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Suspected Case of Combined Immunodeficiency

—— Autopsy Case ——

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An autopsy case of combined immunodeficiency of 3 year 11 months old boy is presented. He had stomatitis, chronic protracted diarrhea, fever, growth retardation which are characteristic symptomes in severe combined immunodeficiency (SCID). IgA was low and IgG and IgM decreased towards the terminal stage. SK-SD skin reaction was negative and DNCB sensitized skin reaction was negative indicating the lack of delayed type of hypersensitivity. Compared to the typical case of primary immunodeficiency, the degree of abnormality of humoral and cellular immune functions is milder in this case. Autopsy revealed the remaining lymph nodes and histologically formation of lymphoid follicles was not distinct and lymphocyte depletion in thymic-dependent area was seen. These lesions varied in degrees depending on the location within the lymph nodes. Thymus weighed 2.2g and had Hassal's corpuscles with calcification. These findings are somewhat different from the typical case of primary immunodeficiency. We consider this case as suspected case or borderline case of combined immunodeficiency by correlation of clinical and histopathological aspect. Generalized cytomegalic inclusion disease is this direct cause of death.

INTRODUCTION

With the remarkable advances of immunologic function tests in recent years, various types of immunodeficiency have been reported and the etiology and clinical picture are gradually being clarified[3)4)13)-16)18)-20)27)28)].

Nevertheless, it has been pointed[8)27)28)]out that there are some patients who can not be classified into any type of immunodeficiency so far described[9)11)12)18)23)27)28)] but appear to have a type of immunodeficiency. Compared to the typical immunodeficiencies so far described, the degree of abnormality of immune function is milder in these pa-

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tients, making it difficult to classify these patients in the same manner as the typical immunodeficient patient. Consequently, it is expected to attemt the sufficient clinicopathological analysis of immunodeficiency, so that the patients with these diseases can be included.

From this point of view, an autopsy case of 3 year 11 month old boy with fever of unknown etiology and diarrhea which proved refractory to the treatment is presented. Clinically, a diagnosis of severe combined immunodeficiency (subsequently abbreviaed SCID) was suspected. A few questions are raised by comparison with typical cases of immunodeficiency previously reported.

CASE REPORT

A 3 year 11 month old boy presented with a chief complaint of fever and diarrhea. Family history was unremarkable without history of susceptibility to infection. He was born after an uneventful pregnancy on the 40th week of gestation. Body weight was low, 2100 g, at birth. Growth and development during the first year was uneventful without history of lactose intolerance. At about the age of 1 year, stomatitis developed and gradually progressed throughout the entire course without improvement, with the eventual development of erosion and ulceration. At about the age of 2 years and 8 months, fever developed, occasionally as high as 38−39°C, and he was admitted to the Department of Pediatrics of the Nagasaki University Hospital for further evaluation.

On admission, the patient was noted to have poor physical development (82 cm in length, 8.3 Kg in weight) and anemia with a pancytopenia. Fanconi's anemia was suspected but neither chromosomal abnormalities nor abnormal bone marrow findings were noted. Therefore, Fanconi's anemia was ruled out. Fever was of an intermittent type, persisting despite the use of multiple antibiotics. At the age of 2 years and 11 months, watery diarrhea developed, initially 5−8 times daily, gradually increasing in frequency, and he was again admitted at the age of 3 years and 3 months. Compared to the patient's condition during the previous admission, the fever was higher, spiking to 38–39 °C with diarrhea 7−8 times a day, and the general condition was worse. At the age of 3 years and 5 months, the fever subsided and he was discharged. At the age of 3 years and 8 months, however, the general condition took a marked turn for the worse and he was admitted for the third time. Various forms of therapy, including antibiotics, IgA, γ-gl, transfactor and steroids were tried without success against the fever and diarrhea and anemia became progressively worse. Bloody stool developed and the general condition worsened. He died of general weakness and respiratory failure at the age of 3 years and 11 months.

