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Subacute Necrotizing Lymphadenitis
– A Collective Clinicopathological and Immunohistochemical Study –

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Subacute necrotizing lymphadenitis (SNL) is a well documented and unique clinicopathologic entity, although its etiology and pathogenesis have not been clearly established. Microscopically, cortical and paracortical necrotizing lesions with karyorrhexis, abundant nuclear debris and infilfration of large mononuclear cells are characteristic. This study analyzed the common clinical and pathological features of 118 patients with SNL and the nature of the mononuclear cells. Patients were generally young women and revealed cervical lymphadenopathy with tenderness, fever, leukopenia and elevation of the erythrocyte sedimentation rate. Features of the adjacent uninvolved area in the lymph node included a starry sky pattern, follicle centers, sinus histiocytosis or aggregation of foamy histicoytes. There was an inverse relationship between the extent of necrosis and of histocytic infiltration but not between the extent of necrosis and the duration from the onset of symptoms to the diagnosis. Immunohistochemically the infiltrated mononuclear cells of the affected foci were T lymphocytes and histiocytes. The clinical, histological and immunohistochemical features suggest that SNL represents a hypersensitivity reaction to certain infectious agent without forming granuloma.

Key Words : Subacute necrotizing lymphadenitis, lymph node, histiocytic necrotizing lymphadenitis, hypersensitivity, immunohistochemistry

Subacute necrotizing lymphadenitis (SNL) was first documented in 1972 by Kikuchi and independently by Fujimoto *et al.* and has been frequently described in Japanese literature (Kikuchi *et al.* 1977; Michaeleck and Henzan 1983; Kikuchi 1978) and later in other countries including Korea (Suseelan *et al.* 1981; Pileri *et al.* 1982; Feller *et al.* 1983; Turner *et al.*1983; Ali and Horton 1985; Evans *et al.* 1985; Koh *et al.* 1985; Carbone *et al.* 1986; Park *et al.* 1987; Unger *et al.* 1987; Hahn *et al.* 1989). Clinically SNL has a remarkable predilection for young women and is usually found in the cervical lymph node, although rarely is there a generalized lymphadenopathy. Because of the characteristic histologic finding which consists of proliferation of lymphoreticuar cells, karyorrhexis and variable degree of necrosis, this self-limiting reactive process must be differentiated from malignant lymphoma, systemic lupus erythematosus (SLE) and viral lymphadenopathy (Takahiro *et al.* 1981; Pileri *et al.* 1982; Turner *et al.* 1983; Ali and Horton 1985; Dorfman and Berry 1988).

Although Ebstein-Barr virus or Toxoplasma have been proposed as the causative agent (Kikuchi *et al.* 1977; Kikuchi 1978; Scheibani *et al;* 1984), some investigators thought that it was a kind of hyperimmune lymphadenitis induced by sensitized T cells (Feller *et al.* 1983; Turner *et al.* 1983; Ali and Horton 1985; Carbone *et al.* 1986; Kikuchi *et al.* 1986; Rivano *et al.* 1987; Unger *et al.* 1987; Dorfman and Berry 1988). But the nature of the proliferating mononuclear cells has not been clarified.

The purpose of this study is to investigate the common clinicopathologic features of 118 patients with SNL and the immunohistochemical nature of

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proliferating mononuclear cells.

MATERIAL AND METHODS

One hundred and eighteen patients with SNL were selected from the records of the Department of Pathology, Yonsei University, Wonju College of Medicine and Youngdong Severance Hospital during the period from January 1978 to December 1988. Clinical records and laboratory findings including the age, sex and chief complanits of patients, the duration of symptoms, the site and size of lymphadenopathy, laboratory findings such as white blood cell count and serologic data and the follow-up data were reviewed. Tissue for light microscopy was processed by preparing paraffin sections from 10% formalin and staining with hematoxylin and eosin. The following histologic features were evaluted; extent of lymph node involvement and necrosis, large mononuclear cell proliferation, number of mitotic figures counted in 10 random high-power fields (HPF) of pathologic area, presence of karyorrhectic nuclear debris, neutrophis and plasma cells, vascular changes, and pattern of remaining lymphoid tissue. Formalin-fixed, paraffin-embedded tissues were stained for the various antigens(Table 1) by the avidin-biotin-peroxidase complex method. Appropriate positive and negative controls were included. The statistical significance between various factors was tested by X^2-test using SPSS/PC + program.

