
Gallium-67 uptake by cutaneous lesions in a patient with Burkitt-like non-Hodgkin's lymphoma

Sir,

Although Gallium-67 (67Ga) uptake by primary cutaneous B-cell lymphoma has been reported, this technique does not have a defined role in monitoring cutaneous lymphoma lesions.1 We present a 67Ga scintigraphy and confirmatory SPECT study (Figures 1 and 2) showing uptake by a secondary skin lymphoma two months before clinical relapse of a Burkitt-like non-Hodgkin's lymphoma with typical diffuse large B-cell lymphoma translocations involving BCL-6 and p53 genes. 67Ga scanning and SPECT may be useful for monitoring cutaneous lesions in Burkitt-like and diffuse large B-cell lymphomas.

Figure 1. Cranial projection image of a Gallium planar study. A diffuse Ga-67 uptake in the scalp region was noted.

Figure 2. Axial projection of SPECT study showed a pathologic uptake in right parotid gland and homolateral submaxillary region, as well as a visualization of the multiple and nodular scalp lesions.

Key words
Cutaneous lesions, Gallium-67 uptake, Burkitt-like non-Hodgkin's lymphoma

Correspondence
Antonio Gutiérrez-García, M.D., Department of Hematology and Medical Oncology, Hospital Clínico Universitario, Av Blasco Ibáñez 17, Valencia 46010, Spain. E-mail: aguti@ene.es

References

Simultaneous occurrence of multiple myeloma and Hodgkin's disease. A case report

We report the case of a 52-year old male diagnosed as having multiple myeloma (MM) and Hodgkin's disease (HD) simultaneously, in the absence of any prior treatment. His plasma cell dyscrasia did not progress autonomously but was associated with HD.

Sir,

Our patient presented in June 1993 with a 3-month history of relapsing-remitting fever. Physical and laboratory findings were normal except for anemia (hemoglobin 9 g/dL), erythrocyte sedimentation rate of 102 mm/h, and presence of an IgG-k monoclonal component with Bence-Jones protein in urine. The bone marrow was infiltrated by plasma cells up to 60%, but a bone ray survey was normal. The patient was diagnosed as having MM and was started on melphalan-prednisolone. Two months later, all findings remained unchanged and he developed left axil-
lary lymphadenopathy. Biopsy revealed a mixed cellularity HD (Stage IIb). He attained a complete remission (CR) of both malignancies after six courses of COPP/ABVD and involved-field radiotherapy. One year later, his HD relapsed (stage IIIIB). A 4-month CR was achieved with CHVPP but both diseases relapsed. Two courses of intermediate dose melphalan failed to control either of them. Bone marrow biopsies showed infiltration with Hodgkin and plasma cells. Cytogenetic and FISH analysis on marrow samples demonstrated cells with a gain or loss of Y chromosome, findings which are more compatible with constitutional mosaicism. A third CR of both diseases was achieved with three ABVD courses but the patient succumbed to overwhelming acute hepatitis B and hepatic failure.

Patients with HD who also presented monoclonal gammapathy without overt MM, or who developed MM after radio- or chemotherapy have been reported, but the simultaneous occurrence of MM and HD has also occasionally been reported. It is unclear whether any pathogenetic association between the two diseases exists. Both are believed to have lymphoid, probably B-cell, origin, and similar karyotypic abnormalities have been detected in them, while the same cytokines, such as IL-6, could play a major role in both cases. IL-6 acts as a factor for growth and apoptosis for plasma cells, while Reed-Sternberg cells (infiltrating the marrow) express IL-6 mRNA and high IL-6 levels are found in patients with advanced HD. Although this stimulus does not seem enough to promote monoclonality, it is possible that the excess IL-6 in the marrow microenvironment might directly stimulate the long-living plasma cells there. Although MM criteria were fulfilled in our patient, the course of this disease was not autonomous, it did not cause bone lytic disease and was always associated with the progression of HD (Figure 1). Consequently, these two distinct malignancies may not be irrelevant to each other. Questions remain to be answered in the future when the pathogenetic mechanisms of malignancy are better understood.

References


Demonstration of Epstein-Barr virus in a case of multiple myeloma after renal transplantation

Immunosuppressed organ transplant recipients have an increased risk of developing lymphoproliferative disorders that are often associated with Epstein-Barr virus (EBV) infection. We report a patient who developed multiple myeloma after renal transplantation. EBV-RNA was demonstrated in the neoplastic cells suggesting the implication of this virus genome in the pathogenesis of this posttransplantation lymphoproliferative disorder.

Sir,

Multiple myeloma (MM) represents no more than 4% of all post-transplantation lymphoproliferative disorders (PTLDs) and is associated with a poor response to discontinuation of immunosuppression and conventional therapy and a short median survival. In March 1997 a 47-year old man was admitted to our hospital with a 6-week history of fatigue, anorexia, left eye progressive blindness and left thoracic pain. He had required a cadaveric renal transplantation in 1988 because of idiopathic chronic glome-