

CASE REPORT

Positional dyspnoea due to aneurysm of the thoracic aorta

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Positional dyspnoea due to aneurysm of the thoracic aorta. G.D. Phillips, E.E.J. Smith, F.J.C. Millard. ©ERS Journals Ltd 1994.

ABSTRACT: Thoracic aortic aneurysms may produce breathlessness by compressing the tracheobronchial tree. We report a patient whose shortness of breath demonstrated a marked positional component, due to varying compression of her major airways by the lesion.

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Chronic aneurysm of the thoracic aorta may present as a chance finding on X-ray in an otherwise asymptomatic patient, as pain from aortitis, erosion of the sternum or vertebral column and compression of the spinal nerves, or due to symptoms arising from compression of adjacent structures [1]. The latter is particularly common in those aneurysms which involve the arch or proximal descending thoracic aorta. Respiratory symptoms, such as dyspnoea, wheezing, stridor, cough, haemoptysis and recurrent infections have been described in textbooks as being due to compression of the tracheobronchial tree, although few case reports exist. We describe a case of positional dyspnoea due to compression of the tracheobronchial tree by an extensive thoraco-abdominal aortic aneurysm.

Case report

A 67 year old woman, with mild smoking-related chronic airflow limitation, presented with a 2 yr history of aching pain in the lumbar spine, which had worsened over the last 2 months. For 2 weeks prior to presentation, she had noted progressively increasing exertional dyspnoea, which finally culminated in breathlessness at rest and an exercise tolerance of only a few yards. The breathlessness was markedly worse when she lay on her back or on her left side, such that she was forced to sit propped up in bed, leaning on her right side. There was no chest pain or cardiac history, and there were no other respiratory symptoms.

On examination, she was thin and breathless at rest. The inspiratory phase of respiration was prolonged, and there was mild audible inspiratory stridor. There were

no abnormal signs in the cardiovascular or respiratory systems, apart from mild hypertension (170/110). She had a moderate sized abdominal aortic aneurysm, but all peripheral pulses were present and symmetrical.

Chest X-ray (fig. 1) showed a widened aortic arch and widening and tortuosity of the descending thoracic aorta. Echocardiography confirmed aneurysmal dilatation of the ascending and descending aorta, with moderately good left ventricular function. On abdominal ultrasound examination, the maximum aortic diameter was 4.7 cm.

On lung function testing peak expiratory flow rate (PEFR) was 170 l·min⁻¹ (43 % predicted), forced expiratory volume in one second (FEV₁) was 1.25 l (81 % pred) and forced vital capacity (FVC) 1.84 l (75 % pred), giving an FEV₁/FVC ratio of 68%. The maximum flow-volume curve showed gross reduction of both inspiratory and expiratory flows at all lung volumes. The peak inspiratory flow rate (PIFR) was 156 l·min⁻¹ (52 % pred). There was no response to an inhaled bronchodilator. Arterial blood gases performed with the patient breathing room air at rest revealed an arterial oxygen tension (Pao₂) of 9.4 kPa (71 mmHg) and an arterial carbon dioxide tension (Paco₂) of 4.7 kPa (36 mmHg).

Computerized tomographic (CT) examination of the thorax, performed with the patient lying on her right side, confirmed the dilatation of both the ascending (maximum diameter 5 cm) and descending (maximum diameter 7 cm) aorta, and demonstrated marked compression of the major intrathoracic airways, particularly on the left side (fig. 2). Owing to the patient's extreme breathlessness, it was not possible to repeat the procedure with the patient lying on her left side. There were

