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 $5.1 \times 10^9/l$ (5100/mm³) (48% neutrophils, 50% lymphocytes). 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), alanine transferase 46 IU/l, and alkaline phosphatase 155 IU/l. Arterial blood gases when breathing air were: P_{aO_2} 7.8 kPa, P_{aCO_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 $9 \times 10^9/l$ (9000/mm³) with 89% lymphocytes. Many blood and sputum cultures gave negative results. The Paul-Bunnell 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: P_{aO_2} was 11.2 kPa, P_{aCO_2} 4.1 kPa, and hydrogen ion 38 nmol/l. The alveolar-arterial P_{O_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's⁴ 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.

¹ Mundry, G R, *British Medical Journal*, 1972, **1**, 219.

² Wechsler, H, F, Rosenblum, A H, and Sills, C T, *Annals of Internal Medicine*, 1946, **25**, 113.

³ Liebow, A A, Steer, A, and Billingsley, J G, *American Journal of Medicine*, 1965, **39**, 369.

⁴ Hoagland, R J, *American Journal of the Medical Sciences*, 1960, **240**, 21.

⁵ Allen, F H, and Kellner, A, *American Journal of Pathology*, 1947, **23**, 463.

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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

"normal." No murmurs were heard and there was no clubbing, splinter haemorrhages or splenomegaly. Thus, at this stage there was no clear evidence that the heart was infected. The haemoglobin on admission was 12.5 g/dl, WBC $5.9 \times 10^9/l$ (5500/mm³), ESR 68 mm in the first hour, and blood urea 5.6 mmol/l (34 mg/100 ml). Blood, faeces, and urine cultures all yielded a growth of *S. enteritidis* sensitive to cephalosporins, ampicillin, tetracycline, gentamicin, trimethoprim, sulphonamides, chloramphenicol, and, on later testing, mecillinam (FL 1060).

Many antibiotics were given without clinical response (see figure). As his general condition was worsening chloramphenicol was started. This was followed by a prompt clinical improvement and he became afebrile. Nevertheless, an early diastolic murmur was noted on 3 October and signs of rapidly increasing aortic reflux appeared over the next few days. Endocarditis was now diagnosed. Replacement of the prosthetic valve was considered necessary, but surgery postponed because the patient developed bone marrow depression with a low platelet count. Chloramphenicol was stopped; blood and platelet transfusions were given. The fever recurred and mecillinam was started on 21 October. This was followed by a fall in the temperature.

Effects of antibiotics on the course of *Salmonella enteritidis* infection. Cot Cotrimoxazole, four tablets daily. Figures in parentheses indicate intravenous daily dosage of the drug, changes of which are explained in the text.

Ampicillin was added two days later as there was in-vitro evidence of synergy between mecillinam and ampicillin against the salmonella. On this combination of antibiotics the patient remained afebrile, the bone marrow depression gradually resolved, and open heart surgery was performed on 28 October.

At operation infected granulations were observed on the struts and sewing ring of the Starr-Edwards valve. A large false aneurysm was also present posterior to the prosthetic valve suture line and this contained a mass of infected clot. The valve and clot were removed and an aortic Björk-Shiley valve inserted; but the patient died without regaining consciousness. The excised Starr-Edwards valve was cultured in nutrient broth containing serum and *S. enteritidis* was isolated.

Comment

There are only two reports, both from America, of *S. enteritidis* endocarditis. The first is mentioned in a review of 7779 patients with salmonella infection.¹ The second describes "probable" salmonella endocarditis affecting a prosthetic mitral valve.² In our case *S. enteritidis* was isolated from the prosthetic valve and so endocarditis due to this organism was proved. The source was clearly food poisoning.

Salmonella endocarditis is difficult to treat successfully with antibiotics alone, and there is a high mortality rate. The only antibiotics associated with a clinical improvement in our case were chloramphenicol and mecillinam. Chloramphenicol may improve the patient's general condition but it cannot be expected to cure endocarditis since it is only bacteriostatic. Furthermore, it may cause bone marrow depression and thus make surgery difficult.

Mecillinam is a new beta-lactam antibiotic with great anti-salmonella activity.^{3,4} Before our case it had not been used in treating salmonella endocarditis and it clearly requires further evaluation. Although our patient died, we conclude that a combination of mecillinam and ampicillin and removal of the infected valve should be considered very early in the disease.

¹ Saphra, I, and Winter, J W, *New England Journal of Medicine*, 1957, **256**, 1128.

² Yamamoto, N, *et al*, *Surgery*, 1974, **76**, 678.

³ Reeves, D S, Wise, R, and Bywater, M J, *Journal of Antimicrobial Chemotherapy*, 1975, **1**, 337.

⁴ Williams, J D, *et al*, *Journal of Antimicrobial Chemotherapy*, 1976, **2**, 61.

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Bicycle injuries in children

In the mid nineteen-sixties a new type of children's bicycle was introduced in the United States. This high rise bicycle with a smaller front wheel than back, high wide handlebars, and a long seat was immediately popular. This popularity was repeated when high rise bicycles were introduced into Great Britain somewhat later. In 1968 Howell¹ expressed doubt whether these bicycles were as safe as the ordinary type; nevertheless, Waller² did not find any difference in the accident rate between them. Craft³ found that abrasions, fractures, and head injuries were significantly more common with high rise bicycles. Because of this conflicting evidence we undertook a study comparing the pattern of bicycle ownership in (a) children who were admitted to hospital or died; and (b) the community.

Methods and results

We obtained information on children from the ages of 5 to 12 years who were admitted to hospital or died in South Glamorgan after bicycle accidents over the period May 1974-April 1975. We asked the children's parents the type of bicycle involved, if it was borrowed or not, and how long it had been owned. We also noted the type of injury sustained and any sequelae. We estimated the pattern of bicycle ownership among children in South Glamorgan by visiting infant, junior, and comprehensive schools, in representative areas in the county. Children were visited in the classroom, and, if they owned a bicycle, were asked whether it had equal size wheels or not.

Fifty-six children (41 boys and 15 girls) were admitted after bicycle injuries in the period in question. In addition two boys were killed (who were riding ordinary bicycles). The data on these children and the controls are shown in the table.

Of the injured children, 45% were riding high rise cycles compared with 25% of controls. This difference is significant ($P < 0.003$) and remains so after adjustment for age and sex differences ($P < 0.03$). Injuries were commoner in boys than girls ($P < 0.02$) and in younger children than older children (NS). High rise cycles were used more often by boys ($P < 0.001$), and by older children (NS, $0.05 < P < 0.1$). Most of the children (21 on ordinary bicycles, 24 on high rise) were admitted with head injuries and concussion. All but three had facial abrasions, and 26 had body abrasions in

Bicycle injuries in children

	Injuries		Controls	
	Ordinary	High rise	Ordinary	High rise
Boys 5-8	12	9	94	36
9-12	12	10	131	73
Girls 5-8	5	3	112	21
9-12	3	4	106	20
Total	32	26	443	150