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Adult T-Cell Leukemia/Lymphoma in a Korean
— A case report —

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The clinicopathologic features of a Korean patient with adult T-cell leukemia/lymphoma(ATLL) are presented. A 51-year-old man, who has lived in Korea since birth, had multiple cutaneous nodules and multiple lymphadenopathy for the previous two months. A histopathologic study of the lymph node and skin lesion revealed T-cell non-Hodgkin's lymphoma of pleomorphic type, medium and large cell type. Peripheral blood examination showed leukemic features with 30% of abnormal lymphoid cells. HTLV-I proviral DNA pX region was detected in the DNA from peripheral blood mononuclear cells(PBMC) and the specific gag, pol, and env HTLV-I sequences were detected in the lymph node using polymerase chain reaction technique. Human T-cell leukemia/lymphoma type I (HTLV-I) antibodies were present in the serum. An immunophenotypic study of the lymph node revealed CD4 positive and CD8 negative helper/inducer T cell type surface markers. This case is the acute type, i.e. prototypic ATLL. He was treated with an intensive chemotherapy including cyclophosphamide, etoposide, doxorubicin, vincristine, and prednisone. Despite initial transient improvement, the tumor progressed after three cycles of the regimen and became refractory to further chemotherapy. These clinicopathologic findings, including the immunophenotypic analysis, established with certainty the diagnosis of HTLV-I-induced adult T-cell leukemia/lymphoma.

Key Words : *Adult T-cell Leukemia/Lymphoma, Human T-cell Lymphotropic Virus Type I (HTLV-I), Lymph node, PCR.*

INTRODUCTION

Adult T-cell leukemia/lymphoma(ATLL) is one of the several clinical entities linked to human T-cell lymphotropic virus(HTLV-I) (Rosenblatt et al., 1988, Urba and Lango, 1985). HTLV-I is etiologically associated with adult T-cell leukemia/lymphoma, which was first reported by Uchiyama et al.(1977) in Japan as a specific T-cell leukemia with unusual clinical, pathological, and epidemiological features. The HTLV-I virus is endemic to Southern Japan (Tamura et al., 1986; Tokunaga et al., 1993), the Caribbean area(Blattner et al., 1982), and Central Africa(Verdier et al., 1989). ATLL is characterized by a rapidly progressive fatal course and frequent cutaneous involvement. The prevalence of ATLL associated with HTLV-I in Korea has not yet been

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described, although Korea has a similar disease pattern to Japan in several malignancies. Lee et al.(1986) performed an epidemiologic study for HTLV-Ⅰ in Korea, which revealed low positivity(0.25%) of serum antibodies for HTLV-Ⅰ without evidence of ATLL and there was no endemic area in Korea. Only two cases of ATLL have been reported in Korea(Lee et al., 1987 ; Kim et al., 1994), of which HTLV-Ⅰ was proven in the serum.

We report a case of Korean ATLL of which HTLV-Ⅰ proviral DNA was proven in both peripheral blood mononuclear cells(PBMC) and lymph node by means of polymerase chain reaction(PCR) and by the presence of antibodies to HTLV-Ⅰ in the serum, having leukemic features, multiple superficial lymphadenopathy and skin lesions with serological and immunopathological studies.

CASE REPORT

Case History

A 51-year-old Korean man was admitted to the Department of Internal Medicine in Korea Cancer Center Hospital in March 1994 for pain in the right neck and multiple skin lesions which he had had for two months. He was born in Korea where he had spent his whole life. He gave no history of previous blood transfusion or other parenteral therapy. Physical examination revealed multiple superficial lymphadenopathy in both sides of the neck, the axilla, and inguinal region. Multiple small papular skin eruptions were observed on the anterior chest, abdomen and upper extremities. He had never complained of fever, sweating or weight loss. An abdominal CT scan showed neither hepatosplenomegaly nor abdominal lymphadenopathy.