RESULTS

Age and sex

The age of the patients varied from 1 month to 63 years of age with a mean of 27.7 years, the majority (41.5%) belonging to the third decade. Ninety two patients were female and twenty six male (M:F =1:3.5) (Table 2).

Symptoms and signs

Common chief complaints were neck mass (98.2 %) with or without tenderness (35.4%), fever (26.4 %) and myalgia (11.8%) (Table 3). The duration of symptoms and signs prior to diagnosis ranged from 5 days to 6 months with a mean of 38.5 days, the majority (72.1%) occurring within 4 weeks (Table

Table 2, Distribution of age and sex of patients with SNL

Age (years)	Male	Female	Total
~9	0	2	2 (1.7%)
10~19	4	14	18 (15.3%)
20~29	7	42	49 (41.5%)
30~39	12	25	37 (31.4%)
40~49	1	7	8 (6.8%)
50~59	2	1	3 (2.5%)
60~	0	1	1 (0.8%)
Total	26	92	118 (100.0%)

Mean age: 27.7 years
Range of age: 1 moth~63 years, M:F=1:3.5

Table 3. Chief complaints of patients with SNL

Symptoms	No. of cases (%) (n = *110*)
Neck mass	108 (98.2)
Fever	29 (26.4)
Tenderness	39 (35.4)
Myalgia	13 (11.8)
Sore throat	7 (6.3)
Chill	5 (4.5)
Night sweat	3 (2.7)
Weight loss	2 (1.8)
Easy fatigability	2 (1.8)

Table 1. Antibodies using immunohistochemical stain

Reagent	Major specificity	Source
Lysozyme	Macrophages and granulocytes	Dako patts. Co., Inc. (USA)
S-100 protein	Interdigitating reticular cells	Dako patts. Co., Inc. (USA)
Alpha-1-antichymotrypsin	Macrophages and granulocytes	Dako patts. Co., Inc. (USA)
MT 1	Tcells, myeloid cells and macrophages	Biotest (U. K.)
MB 2	B cells and some T cells	Biotest (U. K.)

4). On physical examination, all cases showed cervical lymphadenopathy but neither significant hepatomegaly, splenomegaly nor abdominal lymphadenopathy. The clinical diagnosis was lymphadenopathy in 97 cases, tuberculous lymphadenitis in 12, malignant lymphoma in 2, and fever of unknown origin in the remaining 7 cases.

Distribution of anatomical site and season of occurrence

One hundred eight of the 118 patients had lymphadenopathy confined to the cervical lymph node. Axillary, subclavicular and inguinal lymph nodes were occasionally involved. Sixty nine patients presented with multiple lymphadenopathy but only 6 patients had lymphadenopathy at more than one anatomical site. The size of involved nodes varied, but the majority (89.7%) were less than 2 cm in the greatest diameter (Table 5).

Seasonal distribution of occurrence was not remarkable. But the disease developed more often in January and June (Fig. 1).

Table 4. Symptom duration of patients with SNL

Duration	No. of cases (%) (n = 64)
~1 wk	15 (22.1)
1~2 wks	4 (5.9)
2~3 wks	11 (16.2)
3~4 wks	19 (27.9)
1~2 m	10 (27.9)
2~3 m	4 (5.9)
3~4 m	3 (4.4)
5~6 m	2 (2.9)

Mean; 38.5 days
wk: week, m: months

Table 5. Features of lymphadenopathy in SNL

Lymphadenopathy	No. of cases (%)
Site (n=118)	
Cervical	108 (91.5)
Inguinal	4 (3.4)
Axillary	2 (1.7)
Mesenteric	2 (1.7)
Submandibular	2 (1.7)
Multiple	6 (5.1)
Size (N=97)	
~1 cm	43 (44.3)
1~2 cm	44 (45.4)
2~3 cm	7 (7.2)
3~ cm	3 (3.1)

