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Short report

Airways obstruction in a case of disseminated lupus erythematosus

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The lungs and pleura are commonly affected in disseminated lupus erythematosus (DLE) (Dubois, 1966). Patients with airways obstruction associated with DLE have rarely been mentioned. We report a case of DLE in a non-smoker with recent onset of dyspnoea and severe airway obstruction.

Case report

A 59-year-old white woman was admitted to the Johannesburg Hospital in July 1976. A generalised rash had been present intermittently since 1973, and she had become increasingly dyspnoeic since 1975. She was cyanosed with an erythematous rash on the face and trunk. Hyperinflation of the chest was present, with diffuse diminution of breath sounds and prolonged expiration but no wheezes.

Results of routine investigations of blood, sputum, and urine were normal. Antinuclear factor was present (titre 1/320), and anti-DNA antibody titre was 92 units (normal 0-20). Serum complement was low: total complement level titre 1/9 (normal >1/25); beta₁C (C3) level 40 mg/100 ml (normal >75 mg/100 ml); C4 level 20 mg/

100 ml (normal 25-45 mg/100 ml). Alpha₁-antitrypsin level (immunodiffusion technique) was 252 mg/100 ml (normal >180 mg/100 ml). Skin biopsy was non-specific, but immunofluorescence studies showed deposits of IgG in the basement membrane highly suggestive of lupus erythematosus. Chest radiograph showed markedly hyperinflated but clear lung fields.

Pulmonary function studies (see table) showed airways obstruction with hyperinflation, raised airways resistance, and diminished transfer factor. There was no improvement after a bronchodilator aerosol. Static lung compliance was increased, and elastic recoil pressure at TLC was decreased.

Oral prednisone (15-60 mg daily) and intensive bronchodilator treatment have resulted in no improvement in pulmonary function over 18 months. The rash has disappeared.

Discussion

Pulmonary involvement in DLE usually results in a restrictive ventilatory pattern (Gold and Jennings, 1966; Huang and Lyons, 1966; Gibson et al.,

Table Pulmonary function tests

	Measured	Percentage predicted			
Lung volumes (I)			Flow measurements		
Total lung capacity (TLC)	6.67	120	FEV ₁ (l)	0.87	
Vital capacity	2.62	73	FEV ₁ /FVC%	43	
Functional residual capacity	5.25	153	MMF (1/s)	0.35	
Residual volume (RV)	4.05	188	PEFR (1/s)	2.95	
RV/TLC%	61	165	MEF _{50%} (1/s)	0.45	
ransfer factor (steady state)			MEF _{75%} (1/s)	0.24	
(mmol/min/kPa)	2.3	56	∆MEF _{50%}	0	
(ml/min/mmHg)	6.9		Viso V %	53	
Airways resistance (cm H ₂ O/I/s)	6.26		Arterial blood gases*	kPa	(torr)
Static lung compliance (l/cm H ₂ O)	0.34		Po,	7.8	(59)
Elastic recoil pressure at TLC (cm H ₂ O)			Pco ₂	4.7	(35)

MMF = Maximal mid-expiratory flow rate; PEFR = Peak expiratory flow rate; MEF₁₀% = Forced expiratory flow after exhalation of 50% of the FVC; MEF₁₀% = Percentage change in maximal expiratory flow at 50% FVC after 80% helium-20% oxygen mixture; VisoV = Volume of isoflow with helium-oxygen mixture compared with air expressed as a percentage of the FVC.

^{*}Normal values for Johannesburg (altitude 1750 m): arterial Po₂=10 kPa (75 torr); arterial Pco₂=4.7 kPa (35 torr).

1977). A degree of airways obstruction associated with the features of pulmonary restriction has been reported in interstitial lung disease due to various causes and has been attributed to the diffuse inflammatory, fibrotic, or granulomatous process affecting the airways. Our patient, however, had no evidence of restrictive lung disease.

Two of the 29 cases of DLE studied by Huang and Lyons (1966) and two of the 20 of Gold and Jennings (1966) had evidence of airway obstruction alone. In neither of these studies is there any reference to the tobacco consumption of the patients, and comprehensive clinical details are not given. Obstructive lung disease caused by smoking remains a possibility in these cases. Our patient had never smoked, coughed, or produced sputum. A detailed history showed no exposure to noxious fumes or possible causes of allergic alveolitis. Her only respiratory symptom was breathlessness, which appeared about 18 months after her presentation with a rash. The possibility of airways obstruction related to homozygous alpha₁antitrypsin deficiency was excluded by the normal concentration in her serum. The lack of blood and sputum eosinophilia, the poor response to corticosteroid treatment, and the abnormal carbon monoxide transfer factor and static lung compliance make bronchial asthma an unlikely diagnosis.

The pulmonary dysfunction in our patient suggests emphysema with obstruction at the level of the small airways (Δ MEF_{50 %} =0; VisoV=53% (see table). The raised airways resistance suggests additional large airways involvement, the degree of which is difficult to assess.

Gross et al (1972) found bronchiolar dilatation, loss of alveolar septa, or foci of panacinar emphy-

sema in each of 44 cases of DLE. The clinical and pathophysiological abnormalities in this case could be caused by such lesions. Treatment with prednisone for 18 months has not improved respiratory symptoms or function in our patient, but if the airway obstruction is caused by changes such as those described by Gross *et al* (1972) it is unlikely to be improved by corticosteroids.

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