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Multiple Subcutaneous Nodules, Persistent High Fever and Lymphadenopathy*
— Case SNUCH CPC-33 —

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PRESENTATION OF A CASE

This 15-year-old boy was admitted for the second time to Seoul National University Children's Hospital (SNUCH) on February 28, 1988, because of intermittent high fever and lymph node swelling.

His illness started in 1985 as repeated sore throat and high fever, for which he was brought to Korea University Hospital. There he underwent an oropharyngeal biopsy that was read as acute necrotizing inflammation. In August 1987, neck lymph node swelling and splenomegaly were noted in addition to the intermittent fever. On February 29, 1988, he was admitted to SNUCH to receive a lymph node biopsy, which revealed necrotizing and granulomatous inflammation with heavy eosinophilia.

He was born via normal full term spontaneous delivery, and his immediate postnatal course was uneventful. Although no specific disease could be recalled by the parents, intermittent high fever, otitis, and sore throat were recurrent symptoms and signs through his infancy and early childhood.

Laboratory findings were not remarkable except for anemia (Hgb 10.9 g/dl, hematocrit 35.8%). Total protein was 8.9 g/dl with 4.1-4.8 g/dl of albumin. Immunoelectrophoresis showed IgG 3770 mg/dl, IgA 603 mg/dl, IgM 138 mg/dl, IgD 40 mg/dl and IgE 950 IU/ml. No specific treatment was given. A chest X-ray showed a prominent left hilum with perihilar lymphadenopathy.

In January 1989, he was admitted to Korea University Guro Hospital. Physical examination revealed growth retardation and 2-fingerbreadth-palpable spleen. Laboratory data showed no remarkable finding except for ASO titer > 820 units and positive Rh factor. With a diagnosis of juvenile rheumatoid arthritis he was managed with aspirin. Bone marrow examination was done and was unremarkable. Chest X-ray was also normal. Abdominal sonography revealed diffusely enlarged echogenic kidneys. Electrophoresis revealed a broad globulin band of polyclonal nature. Hepatitis B surface antigen and VDRL were negative. During the prednisone treatment he developed edema and proteinuria. In April 1989, a kidney biopsy was done, which showed findings consistent with light chain nephropathy.

On June 18, 1989, he was admitted again to Korea University Hospital because of headache and generalized seizure. He was in a semicomatous state for several days, and a brain CT revealed suggestive cerebral infarction in the right basal ganglia. At that time herpes zoster encephalitis was strongly considered, and an anti-viral agent was prescribed. Over the coming weeks he became much improved and to have alert mentation. However, left hemiparesis and suggestive cerebral infarction on radiograph still remained. At that time he was known to have hypertension with cotton-wool fundic change and left ventricular hypertrophy.

He was relatively good until July 1990, when a left flank mass developed. He visited Korea University Hospital again. A biopsy of the mass show-

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ed chronic necrotizing inflammation with fat necrosis. After the biopsy multiple subcutaneous nodules were noted in the arms, legs, abdomen, and head, associated with local heat and tenderness. He was given prednisolone 20 mg and maintained until the last admission.

The last admission was on October 23, 1990, to Seoul National University Hospital Department of Dermatology for an ill-defined purpuric, erythematous lesion in the lower extremities. The patient, now 17 years of age, was transferred to Internal Medicine Service because of fever, aggravated azotemia (BUN 99 mg/dl and Creatinine 7.5 mg/dl), and hyperkalemia (6.8 mmol/l). The patient was drowsy and edematous and acutely ill looking.

Physical examiantion revealed moon face, pale conjunctivae, slightly icteric sclerae, and dry tongue. The neck showed no lymphadenopathy. The chest revealed a clear breathing sound with inspiratory crackles in the left lower lung field. Systolic murmur was heard along the left lower sternal border. The spleen tip was palpable. The liver was 3FB palpable and hard and slightly tender. The back and extremities were unremarkable. After admission his general condition became progressively worse with myalgia, fever, dyspnea, and blood-tinged sputum. Prothrombin time was prolonged, and fibrin degradation product was positive. He died on November 3, 1990.