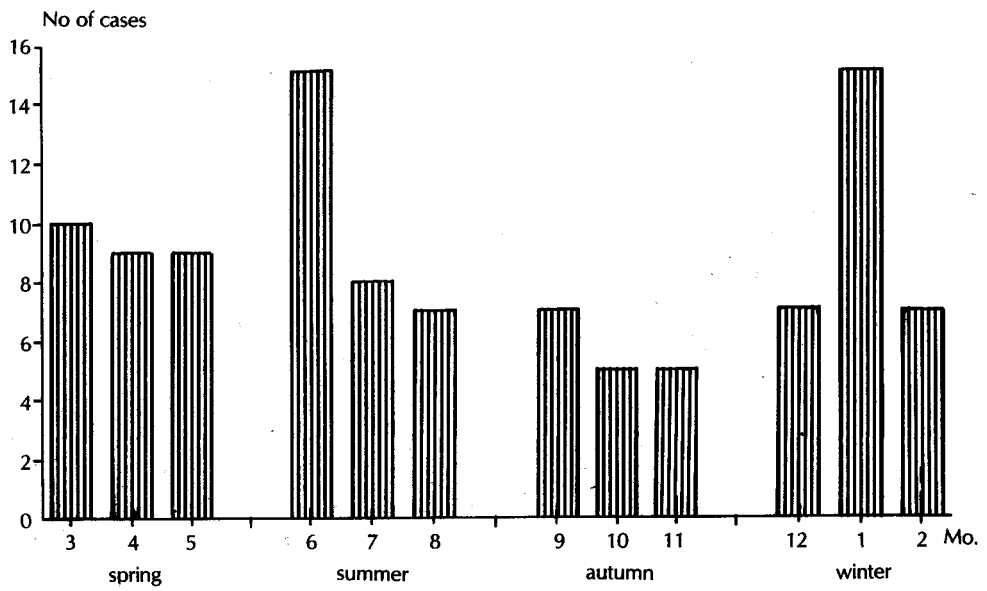

Fig. 1. *Seasonal distribution of SNL.*

Laboratory findings

Thirty four cases (54.8%) had leukopenia (< 4,000/ mm³), and relative lymphocytosis (>60%) was noted in 15 cases (28.3%). The erythrocyte sedimentation rate (ESR) was elevated in 12 out of 20 cases. Among cases where serologic tests were performed, C-reactive protein was positive in 3 out of 23 cases, but Widal test, various virus studies including Herpes simplex virus, cytomegalovirus and Varicella-zoster virus, and serology for Mycoplasma were all negative. Of the patients in whom studies of ANA and rheumatoid factor were performed, all cases, except one which was diagnosed as having SLE by the renal biopsy, were negative (Table 6). The T cell subset of blood in 4 cases was also normal.

Treatment and clinical course

Fifty seven patients received no therapy. Twenty patients were treated with antibiotics, four with steroid agent and five with antituberculosis medication. A follow-up study was possible in 10 cases, and 5 of those complained of fever after medication.

Pathological findings

The histological findings are shown in Table 7.

Table 6. Laboratory findings in patients with SNL

Laboratory finding		No. of cases (%)
WBC		
Total count(x10³/mm³)	<4	34 (54.8)
(n=62)	4~10	28 (45.2)
	10<	0 (0.0)
Granulocyte(%)	<40	14 (26.4)
(n=53)	40~75	38 (71.7)
	75<	1 (1.8)
Lymphocyte(%)	<20	8 (15.1)
(n=53)	20~60	30 (56.7)
	60<	15 (28.3)
ESR[1] (n=20)	increase	12 (60.0)
CRP[2] (n=23)	positive	3 (13.0)
ANA[3] (n=23)	positive	1 (7.7)
Rheumatoid factor (n=10)	positive	1 (10.0)

1) ESR=erythrocyte sedimentation rate increase; >10 mm/hr(male), 20> mm/hr(female).
2) CRP=c-reactive protein. Positive;>6 mg/dl.
3) ANA=antinuclear antibody. Positive; >1: 20.