Laboratory tests revealed a pancytopenia as shown in the Table 1 and the anemia became progressively worse. There is a great variation in the level of γ-gl. In the early stage decrease of γ-gl was slight and marked decrease of γ-gl developed towards the end. Peripheral lymphocytes count were decreased. Blood cultures were negative

Table 1.

Laboratory Data					
Age	2yrs 10m	3yrs 3m	3yrs 5m	3yrs 8m	3yrs 10m
RBC $\times 10^4$	302	292		223	197
Hb g/dl	8.8	9.6		6.2	
Ht %	29.2	27.2		25.0	25.0
WBC /mm^3	2900	9000	5800	2000	3900
Ly. %	35	61	49	20	
Plate $\times 10^4$	7.9	2.0			
T.P g/dl	6.4	4.8		5.4	5.9
γ-gl %	10.6	16.0		7.7	5.3
S-Fe γ/dl	130				
ULBC γ/dl	110				
CRP	(−)	2+		6+	
Sedim. Rate mm/h	65	140			
Blood culture		(−)			
Feces culture		(−)			(−)

and stool cultures failed to yield any pathogenic organisms (Table 1).

Immunological function tests (Table 2) revealed that IgA was low and IgG and IgM fell towards the terminal stage. The B-cell function tests revealed no remarkable quantitative changes exception the terminal stage. B-cell insufficiency was not evident in the initial stage. Regrettably, no tests were done on the antibody function.

Cellular immunity tests revealed the T-cell count normal, the cell stimulation by the phytohaemagglutinin (PHA) normal, SK-SD skin reaction negative and DNCB sensitized skin reaction negative, indicating the lack of delayde type hypersensitivity with definite insufficiency of T-cell function. The nitroblue tetrazolium test (NBT-test) for granulocyte function was normal and Adenosine Deaminase of red cells was normal (Table 2).

Table 2.

immunological function tests			
Age	2yrs 10m	3yrs 5m	3yrs 8m
Ig G mg/dl	950	110	160
Ig A mg/dl	83	95	34
Ig M mg/dl	157	130	36
ASLO		512×	64×
T cell %		59	
Delayed-type Skin Reaction			
SK-SD		(−)	
DNCB		(−)	
PHA			normal
NBT-test		normal	
Adenocine deaminase (ADA)		normal	

In view of the diarrhea which was refractory to treatment, fever of unknown cause and functional insufficiency of T-cell, functional insufficiency or enzymic defect at B-cell level was suspected, and a diagnosis of combinied immunodeficiency was suggested.

AUTOPSY FINDINGS

Autopsy revealed a small boy with a height of 82.0 cm and weight of 7.0 Kg. The scalp hair consisted of lanugo and growth was retarded. Homorrhage was seen around the lips, with ulceration and marked findings of stomatitis. Neither external surface anomaly nor skin eruptions were noted.

The thymus was found in the normal position, but was very small. The weight of the thymus was 2.2 g including the surrounding tissue and small sized lymph nodes. Grossly, thymic parenchymal tissue could not be clearly distinguished, but consisted of fat and connective tissue (Fig. 1). Histologically, thymic parenchymal tissue had indistinct lobular structure and connective tissue proliferation partially accompanied by fatty infiltration. The cortico-medullary junction was indistinct, with marked lymphocyte depletion, comprising oval or short spindle-shaped thymocytes (Fig. 2). Hassal's corpuscles were decreased in number and were of relatively large size. The largest Hassal's corpuscles was measrued 250 μ in diameter with marked calcification. This histological appearance of the thymus resembles the picture of acute severe involution (Fig. 3).

Several lymph nodes measuring up to 1.5×0.5 cm were noted on the mesentery, around the aorta and pancreas, along the trachea and near the bifurcation. Histologically, formation of lymphoid follicles in the cortex was not distinct, with lymphocyte depletion in the deep cortical thymus-dependent areas (Fig. 4). Plasma cells also decreased in number. These lesions varied in degrees depending on the location within the lymph nodes. In the deep cervical lymph nodes, lymphocytes formed a mass with a follicular structure, and the depletion of lymphocyte in the deep cortex was less pronounced than in other parts. Germinal centers, however, were seen nowhere (Fig. 5 and 6). In other words, the lymph nodes were noted to have poor formation of lymphoid follicles without germinal centers, and to decrease in plasma cells, probably reflecting abnormal B-cell function. Lymphocyte depletion in the thymusdependent areas was probably reflecting abnormal T-cell function.