DISCUSSION

Dr. Ahn: This patient had the following clinical problems: recurrent intermittent fever, lymphadenopathy, splenomegaly, and multiple subcutaneous nodules with purpuric erythematous skin lesions. He also had the following pathological problems: acute necrotizing inflammation (oropharynx), necrotizing and granulomatous inflammation with heavy eosinophilia (lymph node), light chain nephropathy, and chronic necrotizing inflammation with fat necrosis (flank mass). His laboratory abnormalities were as follows: anemia, reversion of albumin/globulin ratio, hyperglobulinemia (polyclonal), hyperimmunoglobulin G, increased ASO titer, positive Rh factor, proteinuria, and prominent left hilum and perihilar lymphadenopathy in the chest X-ray. At the terminal stage he had azotemia, hyperkalemia, fibrin degradation product, and prolonged prothrombin time.

With the list of the above problems this patient seemed to have a lymphoproliferative disorder.

Table 1. Causes of Lymphadenopathy in Children

Nonspecific reactive hyperplasia
Infections: bacterial, fungal, parasitic (toxoplasmosis), viral (EBV, HSII, HIV, adenovirus), spirochetes (secondary syphilis), cat scratch fever
Postvaccination including postvaccinial lymphadenitis
Autoimmune: rheumatoid arthritis, systemic lupus erythematosus, serum sickness
Sarcoidosis
Drugs
Histiocytosis X
Reactive lymphohistiocytosis: XLP, lymphomatoid granulomatosis, sinus histiocytosis with massive lymphadenopathy
Angioimmunoblastic lymphadenopathy with dysproteinemia
Giant lymph node hyperplasia
Primary and metastatic malignancy
Mucocutaneous lymph node disease
Hyperthyroidism
Storage diseases: Niemann-Pick, Gaucher's
Beryllium exposure
Autoimmune hemolytic anemia

Lymphoproliferative disorders represent a heterogeneous array of diseases involving B-cell proliferations that range from reactive polyclonal hyperplasia to true monoclonal malignant lymphomas.

Lymphadenopathy in children is a common finding and usually represents a transient proliferative response to localized infection. Table 1 shows causes of lymphadenopathy in children (Siebel *et al.*, 1989), but consideration of infectious origin is beyond the scope of this discussion. Therefore conditions that can resemble lymphoproliferative disorders in pediatric patients will be discussed.

Among the granulomatous lesions with eosinophilia, angiolymphoid hyperplasia with eosinophilia (ALHE) or Kimura disease is a granulomatous disease in the dermis, soft tissue, and lymph node, and is characterized histologically by the presence of lymphoid follicles, vascular proliferation and infiltration of eosinophils (Qunibi *et al.*, 1989). This disease has a predilection site such as the head and neck (110 out of 118 cases; Olsen & Helwig, 1985). The duration of the lesion ranges from 3 weeks to 4 years, but some of them had a history of 5 years of more. The frequency of renal involvement with this disease is apparently high, although the pathogenesis of this association remains unknown (Yamada *et al.*, 1982). In a recent review of the literature, it was noted that 21 out of 175 patients (12%) had proteinuria. Thirteen of these had nephrotic syndrome. Although some patients develop proteinuria years before the mass becomes clinically apparent, the majority develop proteinuria either simultaneously or following the onset of the tumor by months or years. Histopathologic findings describe that most of the reported cases had membranous glomerulonephritis (Yamada *et al.*, 1982), and diffuse proliferative glomerulonephritis with IgE, IgG and complement deposition along the paramesangial area and capillary wall were reported. Also a mesangio-proliferative glomerulonephritis has been reported (Yamada *et al.*, 1982). This disease is responsive to steroid or radiation therapy. The prognosis of this disease is benign even though some patients showed local recurrence.

Among lymphadenopathies with renal involvement, giant lymph node hyperplasia or Castleman's disease is a benign lymphoproliferative disorder and refers to an accumulation of nonneoplastic lymphoid tissue interspersed with plasma cells and blood vessels. The most frequent site is the mediastinum, but lesions may also be found in the abdomen, pelvis, and cervical and axillary regions. Two histologic types exist: hyaline vascular and plasma cell. The hyaline vascular type is more common, solitary, and generally asymptomatic. In contrast, the plasma cell type makes up 10% to 20% of the lesions, involves multiple lymph nodes, and is usually associated with systemic symptoms. The latter is associated with a syndrome characterized by fever, anemia, hypergammaglobulinemia, growth retardation and bleeding tendency. These were resolved after surgical excision. Renal involvement has been reported, such as minimal change lesion, membranous nephropathy, interstitial nephritis, heavy chain proteinuria, monoclonal gammopathy, hematuria or renal failure. Altered renal function improved after tumor resection (Ruggieri *et al.*, 1990).

