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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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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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

time) after the fourth dose. This cleared within 24 hours after stopping EACA. The patient remained unconscious for about three months before she died.

Comment

The rash in this patient seemed to be causally related to the drug. The purpuric element of the rash was probably part of the hypersensitivity reaction and not related in any way to the antifibrinolytic effect of EACA. We do not think there has been a British report of a rash associated with EACA treatment, but we have heard that two such cases have been reported to Swedish manufacturers of the drug.

We thank Kabi-Vitrum (UK), the manufacturers of Epsikapron, for their co-operation, and Dr J M S Pearce for permission to report this case.

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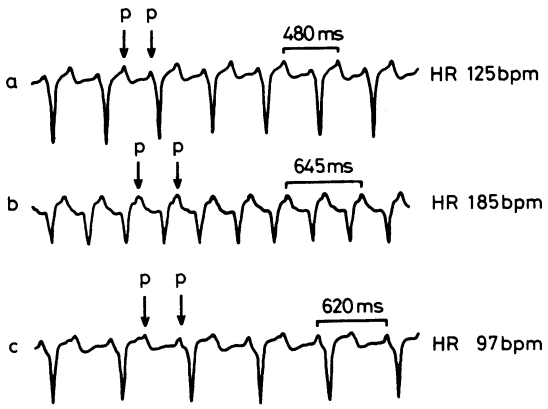
Potentially dangerous effect of disopyramide on atrioventricular conduction in a patient on digitalis

The antiarrhythmic drug disopyramide prolongs the refractory period of atrial myocardium.[1] It is therefore sometimes useful in treating ectopic atrial tachycardia. Disopyramide also has atropine-like properties[2] which may lead to enhanced atrioventricular (AV) nodal conduction. The combination of slowing the atrial rate with "improved" AV nodal function may lead to 1:1 conduction of the tachycardia when previously it had been 2:1. This causes a potentially dangerous increase in ventricular rate. A similar increase may occur when disopyramide is given to patients in atrial fibrillation.[3] For this reason patients should be digitalised before attempted conversion of atrial tachycardia with disopyramide. This report concerns the failure of digitalis to protect a patient from this unwanted effect.

Case report

The patient, a 68-year-old woman, had a long history of rheumatic mitral stenosis. She had been well having no treatment other than warfarin until two months before admission, when she developed a fast heart rate and dyspnoea on minimal exertion. An electrocardiogram (ECG) showed atrial tachycardia with 2:1 conduction. Digoxin and propranolol failed to control the rhythm so she was admitted for a trial of disopyramide. On admission she was taking digoxin 0·25 mg and frusemide 40 mg once a day, potassium supplements, and warfarin. On examination she was euthyroid: pulse rate 125/min, regular; jugular venous pressure raised 3 cm; blood pressure 140/80 mm Hg. Both ventricular impulses were slightly increased and the murmurs of moderate mitral stenosis and incompetence were present. There were no signs of pulmonary oedema. Blood urea concentrations, electrolytes, and thyroid function tests were normal; haemoglobin concentration was 14·7 g/dl, and serum digoxin 1·4 mg/l. Chest radiographs showed mitral cardiac contour with clear lung fields, and an ECG showed atrial tachycardia with 2:1 conduction (figure (*a*)).

Disopyramide was given intravenously in a total dose of 1·5 mg/kg over five minutes with continuous ECG monitoring. Thirty seconds after the injection she developed 1:1 conduction (figure(*b*)). The atrial rate had slowed from 250 to 185. She became breathless and hypotensive. Three minutes later she reverted to 2:1 conduction with an atrial rate of 194 (figure (*c*)).



Electrocardiogram before and after intravenous disopyramide: (a) before; (b) immediately after; (c) three minutes after. Lead VI shown at paper speed of 25 mm/s. p Represents p waves. Time for two atrial cycles shown.

Comment

The feature of interest in this case is that digitalis failed to protect the patient from the atropinic effect of disopyramide. Without an intracardiac study the relative contributions of enhanced AV conduction and slowing of the atrial rate cannot be disentangled nor can the effect of the digitalis on the AV node be assessed. Although the serum digoxin concentration may not be clearly related to its effect on AV nodal conduction, it is the only means available to most physicians to ensure adequate digitalisation. Caution should therefore be exercised when giving disopyramide to patients with atrial tachycardia even when they are taking digitalis. The same caution probably applies equally to treatment with quinidine, whose affect on electrophysiological pathways is very similar to that of disopyramide. This potential hazard should not detract from the usefulness of disopyramide in a condition which may be difficult to treat.

We thank Dr D Verel for permission to report this case.

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Corrections

Anabolic effect of human parathyroid hormone fragment on trabecular bone in involutional osteoporosis: a multicentre trial

We regret that in the first paragraph of the Patients and methods section of the above paper (7 June p 1340), 50 mg (2000 IU) daily should have read 50 μg (2000 IU) daily, and 12·5 mg (500 IU) daily should have read 12·5 μg (500 IU) daily. In the table the column referring to urinary hydroxyproline mmol should have read μmol.

Peroperative venography to ensure accurate sapheno-popliteal vein ligation

An error occurred in the paper by Dr J T Hobbs (28 June, p 1578). The measurements in the sixth line of the methods section should be 30 cm × 40 cm.