The spleen weighed 22.5 g, normal being 37 g. Grossly, splenic follicles were indistinct with congestion. Histological examination revealed marked decrease of lymphocytes in the lymphatic tissue around small arteries probably representing the thymus-dependent area. No structure resembling a lymphoid follicle was present. Functional insufficiency of T-and B-cell was thus also found in the spleen (Fig. 7).

The lymphatics of the intestinal tract showed extremely poor development, without any formation of lymphoid follicles in the appendix and ileum. Only masses of lymphocytes were found (Fig. 8).

In the terminal ileum, coagulated blood with a diameter of 3 cm was found within the lumen. Numerous hemorrhagic spots measuring 0.5×1.5 cm were distributed towards the lower colon and rectum, accompanied by shallow ulcers (Fig. 9). Histologically, the mucous membrane was hemorrhagic and necrotic with desquamation, accompanied by neutrophilic infiltration and numerous inclusion bodies, giving the picture of cytomegalic inclusion enteritis (Fig. 10).

The left lung weighed 90 g and the right lung 150 g with increased weight and consistency. Hemorrhagic spots and small yellow-white nodules were scattered over the surface. Histological examination revealed interstitial pneumonia with numerous cytomegalic inclusion bodies, interspersed with foci of bronchopneumonia (Fig. 11).

In addition, cytomegalic inclusion bodies were seen in the liver, kidney, adrenals, spleen and lymph nodes, giving a picture of advanced infection due to cytomegalic inclusion disease, probably representing the direct cause of death. The final pathological findings are summarized in the Table 3.

Table 3.

Pathological Diagnosis
1. Combined immunodeficiency
1) Severe involution and dysplasia, thymus, 2.2 g.
2) Hypoplasia of lymph nodes and lymphatic tissue.
i. Lymphocyte depletion in T-cell areas of lymph nodes and spleen
ii. Hypoplasia of lymphoid follicles
iii. Hypoplasia of Peyer's patches
iv. Plasma cells depletion
3) Growth retardation (7.0 kg in weight, 82.0 cm in length)
4) Generalized cytomegalic inclusion disease, both lungs, intestine, esophagus, liver, both kidneys etc.
5) Stomattiis
2. Fatty infiltration of liver, others.

COMMENTS

Immunodeficiency has so far been classified based on the abnormalities of differentiation and maturation of immunologically competent cell (immunoblast). These classifications are depend on the presence of disturbance in the T-cell, B-cell, or both T-cell and B-cell, also with reference to the presence or absence of hereditary occurrence, clinical symptoms and pathological changes in the thymus and lymphatic tissue[4)13)-16)18)20)27)28)].

This patient had almost characteristic clinical manifestations of SCID[1)18)19)21)] such as stomatitis, chronic protracted diarrhea, fever, growth retardation. Stool cultures frequently fails to identify the pathogenic bacteria[1)], as was the case of this patient.

In this patient, the degree of immune insufficiency was not remarkably pronounced by the immunological function tests. The decrease of gammaglobulin and immunoglobulins were not so pronounced, raising a question of presence or absence of primary insufficiency of B-cell function. Lymphopenia also varied in degrees depending on the date of the clinical course showing remarkable decrease or not. Since the degree of abnormality of humoral and cellular immune function is milder, this case cannot be unequivocally classified as the typical case of SCID.

Pathological examination revealed the remaining lymph nodes and lymphatic tissues and Hassal's corpuscles in the thymus, which distinguish it from the typical case of primary immunodeficiency so far reported[7)8)10)18)21)]. Histological pictures of lymphoid tissues in this case disclosed the characteristic appearance of the combined immunodeficiency having lymphoid hypoplasia in the thymus-dependent areas and B-cell system[2)7)8)12)].

