Bronchial compression as a result of lung herniation after pneumonectomy

K F Whyte, G McMahon, A J A Wightman, E W J Cameron

Abstract
A patient developed severe exertional dyspnoea and stridor eight months after a right pneumonectomy for a carcinoid tumour, with a progressive loss of lung function. These events were the result of compression of the left main bronchus against the vertebral column by the mediastinal contents, which had shifted into the right hemithorax with the herniated lung.

Pneumonectomy is a common surgical procedure with a low mortality and morbidity. We report a patient in whom gross herniation of the left lung into the right hemithorax after a right pneumonectomy led to progressive exertional dyspnoea, stridor, and serial loss of lung function.

Case report
A 40 year old housewife presented in December 1984 with recurrent right middle and lower lobe pneumonia. There was a history of mild asthma since 1978, which had been treated with salbutamol and beclomethasone.

Bronchoscopy disclosed a carcinoid tumour obstructing the right intermediate bronchus. Enlarged, matted right hilar glands were found at thoracotomy. She underwent a right pneumonectomy and made a rapid recovery. The resected specimen showed a carcinoid tumour with no evidence of local or distant spread. There were numerous granulomas in the hilar glands, consistent with inactive tuberculosis.

Postoperatively her FEV₁ was 1·75 (predicted 3·05) litres. Eight months after operation she noted increasing cough, wheeze, and exertional dyspnoea. On examination she had inspiratory and expiratory stridor and tracheal deviation. Her FEV₁ was 0·95 l (table), with no reversibility after inhalation of terbutaline or three weeks’ treatment with oral prednisolone (40 mg/day). A chest radiograph showed that the mediastinum and left lung had herniated into the right hemithorax (fig 1). Bronchoscopy showed a bowing indentation causing pronounced narrowing half way along the left main bronchus.

Static lung volumes (table) were 0·75–1·00 litres below the predicted values, in keeping with a previous pneumonectomy, and transfer factor for carbon monoxide was proportionately reduced. Arterial blood gases were normal. In a progressive exercise test she did not develop oxygen desaturation but was stopped by dyspnoea at a work rate of 32 watts. Flow-volume curves were characteristic of intrathoracic airflow obstruction, with no evidence of fixed tracheal or extrathoracic obstruction.

Serial respiratory function values

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<tbody>
<tr>
<td>FEV₁ (l)</td>
<td>2·1</td>
<td>1·7</td>
<td>0·95</td>
<td>1·1</td>
<td>0·85</td>
<td>0·75</td>
<td>2·8</td>
</tr>
<tr>
<td>FVC (l)</td>
<td>3·1</td>
<td>2·3</td>
<td>2·2</td>
<td>2·45</td>
<td>2·45</td>
<td>2·45</td>
<td>3·6</td>
</tr>
<tr>
<td>FEV/FVC (%)</td>
<td>68</td>
<td>74</td>
<td>42</td>
<td>46</td>
<td>36</td>
<td>31</td>
<td>77</td>
</tr>
<tr>
<td>FRC (l)</td>
<td>3·4</td>
<td>—</td>
<td>2·9</td>
<td>3·4</td>
<td>3·6</td>
<td>4·5</td>
<td>3·6</td>
</tr>
<tr>
<td>TLC (l)</td>
<td>5·5</td>
<td>—</td>
<td>4·4</td>
<td>4·6</td>
<td>5·1</td>
<td>6·0</td>
<td>5·5</td>
</tr>
<tr>
<td>RV (l)</td>
<td>2·4</td>
<td>—</td>
<td>2·05</td>
<td>2·4</td>
<td>2·6</td>
<td>2·6</td>
<td>1·8</td>
</tr>
<tr>
<td>RV/TLC (%)</td>
<td>44</td>
<td>—</td>
<td>47</td>
<td>49</td>
<td>51</td>
<td>57</td>
<td>33</td>
</tr>
<tr>
<td>TLCo (mmol min⁻¹ kPa⁻¹)</td>
<td>6·00</td>
<td>—</td>
<td>6·85</td>
<td>6·15</td>
<td>6·7</td>
<td>8·6</td>
<td></td>
</tr>
<tr>
<td>Kco (mmol min⁻¹ kPa⁻¹)</td>
<td>1·45</td>
<td>—</td>
<td>1·65</td>
<td>1·55</td>
<td>1·90</td>
<td>1·75</td>
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<tr>
<td>Max workload (w)</td>
<td>—</td>
<td>—</td>
<td>48</td>
<td>32</td>
<td>32</td>
<td>16</td>
<td>—</td>
</tr>
<tr>
<td>V̇e (l/min)</td>
<td>—</td>
<td>—</td>
<td>17·2</td>
<td>16·3</td>
<td>13·3</td>
<td>NA</td>
<td>102</td>
</tr>
<tr>
<td>Max heart rate (beats/min)</td>
<td>—</td>
<td>121</td>
<td>119</td>
<td>120</td>
<td>106</td>
<td>100</td>
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</table>

FEV₁—forced expiratory volume in one second; FVC—forced vital capacity; FRC—functional residual capacity; TLC—total lung capacity; RV—residual volume; TLCo—carbon monoxide transfer factor; Kco—transfer coefficient; V̇e—minute volume; NA—not available.
Computed tomography showed gross herniation of the left lung into the right hemithorax; the left main bronchus can be clearly seen, stretched across the vertebral column and compressed by the mediastinal organs and vessels (fig 2).

