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Histology and Histopathology

IBL-like T cell lymphoma expressing monoclonal gammopathy (macroglobulinemia) in the serum

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Summary. A case of IBL-like T cell lymphoma with serum monoclonal gammopathy was reported. A 58-year-old woman, who had suffered from heart failure, was admitted because of asthma attack, fever and lymphadenopathy. Leucopenia with a small amount of atypical lymphocytes was detected. Serum analysis showed monoclonal elevation of IgM-κ (M-protein) and hyperviscosity. Urinary Bence-Jones protein was detected. Lymph node biopsy revealed the disappearance of normal structure and proliferation of T cells with pale cells which characterized IBL-like T cell lymphoma. Immunocytochemistry revealed the pale cells to bear T cell markers (MT-1, CD 5, CD 8 or CD 4) and IgM-positive cell distribution. Tonsilar biopsy showed the infiltration of atypical lymphoids and pale cells. Bone marrow biopsy showed moderate lymphoplasmacytoid proliferation with lymph follicles. Clinical data and serum analysis suggested macroglobulinemia. Additional lymph node biopsy was performed and revealed IBL-like T cell lymphoma. IBL-like T cell lymphoma is characterized by polyclonal hypergammaglobulinemia. The present case probably occurred initially as IBL-like T cell lymphoma and lymphoplasmacytoid cell proliferation might have followed due to an excess of CD 4^+ cells.

Key words: IBL-like T cell lymphoma, Monoclonal gammopathy, Macroglobulinemia, Immunocytochemistry

Introduction

IBL-like T cell lymphoma was initially described by Shimoyama et al. (1979, 1983) as a different entity from immunoblastic lymphadenopathy (IBL) (Lukes et al., 1975), angioimmunoblastic lymphadenopathy with dysproteinemia (AILD) (Frizzera et al., 1975) and other related types lymphadenopathy (Castleman et al., 1956; Lennert et al., 1968; Kojima, 1978). This disease was characterized by polyclonal gammopathy of the serum and systemic lymphadenopathy resembling IBL and AILD. However, pathological characteristics differing from IBL and AILD, included the appearance of pale cells with large, clear cytoplasm and lacking amorphous PAS-positive deposition in the lymph node (Suchi et al., 1987; Segami et al., 1988). In addition, the nature of this disease was considered to be T cell malignancy as opposed to B cell proliferation in IBL and AILD.

In this paper, we present a 58-year-old woman who suffered from systemic lymphadenopathy and monoclonal gammopathy who was diagnosed as having IBL-like T cell lymphoma by biopsy and macroglobulinemia by serum analysis. Immunocytochemical examination results and some points of interest are discussed.

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Materials and methods

A 58-year-old woman had been well until the age of 30. She suffered from pulmonary tuberculosis (1953-1974), glaucoma of the left eye (1960), retinal bleeding of the right eye (1970), appendicitis with surgical removal (1977) and right ovarian cyst with resulting surgical operation (1983). In 1983, she experienced palpitations and was treated under a diagnosis of paroxymal atrial tachycardia. In July 1987, she was admitted to the Daini Hospital of the Tokyo Women's Medical College because of additional symptoms, including asthma attack, respiratory distress, herpes zoster and weight loss with systemic lymphadenopathy.

On admission, systemic lymph node swelling and tenderness, especially in the cervical and inguinal regions, up to $2 \times 1 \times 1$ cm in size, was noted, but there was no skin rash, swelling of liver or spleen, nor were there any neurological abnormalities.

Lymph nodes were examined by routine methods and by immunocytochemical methods including the PAP procedure by paraffin sections and direct/indirect immunoperoxidase staining of PLP-fixed frozen sections including electron immunomicroscopy as previously reported (Kasajima et al., 1986, 1987).

Results

Laboratory examinations showed a decrease of peripheral white cells, with 1-4% atypical lymphocytes. ESR was 67mm/hr and blood pressure was 140/80mmHg. Biochemistry revealed a slight disturbance of liver function. Serum analysis showed a marked increase in IgM and of blood viscosity. Immunoelectrophoresis disclosed M-protein which was monoclonal IgM kappa light chain (Fig. 1) and also the urinary kappa type of Bence-Jones protein. Moreover, serum tests were positive for cryoglobulin. However, there were no antiautonuclear antibodies or anti-human T cell leukemia virus type I antibodies (ATLA). After admission, cardiac and respiratory symptoms were slightly controlled with drug therapy, but weight loss, lymphadenopathy and the high level of serum IgM-κ still continued. Bone marrow biopsy revealed increasing lymphoplasmacytoid cells with formation of lymph follicles. In addition, enlargement of liver and spleen were noticed. In July 1987, lymph node biopsy was performed. Pathological examination disclosed IBL-like T cell lymphoma. In September 1987, tonsillectomy was also performed and specimen showed atypical neoplastic lymphoid cell infiltration with pale cells which were the same kind of cells as in the lymph node. Additional lymph node biopsy were carried out in December 1987 and also showed morphological features similar to the previously biopsied lymph node. Immunogenetical analysis was simultaneously performed and showed the rearrangement of the T cell receptor ß chain constant region with Southern blotting (Fig. 2). Based on the above mentioned clinical signs, and laboratory data, such as IBL-like T cell lymphoma accompanied by macroglobulinemia (Tables 1, 2) was diagnosed.

