

she had a complete response to high dose carboplatin, etoposide and cyclophosphamide. Our patient had received a total of nine previous chemotherapy courses and three operations in 2 years: this was the reason for not employing higher doses of chemotherapy. Furthermore, since at that time, growth factors were not commercially available, we administered thymopentin after ABMT for its pleiotropic properties and, in particular, for its stimulation of T cell function [7]. The present case demonstrates that metastatic low grade uterine sarcoma can be considered a chemotherapy-sensitive neoplasm. Further experiences should be performed with high dose carboplatin, cyclophosphamide and etoposide in the setting of metastatic uterine sarcoma.

1. Kempson RL, Bari W. Uterine sarcomas: classification, diagnosis and prognosis. *Hum Pathol* 1970, 1, 331-39.
2. Wheelock JB, Krebs HB, Schneider V, Cloperud D. Uterine sarcoma: analysis of prognostic variables in 75 cases. *Am J Obstet Gynecol* 1985, 151, 1016-1022.
3. Echt G, Jepson J, Steel J, et al. Treatment of uterine sarcomas. *Cancer* 1990, 66, 35-39.
3. Larson B, Silfersward C, Nilsson B, Pettersson F. Endometrial stroma sarcoma of the uterus. A clinical and histopathological study. *Eur J Obstet Gynecol Reprod Biol* 1990, 35, 239-249.
4. Levenback C, Rubin SC, McCormack PM, Hoskins WJ, Atkinson EN, Lewis JL Jr. Resection of pulmonary metastases from uterine sarcoma. *Gynecol Oncol* 1992, 45, 202-205.
5. Wade K, Quinn MA, Hammond I, Williams K, Cauchi M. Uterine sarcoma: steroid receptors and hormonal therapy. *Gynecol Oncol* 1990, 39, 364-347.
6. Gershenson DM, Kavanagh JJ, Copeland LJ, Edwards CL, Stringer CA, Wharton T. Cisplatin therapy for disseminated mixed mesodermal sarcoma of the uterus. *J Clin Oncol* 1987, 5, 618-621.
7. Goldstein G, Holmann WW. Polypeptides regulating lymphocyte differentiation. *J Neurol Neurosurg Psychiatr* 1968, 31, 455-458.

European Journal of Cancer Vol. 32A, No. 3, pp. 554-555, 1996.
Copyright © 1996 Elsevier Science Ltd. All rights reserved.
Printed in Great Britain
0959-8049/96 \$15.00 + 0.00

0959-8049(95)00595-1

Skin Lesions and G-CSF in Patients with Malignant Diseases. Malignancy or Cutaneous Side-effect?

A. Loraas, S.D. Fosså, S. Franzen, G. Sæter and L. Rode

The Norwegian Radium Hospital, Montebello, 0310 Oslo, Norway

TREATMENT WITH haematopoietic colony-stimulating factors is occasionally associated with neutrophilic dermatoses and vasculitis [1, 2]. Furthermore, pre-existing factors may be exacerbated during such therapy.

We report 2 patients who illustrate a diagnostic problem which may arise during G-CSF treatment of cancer patients: metastases to the skin or a cutaneous side effect of G-CSF?

Case I. A 26-year-old man with an angiosarcoma of the right atrium and bilateral pulmonary metastases was treated with cycles of etoposide/doxorubicin/ifosfamide (VIGA) and support with filgrastim (Neupogen®, Roche). The pulmonary metastases gradually decreased during treatment. After the sixth course of VIGA, he noticed a 1 cm indurated plaque in the skin of his right forearm. The leucocyte count was $36.8 \times 10^9/l$. A fine needle aspiration was performed and the cytological picture was described as "probable mesenchymal malignant tumour with numerous mature neutrophil granulocytes". The mesenchymal cells were similar to the malignant cells in the primary biopsy. Immunocytochemistry of these cells was not carried out. Neither was a biopsy performed, but the patient's treatment was changed to high dose ifosfamide. The tumour disappeared gradually. A subsequent review of the cytological report altered the diagnosis to "possible inflammatory process combined with mesenchymal cells of uncertain origin".

Case II. A 33-year-old man with a non-seminomatous germ cell tumour of the right testicle and metastases to the retroperitoneum, stomach and left supraclavicular fossa was treated with bleomycin/etoposide/cisplatin (BEP) every 3 weeks. After the third course he received filgrastim 390 µg subcutaneously from day 6 to 14 because of severe leucopenia. On day 17, he noticed a tumour under his right mandibula, and on day 21, the tumour was erythematous and 6 cm in diameter. His leucocyte count was $6.8 \times 10^9/l$ with neutrophil count $4.8 \times 10^9/l$. A fine needle aspiration was performed, and the cytologist's conclusion was "dominance of mature neutrophil granulocytes with a few groups of confluent large malignant cells" (Figure 1). The malignant cells were assumed to represent metastatic spread from his non-seminomatous germ cell tumour. No biopsy was performed. As there was no other clinical or serological suspicion of progression of the patient's malignancy, the treatment was not altered. The skin lesion was treated with benzylpenicillin sodium and metronidazol, and the tumour diminished in size. A new fine needle aspiration 5 days later displayed the picture of a typical inflammatory disorder.