The changes of the thymus in the primary immunodeficiency are aplasia, hypoplasia, dysplasia, and involution according to the type of the disease. In many of these types, Hassal's corpuscles are absent, with hypoplasia or dysplasia[2)7)8)10)13)14)17)18)24)25)26)]. In this case the decrease in number of Hassal's corpuscles and the appearance of large sized Hassal's corpuscles with calcification are somewhat different from simple involution caused by the repeated infection[13)17)] and rather suggest the dyspl a sia[25)]. When the changes of thymus in the Down's syndrome or congenital biliary atresia are also taken into consideration[5)17)22)24)25)], the picture probably represents an involution due to the abnormality towards the end of the fetal life[25)]. The possibility of the severe involution of the thymus due to the repeated infection cannot be completely ruled out and the differentiation between primary and secondary immunodeficiency is difficult based on the morphology of the thymus alone.

Distinctive figure of infection in immunodeficiency is considered to vary depending the picture of infection[6)26)]. In patient mainly with deficiency of cellular immunity, infection by virus and fungi are the overwhelming majority, with characteristically high sensitivity to cytomegalovirus, candida and pneumocystis carinii. In this case, autopsy revealed generalized cytomegalic inclusion disease suggesting the presence of functional insufficiency of T-cell, in agreement with the results of the immunological function tests.

The problem, however, remains in this case as to the primary or secondary nature of immunodeficiency which caused by infection. No basic disease, however, accounted for the entire clinical picture which lasted 1 year and 3 month and consisted of retarded growth, fever of unknown etiology, and severe refractory diarrhea. Therefore, this disease should probably be classified as one of the types of immunodeficiency, despite the failure of attempts to fit into any of the types so far described. It is difficult to explain the whole clinical course with cytomegalovirus infection, and there were no proof of past history of rubella, measles and variola. So the presence of some degree of congenital immunodeficiency must be assumed, with last manifestation and progression in response to viral infection, finally leading to death.

Table 4. Suspected Case of Primary Immunodeficiency

Case	Type of Immunodeficiency	Clinical Symptoms	Serum Protein				Lymphocyte			Pathological findings			
			Y-gl %	IgG mg/dl	IgA mg/dl	IgM mg/dl	count /mm³	T-cell %	PHA	Thymus	Lymphoid tissue	Plasma cell	Immediate cause of death
① 86 days ♀ [Kato, 1968]	Variants of Swiss Type	thrush, abscess, fever	?	?	?	?	4200	?	?	2.0 g Hypoplasia + Involution	Hypoplasia	Hypoplasia	Sepsis
② 67 days ♂ [Kato, 1968]		abscess, fever, exanthem	12.2 (660 mg/dl)	?	no sinking line		4900	?	?	7.0 g Hypoplasia + Involution	Hypoplasia	None	Sepsis
③ 2yrs 3m ♂ [Ze, 1972]	Suspected congenital sporadic hypogammaglobulinemia	fever, recurrent respiratory infection	6.0 ~ 10 (430 mg/dl)	960	72	1625	6240 ~ 9520	?	↓↓	?	?	?	(Surviving)
④ 3m ♀ [Konno, 1977]	Variants of combined immunodeficiency	diarrhea, meningitis, ulcerative colitis	7.0	350	0	50	1190	59	?	?	?	?	Severe water pox
⑤ 3m ♂ [Konno, 1977]		thrush, otitis media, pneumonia, periproctal ulcer	5.6	380	0	110	396	43	?	Hypoplasia	Hypoplasia	?	pneunocytis carinii pneumonia
⑥ 10m ♂ [Konorza, 1978]	Variant of Fireman's disease	rhinitis, pharyngitis, bronchitis	?	370	1200	90	?	?	?	2.5 g dysplasia	Hypoplasia	↓	Generalized septic aspergillosis
⑦ 3m ♀ [Konorza, 1978]		otitis media, seborrheic dermatitis, pyodermia	?	500	7	900	?	?	?	0.9 g dysplasia	Hypoplasia	moderate ↑	Purulent meningitis
⑧ 1yrs 1m ♂ [Shimizu, 1978]	Suspected	stomatitis, ulcer of external genital and thigh	?	220 ~ 310	19 ~ 28	49 ~ 65	normal	?	?	3.0 g involution	Hypoplasia	?	sepsis
⑨ 10m ♀ [Shimizu, 1978]	Primary immunodeficiency	suppurative arthritis, abscess of back, icterus	?	1800 ~ 2300	29 ~ 44	178 ~ 190	normal	?	?	3.8 g involution	Hypoplasia	?	Subacute hepatitis
⑩ 6m ♀ [Shimizu, 1978]		fever, dyspnoe	?	440 ~ 540	70 ~ 105	230 ~ 280	normal	?	?	4.0 g involution	Hypoplasia	?	Cytomegalic inclusion disease
⑪ 3yrs ♂ [Kawai, 1980]	Variant case of combined immunodeficiency	stomatitis, growth retardation, diarrhea, fever	5.3 ~ 16.0	110 ~ 950	34 ~ 83	36 ~ 157	250 ~ 5500	59	normal	2.2 g involution + dysplasia?	Hypoplasia	↓	Generalized cytomegalic inclusion disease