Pathological and immunocytochemical findings of biopsy specimens

1. *Lymph nodes.* Lymph node biopsies were performed twice (July and December 1987). Both lymph nodes disclosed similar histological features; i.e. disappearance of normal architecture and proliferation of atypical lymphoid cells with small arborizing blood vessels. Lymph follicles were indistinct or had disappeared at the light microscope level (Figs. 3, 4). Medium and large lymphoid cells with round or slight convoluted nuclei, and large lymphoid cells with prominent cytoplasm (so-called pale cells) were zonally or focally distributed (Fig. 5A). Moreover, lymphoplasmacytoid and plasma cells were located

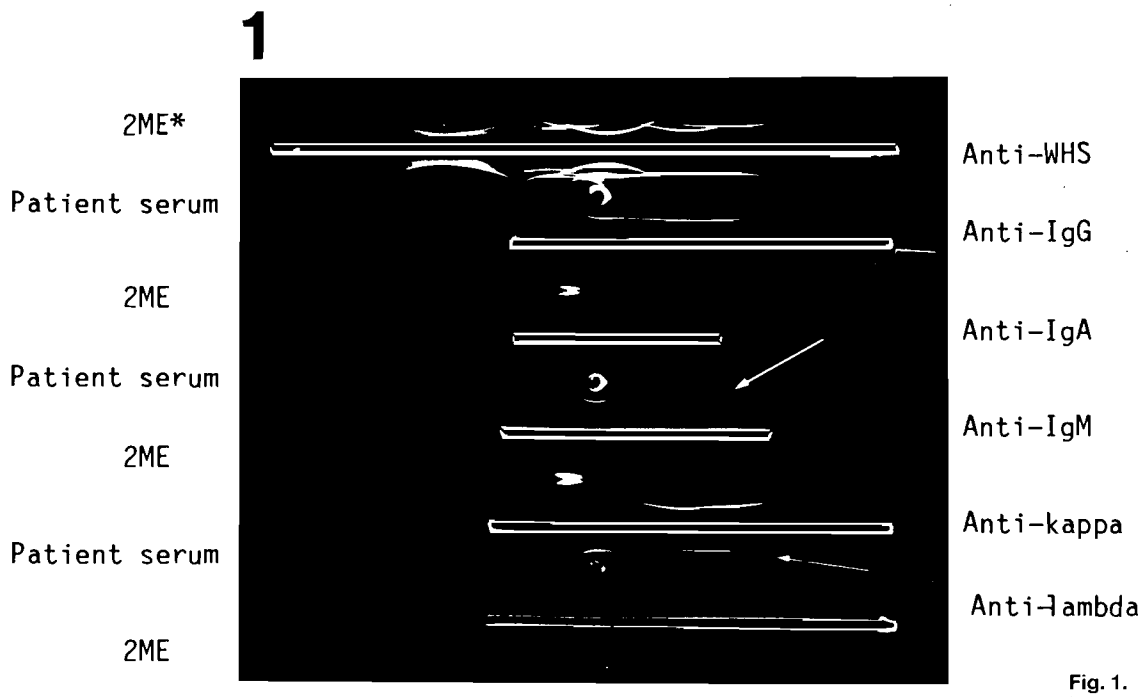
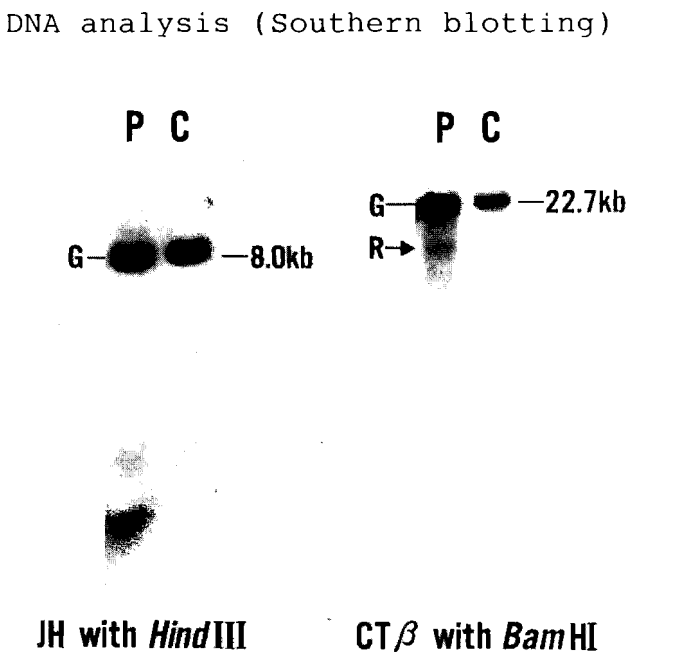


Fig. 1. Immunoelectrophoresis of the serum, showing reaction of the M-component with anti-IgM and anti-K antisera (arrow).



CTβ with Bam HI :R/G faint
JH with Hind III : G
CTβ; T cell receptor β-chain constant region gene
JH ; Immunoglobulin heavy chain joining region gene
R ; rearrangement, G; germ line

Fig. 2. DNA analysis of the second biopsied lymph node, showing rearragement of CTβ (arrow).