The common feature was mononuclear cell proliferation with partial or complete effacement of the normal nodal architecture. The pathologic areas were pale and usually located in the cortex or paracortex in a nodular pattern (Fig. 2). Necrosis was usually present either focal or confluent, and

Table 7. Histologic features of SNL

Histologic features	No. of cases (%) (n=118)
Extent of lymph node involvement	
complete effacement	20 (16.9)
partial effacement	84 (71.2)
focal effacement	14 (11.9)
Extent of necrosis	
confluent	23 (19.5)
focal	61 (51.7)
none	34 (28.8)
Large mononuclear cell proliferation	
+	19 (16.1)
++	54 (45.8)
+++	45 (38.1)
Pattern of lesion	
diffuse	19 (16.1)
nodular	99 (83.9)
Karyorrhectic nuclear debris	118 (100.0)
Polymorphous leukocytes	0 (0.0)
Plasma cells	25 (21.2)
Vessel change	
large mononuclear cells within the vessel	26 (22.0)
angiocentricity of large mononuclear cells	8 (6.8)
Mitotic figures	
0~5/10HPF	99 (83.9)
>5/10HPF	19 (16.1)
Perinodal extension of large mononuclear cells	12 (10.2)
Perinodal extension of karyorrhetic particles	0 (0.0)
Uninvolved lymphoid structure	
follicle center	68 (57.6)
sinus histiocytosis	30 (25.4)
mottled pattern	104 (88.1)
aggregates of foamy histiocyte	16 (13.6)

+: Large mononuclear cells constitute less than 50% of the invovlved area.
++: Large mononuclear cells constitute more than 50% but not total of the involved area
+++: Large mononuclear cells constitute almostly total of the involved area.

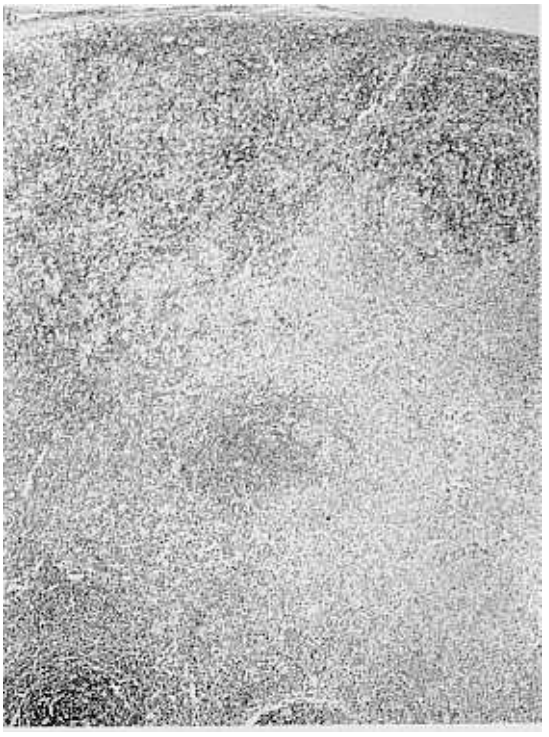

Fig. 2. The lesion is located in the cortex and paracortex. The remaining follicles and starry-sky pattern are seen in the intervening area. (H&E ×40)

karyorrhectic nuclear debris was always present (Fig. 3) at the center of the lesion. Proliferating cell components were dominated by histiocytes, macrophages phagocytosing nuclear debris, reactive immunoblasts, atypical lymphoid cells and occasional foamy histiocytes. A consistent histologic feature of these lymph nodes was the absence of granulocytes and the paucity of plasma cells. The capsule was intact in the majority (89.8%), but perinodal extension of the large mononuclear cells was often present. No karyorrhetic particles were seen outside the lymph node. Mitotic figures ranged 2~15 per 10 HPF. The intervening regions usually showed reactive follicles, a mottled or starry sky pattern and occasionally sinus histiocytosis and aggregation of foamy histiocytes. The degree of necrosis, the proliferation of large mononuclear cells and the duration of symptoms were shown in Table 8 and 9. There was a relatively inverse relationship between the extent of necrosis and of monuclear histiocytic infiltration ($p<0.001$) but this was not true when comparing the extent of necrosis and duration of symptoms ($p<0.1$).