Laboratory findings

Laboratory data revealed a leukocyte count of 28,040/mm^3, hemoglobin 10.6 g/dl, and platelet count 600,000/mm^3. No hepatic or renal function test abnormalities were found and serum calcium was normal. Serum lactate dehydrogenase had been elevated up to 1,054 U/L with subsequent spontaneous decrease. Blood and bone marrow smears showed an abnormal population of pleomorphic lymphocytes varying considerably in size, with a homogenous and condensed nuclear chromatin. Lymphoid cells with pleomorphic nuclei of which the nuclear outiline was convoluted or "clover-leafed"

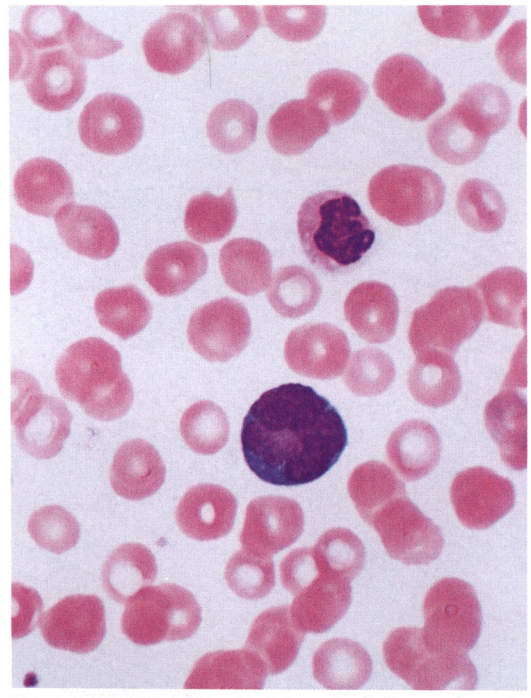


Fig. 1. Leukemic cells with "clover-leaf" like configuration in peripheral blood(Giemsa, ×1,000).

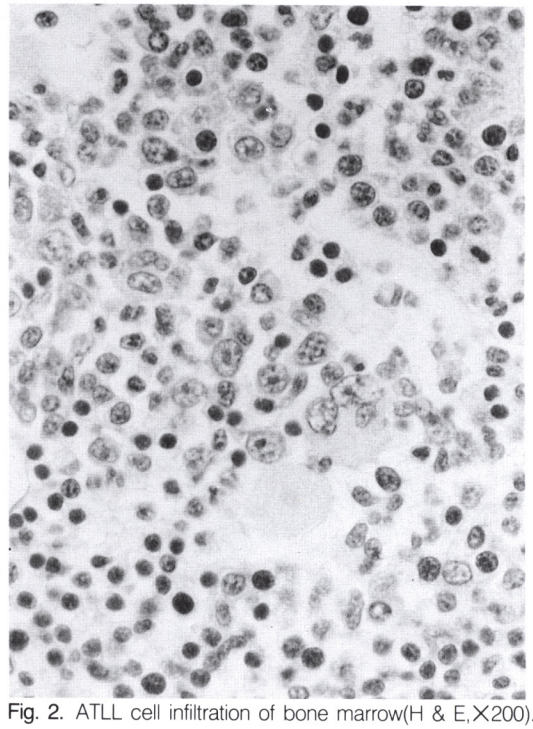


Fig. 2. ATLL cell infiltration of bone marrow(H & E, ×200).

accounted for 30% of the white blood cells on the peripheral blood smear(PBS) (Fig. 1). Blasts(1%) and immature cells(6%) were also found on PBS. In a bone marrow aspiration smear, malignant lymphoid cells which were seen in the PBS, were observed and counted for approximately 12.8% of nucleated bone marrow cells. These cells were variable in size, from large to small, had fine chromatin pattern, some of them showing convoluted nuclear pattern, and several small nucleoli and basophilic cytoplasm without granules. Granulocytic and erythroid series looked normal in maturation and distribution. Bone marrow biopsy showed the interstitial infiltration of malignant lymphoid cells which were intermingled with normal hematopoietic cells(Fig. 2). These PBS and bone marrow findings raised the suspicion of ATLL.