m: month
?: unknown
↓: decreased
↑: increased

Recently variant case or suspected case of primary immunodeficiency were reported [9)11)12)23)29)] (Table 4). In each case hypoglobulinemia, dysgammaglobulinemia and lymphopenia were not so pronounced similar to those in the present case. SHIMIZU[23)] reported the results of detailed studies on the child with susceptibility to infection at autopsy. He said that primary immunodeficiency was highly suspected in these child but it was difficult to draw any definite conclusion.

It present a problem that such a case should be grasped as typical case of primary immunodeficiency. We regard these cases as borderline case of primary immunodeficiency. This case is a very suggestive one for the future reevaluation of classification of immunodeficiency, and studies of many cases like thisf are expected to clarify this problem.

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Fig. 1. The weight of thymus is 2.2g. It consists of fat and connective tissue. Thymic parenchymal tissue cannot be clearly distinguished.

Fig. 2. The cortico-medullary junction of thymus is indistinct. The thymic cortex is almost devoid of lymphocytes and contains small number of Hassal's corpuscles with calcification.

Fig. 3. The large sized Hassal's corpuscle with calcification

Fig. 4. The lymph node around the pancreas shows markedly lymphocytes depletion in the deep cortical thymic dependent area. Lymphoid follicle is not distinct.

Fig. 5 and 6. In deep cervical lymph node, lymphocyte forms a mass with follicular structure without germinal centers.

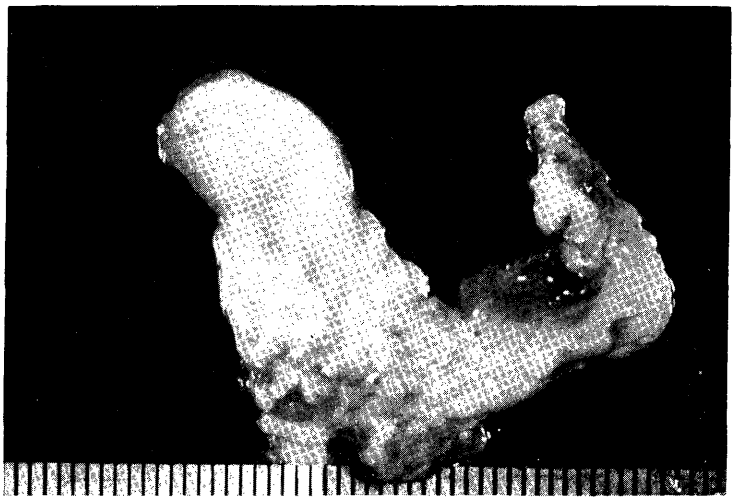
Fig. 7. The spleen reveals marked decrease of lymphocytes around small arteries probably representing the thymus dependent area. No structure resembling a lymphoid follicle.