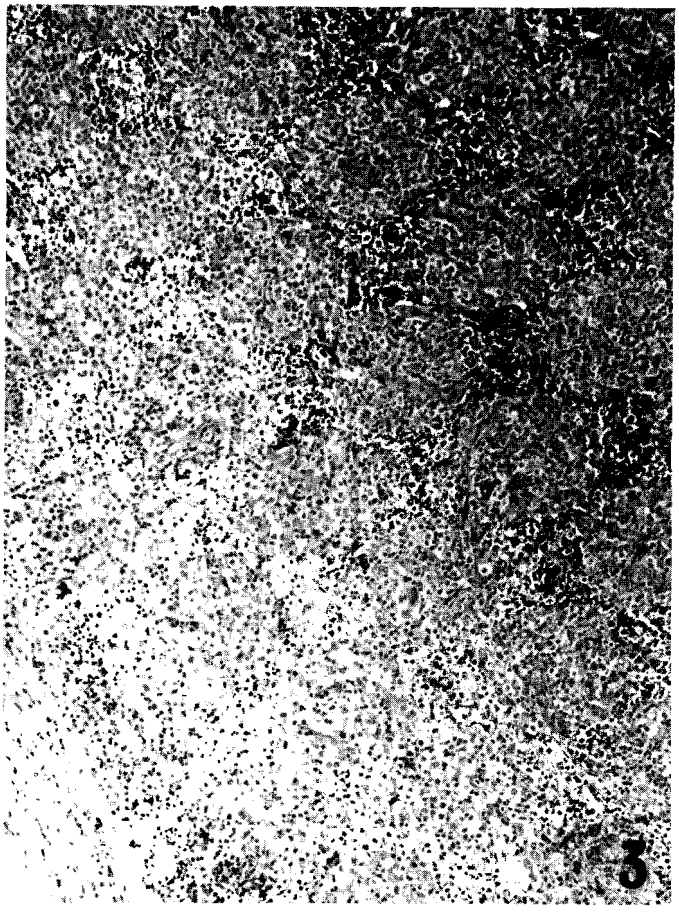


Fig. 3. Lymph node from the first biopsy. Lymph follicles disappear and atypical lymphoid cells distributed diffusely mingl with pale cells. HE × 75

irregularly or focally around the small vessels and beneath the marginal sinus or medullary portion. Deposition of amorphous materials could not be detected.

Immunocytochemically, neoplastic lymphoid cells and pale cells were positive for MT-1 on paraffin sections (Fig. 5B) and positive for CD 5 (Leu 1) in PLP-fixed frozen sections, but were negative for MB-1 and CD 20 (B 1), respectively. On the other hand, some lymphoplasmocytoid cells and mature plasma cells contained IgM more predominantly (Fig. 6) and κ-light chain than other heavy chains and λ-light chain, but neoplastic cells never contained immmunoglobulins. Immunoelectronmicroscopically, both neoplastic cells reacted positively for CD 4 (Fig. 7) or CD 8 (Fig. 8) on each of their cell surfaces. IgM was recognized in the perinuclear spaces and rough endoplasmic reticulum (rER) of lymphoid and plasma cells (Fig. 9). In addition, some reticular areas that reacted with DRC-1 and H107 coincided with the site of lymph follicles (Table 3).

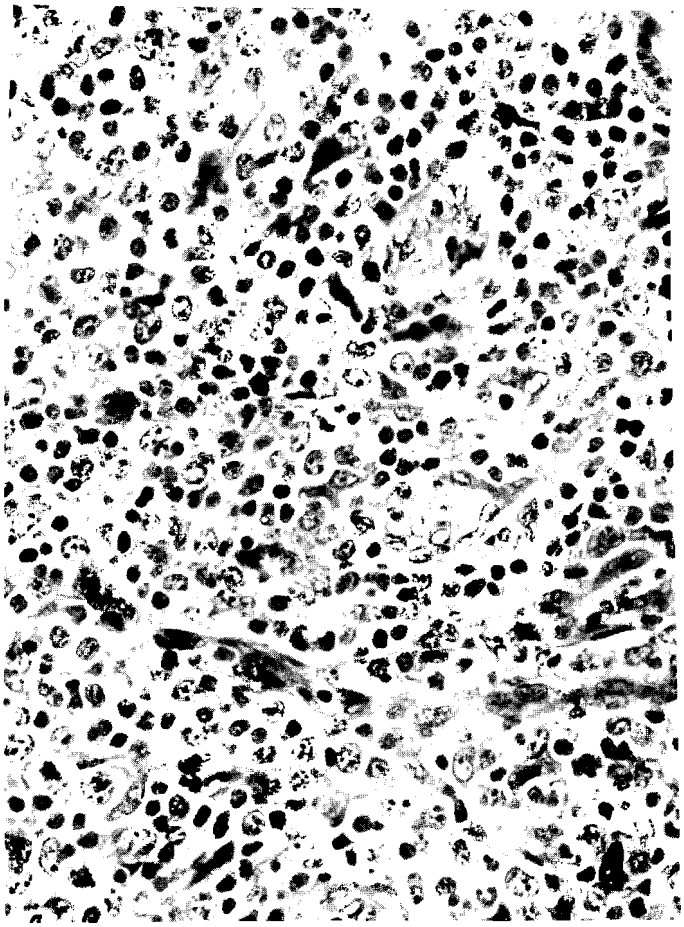
2. *Tonsil.* Lymph follicles disappeared and neoplastic large lymphoid cells with pale cells were distributed throughout the entire part of submucosal area of tonsil. Cytological features of these cells were similar to those of the lymph nodes (Fig. 10).

3. *Bone marrow.* Bone marrow biopsy revealed no lymphomatous condition, but somewhat lymphoplasmacytoid cells increased and lymph follicles with germinal centers were detected (Fig. 11).

Immunocytochemically, cells containing IgM were more predominant than those containing IgA or IgG, but monoclonal distribution of immunoglobulins was hardly recognizable (Fig. 12).

Discussion

IBL-like T cell lymphoma was first reported by Shimoyama et al. (1979). It resembles IBL, AILD and polyclonal immunoblastosis (Kojima, 1978). On the other