Pathologic findings

Light microscopic examination of a cervical lymph node biopsy revealed infiltration of variable-sized neoplastic cells with uneven proportions. Regardless of size, tumor cells showed marked irregularity in nuclear configuration, including convoluted, hyperconvoluted and multilobated forms. Medium-sized cells had dense chromatin. Large cells showed round or oval nuclei with distinct nuclear membrane and two or three large nucleoli. The nuclei of large cells had multiple indentations on one side("jellyfish" appearance) (Fig. 3). The cytoplasm was moderately abundant. These cells were interspersed with giant cells of cerebriform type-(Kikuchi et al., 1986) showing marked nuclear convolution and 2 or 3 prominent nucleoli with a thick membrane(Fig. 4). Some giant cells resembled Reed-Sternberg cells. Many mitotic figures were present. A fair number of macrophages and mature

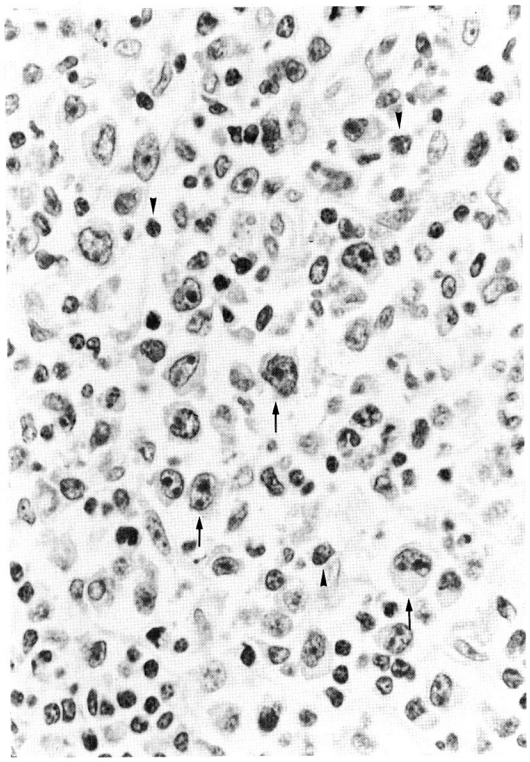


Fig. 3. Photomicrograph showing a lymph node from ATLL composed of cells with a variety of deep indentations and lobulations of the nuclei. There are medium-sized hyperchromatic cell(arrow head) and large cells with distinct nuclear membrane and two or three basophilic nucleoli and abundant cytoplasm(arrow)(H & E,×200).

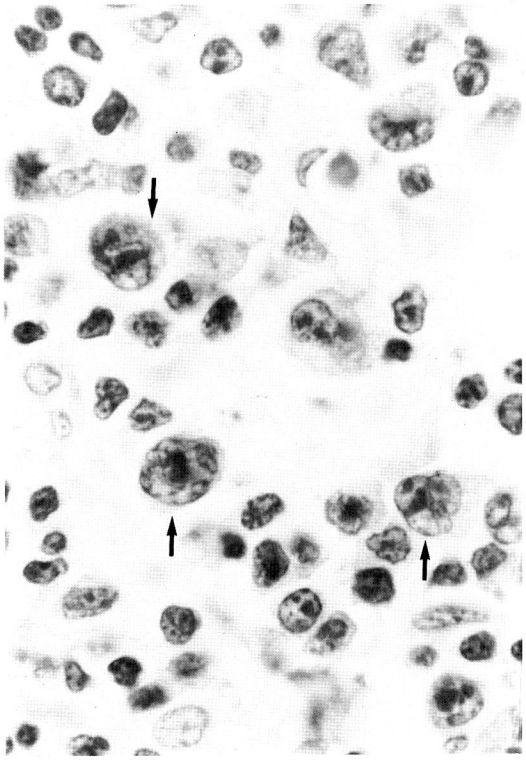


Fig. 4. Giant cell(arrow) corresponds to the cerebriform type with nuclei that have indentations on one side("jellyfish" appearance) are found(H & E,×500).

