SHORT REPORTS

Infectious mononucleosis and fibrosing alveolitis

I report a case of infectious mononucleosis with acute diffuse fibrosing alveolitis. Pulmonary lesions in this disease have been reported but without detailed pulmonary physiological studies.[1 2] The aetiology of acute fibrosing alveolitis is obscure,[3] but in this case the Epstein-Barr virus was implicated.

Case report

A previously fit 21-year-old woman was admitted to hospital with a short history of a "head cold" and sore throat which had responded to oral penicillin. An irritating unproductive cough persisted. She became progressively more dyspnoeic, initially only on exertion but eventually also when at rest. She complained of headache, profuse sweating, and general malaise.

She was cyanosed and feverish (38·8°C). There was no finger clubbing. Cervical and supraclavicular lymphadenopathy was noted but no splenomegaly. Chest expansion was reduced and a few fine crepitations were audible at both bases.

On admission the haemoglobin was 13·1 g/dl, and the white cell count $5{\cdot}1 \times 10^9$/l (5100/mm^3) (48% neutrophils, 50% lymphoctyes). Half of the lymphocytes were atypical. The erythrocyte sedimentation rate was 18 mm in one hour. Results of serum protein electrophoresis were normal. Antinuclear and rheumatoid factor were not detected. Liver function tests showed: bilirubin 20 μmol/l (1·2 mg/100 ml), alamine transferase 46 IU/l, and alkaline phosphatase 155 IU/l. Arterial blood gases when breathing air were: Pao_2 7·8 kPa, $Paco_2$ 3·1 kPa, and hydrogen ion 32 nmol/l. A chest *x*-ray film showed fine, reticular shadowing in mid and lower zones bilaterally.

Treatment was started with 60% oxygen and oxytetracycline because of suspected viral pneumonia. She remained dyspnoeic and feverish and three days later the lymphadenopathy increased, the spleen became palpable, and she became disorientated. The white cell count was 9×10^9/l (9000/mm^3) with 89% lymphocytes. Many blood and sputum cultures gave negative results. The Paul-Bunnel test gave a positive result at a titre of 1/320, and subsequent virological studies over the course of the illness showed a rise in Epstein-Barr virus M antibody titre from 10 to >160 and EBV (fat) IgG from 10 to >160. Other viral and mycoplasma titres remained normal. The tine test gave a grade I result and screening of extrinsic allergic alveolitis precipitins was negative. During the next week her clinical condition slowly improved in every respect except that she remained dyspnoeic. After treatment with 35% oxygen, however, her blood gases improved: Pao_2 was 11·2 kPa, $Paco_2$ 4·1 kPa, and hydrogen ion 38 nmol/l. The alveolar-arterial Po_2 difference, however, rose from 19 to 26 mm Hg and her chest *x*-ray pictures remained unchanged. Serial physiological recordings showed a restrictive gas-transfer defect compatible with acute diffuse fibrosing alveolitis (see table). Lung biopsy was not performed.

Treatment with prednisolone was started on the 10th day with an initial dose of 60 mg/day. There was immediate clinical response confirmed by serial physiological recordings. Progressive slow improvement continued for a year and steroids were slowly reduced before being finally stopped after 13 months' treatment, when pulmonary physiological recordings were close to one standard deviation from normal. She remained well over a year later with normal lung function tests and a normal chest *x*-ray picture.

Comment

Hoagland's[4] major review of infectious mononucleosis emphasised the variety of clinically important pulmonary disease but there are no reports of prolonged interstitial involvement. In this case the accepted diagnostic criteria were satisfied and the clinical, radiological, and physiological evidence of pulmonary involvement was clear-cut. This overall pattern was compatible with acute fibrosing alveolitis and specifically with "acute desquamative pneumonia" as described by Liebow,[3] although no confirmatory histological specimen was available. Prolonged pulmonary infiltrates have, however, been shown pathologically in infectious mononucleosis.[5] The response to prednisolone, the prolonged course of the illness, and the delay of almost a year before the return of physiological values to normal support this premise, and suggest that Epstein-Barr virus is one possible cause of the condition. This positive identification supports Liebow's observation that frequent infections of presumed viral aetiology precede acute desquamative pneumonia. Pulmonary involvement in infectious mononucleosis may also indicate the use of steroids.

I thank Dr N W Horne for permission to report this case.

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[4] Hoagland, R J, *American Journal of the Medical Sciences*, 1960, **240**, 21.
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(*Accepted 12 November 1976*)

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Salmonella enteritidis endocarditis

Salmonella enteritidis commonly causes enteritis, occasionally septicaemia, and rarely endocarditis. We describe the first reported case in Britain of *S enteritidis* endocarditis in a patient with a prosthetic heart valve.

Case report

The patient was a 52-year-old man who had had syphilitic aortic valve disease. In 1968 the aortic valve had been replaced by a homograft. The homograft failed four years later and was replaced by a Starr-Edwards prosthesis. He had remained well for the next three years. On 10 September 1975 he was admitted with a four-day history of headache and vomiting without diarrhoea or abdominal pain. Two days before his illness he had eaten a cold meal of sausages and chicken which had been prepared two or three days previously. On examination he was febrile; his blood pressure was 110/80 mm Hg; and there was sinus tachycardia (150/min). There were no signs of congestive cardiac failure and the prosthetic valve sounds were

Serial respiratory values from three days to 13 months after admission

	Predicted values	3 d	8 d	10 d	24 d	38 d	4 m	7 m	13 m
Carbon monoxide transfer factor (mmol/min/kPa)	9·88 ± 1·2	3·15	4·92	3·52	6·03	6·70	6·70	7·20	8·21
Total lung capacity (l)	5·19 ± 0·5			3·23	4·02	4·53	4·26	4·64	
Vital capacity (l)	3·34 ± 0·4			2·09	2·97	3·36	3·39	3·62	
Functional residual capacity (l)	2·44 ± 0·4			2·03	1·86	1·92	1·76	1·92	
Residual volume (l)	1·53 ± 0·3			1·14	1·05	1·17	0·87	1·02	
Residual volume/total lung capacity (%)	27 ± 5			35	26	26	20	22	
Forced expiratory volume (l)	3·35 ± 0·4			2·64	3·25	3·23	3·37	3·20	3·39
Forced vital capacity (l)	3·65 ± 0·4			2·89	3·75	3·71	3·98	3·95	4·02
Forced expiratory volume/forced vital capacity (%)	86 ± 5			91	87	87	85	81	84