also be of benefit to a certain extent [6].

The patient in the present study developed cerebral arterial air embolism during the induction of artificial pneumothorax possibly due to the increase in intrapleural pressure. Air molecules easily enter into the veins under the action of a pressure gradient. If there is a malignant tumor in the lungs of the patient, the permeability of the vessels of the visceral pleura and the parietal pleura at the lesion sites will significantly increase [7]. Therefore, air emboli are more likely to enter into the venous system. Air emboli may enter into the left heart artery system through the pulmonary veins and cause cerebral arterial air embolism; or the air emboli may enter into the systemic venous circulation through the veins of the parietal pleura and then into the right heart system via the systemic venous circulation and form pulmonary artery emboli. This would subsequently induce an increase in pressure in the right atrium (ventricle) and make the foramen ovale open, thereby allowing air emboli to enter the left heart artery system and cause cerebral arterial air embolism. Air emboli can enter into any part of the brain. However, during the induction of artificial pneumothorax, the patient was placed in the left lateral position, allowing the air emboli to preferentially enter the right cerebral artery.

Acute cerebral arterial air embolism is very serious. In our patient, the clinical accident was discovered in a timely manner and subsequent treatment was given urgently, but she still developed poor activity of the left extremities. During the induction of artificial pneumothorax, changes in pressure in the thoracic cavity should be closely monitored. If the air injection resistance is found to increase, the operation should be terminated in time. This is very important for preventing air embolism.

Conclusion

Artificial pneumothorax may induce cerebral arterial air embolism, and the application of carbon dioxide to establish artificial pneumothorax can effectively reduce

the risk of air embolism, which requires a high degree of vigilance. If cerebral arterial air embolism occurs, it is recommended that the patient be placed in the left lateral or supine position, in order to eradicate the air in the thoracic cavity in time, and be treated with high-flow oxygen or hyperbaric oxygen therapy.

Statement of Ethics

This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of the First Central Hospital of Baoding. Written informed consent was obtained from all participants.

Disclosure Statement

The authors declare that they have no competing interests.

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