Unusual lung consolidation in SLE

Pulmonary manifestations of systemic lupus erythematosus (SLE) include acute lupus pneumonitis, chronic interstitial pneumonitis, diaphragmatic dysfunction, atelectasis, pulmonary vascular disease, upper airway dysfunction, and bronchiolitis obliterans. Pneumonia due to infection also develops because infection is a major cause of death of patients with SLE. Here, we report a patient with SLE who suffered from an ambulatory lung consolidation devoid of pathogens.

A 61 year old woman with SLE had been treated with 10 mg prednisolone every other day since 1990 without deterioration in the clinical course of end-stage renal disease in systemic lupus erythematosus. European Working Party on Systemic Lupus Erythematosus. Medicine (Baltimore) 1999;78:167–75.

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Authors’ reply

We would like to thank Dr J Balba for the thoughtful comments on our paper. As the prevalence of atypical mycobacterial infection was not systematically evaluated in the studies we reviewed, we could not integrate this into our analysis. From a practical point of view both BCG and non-BCG groups are likely to have similar exposures to atypical infection, and clinicians would not be able to assess routinely for such infections. Similarly, information on nutrition was not systematically available but, as a surrogate, we have looked at the impact of BCG as one moves further from the equator, this suggests that nutrition is not a significant factor in the sample size we calculated. Such an effect on nutrition is consistent with the findings of the World Health Organization.
The patient was admitted with a dry cough, shortness of breath, back pain, and progressive infiltrates on chest radiographs. He had no history of risks for BALT lymphoma.  

No rash or lymphadenopathy or organomegaly was detected. A CT scan of the chest showed a right mid lung bulky mass with a diameter of 10.9 × 10.6 cm and infiltrations in both lung fields (Fig. 1). A transbronchial biopsy specimen was compatible with low grade (B cell lymphoplasmocytoid type) lymphoma. Immunohistochemical examination showed a monoclonal membrane surface κ light chain positive. The patient underwent combined chemotherapy (CHOP) which was repeated every 3 weeks. He tolerated the treatment without difficulty, his symptoms improved, and CT scans after completion of six courses of treatment showed a marked reduction in the lesions in both lung fields (Fig 2).

BALT lymphoma shows an indolent course and remains localised for a prolonged period of time, with systemic dissemination occurring late in the clinical course. Recommended treatment options include complete surgical resection, radiotherapy, or chemotherapy. The role of surgery in the management of primary lymphoma of the lung is twofold: (1) to obtain diagnostic tissue and (2) to obtain a therapeutic resection. In our case we used combined chemotherapy because surgical intervention is of limited use in patients with a large non-resectable lesion or bilateral lung disease.  

We conclude that, in patients with a large or bilateral pulmonary BALT lymphoma, transbronchial or transbronchoscopic biopsy and mediastinoscopy are useful diagnostic procedures for obtaining a definitive diagnosis and treatment with combined chemotherapy should be considered.

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References


Obstructive sleep apnoea can directly cause death

A 52 year old woman was referred for investigation of daytime somnolence. She complained of heavy snoring, disturbed sleep, and had fallen asleep while driving. She had an Epworth score of 24/24, a history of hypertension controlled on losartan, had never smoked, and only took occasional alcohol. She had limited mobility as a result of her extreme obesity (168 kg) with a height of 1.58 m (BMI 67.3 kg/m²). Her chest was clinically clear (FEV₁, 1.81, FVC 2.3 I) and her serum bicarbonate level was 31 mmol/L, implying a degree of hypercapnia. She had a trace of oedema but no evidence of cardiac failure.

She was admitted 1 month later for a sleep study. Data collection included pulse rate, movement and sound (SSI Visilab). At 04.30 hours the nursing staff found her lying dead across the bed. The oxygen saturation by pulse oximetry was 91% at the start of the night in a sitting position. Startling data were obtained for the first 25 minutes of the study, the remaining being highly fragmented with values fluctuating between 90% and the instrument cut off level of 25%. The video showed a repeated but irregular pattern of apnoea, snoring, arousal, sitting up, falling asleep, and lying back into the supine position. From one such apnoea she failed to rouse sufficiently to resume breathing and suffered a cardiorespiratory arrest. Post mortem examination showed some coronary atheroma but, crucially, no occlusion, leading to the conclusion that the death was directly attributable to obstructive sleep apnoea (OSA). Lungs, liver and spleen showed some congestion consistent with the post mortem diagnosis of acute cardiorespiratory failure. The coroner initially expressed concern that the patient was not being directly observed. After discussion it was accepted that a sleep test is not monitoring in the usual sense but is an exercise in data collection performed either in hospital or at home for reasons of organisational convenience.