Fig. 8. The Peyer's patch of the ileum shows poor development with only masses of lymphocyte.

Fig. 9. Numerous hemorrhagic spots are distributed toward the colon accompanied by shallow ulcers.

Fig. 10. Mucous epithelium is desquamated and numerous incluison bodies are seen, giving the picture of cytomegalic inclusion enteritis.

Fig. 11. Lung shows interstitial pneumonia with numerous cytomegalic inclusion bodies.



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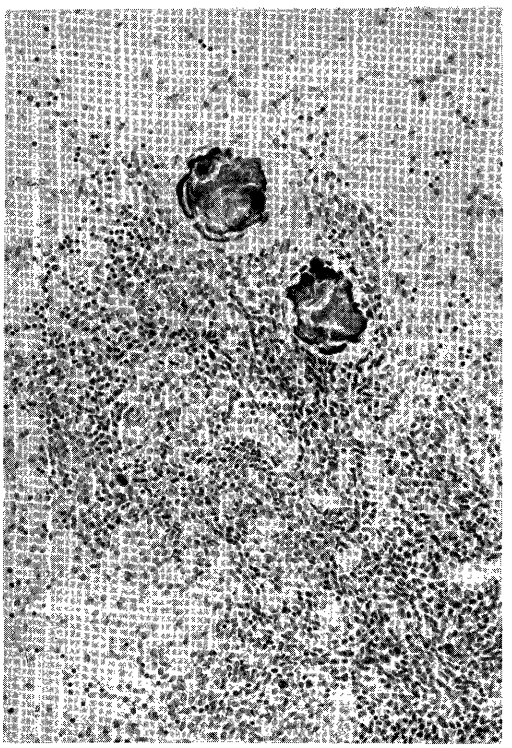

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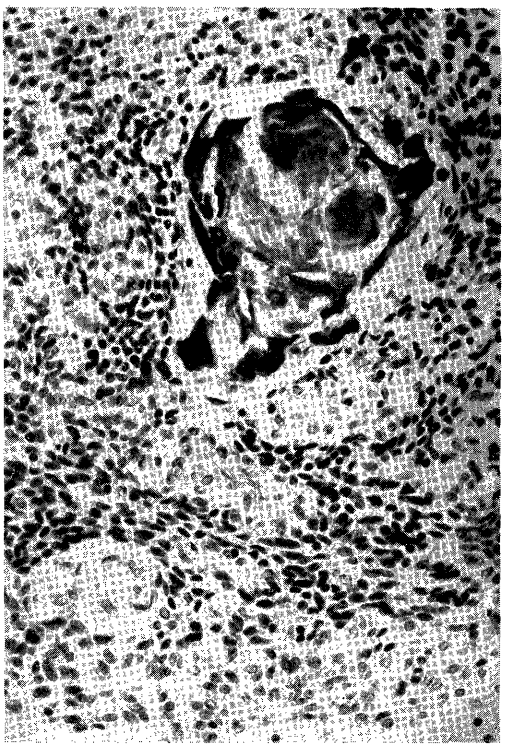

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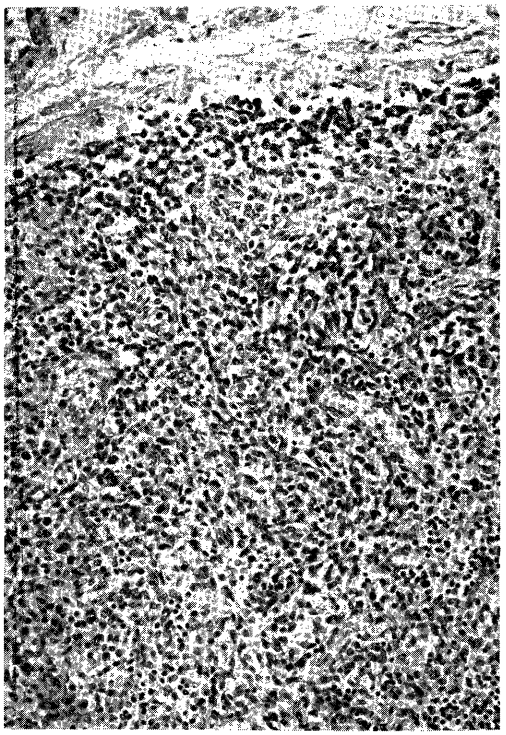