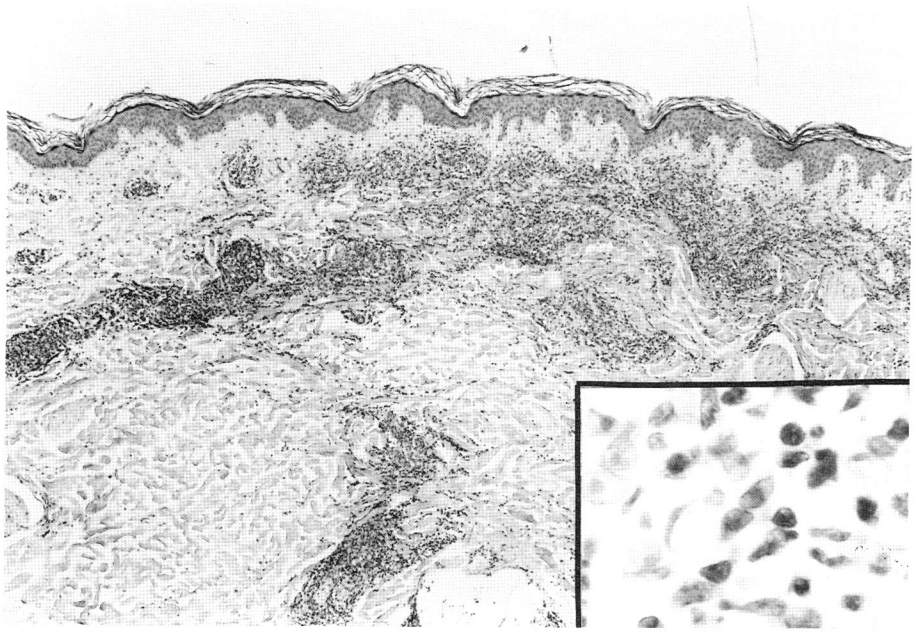


Fig. 5. Diffuse band-like dermal infiltrates of atypical lymphoid cells without epidermotropism(H & E, ×12). Inset shows high magnification of atypical lymphoid cells with irregular-shaped nuclei and prominent nucleoli(H & E,×200).

