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Case report

Lipoid pneumonia; an unsuspected diagnosis

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We describe a case of a woman who presented with a history suggestive of respiratory infection which was felt to be secondary to bronchial carcinoma, but was subsequently found to have exogenous lipoid pneumonia. In retrospect we wonder if this diagnosis could have been made preoperatively, and if this would have altered our management.

CASE REPORT. A 74-year-old female was admitted with a two month history of increasing breathlessness and malaise. Prior to this she had been fit and active. She had begun to feel unwell and developed a cough, initially producing pink sputum which later became green in colour. Two months prior to admission she developed severe right sided pleuritic chest pain on coughing. Initially she was breathless on walking half a mile but gradually became dyspnoeic at rest. Her appetite decreased but she had not noticed any loss of weight. She had smoked 20 – 30 cigarettes daily until the age of 66 years. There was no previous exposure to dust or asbestos. She had had pneumonia at the age of 16 but there was no previous history of tuberculosis.

On admission she was feverish (38·8 C) but had no anaemia, cyanosis, lymphadenopathy or finger clubbing. Her respiratory rate was 20/min. She had signs of decreased chest expansion, dullness to percussion and reduced air entry and vocal resonance in the right mid zone. Pulse rate was 100/min, blood pressure 170/60 mmHg. ESR was 100 mm per hour, haemoglobin 9·7 g/dl, mean cell volume 83 fl and white cell count 29·1 10^9/l (91% neutrophils). Serum albumin was 24 g/l (normal 35 – 50), alkaline phosphatase 474 μ/l (90 – 280) and gammaglutamyl transferase 96 μ/l (5 – 60). Arterial blood pH was 7·50 (7·35 – 7·44), pO2 9·73 KPa (12·0 – 14·6), pCO2 4·2 KPa (4·7 – 6·0) and standard bicarbonate 26·6 mmol/l (22 – 26). Repeated sputum microscopy revealed some polymorphoneuclear leucocytes, epithelial cells and gram positive cocci, but culture grew commensal organisms only. Sputum cytology and microscopy for acid and alcohol fast bacilli were negative. Chest X-ray (Fig 1) showed a large mass in the lateral part of the right midzone. CT scan showed the large mass to be in the right upper lobe extending out to the chest wall and

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involving it at one point. Enlarged lymph nodes were seen in the pre-cranial area. There was no enhancement with contrast. Bone scan was normal.

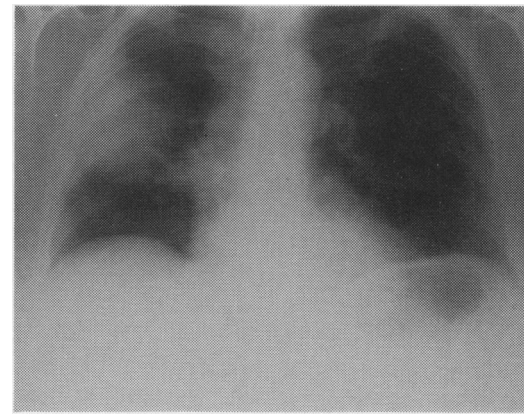

Fig 1. Chest X-ray on admission showing consolidated areas in the right mid zone.

She was initially treated with oral ampicillin. Bronchoscopy was attempted three days later, but mild laryngospasm caused immediate anoxia and the procedure had to be abandoned. Treatment was changed to intravenous erythromycin, her temperature fell after two days and remained normal. Three days later she had a large haemoptysis, and the chest X-ray had deteriorated. During the following week her condition gradually improved again and her haemoglobin rose to 11·6 g/dl, white cell count 8·3 10^9/l and all biochemistry had returned to normal. Bronchoscopy was repeated under general anaesthesia, and biopsy showed occasional inflammatory cells, but no dysplasia or malignancy.

Three weeks after her initial presentation a thoracotomy revealed a dense right upper lobe mass which was removed. She made an uneventful postoperative recovery and remains well with no symptoms.