According to the protocol this patient had light chain nephropathy which was diagnosed at another hospital. This entity is idetified by deposits of monoclonal immunoglobulin light chains and continuous granular electron-dense material within tubular basement membranes in association with the glomerular basement membrane (Tubbs *et al.*, 1981). Most cases are in the fifth to seventh decades of life and usually presented with azotemia and features of glomerular rather than tubulointerstitial disease. Some patients show osteolytic bone lesions and others bone marrow plasmacytosis over 30% consistent with plasma cell myeloma.

One of the pathologic problems is the necrotizing granulomatous lesion. Wegener's granulomatosis (WG) is a distinct clinicopathologic entity that can be diagnosed clinically and confirmed by biopsy having the histologic evidence of a necrotizing granulomatous vasculitis in biopsies of the upper respiratory tract, nose or ears. But in practice, even when there are evident lesions, biopsies often show nothing but nonspecific necrosis even when the base of the lesion is included in the biopsy. For this reason diagnosis will sometimes be provided by blood tests, sometimes by repeated biopsy at a later date, sometimes by the evolution of the clinical picture. Although any organ may be af-

fected in WG, the classic clinical triad consists of intractable rhinitis and sinusitis frequently with epistaxis, nodular, and cavitary pulmonary lesions producing cough and hemoptysis, and hematuria eventually associated with renal insufficiency. This clinical triad correlates with the pathologic triad of necrotizing granulomas in the nose and paranasal sinuses, systemic vasculitis of small arteries and veins, most pronounced in the lungs, and focal necrotizing glomerulitis (Orlowski *et al.*, 1978). A limited form of the disease also presents no evidence of renal involvement and limited or absent systemic vasculitic lesions. WG is being reported with increasing frequency in adults but remains a rare entity in children and adolescents. It is more accurately thought of as a systemic disease with involvement of the joints, skin, and peripheral nerves, skeletal muscles, brain or eyes having been reported in addition to the classic triad (Orlowski *et al.*, 1978). In WG the lungs and upper respiratory tract are nearly always involved early, so the patients present with sinusitis, otitis media or chest infection. Lymphadenopathy is unusual or minor, and splenomegaly is rare.

This patient was presumed to have chronic necrotizing inflammation with fat necrosis from the flank mass. Relapsing nodular nonsuppurative panniculitis (Weber-Christian syndrome) is a rare disorder of unknown cause and probably does not represent a single disease (Schaller & Wedgood, 1987). Infecton, drug reaction (especially to bromides and iodides), abnormal fat metabolism, and hypersensitivity have all been suggested as etiologic factors. It occurs in association with several rheumatic tissue diseases, with pancreatic disease, and with corticosteroid withdrawal. This patient had a history of prednisone treatment. Histologically, there are foci of degeneration and inflammation in the subcutaneous fat. Clinically this disease is characterized by the appearance of crops of subcutaneous nodules in any part of the body: thighs, abdomen, breasts, and arms are the most frequently involved. The nodules vary in size from mm to several cm and may be painful, with redness and warmth of the overlying skin. Nodules regress in days to weeks, usually leaving a pigmented depression (Schaller & Wedgood, 1987), but this patient did not have such a clinical course.

This patient had lymphadenopathy and hyperglobulinemia. Sinus histiocytosis with massive lymphadenopathy is of unknown etiology and refers to a clinical disorder consisting of massive bilateral, painless cervical lymphadenopathy. All lymph node groups of the neck can be involved, as well as the axillary and inguinal regions. A progression is seen from the early stages, in which the lymph nodes are mobile and discrete, to the later stages, in which they adhere each other, resulting in a multinodular mass. Extranodal sites such as the skin, eyelid and orbit, salivary gland, bone, and respiratory tract have been reported. Fever, leukocytosis with neutrophilia, mild normochromic anemia, elevated erythrocyte sedimentation rate, and polyclonal hypergammaglobulinemia are commonly associated findings. The condition usually persists for 3 to 9 months. However, one case reportedly has lasted for 11 years. The course of the disease is not influenced by various treatments but usually resolves spontaneously. Evolution into amyloidosis or malignant lymphoma has been described in individual patients. Histologically the disorder shows distinctive features, including capsular and pericapsular fibrosis and dilatation of the sinuses filled with large granular or vacuolated histiocytes. Phagocytosis of the lymphocytes, plasma cells, and erythrocytes by sinus histiocytes is the most striking feature.

