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Hodgkin's Disease as Pyrexia of Unknown Origin in a Patient with Chronic Lymphocytic Leukaemia

M. Hatjiyanni, M. Giliayos, A. Megalakaki,
 B. Daskalaki and B. Seitanides

AS HYPOGAMMAGLOBULINAEMIA and neutropenia develop eventually in almost all patients with chronic lymphocytic leukaemia (CLL), infections become the most common and important complication [1]. For this reason and also because fever is rarely produced by the disease itself, the presence of fever in CLL is usually considered as manifestation of infection and is treated as such even if there are no clinical signs of infection.

In this report we present the case of a patient with CLL who developed fever of unknown origin which finally proved to be due to Hodgkin's disease (HD).

A 54-year-old man, with past history of pulmonary tuberculosis and diabetes mellitus, presented in September 1989 with peripheral lymphadenopathy, enlarged spleen (12 cm), liver (7 cm), white blood cell count of $320 \times 10^9/l$ (96% mature-looking lymphocytes), haemoglobin of 10.6 g/dl, platelets of $148 \times 10^9/l$ and heavily infiltrated bone marrow with small lymphocytes. He was diagnosed as CLL, stage B. He started treatment with chlorambucil/prednisolone with good clinical and laboratory response. In March 1990, while still on the same treatment, he developed fever, anorexia and weight loss. He presented 1 month later, febrile and in poor general condition, but CLL seemed to be under control. On examination he was found with minimal peripheral lymphadenopathy, liver and spleen both palpable 2 cm. His blood count was quite good with haemoglobin of 12.1 g/dl, white blood cell count of $5.8 \times 10^9/l$ (lymphocytes 29%), platelets of $195 \times 10^9/l$. There was no sign of infection and the investigation disclosed nothing helpful. Computed tomography of the abdomen showed small retroperitoneal lymph nodes and spleen and liver enlargement. Chest X-ray was normal. Empirical treatment with common antibiotics had no effect. Subsequently, there was no improvement with antituberculous treatment but a small lymph node in the right axilla started becoming bigger. This lymph node biopsy revealed HD. He was started on doxorubicin-bleomycin-vinblastin-dicarbazine with excellent response.

Searching the literature we found at least 20 cases [2-4] with firm diagnosis of HD in CLL patients. In conclusion we can say that fever unrelated to infection in CLL patients has not only the grave prognosis of higher malignancy transformation, but could represent B symptoms of HD.

1. Chapel HM, Brunch C. Mechanisms of infection in chronic lymphocytic leukaemia. *Sem Haematol* 1987, 24, 291-296.
2. Han T. Chronic lymphocytic leukaemia in Hodgkin's disease. *Cancer* 1971, 28, 300-305.
3. Greene M, Hover R, Fraumeni J. Subsequent cancer in patients with chronic lymphocytic leukaemia—a possible immunological mechanism. *J Natl Cancer Inst* 1978, 61, 337-340.
4. Davis J, Weiss N, Armstrong B. Second cancer patients with chronic lymphocytic leukaemia. *J Natl Cancer Inst* 1986, 78, 91-94.

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The Prophylactic Use of Fluconazole 50 vs. 100 mg Daily in Haematological Malignancies

P.C. Huijgens, A.C. van Loenen,
 A.M. Simoons-Smit, E. Prooy
 and G.J. Ossenkoppele

SUPERFICIAL AND invasive fungal infections are common and often life-threatening complications during treatment for haematological malignancies. In many studies oral antifungal drugs like amphotericin B, nystatin and ketoconazole decrease the frequency of oral candidiasis but a reduction of invasive infections has not been documented [1, 2, 3]. These agents are usually poorly tolerated because of their taste, quantity and/or hepatotoxicity.

Fluconazole is one of the new azoles which has been shown to be effective in the treatment of oral candidiasis in HIV infected or neutropenic patients using daily doses of 50 to 400 mg [4-9]. It is not clear whether the higher doses add to the prevention of oral candidiasis a decrease of the frequency of invasive fungal infections. We choose to compare two low dose regimens [3, 9] to see whether there is a relevant clinical difference in efficacy for the prevention of oral candidiasis and whether there is any difference in the occurrence of invasive fungal infections and/or the need for empirical intravenous antifungal therapy.

In a pilot study, we entered 60 consecutive patients with acute leukaemia or malignant lymphoma needing chemotherapy which would lead to granulocytopenia (granulocytes $<0.5 \times 10^9/l$) for at least 15 days. Patients were excluded if life expectancy was less than 30 days, age was over 80 years, or if severe liver function disturbances were present. Patients were randomised to receive 50 or 100 mg fluconazole daily in identical appearing capsules from the start of chemotherapy throughout the whole period of

Correspondence to B. Seitanides at the Department of Clinical Haematology, Metaxa Cancer Hospital of Piraeus, 51 Botassi Str, 18537 Piraeus, Greece.

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Correspondence to P.C. Huijgens.

P.C. Huijgens and G.J. Ossenkoppele are at the Department of Haematology; A.C. van Loenen is at the Department of Pharmacy; A.M. Simoons-Smit is at the Department of Clinical Microbiology and Hospital Hygiene, Free University Hospital, De Boelelaan 1117, 1081 HV Amsterdam, The Netherlands; and E. Prooy is at the Medical Department, Pfizer B.V., Rotterdam, The Netherlands.

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