Case report

Recurrent inflight chest pain due to a solitary bulla

Carlos Echevarria*, Richard N. Harrison

Department of Lung Health, University Hospital of North Tees, Hardwick Road, Stockton TS19 8PE, United Kingdom

ARTICLE INFO

Article history:
Received 1 November 2011
Accepted 28 November 2011

Keywords:
Solitary bulla
Flight
Inflight
Bullectomy

ABSTRACT

Ms L is a 47-year-old lady who was referred with severe, left-sided pleuritic chest pain and painful left arm weakness that occurred predictably during consecutive commercial flights. Subsequent investigations diagnosed a large left-sided, isolated bulla. A VATS bullectomy was performed with no complications, and a symptomless flight followed. We discuss here the physiological explanation for her symptoms and the treatment of bulla in this unusual case.

1. Case report

Ms L is a 47-year-old lady who was referred to respiratory medicine with recurrent, predictable symptoms occurring during flight, for assessment and flight testing. She recalled having flown in the past without incident, but had started having symptoms in 2003: at altitude she developed a severe, left-sided, stabbing, pleuritic chest pain that radiated through to her back. This was associated with a sudden, severe tightness across her forehead and painless left arm weakness, sufficiently severe to prevent her from using it to lift a cup. There were no associated neurological symptoms, such as facial weakness, dysphasia, or visual disturbances and she did not recall any associated pallor or discolouration in the arm.

The symptoms occurred on 5 flights, the shortest of which lasted around 2.5 h, the longest 4.5 h. After the first episode, all subsequent flights resulted in symptoms that appeared to increase as the aeroplane reached altitude: they then fully resolved as the plane descended. The pain in the forehead was only prominent during one episode, but the pleuritic chest pain and arm weakness were always the same. She never received any inflight treatment: she never received oxygen.

She had a previous history of eczema diagnosed in 1994, and had previously been treated for depression with fluoxetine, though had never suffered with anxiety attacks or claustrophobia. She had a history of mild asthma as a child, and smoked one cigarette a month on social occasions. There was no significant family or occupational history.

Respiratory examination, cardiovascular and neurological examinations were normal. Her oxygen saturations were 100% breathing room air and did not change with posture or exertion.

The chest radiograph showed a subtle reduction of vascular markings in the left mid and upper zone. A CT pulmonary angiogram showed a solitary left apical bulla measuring 10 × 8 × 8 cm and mild peripheral middle and right upper lobe bronchiectasis (Fig. 1). Other investigations including a head MRI were normal. Pulmonary function tests showed normal spirometry, lung volumes by Helium dilution and transfer factor.

A 3-port left VATS was performed via lateral thoracotomy and a giant bulla identified arising from the left upper lobe. Apical adhesions were divided and the bulla was stapled off the left upper lobe. Histology showed the bulla measured 6.5 × 6.0 × 2.0 cm; 4.5 cm diameter. Its walls showed fibrosis and a mild chronic inflammatory infiltrate composed of plasma cells and lymphocytes.

15 weeks after her surgery she undertook an uneventful flight to Florida.

2. Discussion

At higher altitudes, there is a fall in atmospheric pressure, and a corresponding fall in the partial pressure of oxygen. To avoid unwanted physiological complications such as severe hypoxaemia, altitude sickness, and barotrauma, commercial aircraft, which travel at a cruising altitude of around 35,000 feet, are pressurised to around 8000 feet above sea level.1 Pressurising to sea level would create issues with regards to plane weight and fuel consumption.
The relationship between the reduction in pressure on a plane and the volume of gas can be described by Boyle’s law, which describes an inverse relationship between volume and pressure. At normal sea level, atmospheric pressure is around 101 kPa or 760 mmHg. A cabin pressurised of 8000 feet will have a pressure of around 35–40% less than atmospheric pressure, which means there will be a resultant increase in gas volume of 35–40%. This is a potential issue for any gas that is in a confined space; hence the common experience of discomfort due to expanding air in the middle ear during flight. Similarly, any large bulla which is not in communication with the rest of the lung will undergo volume expansion.

Symptoms during flight are not uncommon, the most serious of which are cardiac. The predominant in-flight symptoms are neurological, primarily dizziness or vertigo; others include seizures and headaches. The clinical features described in this case (pleuritic pain, neurological symptoms and headache) are manifest in panic disorder. Whilst this must be considered as one of the differential diagnoses at presentation, other explanations must be sought.

We propose that her symptoms were due to the lung bulla which will have expanded in volume by around 35–40% of its original volume, though this could have been greater or smaller depending upon other factors such as the moisture content of the gas. Bulla can be classified according to the surrounding lung tissue (e.g. normal or emphysematous), and whether or not they communicate. Most bullae are poorly ventilated, and the amount of air trapped can be estimated through the difference in functional residual capacity by helium dilution and body plethysmography. The lung surrounding a bulla is less compliant, so that a bulla is preferentially filled before the adjacent lung. The expansion of gas within a non-communicating bulla would exert a force which would account for the chest pain.

The weakness in the arm we believe was likely to be neuropathic in origin. It could be explained through a direct pressure effect upon the brachial plexus. Theoretically, there could be serious consequences to an expanding pulmonary bulla, though empirical evidence is scarce: A young aeroplane passenger who unexpectedly died has been attributed to a lung bulla. The authors postulated that mediastinal compression or a systemic air embolism could explain the sudden death. Pulmonary haemorrhage attributed to in-flight pressure changes in a patient with emphysema and an enlarged bulla has been described.

The support for our explanation of the symptoms is: 1) the recurrence of the symptoms, which were predictable, always came on at altitude, and resolved whilst the plane descended and 2) the resolution of these symptoms with treatment of the bulla.

Conflict of interest statement
No authors have any actual or potential conflict of interest including any financial, personal or other relationships that can influence or bias this case report.

References
1. Smeets F. Travel for technology-dependent patients with respiratory disease. Thorax 1994;49:77–81.
2. Gong H. Advising pulmonary patients about commercial air travel. J Respir Dis 1990;11:484–99.
3. Astin TW, Penman RW. Airway obstruction due to hypoxemia in patients with chronic lung disease. Am Rev Respir Dis 1967;95:567.
4. Chan SB, Hogan TM, Silva JC. Medical emergencies at a major international airport: in-flight symptoms and ground-based follow-up. Aviat Space Environ Med 2002;73(10):1021–4.
5. Sirven JJ, Claypool DW, Sahs KL, Wingerchuk DM, Bortz JJ, Drazkowski J, et al. Is there a neurologist on the flight? Neurology 2002;58(12):1739–44.
6. Katon W. Panic disorder and somatization. review of 55 cases. Am J Med 1984;77(1):101–6.
7. Deslauriers J, Coire JG, LeBlanc P. Chapter 84, Bullous and bleb disease of the lung. In: Shields TW, Ponn RB, LoCicero J, Rusch VW, editors. General thoracic surgery. 6th ed. Lippincott Williams & Wilkins; 2004.
8. Neidhart P, Suter PM. Pulmonary bulla and sudden death in a young aeroplane passenger. Intensive Care Med 1995;11(1):45–7.
9. Chen CW, Perng WC, Li MH, Yan HC, Wu CP. Haemorrhage from an enlarged emphysematous bulla during commercial air travel. Aviat Space Environ Med 2008 Dec;77(12):1275–7.