Immunohistochemical findings

About 60% of nucleated cells of the affected area within the lymph nodes were positive for MT 1 (T cell), and the remaining cells stained positive for

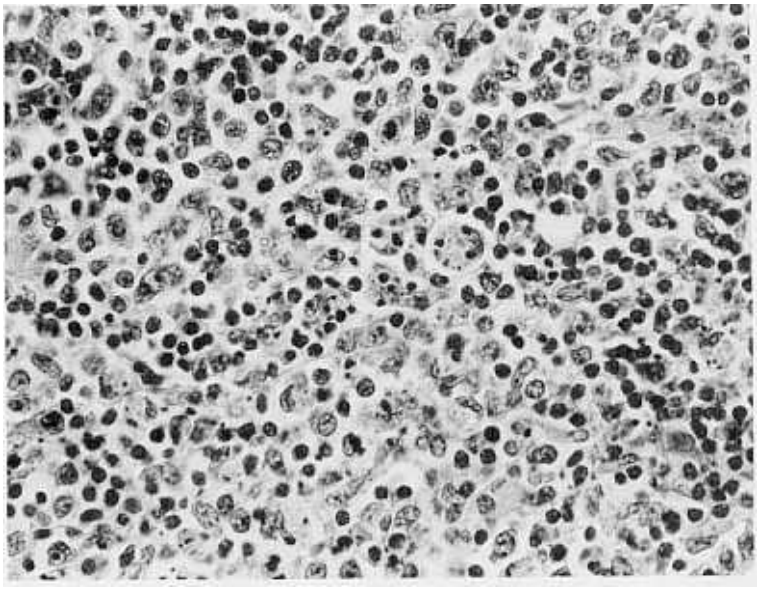

Fig. 3. The lesion consists of proliferation of large mononuclear cells, karyorrhexis and nuclear debris in some phagocytes. (H &E ×400)

Table 8. Degree of necrosis and large mononuclear cell infiltration in SNL (No. of cases)

Necrosis	Histiocytic infiltration*			Total
	+	++	+++	
None	0	0	34	34
Focal	0	50	11	61
Confluent	19	4	0	23
Total	19	54	45	118

+: Large mononuclear cells constitute less than 50% of the involved area

++: Large mononclear cells constitute more than 50 % but not total of the involved area

+++: Large mononuclear cells constitute total of the involved area

*$p<0.001$

lysozyme and alpha-1-antichymotrypsin (monocyte/macrophage) (Table 10) (Fig. 4). Cells positive for S-100 protein were rarely encountered in the affected area (Fig. 5). Staining with the antibody to B cell marker (MB 2) was negative in all cases.

Table 10. Quantitative analysis of proliferating cells in affected areas of SNL. (N=40)

Markers	Positivity
MT 1	60%
MB 2	0%
Lysozyme	45%
Alpha-1-antichymotrypsin	40%
S-100 protein	3%

Table 9. Degree of necrosis and duration in SNL

Necrosis	Duration from onset of symptoms to diagnosis(wks)*						Total No. of cases
	<1	1~2	2~3	3~4	4~8	8<	
None	3	2	1	2	4	2	14
Focal	10	4	10	13	3	2	42
Confluent	2	0	3	4	3	0	12
	15	6	14		10		68

*$p>0.1$

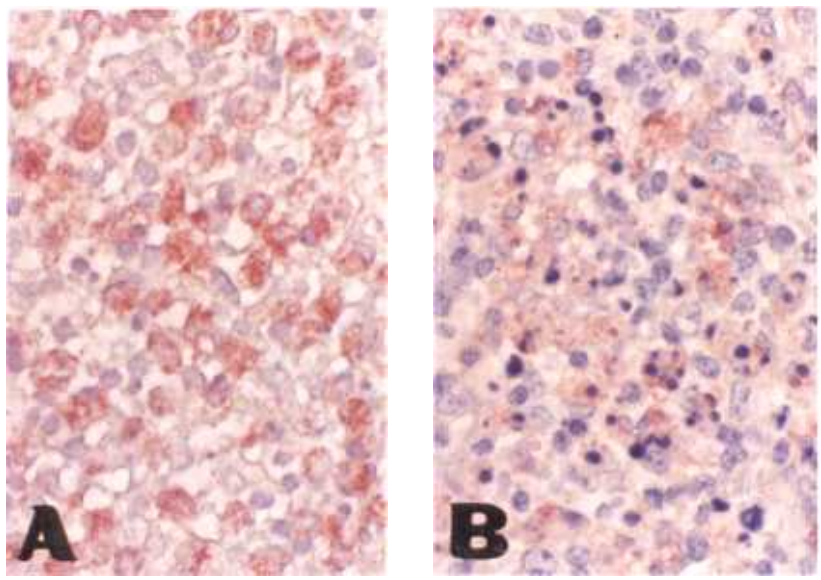

Fig. 4. Immunohistochemical stains for monocyte/machrophage markers. A; alpha-1 antichymotrypsin, B; lysozyme. (methylene blue counterstain ×400)

