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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**University Hospital of Wales, Cardiff**
P S DAVIES, MB, MRCP, senior registrar in medicine
J RHODES, MD, FRCP, consultant gastroenterologist

# 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 hydroxurea.

In July 1974 she suddenly developed fever, extensive purpura, and generalised lymphadenopathy and hepatomegaly. The spleen remained impalpable. 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]

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**Departement d'Hématologie, Clinique Médicale du CHU, Hôpital de Cimiez, 06031 Nice, France**
M V JOYNER, MRCPATH, assistant
P DUJARDIN, MD, assistant
J-P CASSUTO, MD, assistant
P AUDOLY, MD, professor

**Centre Antoine Lacassagne, 36 Avenue de la Voie Romaine, 06 Nice, France**
M SCHNEIDER, MD, professor

# 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 ≈ 00·58 mg/100 ml.