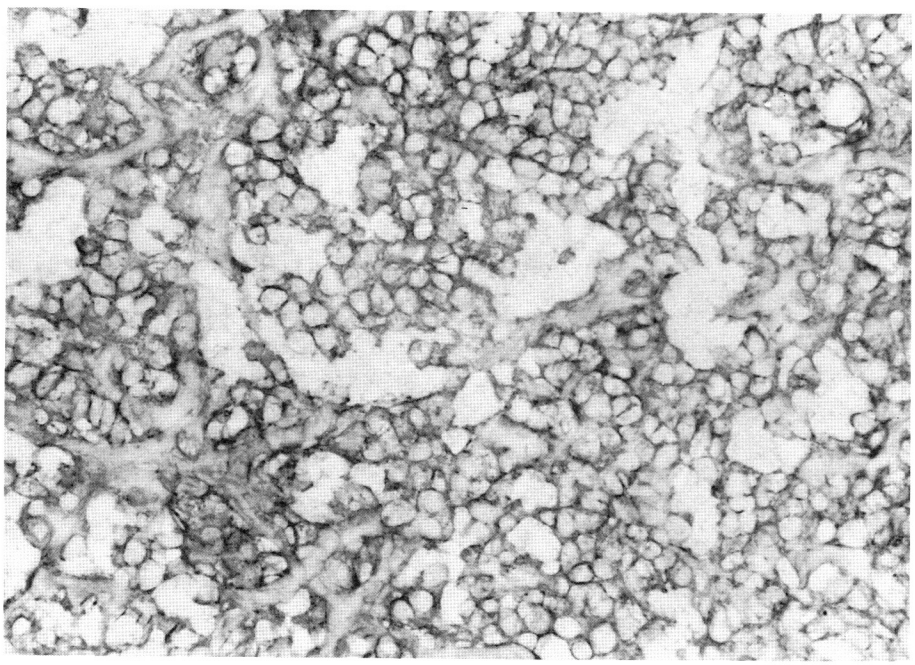


Fig. 6. Immunohistochemical staining for CD4 on frozen section of lymph node showed positivity on the cellular membrane(× 100).

small lymphocytes, and a few plasma cells were interspersed with tumor cells. Epithelioid venules were conspicuous but not arborizing. A biopsy specimen of cutaneous nodules revealed diffuse infiltration of atypical lymphoid cells involving the dermis but preserving the papillary dermis(Fig. 5). Epidermotropism or Pautrier microabscess was not present. The abnormal cells showed the same chracteristics of lymph node, including large or medium-sized abnormal cells with an irregular nuclei, and one or two prominent nucleoli.

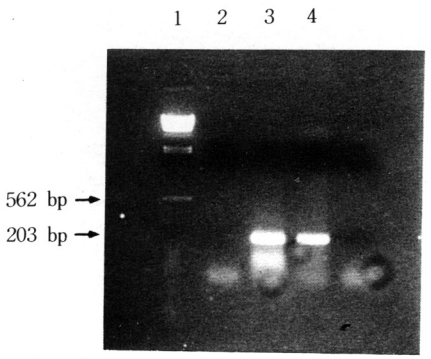
Immunophenotypic Analysis

Immunohistochemical staining was done on paraffin sections and on frozen sections of lymph node. Large and medium-sized cells expressed T-cell subset markers(CD2. CD4, CD45RO) but were negative for CD20 B-cell determinant. Other T-cell antigens(CD1, CD7, CD8) were not detected. Tumor cells show positivity for CD4 with simultaneous expression of the antigens associated with cell activation, CD25(IL-2 receptor) and CD30. EBER-1 in situ hybridization showed a few EBV-positive cells in lymph node.

HTLV-I Serological and Molecular Studies

In PBMC, HTLV-Ⅰ proviral DNA *pX* gene was detected using PCR, showing a specific amplified product of the expected size, i.e. 203 base pairs (Fig. 6). PCR for DNA from lymph node using *pol, gag*, and *env* primers of HTLV-Ⅰ (Innis et al., 1990) showed amplified fragments of 273, 186, and 271 base pair, respectively, with the same size of positive control, which is DNA from the HTLV-1^{+} cell line(Fig. 7).

HTLV-Ⅰ specific antibodies were found in the patient's serum. Antibodies reacting against both HTLV-Ⅰ core proteins(P19, P24, and their precursor, Pr53) and env glycoprotein(gp46) were demonstrated by Western blot.



Lane 1. λHind Ⅲ marker
Lane 2. negative control
Lane 3. positive control
Lane 4. sample DNA from blood

Fig. 7. PCR amplification for HTLV-Ⅰ pX in PBMC. Lane 4 was DNA from PBMC of our case and HTLV-Ⅰ pX amplification product was detected in 203 base pair.

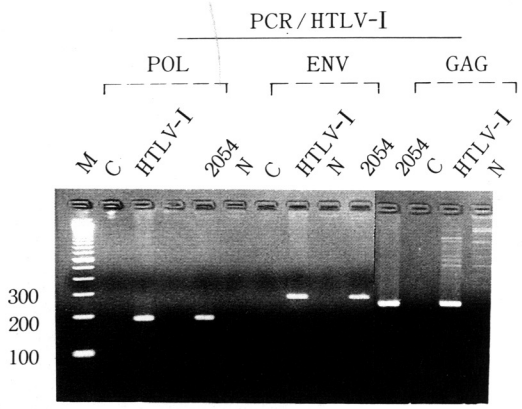


Fig. 8. PCR DNA amplificates using HTLV-Ⅰ specific primers, gag, pol, and env in the lymph node. Our case (2054) showed the same amplified fragments with the positive control of HTLV-Ⅰ. M : marker, C : control, without sample, HTLV-Ⅰ : positive control, 2054 : our case, N : negative control, tonsil.