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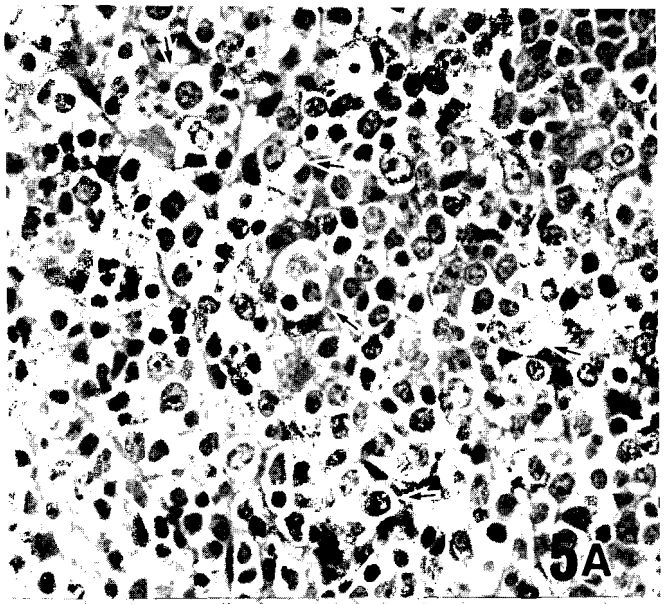
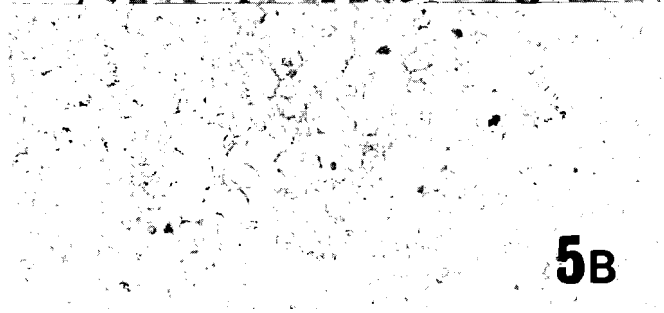


5A



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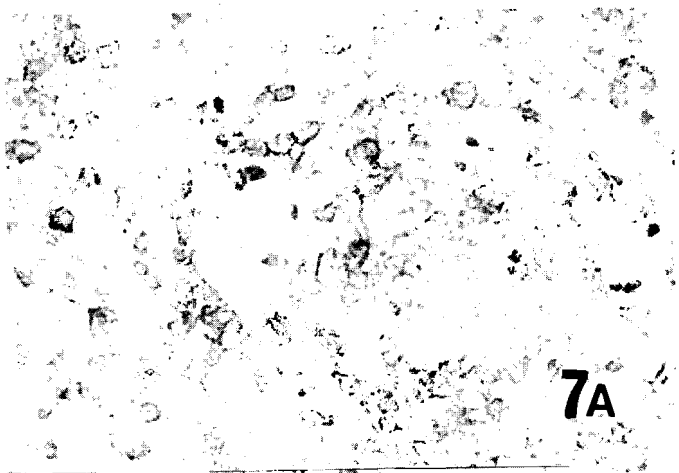
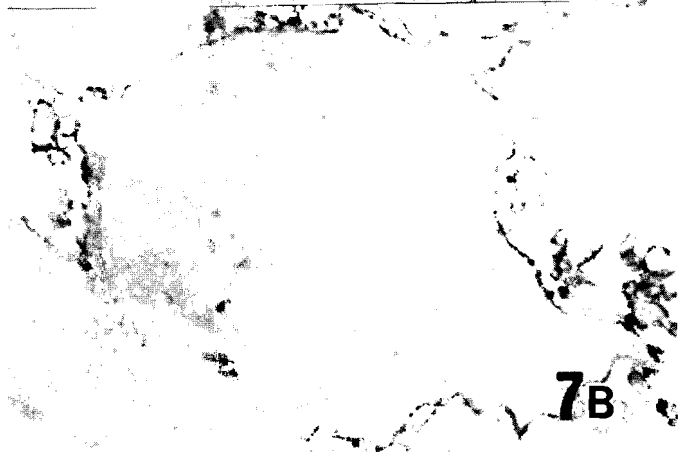


7A



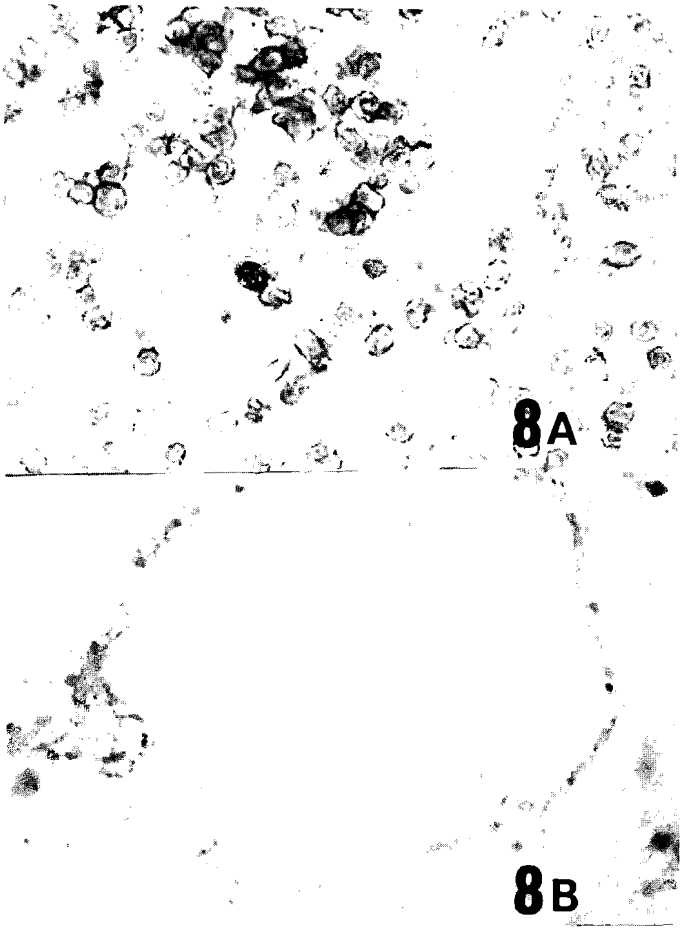
7B

Fig. 4. High power view of Fig. 3. Arborizing blood vessels increase and atypical lymphoids appear intermingling with small lymphocytes. HE × 500

