T-Cell Lymphoma of the Tonsil with Ki-1 Antigen Expression

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A 52-year-old woman was admitted to our hospital with a fever and swelling of the tonsils and lymph nodes. A tonsillectomy was performed and she was diagnosed as having non-Hodgkin’s lymphoma of T-cell phenotype. Genotypic analysis revealed a rearrangement of the T-cell receptor gene, but not of the immunoglobulin genes. The neoplastic cells of the tonsils also expressed the Ki-1 antigen. The clinical course was aggressive with peripheralization of the neoplastic cells, and the patient died of respiratory failure.

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Introduction

Most cases of non-Hodgkin’s lymphoma (NHL) of Waldeyer’s ring are of B-cell phenotype. The T-cell phenotype is rare, and generally indicates a poor prognosis (1, 2). Recently, the Ki-1 antigen, which was first recognized on Hodgkin’s and Reed-Sternberg cells, has been believed to be an activation-associated lymphocyte antigen that is not lineage specific (3, 4). However, there is no case report of T-cell lymphomas of the tonsils with Ki-1 expression. Here, we report a case of T-cell lymphoma arising from the tonsils with Ki-1 antigen expression.

Case Report

A 52-year-old woman was admitted to our hospital on November 20, 1992 with a fever and swelling of the tonsils and lymph nodes. The patient had first noticed the swelling of her tonsils and cervical lymph nodes in March 1992. Upon admission, her body temperature was 40°C. The conjunctiva was slightly anemic. She had elastic firm lymph node swelling, which measured 1 to 2 cm in diameter, at the cervical and inguinal regions. Her tonsils were bilaterally enlarged. There was no hepatosplenomegaly. The blood cell counts were as follows: red blood cells 286×10^6/μl, Hb 9.4 g/dl, white blood cells 40,100/μl with 97.5% neutrophils, 1.5% lymphocytes, and 1.0% monocytes. The erythrocyte sedimentation rate was 51 mm/hr. The total serum protein was 6.5 g/dl, with an albumin to globulin ratio of 1.17. The LDH was elevated to 724 IU/l, but liver function tests were within normal range. The serum concentrations of IgG, IgA and IgM were 1,410, 78, 234 mg/dl, respectively. C-reactive protein (CRP) was 11.3 mg/dl. Anti-HTLV-1 antibody was negative. A gallium scintigram revealed accumulations at the cervical and inguinal regions. Bone marrow aspirate was hypercellular (NCC 40.9×10^6) with myeloid cell predominance, but there was no abnormal myeloid cells, erythroid cells or megakaryocytes. Antibiotic therapy for 2 weeks improved her fever, and CRP declined to 0.4 mg/dl. Thereafter, the patient underwent a tonsillectomy and a biopsy of her cervical lymph node. The histology of her tonsils revealed non-Hodgkin’s lymphoma of the diffuse large cell type according to the LSG classification. The neoplastic cells were large in size, but different from anaplastic large cell lymphoma which is common in Ki-1 lymphoma. The specimen of the cervical lymph node showed a marked infiltration of neutrophils and a disruption of the architecture of the lymphoid follicle. The immunophenotype of the lymphoma of the tonsil was T-cell lymphoma. Immunocytochemistry showed that neoplastic cells from the tonsils were immunoreactive for the Ki-1 antigen (Fig. 2). Genotypic analysis demonstrated a rearrangement of T-cell receptor (TCR) β gene (Fig. 3), without any rearrangement of the immunoglobulin heavy or light chain genes. Figure 4 shows her clinical course. She was treated with etoposide at 50 mg/day for 3 months, but her disease progressed aggressively with the appearance of Ki-1 positive neoplastic cells in the peripheral blood. An immunophenotypic analysis of peripheral blood showed the following distributions: CD2 86.4%, CD5 3.1%, CD7 56.7%, CD3 91.7%, CD4 1.8%, CD8 2.7%, CD10 5.5%, CD13 25.1%, CD14 19.4%, CD19 1.0%, CD20 0.7%, CD33 32.5% and HLA-DR 97.7%. She died of respiratory failure on May 25, 1993.
Tonsil Neoplasm with Ki-1 Expression

Fig. 1. Specimen from the tonsils showing non-Hodgkin’s lymphoma, diffuse large cell type (arrow) (HE stain, a ×16; b ×160).

Fig. 2. Immunostaining for Ki-1 antigen on neoplastic cells from the tonsils (arrow) (×200).

Fig. 3. Southern blot hybridization analysis for the TCR β gene rearrangement. Hybridization of DNA digested with EcoRV and BamHI to a constant region of a TCR β gene probe shows a distinct, high intensity rearrangement band. C: control, P: patient, arrow (R): clonal rearrangement band.

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Discussion

Waldeyer's ring is frequently involved with NHL, in which B-cell lymphomas occur far more frequently than T-cell lymphomas. Yamanaka et al (1) reported that in cases of Waldeyer's non-Hodgkin's lymphoma, 86% have B-cell markers and 14% have T-cell markers. The prognosis of Waldeyer's NHL is associated with the surface phenotype of the tumor cells, with B-cell lymphomas exhibiting a good prognosis, and T-cell lymphomas having a poor prognosis (1, 2). In the present case, the T-cell lymphoma arose from the tonsil. Upon admission, infection was suspected because the administration of antibiotics improved her fever, and a marked infiltration of neutrophils was observed morphologically in a specimen of her cervical lymph node. The clinical course was aggressive, with the peripheralization of neoplastic cells which expressed CD2+, CD3+, CD7+, HLA-DR+, CD4−, CD5−, and CD8−. Moreover, genotypic analysis revealed rearrangement of the TCR β gene without any rearranged immunoglobulin genes, suggesting the monoclonality of T-cell lineage. In Japan, adult T-cell leukemia/lymphoma is a well-known pathology, and several cases arising from the tonsils have been reported (5). However, this was not the case in this patient, since no anti-HTLV-1 antibody was detected.

The Ki-1 antigen was first recognized on Hodgkin's and Reed-Sternberg cells by Stein and colleagues (3), and was originally considered to be a specific marker for Hodgkin's lymphoma. Subsequently, the Ki-1 antigen was believed to be an activation-associated lymphocyte antigen that was not lineage specific, e.g., most frequently associated with T-cells and less commonly with B-cells (4). In addition to neoplastic disorders, variable numbers of Ki-1 immunopositive cells are observed in angioimmunolymphoblastic lymphadenopathy, and EB virus and HTLV-1 virus infected lymphocytes (3). To our knowledge, there has been no previous report on T-cell NHL of the tonsils with Ki-1 antigen expression. Further accumulation
of such cases will be valuable to elucidate the cause and mechanisms underlying Ki-1 expression.

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References


