no support to the concept that a high-fibre diet is of value in maintaining clinical remission in patients with ulcerative colitis, although this does not exclude its value in patients who are particularly apt to develop colonic obstruction with recurrence of disease activity.

Most of our patients tolerated the diet and at the end of the study many said that they wished to continue with it in addition to sulphasalazine.

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¹ Dissanayake, A S, and Truelove, S C, *Gut*, 1973, **14**, 923.

² Misiewicz, J J, *et al*, *Lancet*, 1965, **1**, 185.

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Long remission after lymphoblastic transformation of chronic granulocytic leukaemia

Blastic transformation is common in chronic granulocytic leukaemia (CGL) and usually represents a terminal aspect of the disease owing to its resistance to chemotherapy, mean survival time being a few weeks or months. Attention has been focused on the different cell morphologies seen in transformation^{1,2} and their possible relevance to the forms of chemotherapy employed and subsequent survival.³ We therefore thought this case report was of interest.

Case report

A 25-year-old woman presented in December 1970 with left hypochondrial pain and pronounced splenomegaly. Her haemoglobin concentration (Hb) was 11.9 g/dl; white cell count (WBC) $125 \times 10^9/l$ ($125\,000/mm^3$), composed predominantly of neutrophils but with numerous myelocytes and metamyelocytes; and platelet count $785 \times 10^9/l$ ($785\,000/mm^3$). Considerable granulocytic hyperplasia was found on bone marrow examination, cytogenetic analysis of which showed the Philadelphia chromosome in all mitoses examined. Leucocyte alkaline phosphatase (LAP) score was 5. CGL was diagnosed. The splenomegaly disappeared and the blood count returned to normal after a six-week course of busulphan. Over the subsequent two years two short courses of busulphan were required, after which maintenance was continued with hydroxyurea.

In July 1974 she suddenly developed fever, extensive purpura, and generalised lymphadenopathy and hepatomegaly. The spleen remained palpable. Hb was 8.4 g/dl; WBC $20 \times 10^9/l$ ($20\,000/mm^3$) with 14% neutrophils and 70% microblasts; and the platelet count $80 \times 10^9/l$ ($80\,000/mm^3$). The bone marrow was hyperplastic with a 70% infiltration of small peroxidase-negative blast cells of lymphoblastic morphology. LAP score was 180. No mitoses were obtained for cytogenetic analysis. Cerebrospinal fluid examination was normal. A conventional antilymphoblastic treatment regimen was initiated—namely, intravenous vincristine 2 mg weekly, intramuscular L-asparaginase 20 000 U twice weekly, and prednisone 60 mg daily by mouth. Full haematological remission was obtained after six weeks. Central nervous system prophylaxis was carried out six weeks after remission with cranial irradiation and intrathecal methotrexate.

Maintenance treatment with daily 6-mercaptopurine by mouth and twice-weekly intramuscular methotrexate was given until April 1977, when hydroxyurea was reinstituted for a thrombocytosis of $1000 \times 10^9/l$ ($1\,000\,000/mm^3$). The blood count became normal and the patient remained well and asymptomatic 39 months after transformation, 82 months after the original presentation. All cytogenetic analyses performed since transformation have shown persistence of the Philadelphia chromosome.

Comment

The clinical and laboratory features of CGL in this patient are unremarkable and little maintenance treatment was required in the chronic phase. Acute transformation was clinically unusual only in the absence of splenomegaly, and it was morphologically indistinguishable from a "de-novo" acute lymphoblastic leukaemia.

Subsequent remission induction and duration, however, contrast with results currently obtained in transformed CGL, although response to chemotherapy may be superior in "lymphoblastic" varieties, especially if anti-ALL regimens are employed.^{3,4} That long remissions may be obtained, as exemplified by this young patient, underlines the importance of both recognising the morphological type of transformation and subsequently instituting appropriate treatment. This includes CNS prophylaxis, since CNS relapse often occurs during remissions obtained in lymphoblastic and myeloblastic transformations.⁵

¹ Rosenthal, S, *et al*, *Blood*, 1977, **49**, 705.