Among the lymphadenopathies with hyperglobulinemia, angioimmunoblatic lymphadenopathy with dysproteinemia can be considered. Angioimmunoblastic lymphadenopathy with dysproteinemia is a potentially fatal disease of unknown etiology, more frequently seen in adults but which has been reported in children as young as 5 years of age (Siebel *et al.*, 1989). In the American literature, this entity was referred to as immunoblastic lymphadenopathy (IBL) or angioimmunoblastic lymphadenopathy with dysproteinemia (AILD) (a term also adopted in France), while the Kiel lymphoma group called it lymphogranulomatosis X (LgX) (Knecht, 1989). In AILD, IBL, and LgX, the histopathologic criteria for diagnosis are identical in the main findings-obliteration of the lymph node architecture, vascular proliferation, and polymorphous cytology-but dif-

Table 2. Morphologic criteria for diagnosis of AILD, IBL, and LgX

Common findings
Diffuse obliteration of the lymph node architecture
Proliferation of small vessels (mainly postcapillary venules)
Polymorphous cytology
Absence of florid germinal centers
Differences
Immunoblasts and plasma cells are abundant in AILD and IBL; in LgX either immunoblasts, plasma cells, lymphocytes, or epithelioid cells may prevail.
In IBL, the lymph node is lymphocyte-depleted, and hypocellular
PAS-positive interstitial material is always present in IBL, but is not a condition in AILD and LgX.
Burned-out germinal centers may occur in AILD and LgX.

Table 3. Clinical findings at presentation in AILD

Clinical Findings	Patients (%)
Generalized lymphadenopathy	79
Localized lymphadenopathy	21
Hepatomegaly	73
Splenomegaly	63
Fever	72
Skin rash	44
Pruritus	48

Table 4. Serological Profile of AILD

Serological Profile	Patients (%)
Polyclonal hypergammaglobulinemia	70
Hypogammaglobulinemia	7
Monoclonal component	5
Normal gammaglobulins	23
Positive Coombs test	39

fer in some additional features (Table 2). Subtle morphological differences between AILD, IBL, and LgX have been recently reviewed (Frizzera *et al.*, 1989). However, the identical mode of clinical presentation, the propensity to develop malignant lymphomas, the susceptibility to infections, and often rapid death justify treatment of the histologically-defined entities of AILD, IBL, and LgX as a single entity. The onset of the disease is usually rapid, with patients developing signs and symptoms of a systemic disease within a few days or weeks. Generalized lymphadenopathy, hepatosplenomegaly, and fever, accompanied by weight loss and general malaise, are regularly seen (Table 3). Generalized or localized edema, ascites, pleural effusion, pulmonary infiltrates, or enlarged parotid glands are less frequent. At first presentation, localized lymphadenopathy tends to generalize within a few weeks, accompanied by skin manifestations if treatment is delayed. However, in some cases the onset is insidious, with anorexia and lymph node swelling occurring up to 2 years before diagnosis. Bone marrow infiltration by plasma cells and immunoblasts is detected by aspiration technique in 60 % of the cases and is of a polyclonal nature. Eosinophilia, increased left shift in myelopoiesis, and erythropoiesis are not infrequent. Focal fibroblastic and vascular proliferations, isomorphic to those of lymph nodes, are identified on 30% to 60% of trephine biopsies. Dysproteinemia is a regular and impressive feature in the analysis of blood plasma components (Table 4). Polyclonal hypergammaglobulinemia as high as 60 g/L, associated with circulating plasma cells and hypoalbuminemia, has been reported. Initial immunoglobulin levels may increase, remain unchanged, fluctuate, or diminish during progression of the disease. Occasionally, monoclonal components are identified in plasma or in urine at diagnosis or during the course of the disease. Lukes and Tindle (1975) were the first to observe the evolution of AILD into malignant lymphoma. Such a progression, with a poor prognosis, may occur both early in the course of the disease and after long-lasting (> 5 years) complete re-

Table 5. Malignant lymphoma and carcinoma at autopsy in AILD

Reference	No. of Patients	Malignant Proliferation (No.)
Ganesan *et al.*, (1987)	7	Immunoblastic lymphoma (1)
Lukes *et al.*, (1975)	12	Immunoblastic lymphomas (2)
Knecht *et al.*, (1985)	38	Immunoblastic lymphomas (3)
		Hodgkin's lymphomas (2)
		Carcinomas (4)

