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Lymphomas Associated with the Endemic (African) Variant of Kaposi's Sarcoma: A Chemosensitive but Fatal Entity

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THERE ARE very few reports of the association between the endemic (African), non-AIDS-related variant (AKS) of Kaposi's sarcoma (KS) and secondary lymphoma compared with reports on classic KS [1–3]. The scarcity of reported cases may be related to under-reporting, due to the acute lack of medical facilities in Africa and a lack of adequate follow-up, as well as to increased morbidity through exacerbation of infectious diseases.

Between 1981 and 1992, 47 HIV-negative Black males were referred to the Johannesburg General Hospital for treatment of their KS. The majority (37/47, 79%) presented with skin disease, and 10/47 (21%) patients had additional gastrointestinal tract, lymph node and lung involvement. Staging and treatment modalities (mainly radiotherapy) have been described previously [4].

2 patients with KS limited to the skin (stage I), which regressed almost totally with 'involved field' radiotherapy (24 Gy), presented simultaneously with generalised lymphadenopathy (peripheral, mediastinal and para-aortic). Meticulous multiple lymph node biopsies revealed follicular lymphoma and peripheral T-cell lymphoma, respectively. Both received COPP (cyclophosphamide, oncovine, procarbazine, prednisone) regimen with a marked response, albeit of short duration (both patients subsequently died).

A third patient with stage IV KS presented with advanced lymphadenopathy due to immunoblastic lymphoma. Both his diseases responded initially to vinblastine/etoposide (plus radiotherapy) and later to a CHOP (cyclophosphamide, doxorubicin, oncovine, prednisone) regimen, but he relapsed 3 months later and died.

The fourth patient presented with advanced KS (skin, peripheral lymph nodes), as well as hepatomegaly, ascites and pleural effusion. Bone marow trephine biopsy demonstrated infiltration with a large cell lymphoma of B-cell origin. Immune function tests showed an absolute T-cell lymphopenia with a low T_4/T_3 ratio (0.78 : 1). Following cytarabine/interferon-α therapy, a significant response was achieved. The patient was then continued on CHOP with a continued response. 4 months after completion of his chemotherapy, he relapsed with massive hepatosplenomegaly and pancytopenia due to extensive lymphomatous infiltration of the bone marrow.

Data concerning AKS-related lymphomas are very rare and are mentioned only sporadically in the literature. All the reported cases lack proper staging, treatment, assessment of response and sufficient follow-up [5].

Recent reports have demonstrated a moderate alteration in cell-mediated immunity in AKS [6]. Protein malnutrition, chronic infections (e.g., hepatitis), tropical infections (e.g., malaria) and the wide use of alkylating agents may contribute to impaired cell immunity. The compromised immunity may lead to T-cell regulatory dysfunction and unopposed proliferation of abnormal B-cells, emerging in malignant lymphomas [7]. Other authors postulate a common pathogenetic mechanism between KS and lymphoma [8].

Another hypothesis is that certain antigenic stimuli may result in the simultaneous proliferation of both lymphoid and endothelial cells which, in the presence of immune dysfunction, may result in the simultaneous appearance of KS and lymphoma.

In conclusion, the improved outcome of patients with AKS due to modern therapeutic methods may unmask an increased number of chemoresponsive but biologically aggressive lymphomas.

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