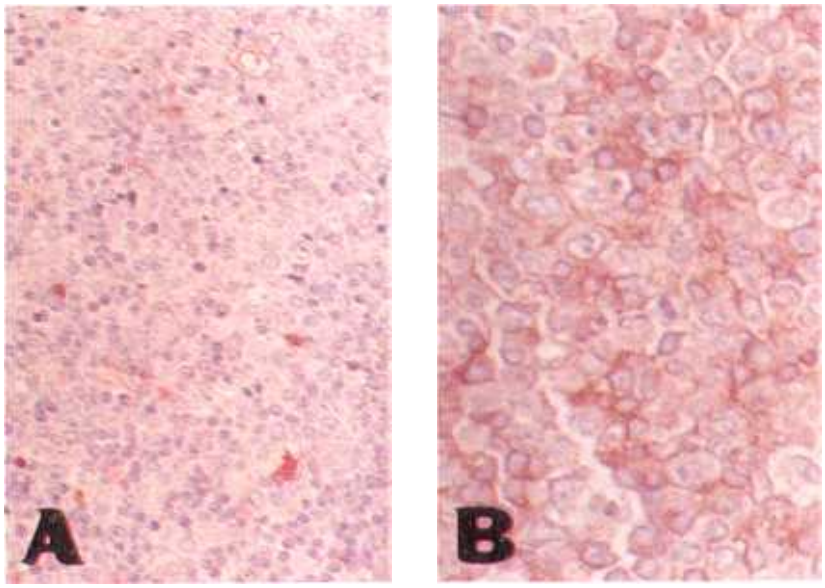

Fig. 5. *Immunohistochemical stains for MT 1, × 200 (A), and S-100 protein, × 400 (B).*

DISCUSSION

After the original description of SNL by Kikuchi and independently by Fujimoto *et al* in 1972, this di-sease entity has been reported under various designations such as necrotizing lymphadenitis (Wakasa *et al.* 1975; Kikuchi *et al.* 1977; Wakasa *et al.* 1978), necrotizing-histiocytic lymphadenitis (Kikuchi 1978) phagocytic necrotizing lymphadenitis(Kikuchi and Uryu 1976) and pseudolymphomatous hyperplasia in the lymph node (Michaeleck 1977; Michaeleck and Henzan 1983).

Clinically, SNL occurs mainly in young women and involves most commonly the neck; it rarely appears as generalized lymphadenopaty (Pileri *et al.* 1982). Fever and tenderness of the nodes are frequently accompanied (Suseelan *et al.* 1981; Turner *et al.* 1983; Ali and Horton 1985; Evans *et al.* 1985). The present study also showed a remarkable predilection for women (86%), most belonging to the third decade (41.5%). Although cervical lymph nodes were affected in most cases, six patients presented with generalized lymphadenopathy.

As shown in this study and by others, SNL frequently accompanies fever of the affected area with tenderness, elevation of ESR and CRP (Turner *et al.* 1983; Ali and Horton 1985) and has a relatively short clinical course, suggesting that it is caused by a certain infectious agent. Its tendency to occur in particular seasons may also indicated the infectious nature (Michaeleck and Henzan 1983).

Because of the uniformly negative cultures and the resolution of the illness without antibiotic treatment in more than two thirds of cases, SNL seems not to be a bacterial disease. Several authors found that some patients with SNL had high antibody titers to Toxoplasma or Ebstein-Barr virus (Kikuchi *et al.* 1977; Takada *et al.* 1980; Scheibani *et al.* 1984), but others failed to support this viral or protozoal etiology (Takai *et al.* 1975; Pileri *et al.* 1982).