Clinical course

The patient was diagnosed as having ATLL based on the clinicopathologic findings and was treated with an intensive chemotherapy including cyclophosphamide, etoposide, doxorubicin, vincristine, and prednisone. Despite initial improvement, the tumor progressed after three cycles of the regimen and became refractory to further chemotherapy.

DISCUSSION

ATLL is a unique peripheral T-cell lymphoma closely related to HTLV-1. The gene encoding for an HTLV-Ⅰ receptor is located at chromosome

17(Sommerfelt et al., 1988). but the receptor itself has not yet been isolated or characterized(Nagafuji et al., 1993). Almost all cases with ATLL are seropositive for anti-HTLV-I antibody, and the tumor cells possess proviral DNA of HTLV-I (Clark et al., 1988). At first, ATLL was characterized by lymphadenopathy, hepatosplenomegaly, skin rash, leukemic change, and hypercalcemia with aggressive clinical course to a fatal termination. Cytopathologically, HTLV-1-positive ATLL was characterized by extreme nuclear pleomorphism with $CD4^+$ helper T-cell type surface markers(Suchi et al., 1979; Hattori et al., 1981). As a rule, the phenotype is that of peripheral helper/inducer T cells($CD3^+$, $CD4^+$, $CD1^-$, $CD8^-$) with simultaneous expression of the antigens associated with cell activation, viz. HLA-DR and CD25(Lennert and Feller, 1992). A few tumors have shown co-expression of CD4 and CD8, but CD1 negative(Shamoto et al., 1984). All cases are CD7 negative. In the Working Formulation the group of HTLV-I-positive T-cell lymphoma is included in the "large cell, immunoblastic" type. This is certainly an oversimplification, because the difference in morphology between HTLV-I-positive T cell lymphoma and T-immunoblastic lymphoma is obvious(Lennert et al., 1985). ATLL is histologically divided into more detailed categories by the Japanese Lymphoma Study Group Classification(Suchi et al., 1979 ; Lennert et al., 1985). Hanaoka(1979) and Lennert (1985) have reported that the pleomorphic type is the most common among them. Histological identification of HTLV-I-positive cases might be possible, based mainly on the findings including intensely basophilic and coarsely aggregated chromatin, extreme irregularity of nuclear outlines and giant cells with hyperconvoluted nuclei(Lennert et al., 1985). Our case showed the characteristic histologic and immunophenotypic findings of HTLV-positive ATLL, and was classified as the pleomorphic type, medium-sized and large cell, based on the variable sized neoplastic cells with marked irregularity in nuclear configuration, including convoluted, hyperconvoluted and multilobated forms and 2 or 3 prominent nucleoli. Our case was positive for CD4 and CD25(IL-2R) and negative for CD8.

Tokunaga et al.(1993) demonstrated the coinfection of EBV and HTLV-I in tumor cells of 21 out of 96 ATLL cases using PCR and in situ hybridization, and hypothesized that both of these two virus might infect the same T cells in early life and might play possible roles in the oncogenesis of ATLL. In our case EBV positive cells were also present.