Fig. 5A. High power view of Fig. 3. Pale cells which have prominent clear cytoplasm (arrow) appear. HE × 500. **Fig. 5B.** Immunocytochemical staining for MT-1, showing positive reaction on the cell surfaces of pale cells and lymphoids. Indirect method on paraffin sections with methyl-green stain. × 250

Fig. 6. Immunocytochemical staining for IgM. IgM-positive cells are distributed on the subcapsular area and cortex, but are not detected on atypical cells of the lesion. Paraffin sections, PAP method and methyl-green stain, × 75

Figs. 7A and 7B. Immunocytochemical staining for CD 8. CD 8 positive cells are scattered in the second biopsied lymph node (A), and immunoelectron-microscopy show the positive reaction on the cell surface (B). A × 500, B × 9,600



Figs. 8A and 8B. Immunocytochemical staining for CD 4. CD 4 positive cells are distributed in the second biopsied lymph node (A), and are positive on the cell surface (B). A × 500, B × 9,600

hand, IBL-like T cell lymphoma differs from IBL, AILD and polyclonal immunoblastosis in that the former is characterized by the proliferation of the T cell series, while the latter involve the proliferation of the B cell series.

Moreover, the prognosis of IBL-like T cell lymphoma was poorer than that of IBL, AILD and polyclonal immunoblastosis. Shimoyama et al. (1983) reported that the mean survival interval of this disease was ten months (3-153 months) and also stated that T suppressor type of this disease had extremely poor prognosis. On the other hand, irrespective of the prolongation of lymph node swelling, abnormal ,gammopathy and other symptoms, the present patient is fortunately alive and well now.

Definite polyclonal hypergammaglobulinemia is one of the main features of this disease as has been reported by many authors (Shimoyama et al., 1979; Maeda et al., 1986; Segami et al., 1988). In our case, twice biopsied lymph nodes disclosed IBL-like T cell lymphoma, whereas serum analysis revealed monoclonal gammopathy to be a macroglobulinemia (Wardenström et al., 1944). Histological examination of lymph nodes showed features compatible with IBL-like T cell lymphoma; i.e. disappearance of lymph follicles, proliferation of neoplastic lymphoid cells and pale cells with remarkable small arborizing blood vessels. In addition, immunocytochemistry revealed that these neoplastic cells were characterized by a T cell nature ($MT\text{-}1^+$, $CD\ 5^+$). There

Fig. 9. Immunocytochemical staining for IgM. IgM-positive reaction is recognized in the rER and perinuclear spaces (arrows) of plasma cells and lymphoplasmacytoid cells. Indirect method, PLP-fixed frozen. × 5,800

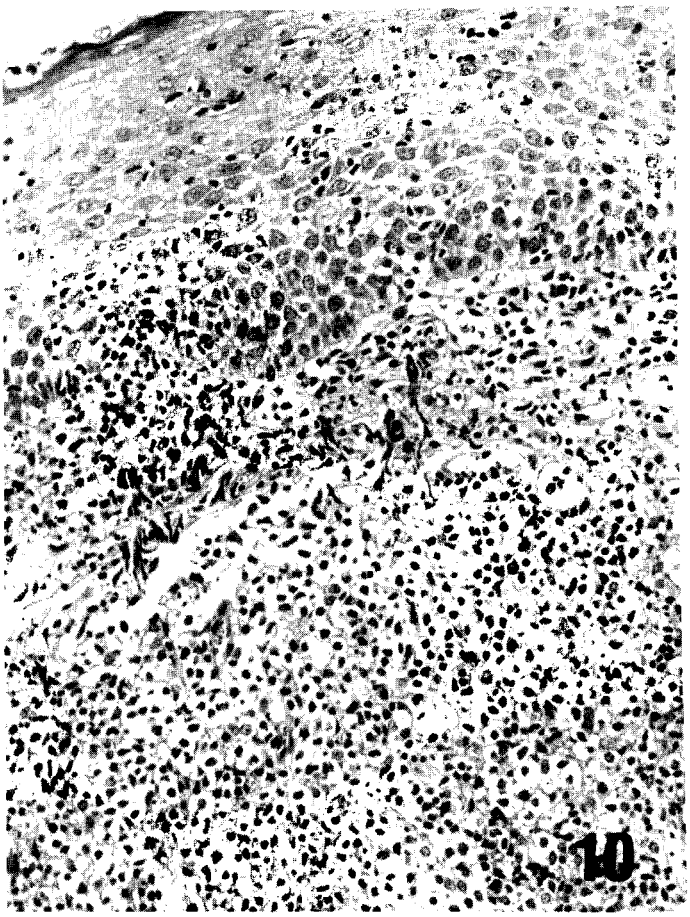
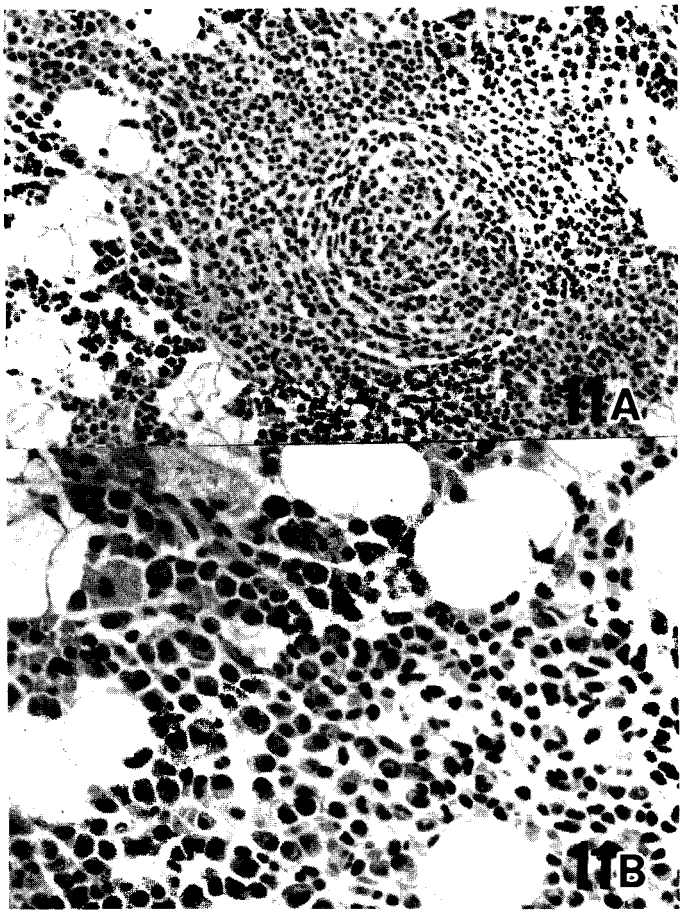