The cells with malignant appearance in our patients were not immature myeloid cells, but they could have been large



Figure 1. Large undifferentiated and multinucleated cell with a background of mature neutrophil granulocytes. Diff Quick stain, oil immersion lense ($\times 100$).

dermal macrophages. It is known that GM-CSF can increase the number and size of dermal macrophages [3]. Our two cases suggest that G-CSF may cause similar cytological changes. Clinicians should be aware of the clinical and cytological features of G-CSF-induced cutaneous toxicity. The demonstration of bizarre, apparently malignant cells in skin lesions should thus not automatically lead to the diagnosis of disease progression and change of treatment.

1. Johnson ML, Grimwood RE. Leukocyte colony-stimulating factors. A review of associated and neutrophilic dermatoses and vasculitides. *Arch Dermatol* 1994, **130**, 77–81.
2. Park JW, Mehrota B, Barnett BO, Baron AD, Venook AP. The Sweet syndrome during therapy with granulocyte colony stimulating factor. *Ann Intern Med* 1992, **116**, 996–998.
3. Scott GA. Report of three cases of cutaneous reactions to granulocyte macrophage-colony-stimulating factor and a review of the literature. *Am J Dermatopathol* 1995, **17**, 107–14.

European Journal of Cancer Vol. 32A, No. 3, pp. 555–556, 1996.
Copyright © 1996 Elsevier Science Ltd. All rights reserved.
Printed in Great Britain
0959–8049/96 \$15.00 + 0.00

0959–8049(95)00594–3

Herpesvirus-like DNA Sequences Selectively Cluster with Body Cavity-based Lymphomas Throughout the Spectrum of AIDS-related Lymphomatous Effusions

A. Carbone,¹ U. Tirelli,² A. Gloghini,¹
C. Pastore,³ E. Vaccher² and G. Gaidano³

¹Division of Pathology; ²Division of Medical Oncology and AIDS program, Centro di Riferimento Oncologico, IRCCS, Aviano I-33081, Italy; and
³Laboratorio di Medicina e Oncologia Molecolare, Dipartimento di Scienze Biomediche e Oncologia Umana, Ospedale S. Luigi, Università di Torino, Italy

DNA SEQUENCES belonging to a putative novel human virus, provisionally termed Kaposi's sarcoma-associated herpesvirus

(KSHV) [1], have recently been identified in tissue samples taken from patients with AIDS-related and -unrelated Kaposi's sarcoma (KS) [2], as well as in non-KS skin lesions of transplant patients [3]. KSHV DNA sequences have also been detected in AIDS-related body cavity-based lymphomas (BCBL), but not in any other non-Hodgkin's lymphoma (NHL) studied thus far [4]. Based on the latter finding, a specific link between AIDS-related BCBL and the KSHV DNA sequences has been suggested [4]. However, data on the distribution of KSHV sequences in AIDS-related BCBL are limited to series of patients from North America. Furthermore, the selectivity of the association between KSHV sequences and BCBL throughout the spectrum of lymphomatous effusions has not been tested. This study was aimed at evaluating the presence of KSHV sequences in a well characterised series of seven lymphomatous effusions, including four BCBL and three effusions secondary to tissue-based lymphoma, in patients infected with HIV-1 from Italy.

The 4 cases of AIDS-related BCBL accounted for 2.9% of a consecutive series of 140 AIDS-related NHL referred to the Division of Pathology of the Centro di Riferimento Oncologico (Aviano, Italy) during a period of 10 years. Detection of KSHV DNA sequences was restricted to cases of BCBL (three of four) (Figure 1). All other AIDS-related lymphomatous effusions secondary to tissue-based lymphomas scored negative for KSHV sequences. The group of patients with KSHV-positive BCBL comprised three males [one homosexual, one intravenous drug user (IVDU) and one

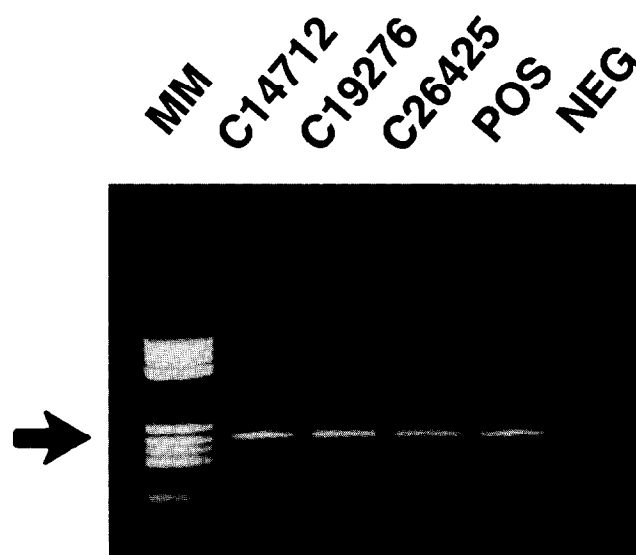


Figure 1. Analysis of Kaposi's sarcoma-associated herpesvirus (KSHV) infection by polymerase chain reaction (PCR). Genomic DNA was enzymatically amplified with primer oligonucleotides derived from KSHV-specific sequences as previously reported [1]. C14712, C19276 and C26425 represent three cases of body cavity-based lymphomas in HIV-infected individuals. A positive (POS) and a negative (NEG) control were included. The positive control was represented by a case of AIDS-related Kaposi's sarcoma. Molecular marker (MM) sizes are as follows (from top to bottom): 587, 540, 504, 458, 434, 267, 234, 213, 192, 124, 104 and 89 bp. The arrow points to the KSHV amplification product (233 bp).