From an epidemiologic point of view, most of the series of ATLL cases have been reported in Japan or in the Caribbean, especially in Jamaica, areas where HTLV-I is highly endemic(Blattner et al., 1982; Clark et al., 1988). More recently, ATLL cases have been described in Central and South American countries where the HTLV-I endemicity begins to be better known(Blattner, 1989). Certain areas of the Middle East such as the Northern part of Iran and surrounding regions appear as a newly described area of high HTLV-I endemicity(Sidi et al., 1990; Kaplan et al., 1991). And ATLL has been reported in immigrants from Iran living in Israel and in Germany. The African continent is considered as the largest endemic area for HTLV-I infection. Seroepidemiologic studies have shown that in tropical and equatorial Africa the overall prevalence of HTLV-I infection in the general population is around 0.5% to 5%(Delaporte et al., 1989; Verdier et al., 1989). However, in Africa, HTLV-I associated disease, i.e. ATLL and tropical spastic paraparesis/HTLV-I associated myelopathy(TSP/HAM), have until now been rarely described and they were mostly sporadic in Central and West Africa(Tubiana et al., 1985; Gwssain et al., 1992). Rare sporadic ATLL cases have been reported(Cunningham et al., 1985). Lee et al.(1986) performed an epidemiologic study for HTLV-I in Korea, which revealed low positivity(0.25%) of serum antibodies for HTLV-I without evidence of ATLL and there was no endemic area in Korea. Only two cases of ATLL have been reported in Korea(Lee et al., 1987; Kim et al., 1994), of which HTLV-I was proven in the serum. In the first Korean case(Lee et al., 1987), both anti-HTLV-I antibody and antigen in the leukemic cells were positive by indirect immunofluorescence test using HUT-102 cells, and the patient's mother was a HTLV-I carrier who had a previous history of transfusion. The second case was also proven in association with HTLV-I in the PBMC. Our patient has been living in Korea from birth and has never been to other countries. This case can be considered as a sporadic case developed in Korea.

HTLV-I proviral *pX* region is well conserved in defective virus despite the frequent deletion of other structural genes such as *gag, pol*, and *env*(Hiramatsu et al., 1986; Ohshima et al., 1991). For this reason, HTLV-I proviral *pX* is the most reliable

target to detect HTLV-I for the purpose of screening. In our case, HTLV-I proviral DNA, *pX*, was detected first in the PBMC by PCR, and proviral *gag*, *pol*, and *env* were all detected in the lymph node by PCR. HTLV-I specific antibodies were positive in the serum.

In general, prototypic ATLL occurs in adults and takes a subacute or acute course. However, ATLL was classified into four types according to the clinical features and course ; smoldering, chronic, crisis, and acute(Kawano et al., 1985). The clinical course is predicted by initial hallmarks, so this classification allows treatment strategy to be determined at diagnosis. In acute type, so-called prototypic ATLL, which progresses acutely or subacutely, the patients suffer from increased ATLL cells, skin disease, systemic lymphadenopathy, and hepatosplenomegaly. Most patients in this group are resistant to chemotherapy and die rapidly. In general, a poor prognosis is indicated by elevations of serum LDH, calcium, and bilirubin as well as by high leukocyte count. For smoldering and chronic cases, no treatment is recommended. However, in crisis and acute cases, aggressive chemotherapy is neccessary, although the acute type cases show extremely poor prognosis despite the most aggressive chemotherapy(Kawano et al., 1985). Poor prognosis of ATLL is probably because ATLL cells frequently develop resistance to chemotherapy, although ATLL cells are initially chemosensitive(Nagafuji et al., 1993). Our case met the diagnostic criteria of acute form of ATLL, presenting with skin lesions, generalized lymphadenopathy of acute onset and 30% of ATLL cells on PBS. But there was neither hepatosplenomegaly nor hypercalcemia in our patient. And our case had the typical clinical course of acute ATLL, i.e., the disease progressed rapidly and became refractory to chemotherapy after initial transient response.

In addition to clinical and pathologic characteristics, serum antibodies to HTLV-I were detected and HTLV-I proviral DNA was proven in PBMC and lymph node by PCR. these clinicopathologic findings, including the immunophenotypical analysis, indicate that there can be little question as to diagnosis.

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