Fig. 10. Biopsy specimen of tonsil. Atypical lymphoid and pale cells occupying submucosal region of tonsil. HE × 150.

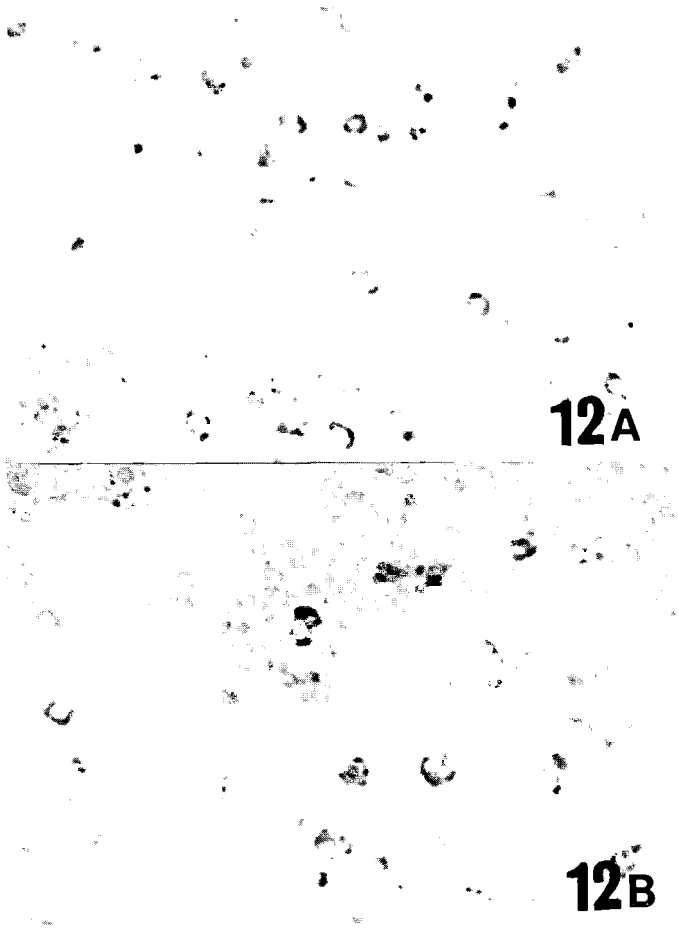


Figs. 11A and 11 B. Bone marrow. Formation of lymph follicle with germinal center (A) and lymphoplasma cells (B) are observed, HE A. × 150, B. × 300

are no case reports of IBL-like T cell lymphoma with monoclonal gammopathy. Hido et al. (1987) reported a case of IBL-like T cell lymphoma with polyclonal gammopathy which had monoclonal elevation of cold hemaglutinin titer (IgM-kappa), and suggested that the tumor cells might stimulate the production of cold hemaglutinin through B lymphocytes.

On the other hand, macroglobulinemia is thought to be the condition of elevation of monoclonal serum IgM, casued by tumor-like proliferation of IgM-producing-B lymphocytes in bone marrow and other lymphoreticular tissues, with appearance of Bence-Jones protein (Wardenström et al., 1944). In the present case there was a moderate increase in lymphoplasmacytoid or plasma cells and formation of lymph follicles in bone marrow, but no leukemic change was noticed. Lymphoplasmacytoid cells distributing in bone marrow and lymph nodes could not be considered as neoplastic cells. Immunocytochemically, IgM and κ-light chain were detected in rER and perinuclear spaces of these cells. Perivascular lymphoplasma cell infiltration is one of the features of IBL-like T cell lymphoma and the present case showed lymphoplasma cell infiltration not only in perivascular areas but also areas apart from the perivascular regions; i.e. the cortex and medullary portions. In spite of extensive distribution of IgM-bearing lymphoplasma cells, there was infiltration by plasma cells bearing other types of immunoglobulins to varying degree. Moreover, immunocytochemistry revealed several areas positive for DRC-1 (Follicular dendritic cell specific antibody) or H107 (FcεR II) (Kasajima et al., 1986, 1987; Masuda et al., 1987, 1988) in which lymph follicles could not be detected at the light microscopical level. In these areas, lysis of lymph follicles was considered to be involved in the vanishing process because of the absence of immunoglobulins. Takagi et al. (1988) reported that these areas were thought to be areas of reaction process.

