orientation, combative delirium, apathy, or coma. Vomiting and faecal incontinence were prominent. Mean rectal temperature was 39·1°C (range 37·3-41·6). Haemoglobin concentrations were normal (mean 17 g/dl), leucocyte counts raised (mean 18 000 × 10^9/l), and initial platelet counts normal. A mild metabolic acidosis was present (mean arterial pH 7·34). The serum urea concentration was slightly raised (mean 8·7 mmol/l (52·4 mg/100 ml)).

Initial treatment was rehydration with oral or intravenous fluids; cooling with iced blankets or baths; and sedation with diazepam or chlorpromazine. In milder cases normal cerebral function returned within hours, but lethargy, anorexia, nausea, epigastric pains, and muscle cramps continued for up to one week. Five patients subsequently developed spontaneous bruising with coagulation abnormalities (thrombocytopenia, prolonged coagulation times) typical of disseminated intravascular coagulation. Tender hepatomegaly was noted in five patients during this phase. The table shows biochemical changes during convalescence.

The worst affected was a 23-year-old competitive swimmer who was in convulsions on admission, and whose rectal temperature was 41·6°C. Initial management was iced baths, rehydration, sedation with phenobarbitone, and curarisation with positive pressure ventilation. Profound disseminated intravascular coagulation developed six hours after admission, which was treated by low-dose heparin infusion. An appreciable rise in bilirubin concentration (peak 120 μmol/l (7 mg/100 ml) on day 3) and SGOT (peak 2100 IU on day 2) with hepatomegaly suggested liver damage. The renal failure persisted for five weeks requiring intermittent haemodialysis, while recurrent pseudomonas septicaemia and hypertension delayed recovery, which was complete.

Discussion

These cases occurred in highly motivated muscular young men, who were active sportsmen but not trained long-distance runners. Toxic hepatitis[2] and disseminated intravascular coagulation[3] have been described as prominent features of heat stroke.

Education of runners alone did not prove effective in preventing heat stroke, and responsibility must be taken by organisers of such events to minimise the risks. Similar events should be run in the cooler parts of the day and year, and adequate fluid intake before and during the run encouraged. First aid facilities must be readily available, with close liaison among groups such as traffic police, ambulances, and hospitals. Such recommendations have been made before,[4 5] but cases of this potentially fatal illness continue to occur in sporting events.

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Disseminated intravascular coagulation and myocarditis associated with Mycoplasma pneumoniae infection

Mycoplasma pneumoniae infection may produce a wide variety of non-respiratory syndromes.[1] We describe a patient with *M pneumoniae* infection who had involvement of the cardiovascular and haematological systems.

Case report

A 65-year-old man was admitted with a 12-day history of intermittent left infrascapular pain, radiating anteriorly and to the right of the chest. On one occasion it had been pleuritic and associated with sweating. Examination showed a pulse rate of 100/min, with a blood pressure of 110/70 mm Hg. Cardiac auscultation showed normal findings. There were fine crepitations at both lung bases and scattered low pitched expiratory rhonchi. Investigations showed a haemoglobin concentration of 15·4 g/dl, and WBC 11·8 × 10^9/l with a normal differential count. The platelet count was 335 × 10^9/l, reticulocytes 1·6%, and ESR 70 mm in the first hour. The blood film was normal. The blood urea concentration was 4·5 mmol/l (27·1 mg/100 ml), Na 134 mmol (mEq)/l, K 3·8 mmol (mEq)/l; aspartate aminotransferase 37 U/l, urea-stable lactate dehydrogenase 280 U/l. Liver function tests gave normal results. Chest *x*-ray film was normal; ECG showed right bundle branch block, with S-T elevation throughout the anterior leads. The results of a lung scan were normal.

He continued to complain of pleuritic pain, which was associated with recurrent fever. Treatment was started with ampicillin and a diuretic. Repeat chest *x*-ray film suggested right basal infection. After three weeks he was much improved. Serial ECGs showed no progression of the initial changes, and serial cardiac enzyme concentrations were normal. He was discharged home to be seen at follow-up. Shortly after discharge a significant rise in the *M pneumoniae* titre (1/32-1/256) was reported. His general practitioner was contacted and oxytetracycline, 250 mg four times daily, prescribed. Two days later the patient was readmitted as an emergency with tiredness, drowsiness, and complete anorexia. His blood urea concentration was 70 mmol/l (422 mg/100 ml), Na 129 mmol/l, and K 6·3 mmol/l. Hb was 13·1 g/dl, WBC 17·6 × 10^9/l, platelets 19 × 10^9/l, reticulocytes 11·0%, fibrinogen 2·7 g/l, fibrin degradation products 80-160 mg/l, partial thromboplastin time with kaolin 43 s (control 26 s). Blood film showed fragmented red blood cells. Cold agglutinins were not detected. The direct antiglobulin test of his red blood cells was positive because of C3 coating, and the anti-C3 titre was 1/256. The immunoglobulin concentrations were: IgG 7·7 g/l, IgA 5·6 g/l, IgM 0·69 g/l. Immunoelectrophoresis gave normal results. Repeated bacterial and viral investigations gave negative results. Chest *x*-ray film showed enlargement of the heart; the ECG remained unchanged. Echocardiography showed a large left ventricular cavity, with no evidence of aneurysm. He was transferred to the medical renal unit and after 60 hours' peritoneal dialysis his blood urea concentration had fallen to 26 mmol/l. With conservative management the renal function subsequently returned to normal. He received erythromycin, 250 mg 6-hourly, and was digitalised. Three weeks later he was discharged. At that time the Hb concentration was 10·3 g/dl, platelets 170 × 10^9/l, partial thromboplastin time 1·0:1, reticulocytes 1·7%, urea 6·2 mmol/l (37 mg/100 ml). Serum electrolyte concentrations were normal. Radiologically the enlargement of the heart persisted although the lung fields were clear.

Comment

This case report illustrates two uncommon complications of *M pneumoniae* infection: myocarditis and disseminated intravascular coagulation, the latter causing acute renal failure. An association between *M pneumoniae* and cardiovascular disease was first described in 1944,[2] but, although disseminated intravascular coagulation has been described with bacterial and viral infections, its occurrence in *M pneumoniae* infection is rare—only two other cases having been described,[3 4] both fatal.

The more usual haematological complication of *M pneumoniae* infection is haemolysis occurring two to three weeks after the onset of symptoms, often subclinical and coinciding with recovery from pneumonia and the peak cold haemagglutinin titre.[5] In our patient cold agglutinins were not detected, although the intravascular coagulation occurred at a time when they would, if present, have been at their maximum titre. The cause of the disseminated intravascular coagulation is unknown, though possibly a toxin released by the organism or thromboplastin released from the damaged lungs could trigger this off.

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