mission. Large clusters of immunoblasts, or clear cells (lymphoid cells with abundant pale cytoplasm), are generally interpreted as the first histological signs of malignancy, when lymphoma is diagnosed by biopsy. The overall malignant transformation rate in AILD is 18%, based on a study with a minimal follow-up of 5 years, including biopsy and necropsy results. Similar transformation rates are found in autopsy studies (Table 5), in which the diagnosis of lymphoma is less difficult. It has been shown that malignant proliferation usually involves multiple lymphatic sites and sometimes extralymphatic organs such as the kidneys, stomach, and lungs. Evolution into B-immunoblastic lymphoma (15-20%) (Lukes & Tindle, 1975; Knecht *et al.*, 1985; Ganesan *et al.*, 1987; Cavanna *et al.*, 1988), peripheral T-cell lymphoma (5 cases, Brice *et al.*, 1987), PDLL (2 out of 16 cases, Jootar *et al.*, 1987), and Hodgkin's disease (Yataganas *et al.*, 1977) has been proven. ALD is also associated with carcinoma (stomach, lung, pancreas, colon, Knecht *et al* 1985, Cavanna *et al.*, 1988). However, it is not malignant lymphoma, but rather overwhelming infections that account for most deaths in AILD.

Renal lesions in lymphoid malignancies are rare, with most lesions observed in association with Hodgkin's disease. In 2 large series of patients with Hodgkin's disease, only 0.4% had a minimal-change lesion, whereas 0.1% had amyloidosis. The nonHodgkin's lymphomas and leukemias comprise large and heterogeneous groups with equally diverse renal lesions. As in Hodgkin's disease, the most frequent lesion is minimal-change nephrotic syndrome. Also recognized are rare reports of renal disease associated with the atypical lymphoid proliferations of angioimmunoblastic lymphadenopathy, giant lymph node hyperplasia syndrome, and acquired immune deficiency syndrome. Broad generalizations regarding the pathogenesis of renal disease in these syndromes are difficult, partly due to the paucity and sporadic reporting of such cases. Mechanisms proposed to explain the renal pathologic findings include autologous nontumor antigens, tumor antigens, fetal antigen expression, immune complex deposition, viral antigens, and disordered T-cell function. A general characteristic of Hodgkin's patients is that the nephrotic syndrome usually occurs early in the course of the disease and may even constitute a presenting symptom. In contrast to the patients with Hodgkin's disease, there is little information about the relationship between onset of the glomerular disease and recognition of the other lymphoma. When documented, the nephrotic syndrome either precedes or occurs simultaneously with the lymphoma (Dabbs *et al.*, 1986).

Cutaneous lymphoma is a rare disease, and the incidence is 1-3.5% of all NHL in children (Grumayer *et al.*, 1988). Immunophenotyping of the lymphomatous infiltration disclosed T, pre-B, pre-pre-B, and non-T/non-B characteristics. I have seen a total of 10 cases of cutaneous lymphoma in this hospital during the last 10 years. Two of them were confirmed to have had a T-cell origin, but an immunologic study has not been done in the remaining cases.

In summary, this patient is presumed to have suffered from AILD since February. 1988 when a lymph node biopsy revealed necrotizng and granulomatous inflammation with heavy eosinophilia. Perhaps he might already have had the same illness in 1985. When he had proteinuria in 1989, renal disease might be associated with AILD or malignant lymphoma. In July 1990, chronic ne-

crotizing inflammation with fat necrosis of the flank mass might have been due to panniculitis or skin involvement of malignant lymphoma, and the final skin lesions can be explained by skin involvement of malignant lymphoma probably of T-cell origin. I thought the cause of death was DIC associated with sepsis.

Dr. Ahn's Diagnosis:

1. Angioimmunoblastic lymphadenopathy, transformed into nonHodgkin's lymphoma (skin &/or internal organ)
2. Disseminated intravascular coagulopathy and sepsis

Pathology Findings

Dr. Chi: This was a very unusual case that bothered us for a long time. The first lymph node biopsy done in 1988 at Children's Hospital showed both necrotizing granulomas and heavy eosinophilia. It was definitely atypical and difficult to categorize into a certain specific entity. However, in retrospect, angioimmunoblastic lymphadenopathy is still hardly considered. Subsequent subcutaneous biopsies done at Korea University Hospital also showed necrotizing panniculitis but with no definite malignant cells (Fig. 1). However, the last skin biopsy done at SNU Hospital did show definite neoplastic lymphoid cells particularly around the dermal blood vessels, as well as in the subcutaneous panniculus (Fig. 2). These large atypical lymphoid cells often had convoluted nuclei with prominent nucleoli and a moderate amount of cytoplasm. These cells were stained positive for Pan-T marker and negative for B-cell and macrophage markers (Fig. 3). Therefore, at the last moment, i.e., only a few days before the patient's demise, one could be able to coin this case as a T-cell lymphoma.