The appearance of pale cells, which were characteristic cells of T cells malignancy and especially IBL-like T cell lymphoma, were shown to be CD 8^+ (Watanabe et al., 1982; Shimoyama et al., 1983), but it has been reported that pale cells do not always express only CD 8 (Tamaki et al., 1984; Hido et al., 1988; Segami et al., 1988). In the present case, pale cells expressed both CD 4 or CD 8 T cell antigens in the lymph node of the first biopsied node. It was reported that IBL and AILD were states of T cell dysplasia possessing the possibility of transition to lymphoma. In addition, the pale cells always



Figs 12 A and 12 B. Immunocytochemical staining for IgM (A) and IgG (B) of bone marrow. IgM-positive cells were predominant over IgG-positive cells with no monoclonal staining results. PAP method, paraffin sections with methyl-green. × 500

appeared when remarkable CD 8 expression was recognized (Watanabe et al., 1986). In the present case, pale cells were recognizable in the lymph nodes of both biopsies in which both phenotypes were expressed. Takami et al. (1988) also reported that a lymphadenopathy with pale cells of T cell nature differed from IBL-like T cell lymphoma or other T cell lymphoma and it was named T clear cell lymphoma which had a relative favorable prognosis. Our patient, up to now, is fortunately alive and well, but this case could not be considered as compatible with Takami's proposal. Recently, the usefulness of examination for DNA arrangement of immunoglobulin and T cell antigen receptor for the diagnostic analysis lymphoproliferative disorders or lymphoma, have been discussed (Sheibani et al., 1987; Weiss et al., 1988). Tobinai (1987) reported the rearrangement of T cell antigen receptor (CTß) in eight cases out of 12 cases of IBL -like T cell lymphoma had possibility of monoclonal growth as a T cell malignancy.

In the present case, Southern blotting hybridization revealed the rearrangement of CTß without rearrangement of the immunoglobulin gene. Results suggested that our case expressed T cell monoclonal growth character in lymph nodes. Although DNA analysis of bone marrow cells was not available, morphological features could not show tumorous change. So, our case probably occurred initially as IBL-like T cell lymphoma and then lymphoplasmacytoid proliferation might follow, or clinically simultaneously occur as macroglobulinemia due to excess CD 4^+ cells in lymph nodes and other tissues. However, problems concerning monogenesis remain unsolved in this case.

Table 1. Laboratoy Data

Peripheral blood		**Bone marrow**	
RBC	426 × 10^4/mm^3	NCC	3.9 × 10^4/mm^3
WBC	2100/mm^3	Megakaryocyte	12.5/mm^3
Hb	12.3g/dl	Myeloblast	0.6 %
Ht	39.7%	Promyelocyte	3.2%
Platel.	20.9 × 10^4/mm^3	Myelocyte	16.6%
ERS	61mm/hr	Metamyelocyte	2.6%
T cell*	84.0%	Band granulocyte	18.0%
CD 8**	20.1%	Segented granulocyte	11.0%
CD 4	47.3%	Eosinophils	1.0%
CD16	15.1%	Basophils	0 %
CD4/CD8	2.35	Monocyte	1.8%
B cell***	4.0%	Lymphocyte	14.6%
		Plasma cell	0.4%
		Proerythroblast	0%
		Basophilic erythroblast	0.8%
		Polychromatic erythroblast	21.0%
		Orthochromatic erythroblast	1.8%
		Atypical lymphocite	4.4%

* T cells were evaluated with SRBC rosette forming method, T-cell phenotypes were analysed with ELISA** and B cells*** were examined with surface immunoglobulin detection.

Tumor markers; CEA 1.3ng/ml, AFP 2.9ng/ml, CA 19-914U/ml Ferritin 6.5ng/ml, Serum $ß_2$microglobulin 4.1mg/dl, Urine polyamine 87.1µmol/L, ATLA (–)

Examined in December, 1987.

Table 2. Main Laboratory Data

Biochemistry	
T.P.	6.5g.dl
Albumin	61.3%
α_1-globulin	4.0%
α_2-globulin	8.5%
ß-globulin	9.3%
γ-globulin	16.9%
IgG	1034* → 977mg/dl
IgA	82.6* → 149mg/dl
IgM	1576* → 1980mg/dl
Cryoglobulin	(+)
Pyroglobulin	(–)
GOT	122 IU/l
GPT	125 IU/l
Al-phos.	146 IU/l
LAP	189 IU/l
TTT	17.1M-U
ZTT	22.5 KU
CRP	0.24mg/dl
CH50	23.3 U/dl
C3	73.3mg/dl
C4	3.8mg/dl
RA	(+)
Anti-DNA Ab.	(–)
Anti-nucl Ab.	(–)
LE	(–)

Viscosity	
10 RPM	5.64 cp
20 RPM	4.86 cp
50 RPM	4.34 cp
Immunoelectrophoresis	
Serum	IgM-kappa (M-protein)
Urine	Bence-Jones' protein (+)
Blastogenesis of lymphocyte	
PHA $^+$	4685 cpm (cont. 332 cpm)
Con A$^+$	2557 cpm (cont. 332 cpm)

Exmined in December 1987,
(* examined on July 1987)

Table 3. Immunocytochemical Staining Results of Lymph Nodes

	Neoplastic Lymphoids & Pale cells	Lymphoplasmacytoid & Plasma cells	CD clone Specificity	Source
Monoclonal antibodies				
LCA	+	+/–	CD 45	DAKOPATTS
MT-1	+	–	T cell & Myelogenous	Bioscience
MB-1	–	–/+	B cell & variable	Bioscience
Leu 1	+	–	CD 5	B-D*
Leu 2a	+	–	CD 8	B-D
Leu 3a	+	–	CD 4	B-D
Leu 7	–	–	LGL, NK/K	B-D
Leu M1	–	–	Myelogenous	B-D
B 1	–	+/–	CD 20	Coulter
DRC-1	#	#	FDC	DAKOPATTS
H107	#	#	FcεR	Nichirei
Polyclonal antibodies				
IgM	–	++	Ig**	DAKOPATTS
IgG	–	+	Ig	DAKOPATTS
IgA	–	+	Ig	DAKOPATTS
IgE	–	–	Ig	DAKOPATTS
κ	–	++	Ig	DAKOPATTS
λ	–	+	Ig	DAKOPATTS
Lysozyme	–	–		DAKOPATTS
S100	–	–		DAKOPATTS

* B-D: Becton-Dickinson, **Ig: Immunoglobulin, # Positive reticular reaction were detected at sites corresponding to lymph follicles which were probably vanishing.

