Human Immunodeficiency Virus (HIV) Infection Associated with Acute Myeloblastic Leukemia in a Low HIV Prevalence Area

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The association of acute myeloblastic leukemia (AML) and human immunodeficiency virus (HIV) infection is rare [1-4]. Previous reports have been from relatively high HIV prevalence areas. We report 2 cases of coexistent HIV infection and AML from a low HIV prevalence area.

Case 1

An 18-year-old male presented in December 1991 with fever and fatigue. Physical examination showed mild cervical lymphadenopathy and hepatosplenomegaly. Laboratory investigations revealed hemoglobin (Hb) 9.9 g/dl, platelets $35 \times 10^9/l$, white blood cell count (WBC) $13.4 \times 10^9/l$, with 82% blasts in the peripheral blood smear. His bone marrow aspirate was markedly hypercellular with 80% blasts. On the basis of morphology and cytochemical reaction of the blasts, a diagnosis of acute myelomonocytic leukemia (FAB classification AML-M4) was made. Viral markers including enzyme-linked immunosorbent assay (Elisa) for HIV antibody were negative. He was treated with daunorubicin 45 mg/m² on days 1, 3 and 5, cytosine arabinoside 100 mg/m² and 6-thioguanine 100 mg/m² on days 1-7. Remission induction therapy was complicated by fever and disseminated intravenous coagulopathy, treated with broad-spectrum antibiotics and blood component transfusions. He achieved complete remission and received two courses of consolidation chemotherapy. Both courses were complicated by myelosuppression and fever and required supportive care including blood component transfusions. In April 1992, he presented with generalized erythematous nodular cutaneous rash and marked cervical lymphadenopathy. Laboratory investigations showed normal hemogram, fine-needle aspiration cytology from the cervical lymph nodes showed many blasts and the bone marrow aspiration revealed 15% blasts. Histopathological examination of the cutaneous nodule revealed leukemic infiltrates. Elisa for HIV antibodies was positive, confirmed by Western blotting assay. He was treated in another hospital with combination chemotherapy and achieved complete remission. While in remission he developed a possible fungal pneumonia and intracerebral infection to which he succumbed.

Case 2

A 70-year-old male presented in February 1993 with cough and expectoration. He was noted to have cachexia, multiple cutaneous branding marks, hepatosplenomegaly and right inframammary crepitations. Chest X-ray showed patchy right mid and lower lobe pneumonia. Laboratory investigations showed Hb 10 g/dl, platelets $65 \times 10^9/l$, WBC $13 \times 10^9/l$ with 9% blasts in the peripheral blood smear, re-ticulocyte count 0.9%, serum albumin 19 g/l, bilirubin $24 \mu\text{mol/l}$, γ -glutamyltransferase $424 \mu\text{mol/l}$, alanine amino-transferase $94 \mu\text{mol/l}$ and alkaline phosphatase $784 \mu\text{mol/l}$. Direct Coombs' test was strongly positive (weakened at 37 °C) anti-C 3d positive, findings compatible with cold agglutinin disease. Bone marrow aspirate was hypercellular with 50% blasts. On the basis of morphology and cytochemistry, the diagnosis of acute myelomonocytic leukemia (FAB classification M4) was made. Bronchoalveolar lavage was negative for acid fast bacilli, Pneumocystis

carinii and fungus. Mycoplasma pneumoniae IgG (Elisa) was strongly positive, Venereal Disease Research Laboratory test (VDRL) and Treponema pallidum hemagglutination assay were positive. HIV antibody screening test (Elisa) was positive, confirmed by Western blotting assay. Erythromycin administration resulted in prompt resolution of pneumonia. The patient declined to receive chemotherapy for AML.

Coexistent AML and HIV infection is rare and only 8 cases have been reported till 1990 [1-4]. This association may be due to chance. Both of our cases had acute myelomonocytic leukemia, this is in conformity with previous reports [1, 4]. Murthy et al. [1] have shown from in vitro culture studies that HIV had infected myelomonoblasts in vivo in their patient and speculated about the possibility of direct or indirect viral transformation. HIV infection is associated with T cell immunodeficiency and may contribute to the development of AML in such patients either due to

defective T cell regulation of hemopoiesis and/or due to failure of immune surveillance [4].

Coexistent HIV infection in patients with AML may be overlooked especially in low HIV prevalence areas. We were able to detect 2 cases due to routine HIV screening, this is in contrast to absence of HIV antibody in 27 AML patients tested by Lindhart et al. [5]. The experience with the management of AML with coexistent HIV infection is limited. However, complete remissions have been documented after low-dose cytosine arabinoside [6] and intensive combination chemotherapy [1]. Complete remission was obtained in 1 of our patients after intensive combination chemotherapy. The patient tolerated chemotherapy like the HIV-negative AML patients, however, unlike them he succumbed to fungal infection while in remission. This report documents the existence of HIV infection with AML in low HIV prevalence area and the usefulness of routine HIV antibody screening of AML patients.

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