² Janossy, G, *et al*, *British Journal of Haematology*, 1976, **34**, 179.

³ Peterson, L C, Bloomfield, C D, and Brunning, R D, *American Journal of Medicine*, 1976, **60**, 209.

⁴ Hoerni-Simon, G, *et al*, *Nouvelle Presse Médicale*, 1976, **5**, 2885.

⁵ Schwartz, J H, *et al*, *American Journal of Medicine*, 1975, **59**, 819.

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Heat stroke in a "run for fun"

Heat stroke may occur with strenuous exercise in hot humid environments and is not usually considered a risk in community sports in a temperate environment. A life-threatening emergency, it is characterised by three cardinal signs: severe disturbance of the central nervous system, hyperpyrexia, and hot dry skin.¹ It occurs with a high work rate in conditions preventing dissipation of heat by convection and radiation. We describe 16 severe cases occurring under temperature conditions where the risk is considered low.

The event

Some 20 000 runners participated in an 11-km "run for fun" in Auckland, New Zealand, in late summer 1977. The run began at 10 am, when the temperature was 21.3°C, humidity 73%, and wind velocity 9 knots. The run was promoted as a community sporting event, and runners were encouraged to enter in social teams (63% of registered runners), though they could also enter individually. Certificates were given to those who completed the course in under 100 minutes.

The organisers gave advice to runners in a pamphlet that described appropriate preparation and training for the run. The need to drink adequate fluid before and during the run was emphasised. Runners were advised to stop if they became unwell or developed excessive breathlessness, dizziness, or chest pains. Ambulance personnel estimated that they treated 200 with heat illness of varying severity; 16 patients were admitted to local hospitals.

Clinical observations—The 16 severely affected were men aged 20-44; there were no heat-stroke casualties among older runners or women. All the patients were active in sports other than long-distance running and had trained for the event but in the cooler parts of the day. Subsequent interviews established that they were all highly motivated to finish, to improve on a previous performance, or from team loyalty. Only one patient (taking propantheline for symptoms of peptic ulceration) had been other than completely well before the run. The patients presented with varying degrees of dis-

Substance (and normal concentration)	Median	Range	No tested
CPK (5-115 IU/l)	418	59-2252	10
Bilirubin (5-22 µmol/l)	34	12-192	11
SGOT (10-55 IU/l)	690	165-5000	11
Alkaline phosphatase (30-95 IU/l)	98	65-140	11

Conversion: SI to traditional units—Bilirubin: 1 µmol/l $\approx$ 0.058 mg/100 ml.

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 $18\,000 \times 10^9/l$), 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 $\mu\text{mol/l}$ (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 hepatitis¹ and disseminated intravascular coagulation² 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.

¹ Clowes, G H A, jun, and O'Donnell, T F, *New England Journal of Medicine*, 1974, 291, 564.

² Kew, M, et al, *American Journal of Medicine*, 1970, 49, 192.

³ Shilbolet, S, et al, *New England Journal of Medicine*, 1962, 266, 169.

⁴ Sutton, J R, and Harrison, H C, *Medical Journal of Australia*, 1977, 1, 193.

⁵ Knochel, J P, *Journal of the American Medical Association*, 1975, 233, 513.

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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 $11.8 \times 10^9/l$ with a normal differential count. The platelet count was $335 \times 10^9/l$, 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 $17.6 \times 10^9/l$, platelets $19 \times 10^9/l$, 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 $170 \times 10^9/l$, 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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¹ Murray, H W, et al, *American Journal of Medicine*, 1975, 58, 229.

² Finkelstein, D, and Klainer, M J, *American Heart Journal*, 1944, 28, 385.

³ Nilsson, I M, et al, *Acta Medica Scandinavica*, 1971, 189, 359.

⁴ De Vos, M, Van Nimmen, L, and Baele, G, *Acta Haematologica*, 1974, 52, 120.

⁵ Barrett-Connor, E, *American Journal of Medicine*, 1973, 52, 242.

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