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References

Castleman B., Iverson L. and Menendex V. (1956). Localized mediastinal lymph-node hyperplasia resembling thymoma. Cancer 9, 822-880.

Frizzera G., Morgan E.M. and Rappaport H. (1975). Angioimmunoblastic lymphadenopathy with dysproteinemia. Lancet, 1070-1073.

Hido M., Takai K, Sanada M., Okazaki E. and Honma K. (1987). A case of IBL-like T cell lymphoma (helper T cell phenotype) with marked high titer of cold hemagglutinin. J. Jap. Soc. RES. 26, 265-272 (in Japanese).

Kasajima T., Sato T., Yamakawa M., Masuda A. and Imai Y. (1986). Immunocytochemical study of the immune complex and germinal center of synovial tissue of rheumatoid arthritis with special regard to complement and follicular dendritic cells. Acta Pathol. Jpn. 36, 835-855.

Kasajima T., Yamakawa M. and Imai Y. (1987). Immunohistochemical study of intrathyroidal follicle. Clin. Immunol. Immunopathol. 43, 412-417.

Kojima M. (1978). Immunoblastic lymphadenopathy (Lukes & Tindle) -proposal of polyclonal immunoblastosis. Jpn. J. Clin. Hematol. 19, 412-417.

Lennert K. and Mestdagh J. (1968). Lymphogranulomatosen mit konstant hohen Epitheloidzellgehalt. Virchows Arch. (A) 344, 1-20.

Lukes R.J. and Tindle B.H. (1975). Immunoblastic lymphadenopathy; A hyperimmune entity resembling Hodgkin's disease. N. Engl. J. Med. 292, 1-8.

Maeda K., Matsuda M., Kasajima T. and Imai Y. (1986). An autopsy case of «IBL like T cell lymphoma» developing to diffuse lymphoma with marked elevation of EB virus titer. J. Jap. Soc. RES. 25, 235-246 (in Japanese).

Masuda A. (1988). Immunohistochemical study of Warthin's tumor with special regard to germinal center. Histol. Histopath. 3, 81-91.

Masuda A., Kasajima T., Yamakawa M. and Imai Y. (1987). Immunohistochemical study of Fcε receptor in lymph follicle. Jap. J. Soc. RES. 27, 73-83 (in Japanese).

Segami H., Abe M. and Wakasa H. (1988). Peripheral T cell lymphoma with helper T cell phenotype (Leu $3a^+$ Leu 8^-). Acta Pathol. Jpn. 38, 591-603.

Sheibani A., Wu A., Ben-Ezra J., Stroup R., Rappaport H. and Winberg C. (1987). Rearrangement of K- chain and T cell receptor ß-chain genes in malignant lymphomas of «T cell» phenotype. Am. J. Pathol. 129, 201-207.

Shimoyama M., Minato K., Saito H., Takenaka T., Watanabe S., Nagatani T. and Naruto M. (1979). Immunoblastic lymphadenopathy (IBL) -like T cell lymphoma. Jpn. J. Clin. Oncol. 9 (Supopl), 347-356.

Shimoyama M., Tobinai K., Minato K. and Watanabe S. (1983). Immunoblastic lymphadenopathy (IBL)-like T cell lymphoma. Recent Adv. RES Res. 23, 161-170.

Suchi T., Lennert K., Tu L.Y., Kikuchi M., Sato E., Stanfeld A.G. and Feller A.C. (1987). Histopathology and immunohistochemistry of peripheral T cell lymphomas. A proposal for classification of T cell lymphomas. J. Clin. Pathol. 40, 995-1015.

Takagi K., Fukunaga S., Yamaguchi H., Sugino N., Mori S., Katayama I. and Ishikawa E. (1988). IBL like T cell lymphoma. Immunohistochemical analysis. In: Angioimmunoblastic lymphadenopathy-AILD-IBL type diseases. Suchi Y., Hanaoka M. and Kijuchi M. (eds). Kindai Shuppan. Tokyo. pp 159-163, (in Japanese).

Takami T. and Kikuchi K. (1988). Immunohistochemical study on AILD-IBL related disease-especially on the clear cell type T lymphoma. In: Angioimmunoblastic lymphadenopathy-AILD-IBL type disease. Suchi Y., Hanaoka M. and Kikuchi M. (eds). Kindai Shuppan. Tokyo. pp 58-65 (in Japanese).

Tamaki T., Katagiri S., Kanayama Y., Konishi I., Yonezawa T., Tarui S. and Kikutani T. (1984). Helper T cell lymphoma with marked plasmacytosis and polyclonal hypergammaglobulinemia, A case report. Cancer 53, 1590-1595.

Tobinai K. (1988). Rearrangement of immunoglobulin gene and T cell antigen receptor gene in lymphoid malignancy. Pathol. Clin. Pathol. 6, 725-728.

Wardentröm J. (1944). Incipient myelomatosis or «essential» hyperglobulinemia with fibrinogenopenia--- a new syndrome? Acta Med. Scand. 117, 216-247.

Watanabe S., Nakajima T. and Shimoyama M. (1982). T cell lymphoma in relation to subpopulation of T-lymphocytes. GANN Monograph Cancer Res. 28, 107-120.

Watanabe S., Sato Y., Shimoyama M., Minato K. and Shimosato Y. (1986). Immunoblastic lymphadenopathy, angioimmunoblastic lymphadenopathy, and IBL-like T cell lymphoma ---A pectrum of T cell neoplasia. Cancer 58, 2224-2234.

Weiss L.M., Picker L.J., Grogan T.M., Warnke R.A. and Sklar J. (1988). Absence of clonal beta and gamm T cell receptor gene rearrangment in a subset of peripheral T cell lymphomas. Am. J. Pathol. 130, 436-442.

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