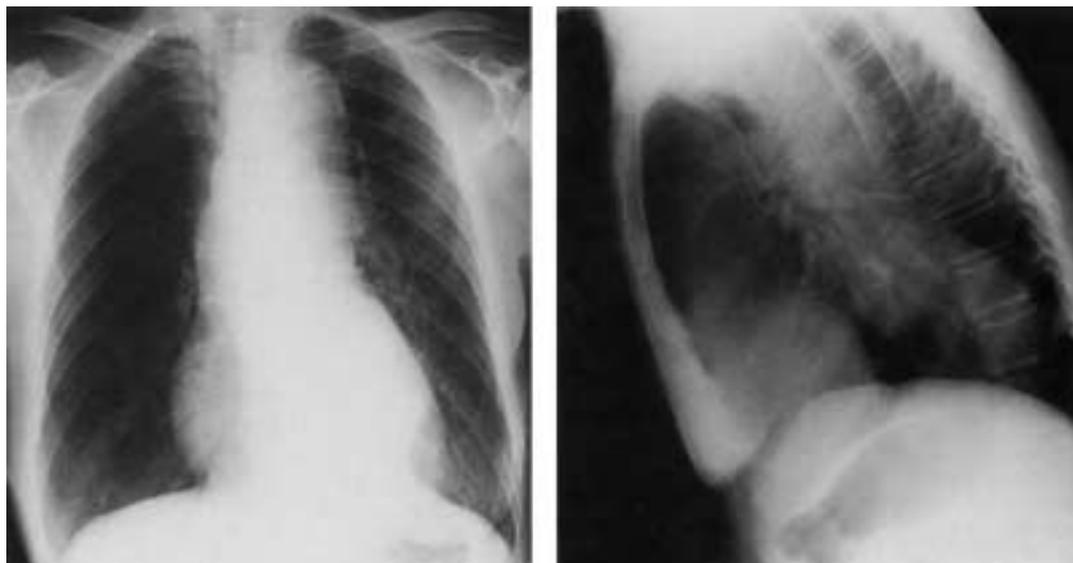


Fig. 1. — Posteroanterior and lateral chest X-ray showing large thoracic aortic aneurysm.



Fig. 2. — Computerized tomographic (CT) scan 2 cm below carinal level, taken with patient lying on her right side. Note the marked compression of the major airways, particularly on the left side.

no pulmonary parenchymal abnormalities. At fiberoptic bronchoscopy, there was severe extrinsic compression of the left sided airways, the lumen of the left main bronchus being reduced to 25% of its normal size. The trachea was deviated to the right and its lumen was diminished by 50%. The right main bronchus was much less severely affected. The patient refused any further investigations.

Discussion

This patient's breathlessness is unlikely to have been cardiac in origin, since there was no clinical or radiological evidence of cardiac failure, and echocardiography showed good left ventricular function, with no evidence of any valvular abnormality. Although she

was a smoker and lung function tests showed an obstructive defect, chronic airflow limitation would not explain the markedly postural nature of her breathlessness. In addition, there was no evidence of a phrenic nerve lesion.

Although dyspnoea is well-recognized as a symptom of thoracic aortic aneurysms, and compression of the trachea and bronchi is mentioned in the literature [2, 3], and in many standard texts [1, 4], we believe that this is the first well-documented case report of marked positional breathlessness caused by such a lesion. The very severe narrowing of the left main bronchus observed at bronchoscopy is the most likely explanation for the marked worsening of this patient's symptoms, particularly noted in the left lateral position, and for her preference for sitting forward leaning to the right. Compression of adjacent structures is especially common in aneurysms of the transverse and descending arch, the so called "aneurysm of symptoms" [2]. Other structures which may be similarly affected include the oesophagus and left recurrent laryngeal nerve.

CHALANT *et al.* [5] reported seven cases of traumatic rupture of the descending aorta, and observed that there is often an anteriorly directed bulge, which compresses the tracheobronchial bifurcation and causes breathlessness. In a separate case report, VARKEY and TRISTANI [6] described a patient who presented with breathlessness and wheeze due to compression of the left main bronchus by an aneurysm of the proximal descending thoracic aorta. Compression of the left pulmonary artery also occurred, and these effects were corrected by successful surgery.

The cause of this woman's aneurysm is probably atherosclerosis, and is no doubt related to her history of mild hypertension and of smoking. Her syphilitic serology was negative. This was a very extensive thoracoabdominal aneurysm, for which surgery would have been extremely hazardous. In any case, the patient refused any further investigation or treatment of her condition.

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