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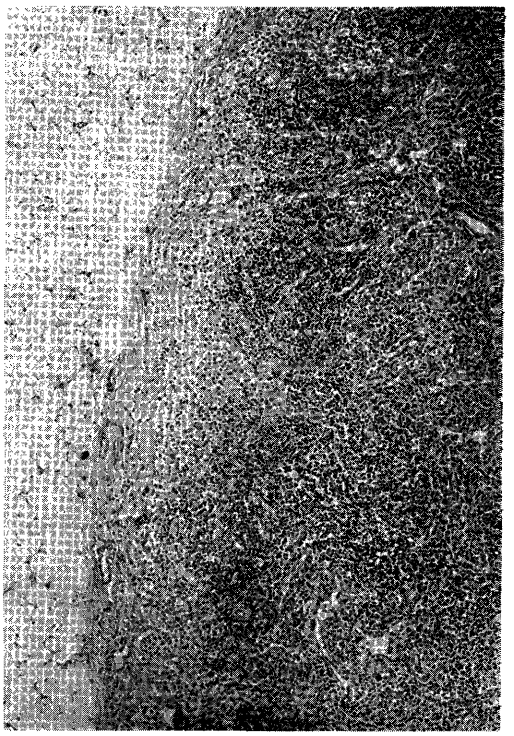

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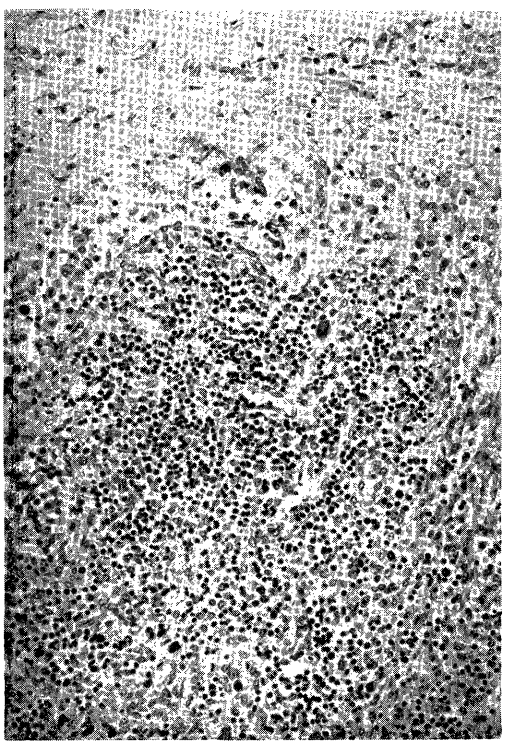

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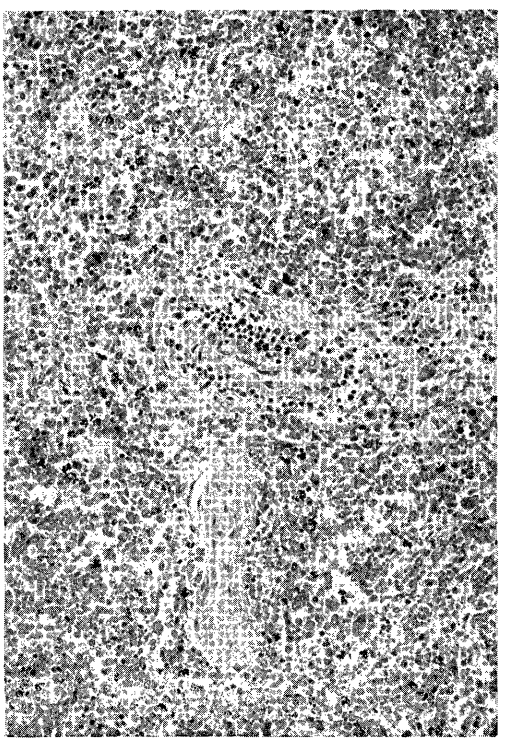