Three years after operation she complained of further loss of exercise tolerance. Though her ventilatory capacity was unchanged, total lung capacity had increased from 4-8 to 6-0 litres. Transfer factor was unchanged. Arterial blood gases remained normal but her exercise tolerance had fallen to 16 watts.

A further thoracotomy was performed to attempt a corrective procedure but was abandoned because of right apical pleural adhesions. The patient has had subsequent admissions with recurrent pulmonary infections and remains severely disabled.

Discussion

Stridor and such severe exertional dyspnoea are not recognised complications of pneumonectomy, a relatively common operation (1500 per year in the UK, thoracic surgery register), though mediastinal shift is common after pneumonectomy and herniation of the remaining lung into the opposite hemithorax, causing an increase in lung volume, has been reported. These shifts of thoracic structures are not usually associated with symptoms. In young children and adolescents there have been isolated reports of lung herniation that has led to cough, dyspnoea, and stridor, resulting both from pneumonectomy and from agenesis of the right lung. Two cases of similar symptoms following right pneumonectomy in adults have been reported. In the first case the authors attributed the stridor to an increase in air turbulence in the elongated and tortuous trachea and the severe exertional dyspnæa to the increased elastic work resulting from overdistension of the left lung superimposed on the increased resistance of the distorted airways. We would suggest, as did the authors of the second report, that the principal cause of the symptoms is compression of the left main bronchus against the vertebral column by the aortic arch and the great vessels as they rotate towards the vertebral gutter, as shown in the computed tomogram (fig 2). Our serial measurements show the relentless loss of lung function, possibly due to tracheobronchial malacia secondary to this compression. Tracheobronchial malacia, the weakening of cartilage and myoelastic elements as a result of prolonged pressure, leading to partial or complete airway collapse, particularly during coughing, has been found in three children after right pneumonectomy. It is complicated by an increased incidence of pulmonary infections. The increased elasticity and compliance of the lung and mediastinum in the young may explain the higher incidence of this rare complication in children. We are not clear why this should have occurred after a right pneumonectomy in our patient.

In the original case report the left lung was returned to the left hemithorax by suturing four silastic implants, similar to those used in augmentation mammoplasty, into the right hemithorax and thus filling the redundant space. An appended editorial comment states that in three similar cases, all following right pneumonectomy, suturing the right side of the pericardium to the left anterior chest wall was successful in correcting the rotation and removing the compression of the airway. At operation our patient had adhesions and the risk of lung damage during mobilisation precluded such an attempt.

The possible use of a silastic tube prosthetic stent was discussed; in a relatively young woman, however, without a malignant condition a decision was made to attempt corrective surgery. The recent description of the use of expandable metal stents in the treatment of bronchial obstruction raises the possibility of an alternative approach in this patient. Such an attempt would be experimental and the patient does not want any further intervention at present.

This patient was under the care of the late Professor David C Flenley and we acknowledge our debt to him.

2. Kristersson S, Lindell SE, Svanberg L. Prediction of
Synchronous double primary lung cancers of squamous and neuroendocrine type associated with cryptogenic fibrosing alveolitis

Jeong-Wook Seo, Jung-Gi Im,
Young-Whan Kim, Joo-Hyun Kim,
Mary N Sheppard

Abstract
A 72 year old man with simultaneously occurring squamous cell and neuroendocrine carcinomas in association with cryptogenic fibrosing alveolitis is reported. The tumours were separate and both were in the fibrotic area of the right lower lobe.

We report a case of two synchronous cancers developing in a 72 year old man with cryptogenic fibrosing alveolitis. The histological types were squamous cell carcinoma and neuroendocrine carcinoma, a combination that has not been reported previously.

Case report
A 72 year old Korean merchant visiting a hospital for a routine check was found to have a nodular density associated with diffuse infiltrates on his chest radiograph. He had smoked 20 cigarettes daily for 50 years. Cough, sputum, and mild dyspnoea were present. Physical examination disclosed end inspiratory cracks at both lung bases. The chest radiograph and high resolution computed tomogram of his chest showed diffuse, irregular, and bilateral reticulonodular densities in the lung, which were more pronounced in the lower lung fields and subpleural areas in keeping with cryptogenic fibrosing alveolitis. Two intrapulmonary masses, 2 and 3 cm in diameter, were also seen in the right lower lobe in association with the reticulonodular densities. Ventilatory function was normal; transfer factor for carbon monoxide was 63% predicted.

A right lateral thoracotomy disclosed a serosanguinous effusion and diffuse pleural fibrosis and nodularity. A right lower lobectomy was performed.

Gross examination of the resected lobe showed diffuse honeycombing, especially in the subpleural areas of the lung. There were also two separate masses within the honeycomb area, measuring when fixed 3.0 x 1.5 x 1.5 cm and 1.2 x 1.0 x 0.5 cm. The larger one had a granular cut surface and

Figure 1. Slice of the right lower lobe showing subpleural honeycombing and a fleshy mass arising in it.
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