This recorded death directly resulting from OSA in combination with severe obesity is unlikely to be unique and may be unusual only in that it was captured on the video recording. In such extreme cases recognising the component of OSA may be difficult as the oximetry recording is erratic rather than the familiar “saw tooth” waveform. A number of mechanisms associating OSA with increased morbidity and mortality have been proposed, significantly obesity and ventilatory failure and vascular disease. However, this case demonstrates a causal connection.

Attributing unexpected deaths to cardiac events rather than to OSA may conceal a number of deaths directly caused by OSA.

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References


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Infections, lymphomas, and metastases are unique causes of Pancoast’s syndrome. The most common aetiological factor is non-small cell lung cancer (NSCLC). Pancoast’s syndrome resulting from metastatic renal cell carcinoma (RCC) has not previously been reported. We present the case of a metastatic Pancoast tumour in the left lung in a patient who had previously undergone resection for RCC.

A 49 year old woman was admitted with a pain in the left shoulder radiating to the arm which had been present for several months, eight years after excision of the left kidney for clear cell RCC. Computed tomographic scans of the chest and abdomen revealed a solitary irregular mass (4.5 cm) in the apical segment of the left lung adhering to the thoracic inlet with no evidence of local recurrence in the abdomen. Extrapleural resection of the tumour with the apical segment of the lung was performed. Histological examination proved the metastatic origin of the tumour from RCC. Nine months after resection of the metastatic tumour the patient had a relapse in the left thoracic inlet. Residual left upper lobectomy with excision of ribs 1–4 and the infiltrated part of the brachial plexus was performed. Seven months later the patient underwent radiation therapy to the tumour bed and supraclavicular region because of metastases in the scalene nodes (total dose 60 Gy in 27 fractions over 41 days).

Twenty five months after the first metstatic resection the patient’s general condition deteriorated due to dissemination of the disease. Radiological examination showed several new foci in both lungs and a metastatic tumour in the brain. The patient died 31 months after the first thoracotomy and 11 years after nephrectomy.

We conclude that metastatic RCC should be considered as a possible cause of Pancoast’s syndrome. The resection should be as radical as in NSCLC—if necessary involving the adjacent structures—which should have been done in our first operation.

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References

BOOK REVIEW

The Clinician’s Guide to Asthma

This is an easy to read and thorough guide to asthma which covers a wide range of topics from prevalence to treatment, pharmacology to pathology. The text is engaging and interesting. It is well illustrated with numerous pictures and diagrams that enhance its readability. The strength of this book lies in its clarity. Even the chapter on the pathophysiology of asthma can be read and understood by those who feel weak at the very mention of cytokines! It is also a contemporary text and provides a useful insight into emerging ideas and novel treatments in this field. The book is suited to those who wish to obtain a broad overview of the subject and would suit medical and MRCP students, respiratory trainees, and other health professionals involved in the care of the asthmatic patient.

Although the management of the asthmatic patient is covered in detail, this is not a handbook to guide individual patient care but is, instead, a concise and thoughtful review of the disease.

In summary, if asthma is not your primary field of interest and you were asked to give a talk on the subject tomorrow, you would be well advised to hunt out this book.

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CORRECTION

Montelukast and inhaled budesonide in mild to moderate asthma
In the paper entitled “Effect of montelukast added to inhaled budesonide on control of mild to moderate asthma” by MJ Vaquerizzo et al which appeared in the March issue of Thorax (2003;58:204–11), there is an error in the first sentence of the abstract which should read “Proinflammatory leukotrienes, which are not completely inhibited by inhaled corticosteroids, may contribute to asthmatic problems”. The publishers apologise for this error.