Comment

Normal blood pressure has been restored by transluminal angioplasty. Renal function was impaired after the angioplasty and was due to either extensive manipulation of the catheter or injection of radio contrast medium.[5] Renal function subsequently returned close to pre-procedure levels. The reduced blood pressure coincided with, and may have been due to, a reduction in total exchangeable sodium, although external sodium balance was not measured. In patients with transplants who develop renal artery stenosis, it may be worth using this technique rather than surgery, which has a high failure rate and associated morbidity.

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Western Infirmary, Glasgow

D CARR, MRCP, FRCR, consultant, *x*-ray department
R O QUIN, MD, FRCS, consultant, department of surgery
D N H HAMILTON, PHD, FRCS, consultant, department of surgery
J D BRIGGS, MB, FRCP, consultant, renal unit
B J R JUNOR, MD, MRCP, consultant, renal unit
P F SEMPLE, MB, MRCP, consultant, MRC blood pressure unit

Myopericarditis associated with farmer's lung

Farmer's lung is an extrinsic allergic alveolitis caused by mouldy hay.[1] Its main immunological mechanism is considered to be the immune-complex-mediated (type III) reaction.[2] Local injury in the alveoli and bronchioles seems to be the predominant feature of the disease.[3] Abnormality in other organs, however, has not been described, even though patients with farmer's lung also have systemic complaints such as malaise, fever, and various aches and pains.

We report a case in which myopericarditis was associated with farmer's lung.

Case Report

A 35-year-old farmer, who was an ex-smoker and otherwise healthy, suffered a clinically proved episode of farmer's lung for the first time in late autumn 1976. All the symptoms cleared within six months. In early spring 1978 he suffered a relapse. A chest radiograph showed a slight increase in the size of the heart when compared with earlier radiographs. Relative heart volume measured from the radiograph, however, was still normal (450 ml/m^2). Five months later the volume was above normal (510 ml/m^2). He had no hypertension, an electrocardiogram was normal, and there were no clinical signs of cardiac failure. At catheterisation of the right side of the heart with pressure measurements, in September, both pulmonary arterial and pulmonary wedge pressures were normal. In December he suffered a second relapse. No further increase in heart size was seen. A resting electrocardiogram was normal, but during exercise a prominent P wave was seen which did not disappear until 10 minutes after exercise. There was no chest pain.

The patient tried to avoid further exposures to mouldy hay, but one evening in May 1979 he worked without a dust respirator for about one and a half hours in a cow shed. That night he had severe shortness of breath, coughed up blood-stained sputum, and had fever and muscular pains. Two days later he was admitted to hospital, where cardiac failure with tachycardia and ventricular gallop rhythm was diagnosed. No pericardial friction rub was heard. Electrocardiograms showed negative T waves in limb leads and left chest leads, and several days later also in right chest leads. There was no laboratory or clinical evidence of myocardial infarction.

Erythrocyte sedimentation rate was 12 mm in first hour and blood leucocyte count $11{\cdot}9 \times 10^9$/l (11 900/mm^3) with 7% eosinophils; otherwise the differential count was normal. Tests for LE cells, antinuclear antibodies, and rheumatoid factor were negative. Antistreptolysin titre and antiviral antibody titres to common respiratory pathogens and complement-fixing antibody titres to *Mycoplasma pneumoniae* were normal. Cold agglutinins were not found. A precipitin test to *Thermoactinomyces vulgaris* gave a positive result. The urine was normal. A chest radiograph showed an enlarged heart shadow (relative volume 655 ml/m^2); pulmonary venous congestion and interstitial pulmonary oedema were also visible. Echocardiography showed an enlarged left ventricle, low ejection fraction, and pericardial effusion. The patient was given digitalis, diuretics, and corticosteroids. The worst symptoms disappeared rapidly. Two weeks later echocardiography showed no pericardial effusion but the function of the left ventricle was still impaired. Over the next few months the heart remained dilated and electrocardiographic signs of left ventricular hypertrophy and strain persisted. In January 1980 function of the left ventricle was still impaired.

Comment

The repeated episodes of farmer's lung suggest that this patient was highly sensitive to moulds. He developed cardiac enlargement gradually with the relapses of alveolitis. Catheterisation excluded pulmonary hypertension as the cause of the enlargement. Drastic deterioration of cardiac function with acute failure and pericarditis occurred several hours after exposure to vegetable dusts, strongly suggesting a causal relation between exposure and cardiac deterioration. Pericarditis alone was not responsible for the failure; repeated check-ups showed that there was also permanent myocardial injury.

That in this case the myopericarditis might have been a manifestation of the farmer's lung syndrome, caused by circulating immune complexes, seems worthy of consideration. Cardiological examination of all patients with extrinsic allergic alveolitis might help to detect other cases of a similar nature.

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Requests for reprints should be addressed to Dr Erkki O Terho, University Central Hospital, Kuopio, SF-71880 Tarinaharju, Finland.

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University Central Hospital, Kuopio, Finland

ERKKI O TERHO, MD, department of pulmonary diseases
RISTO VALTA, MD, department of pulmonary diseases
HANNU TUKIAINEN, MD, department of pulmonary diseases
ESKO LÄNSIMIES, MD, department of clinical physiology

Purpuric rash due to epsilon-aminocaproic acid

Epsilon-aminocaproic acid (EACA) is now widely used to prevent rebleeding after subarachnoid haemorrhage. Side effects and complications[1-4] include diarrhoea, toxic confusional states, arterial and venous thrombosis, and pulmonary embolism. We report a case in which a purpuric morbilliform rash was due to treatment with EACA.

Case report

A woman aged 59 years was admitted to hospital with sudden onset of severe headache and loss of consciousness. A similar headache three weeks before had been followed by dysphasia for a few hours. On admission she was deeply unconscious with neck stiffness and decorticate posture. Computed tomography showed intraventricular and subarachnoid bleeding. She was treated conservatively and given EACA 24 g daily in divided dosage through a nasogastric tube. Her condition remained unchanged and after 12 days she developed a morbilliform rash over the front and sides of the chest and in the axillae. Some of the lesions became purpuric. She was not then on any other drug. EACA was discontinued and the rash completely disappeared within 72 hours. A full blood count was normal but detailed coagulation studies were not done. On further challenge after two months with EACA (6 g six-hourly) she again developed a faintly erythematous rash (not purpuric this