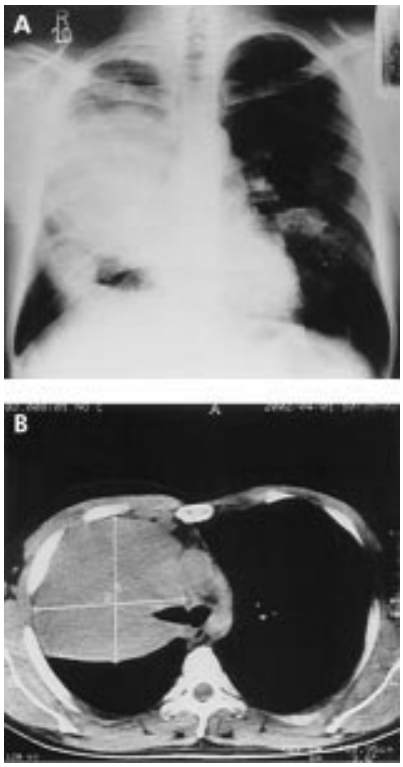

Figure 1 (A) Posteroanterior chest radiograph showing a bulky mass in the right lung, a smaller mass in the lower zone of the right lung, and heterogeneous infiltration in the middle zone of the left lung. (B) CT scan of the chest showing a bulky mass in the right lung.

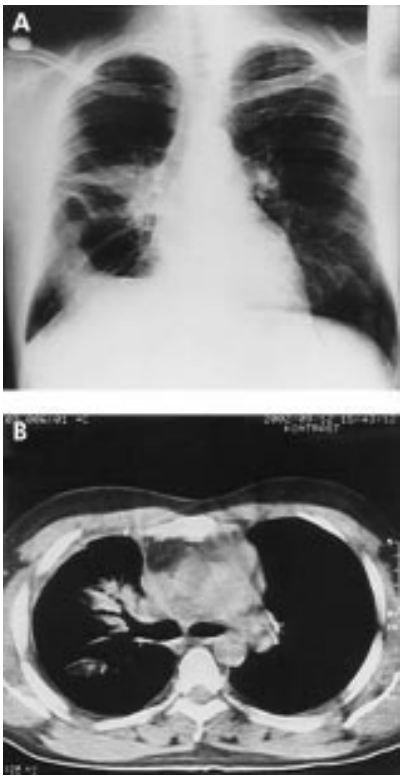

Figure 2 (A) Posteroanterior chest radiograph and (B) CT scan showing marked reduction in the lesions shown in fig 1.

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.[1 3 4] 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.[2] 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.[2 5]

We conclude that, in patients with a large or bilateral pulmonary BALT lymphoma, transbronchial or transthoracic biopsy and mediastinoscopy are useful diagnostic procedures for obtaining a definitive diagnosis[5] and treatment with combined chemotherapy should be considered.

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Obstructive sleep apnoea can directly cause death

A 52 year old woman was referred for investigation of daytime somnolence. She complained of heavy snoring, unrefreshing disturbed sleep, and had fallen asleep while driving. She had an Epworth score[1] 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/m^2). Her chest was clinically clear (FEV_1 1.8 l, FVC 2.3 l) 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 oximetry, 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. Good data were obtained for the first 25 minutes of the study, the remainder 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[2] and vascular disease.[3] 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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