The histologic features we have observed are similar to the previous descriptions (Fujimoto *et al.* 1972; Kikuchi 1972; Michaeleck 1977; Kikuchi 1978; Wakasa *et al.* 1978; Pileri *et al.* 1982; Feller *et al.* 1983; Michaeleck and Henzan 1983; Tuner *et al.* 1983; Ali and Horton 1985; Carbone *et al.* 1986; Kikuchi *et al.* 1986; Dorfman and Berry 1988). The characteristic lesion is usually present in the cortex or paracortical areas and consists of wide areas of patchy or confluent necrosis with numerous karyorrhectic nuclear dusts and the surrounding infiltration of lymphoreticular cells. Absence of neutrophils together with a paucity of plasma cells are characterisic. The degree of necrosis and the proliferation of large mononuclear cells varies. As the amount of karyorrhectic particles increases, the de-

gree of proliferation of the large mononuclear cells was low. An invariable finding is the prominent mottling by histiocytes or transformed lymphoid cells in the intervening areas. Mitotic figures are frequent, but are not seen more than 15 per 10 HPFs.

The differential diagnosis of SNL includes malignant lymphoma, microabscess-forming histiocytic lymphadenitis, systemic lupus erythematosus(SLE) and infectious mononucleosis(Wakasa and Asano 1980; Takahiro *et al.* 1981; Pileri *et al.* 1982; Turner *et al.* 1983; Evans *et al.* 1985; Dorfman and Berry 1988). The proliferation of large mononuclear cells and the increase of mitotic figures can lead misdiagnosis to malignant lymphoma(Turner *et al.* 1983; Dorfman 1987; Rivano *et al.* 1987; Dorfman and Berry 1988; Chamulak *et al.* 1990). However, in contrast to malignant lymphoma, SNL reveals polymorphous proliferation of large mononuclear cells with extracellular nuclear dusts and phagocytic macrophages, moderate numbers of mitotic figures and a mottling pattern in the nonpathologic area (Chamulak *et al.* 1990). In the histiocytic lymphadenitis forming microabscess such as cat scratch disease, Yersinia lymphadenitis and lymphogranuloma venereum, there are numerous polymorphonuclear leukoeytes located in the necrotic center which is surrounded by palisaded histiocytes, imparting the appearance of granulomatous inflammation(Wear et al. 1982). Lymphadenopathy arising in patients with SLE is characterized by numerous plasma cells and the irregular globules of deeply basophilic material (hematoxylin bodies) in the paracortex and on the vessel walls (Dorfman and Berry 1988). But in some cases of SLE, it is difficult to distinguish from SNL, because the hematoxylin body is not always present in SLE. In addition to the profound immunoblastic reaction, infectious mononucleosis shows focal necrosis, karyorrhexis, many plasma cells, polymorphonuclear leukocytes and reactive follicular hyperplasia. Toxoplasmosis shows florid follicular hyperplasia and clusters of epithelioid cells without necrosis, which are not found in SNL.

Imamura *et al.* (1982) identified tubuloreticular structures in the cytoplasm of stimulated lymphocytes and histiocytes of SNL, which had been reported in SLE, SLE-related autoimmune disease, some viral infections and neoplasias, suggesting a hyperimmune reaction. Some authors suggest that SNL could be a self-limited SLE-like autoimmune condition because there are some cases initially thought to be SNL, but developing SLE in the follow-up study (Dorfman 1987; Dorfman and Berry 1988). Recent immunohistochemical studies revealed that the majority of cells within the necrotizing foci reacted to the markers of T cells and histiocytes, particularly to that of T cells. Helper/inducer T cells are predominant within 2 weeks and suppressor/cytotoxic T cells appear in greater numbers after 1 month (Rivano *et al.* 1987). Granulocytopaenia, the absence of neutrophils and the appearance of abundant nuclear debris in the affected foci might be related to some lymphokines and the macrophage migration inhibition factor released from the proliferating T cells. Therefore histologic features of SNL seem to be related to delayed-type hypersensitivity reaction as well as to a defense mechansim against some infectious agents. The results of the present immunohistochemical study also showed that the proliferated mononuclear cells had markers of MT 1, alpha-1-antichymotrypsin and lysozyme. In the early lesion, cells are positive for MT 1; in the enriched lesion, more proportion of cells are positive for lysozyme and alph-1-antichymotrypsin in the areas of karyorrhexis and necrosis. In conclusion, the clinical, histological and immunological data suggest that SNL seems to be related to a delayed-type hypersensitivity against certain infectious agents.

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