Postmortem findings confirmed diffuse lymphoma cell infiltration in the liver, spleen, lymph nodes, kidneys, and lungs (Fig. 4). The involvement pattern was diffuse, but the extent varied considerably by areas and by tissues. It was predominently portal in the liver, associated with fatty change (Fig. 5). The kidneys and lungs showed patchy interstitial infiltrations around the vessels particularly in the latter. The skin and subcutis in many areas in-

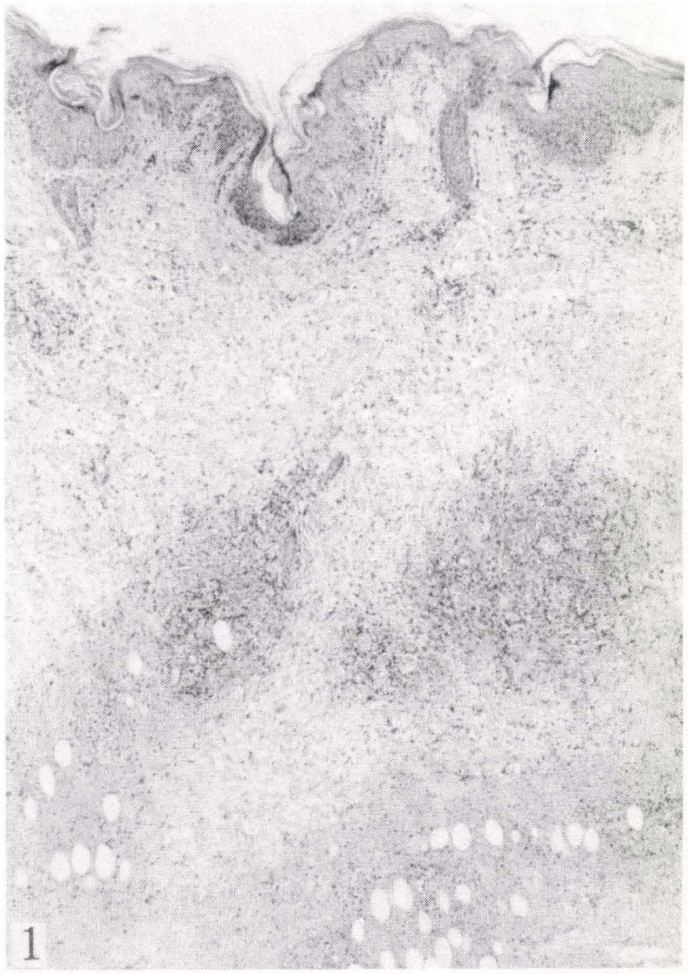

Fig. 1. Skin biopsy at Korea University Hospital shows necrotizing inflammation involving the entire thickness of the dermis and subcutaneous fat. H&E X100

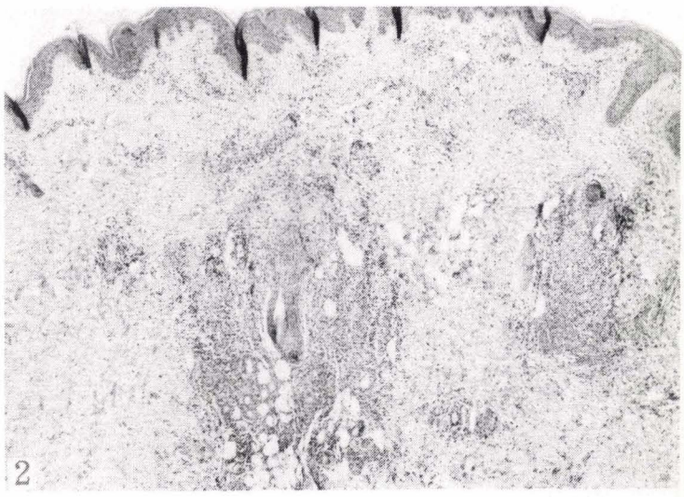

Fig. 2. Skin biopsy at Seoul National University Hospital done several days before death. Perivascular atypical lymphoid cell infiltration is apparent in the dermis. H&E X100

