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.

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

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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 lymhomas (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

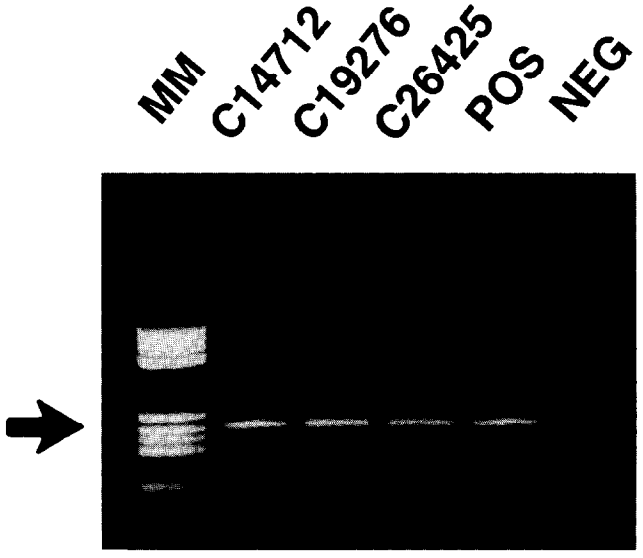

Figure 1. Analysis of Karposi's sarcoma-associated herpesvirus (KSHV) infection by polymerase chain reaction (PCR). Genomic DNA was enzymatically amplified with primer oligonucleotides derived from KSHV-specific sequences as previously reported [1]. C14712, C19276 and C26425 represent three cases of 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).

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homosexual IVDU], ranging in age from 27 to 44 years. In BCBL cases, the patients exhibited exclusive or predominant lymphoma involvement of the body cavities. Their clinical outcome was poor, with a median survival of 4 months after the diagnosis, despite the administration of systemic chemotherapy. No patient had a history of KS. In all cases, the smears and cell blocks prepared from pleural fluid samples showed a tumour cell population greater than 95% as evaluated by morphological and immunophenotypic analysis. Neoplastic cells from BCBL cases showed anaplastic (three of four) or immunoblastic (one of four) features, expressed CD30 (four of four) and tended to display indeterminate phenotypes (three of four), whereas all lymphomatous effusions secondary to tissue-based lymphomas consistently expressed B-cell phenotype. By an *in situ* hybridisation (ISH) technique, monotypic κ mRNA or λ mRNA were detected in all BCBL and other cases, establishing their B-cell clonality. The presence of Epstein–Barr virus (EBV) was detected in two of three KSHV-positive BCBL by EBV-encoded small RNAs (EBER) ISH. In both EBV-positive cases, a fraction of tumour cells expressed latent membrane protein-1 (LMP-1).

Overall, our results indicate that AIDS-related BCBL preferentially associates with peculiar clinical, immunophenotypic and molecular features among lymphomatous effusions and, therefore, should be singled out as a specific clinicopathological entity. Intriguingly, BCBL morphological and immunophenotypic characteristics, as well as EBV phenotype (LMP-1+), are similar to those of AIDS-related immunoblastic or CD30+ anaplastic large cell lymphomas (ALCL) [5]. However, the clinical manifestations of BCBL and its association with KSHV DNA sequences are highly specific for this type of lymphoma among AIDS-related NHL, since KSHV DNA sequences were not detected in a series of 28 systemic AIDS-related NHL, including CD30+ ALCL (our unpublished observation).

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Magnesium Supplements with Cisplatin Chemotherapy

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We recently reported on the use of routine intravenous magnesium supplements in patients receiving cisplatin chemotherapy with continuous infusional 5-fluorouracil and epirubicin (ECF) [1]. Cisplatin is known to cause renal tubular magnesium wasting which can result in symptomatic hypomagnesaemia [2]. In our report, all 14 evaluable patients who did *not* receive routine supplements had at least one episode of hypomagnesaemia (Mg <0.7 mmol/l) and subsequent supplements had to be given with 50% of the cisplatin chemotherapy cycles. In contrast, only 6/14 patients who received routine magnesium supplements were found to have a reduced serum magnesium on admission, and on only one occasion was this <0.6 mmol/l.

Although these data show that cisplatin associated hypomagnesaemia can be avoided by the routine addition of magnesium sulphate to the hydration fluids, this adds considerably to the cost of treatment. The estimated additional cost of 28 mmol magnesium sulphate per chemotherapy cycle was £34.30. In the group of patients who only received supplements when required, the cost per patient per cycle was £17.51.

Table 1. *Mean magnesium concentration* (± *S.E.*) *on day* 1 *of each chemotherapy cycle*

	Average (Mg) mmol/l	
	25 mmol/cycle	12.5 mmol/cycle
Cycle 1	0.85 ± 0.017 (0.81–0.90)	0.89 ± 0.02 (0.84–0.94)
Cycle 2	0.81 ± 0.016 (0.77–0.85)	0.86 ± 0.026 (0.79–0.92)
Cycle 3	0.83 ± 0.018 (0.79–0.88)	0.78 ± 0.043) (0.67–0.89)
Cycle 4	0.78 ± 0.027 (0.71–0.85)	0.79 ± 0.024 (0.73–0.85)
Cycle 5	0.78 ± 0.043 (0.67–0.89)	0.78 ± 0.028 (0.71–0.85)
Cycle 6	0.78 ± 0.030 (0.70–0.85)	0.80 ± 0.035 (0.71–0.88)

95% confidence intervals are given in parentheses. Mean Mg mmol/l.

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