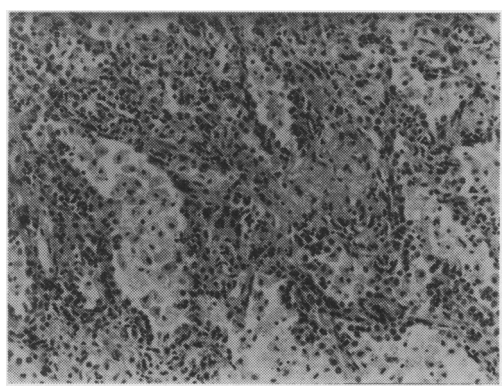

Fig 2. Area showing lipid-laden macrophages. The septa are infiltrated by chronic inflammatory cells. H & E × 630.

The right upper lobectomy specimen was virtually replaced by a solid greyish-white mass (7 × 4 × 4 cm) with overlying pleural thickening. The histological features were dominated by intra-alveolar collections of lipid-filled macrophages associated with marked interstitial fibrosis, and inflammatory infiltrates of lymphocytes and plasma cells (Fig 2). Many of the small arteries showed fibrosis. The major bronchi were not obstructed by malignancy or other lesions. The appearances were those of chronic lipoid pneumonia.

Subsequent questioning of the patient revealed that she had repeatedly used a liquid paraffin laxative at night when on holiday two months previously. She had noticed no dysphagia at that time.

DISCUSSION

Most diagnoses are reached in medicine following clinical history-taking and examination. In some cases patients initially fail to report events which may later

prove to be of importance. This is well demonstrated in the present case where a woman who presented with a respiratory illness and was found to have a mass in her right lung field, gave no history of aspiration, and was felt to have a lung neoplasm. The history of liquid paraffin ingestion was obtained only by specific questioning after the histological diagnosis was obtained.

Lipoid pneumonia was first described by Laughlen in 1925[1] who found consolidated areas of lung, which contained oil, in autopsies of children. This oil was situated in phagocytic vacuoles of mononuclear endothelial leucocytes and stained positively with Sudan III. The children had received oil preparations as part of their treatment. Laughlen then produced similar changes by innoculating rabbits with oil confirming the relationship of lipoid pneumonia with exogenous administration of oil. Since this first report others[2] have noted the diversity of possible presentations of lipoid pneumonia, and its ability to mimic other conditions. Patients may be asymptomatic or diagnosed with acute or chronic pneumonitis, or as in this case may simulate carcinoma of the lung.[2] The clinical presentation depends on the amount and the characteristics of the ingested oil. A high free fatty acid content and a high viscosity stimulates greater pulmonary damage.[3,4] Liquid petroleum is a mixture of refined hydrocarbons of high viscosity, which our patient took as a laxative on retiring to bed on holiday. Fox and Bartlett[5] noted that patients who take mineral oil at bedtime are particularly susceptible to lipoid pneumonia. A study of 389 chronically ill patients found 14·6% incidence of lipoid pneumonia.[6] The commonest reason for ingestion of oil in this group was for constipation. Other reports have described lipoid pneumonia caused by lip gloss,[7] aerosol lubricant[8] and inhalation of liquid paraffins in a female fire eater.[9]

Lipoid pneumonia may be difficult to diagnose. Specific questioning about oil ingestion may be required. Demonstration of a diffuse multilobar infiltrate or a well-circumscribed homogeneous infiltrate in the lower lobes[5] on chest X-ray may suggest the diagnosis, but other authors have required open lung biopsy to establish the diagnosis.[2,7,10,11,12] If lipoid pneumonia is suspected, microscopic examination of sputum or bronchial washings for fat and oil laden macrophages may be useful diagnostically.[6,13] More recently characteristic absorption on computed tomography,[14] or magnetic resonance scan[15] have been described.

Treatment of lipoid pneumonia is to avoid the offending agent. Repeated innoculation may otherwise result in recurrent acute respiratory inflammation.[2] Bacterial superinfection requires antibiotic treatment. Some authors have suggested a role for corticosteroid therapy[10,11] but others have found no benefit.[16,17] Diagnosis of this rare condition using some of the methods reviewed above might have resulted in gradual resolution of lipoid pneumonia and avoided thoracotomy and lobectomy.

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