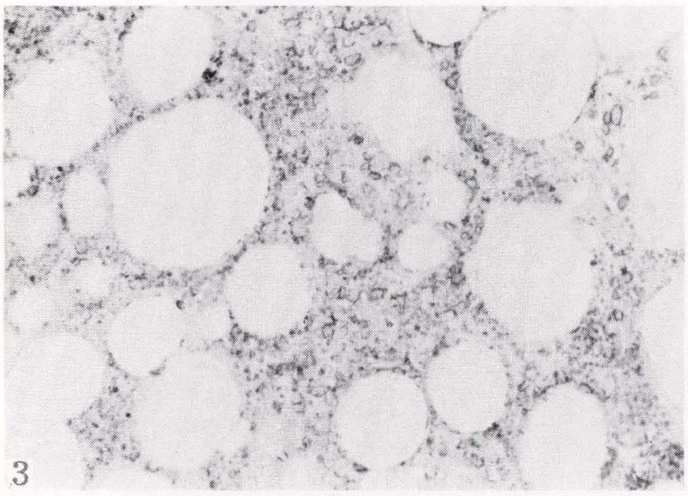

Fig. 3. Subcutaneous fat tissue is actively infiltrated by lymphomatous cells that show positive immunostain of pan-T marker. PAP X250

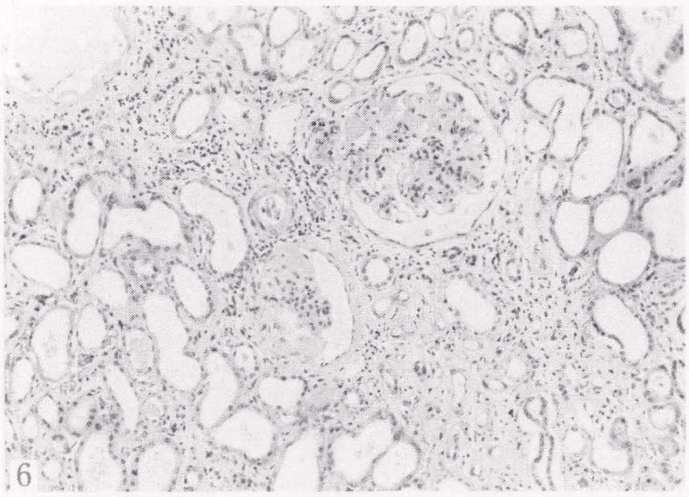

Fig. 6. Kidney tissue showing chronic pyelonephritis and glomerular hyalinization. H&E X100

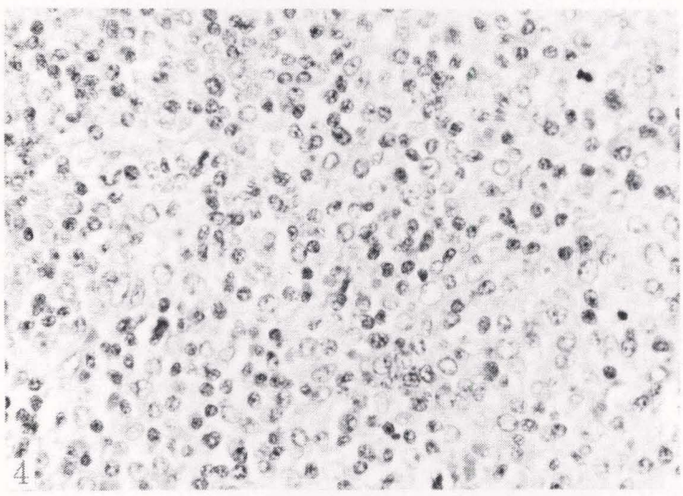

Fig. 4. Malignant lymphoma involving the lymph node. The cells consist of large cells with clear cytoplasm and smaller dark cells. H&E X250

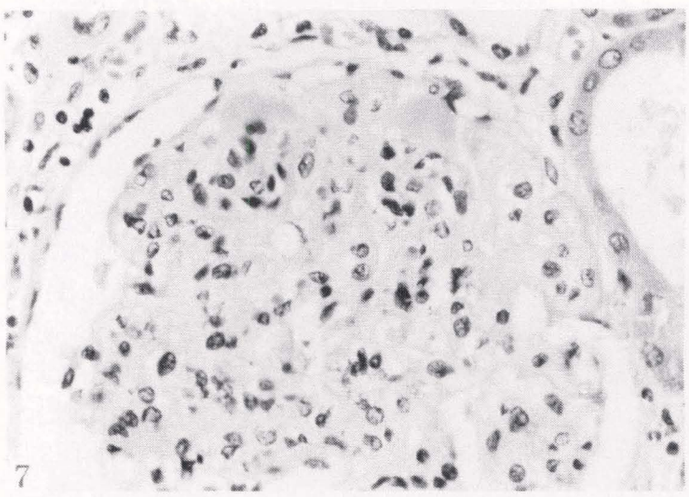

Fig. 7. A glomerulus shows globular fibrin deposits. There was hyaline vasculopathy in the other vessels. H&E X250