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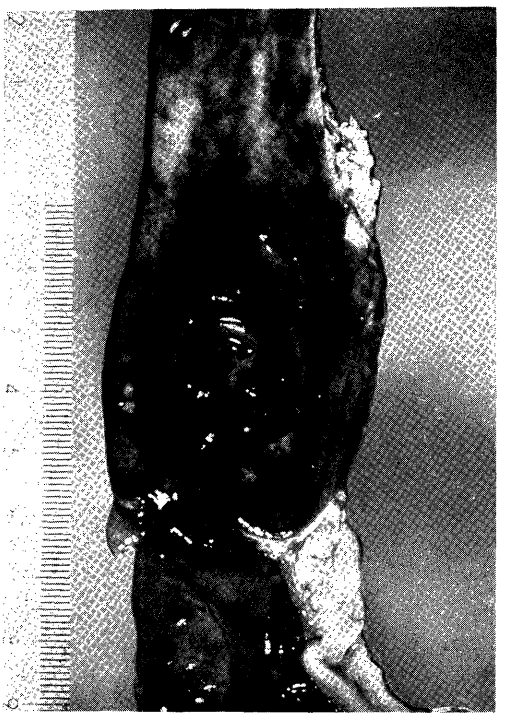

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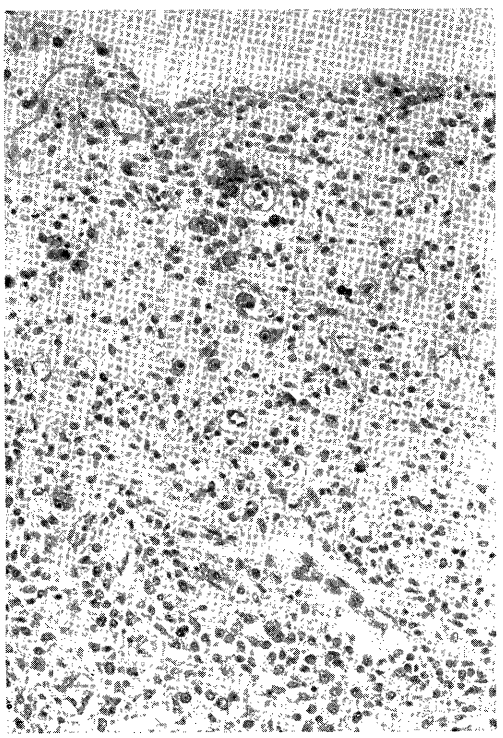

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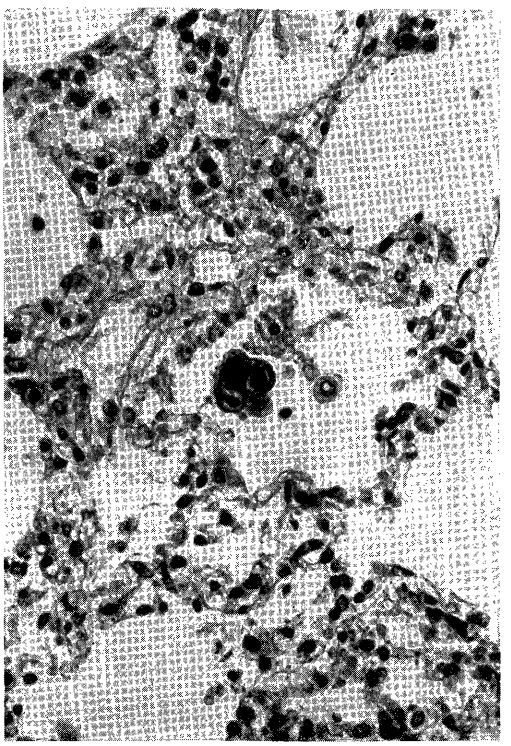

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