Stanford type A acute aortic dissection associated with aircraft depressurization: Cause or coincidence?

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We declare that the patient approved the study by signing an informed consent form and the study followed the ethical guidelines established by the Declaration of Helsinki.

Abstract

We report the case of a 82-year-old woman, known to have hypertension, who was admitted to the emergency department of Luanda Medical Center for chest pain 24 hours after an episode of commercial aircraft depressurization and whose thoraco-abdominal CT revealed a Stanford type A acute aortic dissection.

Key clinical message

Elderly patients who present at emergency department with chest pain after an episode of depressurization in the cabin of a commercial airplane deserve special attention in order to exclude acute aortic syndrome

Keywords: aortic dissection, Airplane cabin depressurization; Cardiac surgery

Introduction
Acute ascending aortic dissection is an uncommon disease that causes tragic loss of life if not treated emergently. Therefore, having a high index of clinical suspicion is crucial for better survival [1]. Here, we present a case of Stanford type A acute aortic dissection (AAD) associated with commercial airplane depressurization.

Case presentation

An 82-year-old female, autonomous in activities of daily living, with a history of arterial hypertension dyslipidemia, and chronic venous insufficiency medicated with Losartan 50mg/day Simvastatin 20mg/day and Daflon was admitted to the emergency department (ED) of the Luanda Medical Center because she woke up complaining of excruciating chest pain radiating to the back, associated with tiredness. A day earlier, he mentions that on an airplane trip, during the flight there was depressurization in the cabin, causing him a lot of stress and anxiety. Since then she started to feel chest discomfort.

On arrival at the ED, the patient was very complaining, tachypneic, respiratory rate = 28 cycles/min, Sat O2 87% in ambient air, rising to 93% with 3 L of O2 through nasal cannula. BP was 120/70 mmHg, with a pulse 70 bpm, and normal cardiac auscultation. Pulmonary auscultation: diminished vesicular sound with wheezes. Slight edema in the lower limbs. Peripheral pulses are present and symmetric.

Labs: Hb 10.0 g/L, D-dimers 32.199, values of CK-MB and troponine were normal. Blood gases: compensated respiratory acidosis with hypoxemia. Chest Rx (AP-view): Apparent widening of the mediastinum. Thoracic-abdominal CT angiography shows a Stanford type A AAD(Figure 1 A). The patient was referred for surgical treatment. The patient underwent surgical treatment (figure 1B,C), and she died on the 26th postoperative day due to respiratory failure.

Discussion

Commercial aircraft generally use flight levels ranging from 30,000 to 40,000 feet (about 9,000 to 12,000 meters). At this altitude, external effects begin to cause changes in the organism [2]. Depressurization can occur when there is a loss of pressure. This can be fast or slow, being the result of a malfunction in the system or a leak in the aircraft fuselage.

In cases of depressurization, the symptoms are headache, fatigue dizziness, nausea vomiting, lack of concentration/confusion extremity paresthesias and chest pain [3]. In the case reported herein, it was a slow depressurization, the patient was subjected to intense emotional stress and a feeling of panic. During this period the patient experienced atypical chest discomfort. 24 hours latter the patient started experiencing chest pain radiating to the back associated with extreme tiredness. The diagnosis of Stanford type A AADD at ED was made. To the best of our knowledge, there is only one clinical case described in a fighter pilot instructor who developed an AAD at a height of 230 m during left drill action of acute spiral down movement from 2000-m high attitude driven by an air force pilot trainee[1J.

Conclusion

This work deals with a very rare association between the depressurization of a commercial plane and a Stanford-type A AAD. Our hypothesis is that cabin depressurization served as a trigger for the development of AAD, which is supported by the fact that the ascending aorta diameter is normal for age.

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References


Fig. 1A - Thoracoabdominal CT scan revealed Stanford type A acute aortic dissection extending below the renal arteries AAo: Ascending aorta, DAo: Descending aorta, Flap (blue arrow), PT: Pulmonary trunk, TL: True lumen, 1B and C - Intraoperative images reveal the thrombosed false lumen (asterisk), and the Gortex conduit placed in the ascending aorta (blue ellipse)