

The Migration of Air into the Aorta from an Aorto-Pulmonary Parenchymal Fistula

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Abstract: A 75-year-old man called his care manager with his cell phone when he suddenly experienced hematemesis. Unfortunately, by the time that the care manager arrives at his house, the patient had already fallen unconscious. When emergency medical technicians (EMTs) reached him, he was in a state of cardiopulmonary arrest. A physician transported via helicopter treated him with tracheal intubation. Massive continuous blood from a tracheal tube was observed when he received chest compression. Advanced cardiac life support failed to obtain spontaneous circulation. A postmortem computed tomography (CT) pan scan immediately after ceasing resuscitation revealed systemic air embolism, air in the aorta and bilateral alveolar hemorrhaging. The cause of death was unstable circulation due to massive hemorrhaging from the aorta and air embolism, and respiratory failure due to bilateral alveolar hemorrhaging by aspiration. In the present case, the mechanism of the air in the aorta and carotid artery may have been the result of a massive hemorrhaging from the fistula, and the introduction of air by spontaneous and mechanical ventilation through the fistula between the aorta and pulmonary parenchyma. This unique case adds one more cause to the list of documented etiologies of air in the aorta.

Keywords: air; aorta; lung; fistula.

INTRODUCTION

Spontaneous acute aorto-pulmonary fistula (APF) is a rare clinical entity and usually results in a postmortem diagnosis. The etiology of APF is an inflammatory mechanism, including a history of chest operation or bacterial infection or adhesion between aortic aneurysm and the lung, e.g. via traumatic induction [1-3]. Recently, iatrogenic causes, such as endovascular treatment, have been increasingly reported [4]. We herein report a rare case of a patient with migration of air into the aorta from an aorto-pulmonary parenchymal fistula.

CASE PRESENTATION

A 75-year-old man called his care manager by his cell phone when he suddenly experienced hematemesis. By the time that the care manager finally arrived at his house, the patient had already fallen unconscious. When emergency medical technicians (EMTs) reached him, he was in a state of cardiopulmonary arrest. The initial rhythm was asystole. He underwent basic life supports by EMTs. He had alcoholic liver cirrhosis. A physician transported via helicopter treated him at the rendezvous zone with tracheal intubation, securing a venous route and administering adrenaline; however, he remained in

cardiac arrest when he was transported to our hospital by air.

Massive continuous blood from the tracheal tube was observed when he received chest compression. The findings of a blood gas analysis (FiO₂ 1.0) on arrival were pH: 6.67, PCO₂: 112 mmHg, HCO₃⁻: 12.4 mmol/l and lactate: 14.1 mmol/l. The main results of a biochemical analysis of the blood were as follows: white blood cell count of 5,600/μl, hemoglobin level of 7.0 g/dl, platelet count of 3.3 × 10⁴/μl, aspartate aminotransferase level of 168 IU/L, alanine aminotransferase level of 68 IU/L, blood urea nitrogen level of 14.5 mg/dl, creatinine level of 1.29 mg/dl, creatinine phosphokinase level of 182 IU/l, C-reactive protein level of 0.3 mg/dl, activated partial thromboplastin time of over 150 (26.3) seconds, international normalized ratio of prothrombin time of 4.19, and fibrin degradation products of 206 μg/mL. Advanced cardiac life support finally failed to obtain his spontaneous circulation. A postmortem computed tomography (CT) pan scan immediately after ceasing resuscitation revealed systemic air embolism, air in the aorta and bilateral alveolar hemorrhaging. The cause of death was unstable circulation due to massive hemorrhaging from the aorta and air embolism and respiratory failure due to bilateral alveolar

hemorrhaging by aspiration. Permission for an autopsy

could not be obtained from his family.

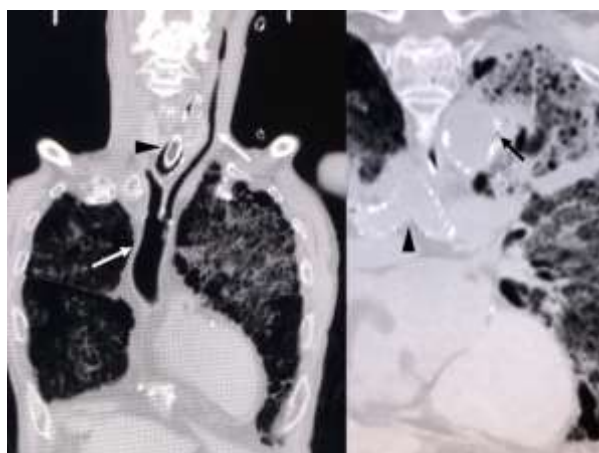


Fig-1: The postmortem computed tomography (CT) pan scan. CT reveals scattered ground-glass attenuations in the lung field, opacification of the tracheal tube and main bronchus (triangle), and air in the ascending aorta and carotid artery (white arrow). CT also depicts a tiny amount of air density at the wall of the aortic arch (black arrow), which might be a fistula between the lung and aorta

DISCUSSION

To our knowledge, this is the first case of the migration of air into the aorta and carotid artery from an aorto-pulmonary parenchymal fistula. The previous main causes of migration of air into the aorta have been iatrogenic, including cardiac or aortic surgery, arterial endovascular management, and a transthoracic lung biopsy [5-9]. However, the present case did not have such a history. In rare cases, the suicidal connection of a peripheral venous catheter with oxygen gas has resulted in the migration of air into the aorta [10]. In patients with decompression sickness, CT can demonstrate intra-arterial gas [11]. In trauma cases, a massive amount of air ventilated with positive pressure in a patient with pneumothorax can enter the site of a penetrating injury of the aorta, resulting in the aorta being filled with air. However, here as well, the present case did not have such a condition. In the present case, the air in the aorta and carotid artery may have been the result of massive hemorrhaging from the fistula, and the introduction of air by spontaneous and mechanical ventilation through the fistula between the aorta and pulmonary parenchyma. This unique case adds one more cause to the list of documented etiologies of air in the aorta.

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