Clinical Image

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

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

Acute ascending aortic dissection is an uncommon disease that causes tragic loss of life if not treated as an emergency. Therefore, having a high clinical suspicion in such cases is crucial for better survival [1]. Here, we present a case of Stanford type A acute aortic dissection (AAD) associated with commercial airplane cabin depressurization. An 82-year-old female, autonomous in her daily living activities, with a history of arterial hypertension, dyslipidemia and chronic venous insufficiency medicated with losartan 50mg/day, simvastatin 20mg/day and flavonoids, was admitted to the emergency department (ED) of the Luanda Medical Center complaining of excruciating chest pain radiating to the back. Patient presented with a history of an airplane trip on the previous day, with depressurization and associated stress, anxiety, breathlessness, and subsequent chest pain.

On arrival at the ED, the patient was in distress, tachypneic, respiratory rate 28 cycles/min, O2 Saturation of 87% in ambient air, rising to 93% with 3 L of O2 through nasal
cannula. BP of 120/70 mmHg, with a pulse of 70 bpm and normal cardiac auscultation. Pulmonary auscultation: reduced vesicular sound with fine wheezes. Abdomen without organomegaly. Lower limbs edema. Peripheral pulses present and symmetric.

Laboratory findings: Hb 10.0 g/dL, D-dimers 32199 ng/mL, CK-MB 28 U/l, Troponin 0.0040 ng/ml, C-reactive protein 14.8 mg/dl, blood gases: pH 7.42, pCO2 58 mmHg, pO2 46.4, HCO3 29 Eq/L, BE 6, Lactate 0.80 mg/dl. CXR (AP-view): Apparent widening of the mediastinum. Thoraco-abdominal CT angiography shows a Stanford type A AAD (Figure 1A). The patient underwent surgery with placement of a Dacron conduit in the ascending aorta (Figure 1B, C) but died on the 26th postoperative day due to respiratory failure.

Commercial aircraft generally fly at altitudes of 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.

Symptoms of depressurization include 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 with a feeling of panic. During this period the patient experienced atypical chest discomfort. 24 hours later the patient started experiencing chest pain radiating to the back associated with extreme tiredness. The diagnosis of Stanford-type A AAD at ED was made. From the research carried out, we did not find any case linking cabin depressurization to aortic dissection, however we found in the literature a case of a fighter pilot instructor who developed an AAD at a height of 230 m during a leftwards sharp spiral down movement from 2000-m high altitude [1].

In the case in question, there was no family history of dissection, and the patient revealed some risk factors associated with the development of AAD: hypertension, dyslipidemia, and advanced age [4,5]. These risk factors combined with a stressful situation such as cabin depressurization may have acted as a trigger for the onset of AAD, which is corroborated by the fact that the ascending aorta diameter was normal for age (41 mm) and the chest pain started after the depressurization episode. In this patient, there is an unmistakable temporal relationship between depressurization and aortic dissection. As it is impossible to completely exclude other causes of dissection present in this patient (age and hypertension), the authors believe that depressurization, if not the main cause, certainly contributed to the dissection.

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References