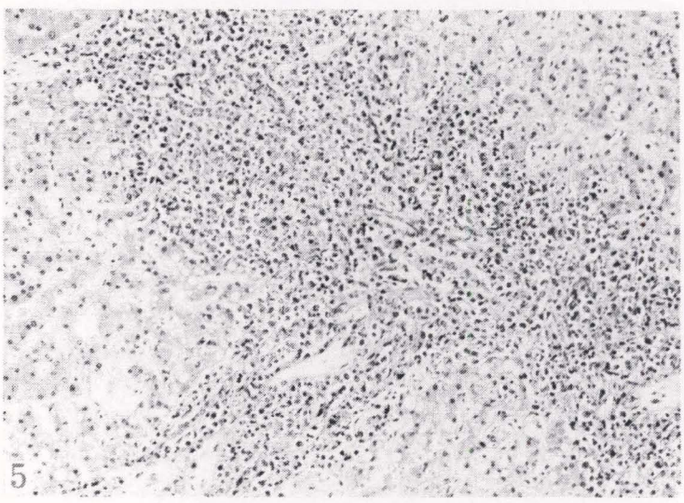

Fig. 5. Photomicrogrph of the liver, showing portal lymphoid cell infiltration. H&E X100

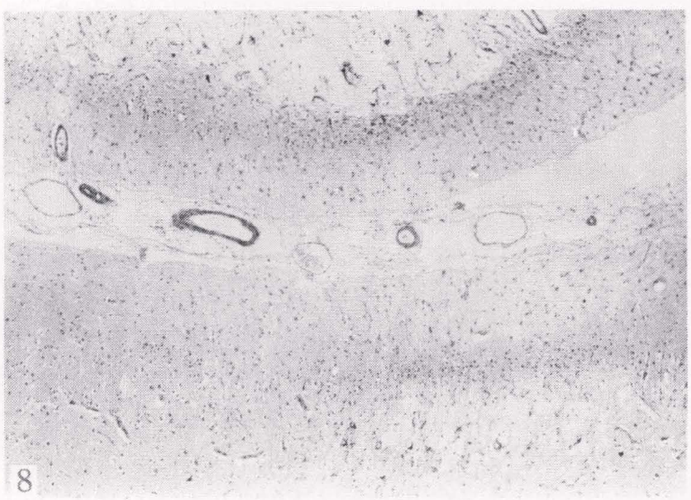

Fig. 8. A brain section shows old ischemic infarction with sparing of molecular layer. H&E X100

cluding the ankle were also involved. The lymphoma cells were accompanied frequently with tingible body macrophages, but no granuloma formation, necrosis or eosinophilic infiltration were demonstrated in any organ. The bone marrow was free of lymphoma cells, but macrophages with engulfed red cells were often seen in the marrow.

The next interesting finding in this case was the peculiar hyaline material deposit in the vessel wall associated with nuclear debris (Fig. 6). It was prominent in the kidney, and the vasculitis was seen in the lungs. This apparent vasculopathy probably accounted for multiple small infarcts, old and recent, seen in the pancreas, spleen, pituitary, and brain (Fig. 7). The brain was the site of numerous old microinfarctions throughout the cortex and basal ganglia. There were large areas of softening in the hemispheres and basal ganglia representing old infarcts. However, neoplastic cell infiltration was not seen in the brain. No evidence of herpes encephalitis was present in the brain, spinal cord or dorsal root ganglia. The kidney showed a considerable tubulointerstitial disease representing chronic pyelonephritis. A more significant finding was a hyaline vascular change of the glomeruli, resembling a nodular glomerulosclerosis of diabetes. There was no immunoglobulin deposit in the glomerulus. We presume that these findings are probably related to the lymphoproliferative lesion that this patient had suffered from.

The terminal event was disseminated intravascular coagulation associated with sepsis, although no organism was cultured in the postmortem blood and lung samples.

Final Diagnosis:

1. T-cell lymphoma involving the lymph nodes, skin, subcutis fat, liver, spleen, kidneys and lungs
2. Old vascular occlusive lesion and hyaline vasculopathy, with gross and microscopic infarcts, multiple, brain
3. Necrotizing hyaline glomerulonephritis
4. Chronic pyelonephritis
5. Splenic infarct, recent
6. Disseminated intravascular coagulopathy

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