Studies on Rama Dewa, the Enzootic Disease of Cattle occurring in Lampung Province of Sumatra, Indonesia – its Histopathology and Critical Views on Name of the Disease

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Studies on Rama Dewa, the Enzootic Disease of Cattle occurring in Lampung Province of Sumatra, Indonesia - its Histopathology and Critical Views on Name of the Disease

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INTRODUCTION

The enzootic disease of cattle broke out in Central Lampung of Lampung Province, Southern end of Sumatra, Indonesia. The disease occurred for the first time at Rama Dewa village on May of 1976, so that this disease has been called the Rama Dewa.

In 1976, the Committee for the Prevention of Rama Dewa was set up in Directorate of Animal Health, Directorate General of Livestock Services, Ministry of Agriculture, and sum of the damage due to this disease, epidemiology, clinical signs and pathological changes of the disease have been discussed. In the same year, Soeharsono and Darmadi (1976) succeeded in the transmission experiments using subctaneous inoculation of whole blood of diseased cattle, and made also experiments for the treatment using antibiotics, but they did not obtain the positive results.

Afterward until 1983, Rama Dewa occurred in every years except 1977, however, during the period only epidemiological and clinical investigations have been done by Marfiatiningsih et al. (1980) and Swastawa et al. (1983), and no histopathological observations were carried out.

Since May of 1980 authors have investigated on the field-occurring and experimental cases of the Rama Dewa. This report dealed with histopathological findings, and the comparison of this disease with some kinds of viral diseases of cattle, especially malignant catarrhal fever. Furthermore, our critical views on name of the disease were described.

INVESTIGATIONS ON FIELD CASES

1. Field observations and source of autopsied cases

According to the Report of the Committee for the Prevention of Rama Dewa (1976), in a period from May of 1976 to January of 1977, the disease occurred at 4 subdistricts such as Raman Utara, Seputih Raman, Rumbia and Seputih Banyak out of 21 subdistricts in Central Lampung, Lampung Province, and 756 out of 6,097 heads of Bali cattle died or condemned (Fig.1). Only Bali cattle were suffered from this disease, but exact numbers of diseased animals were not clear until year of 1980 in that authors begun to investigate.

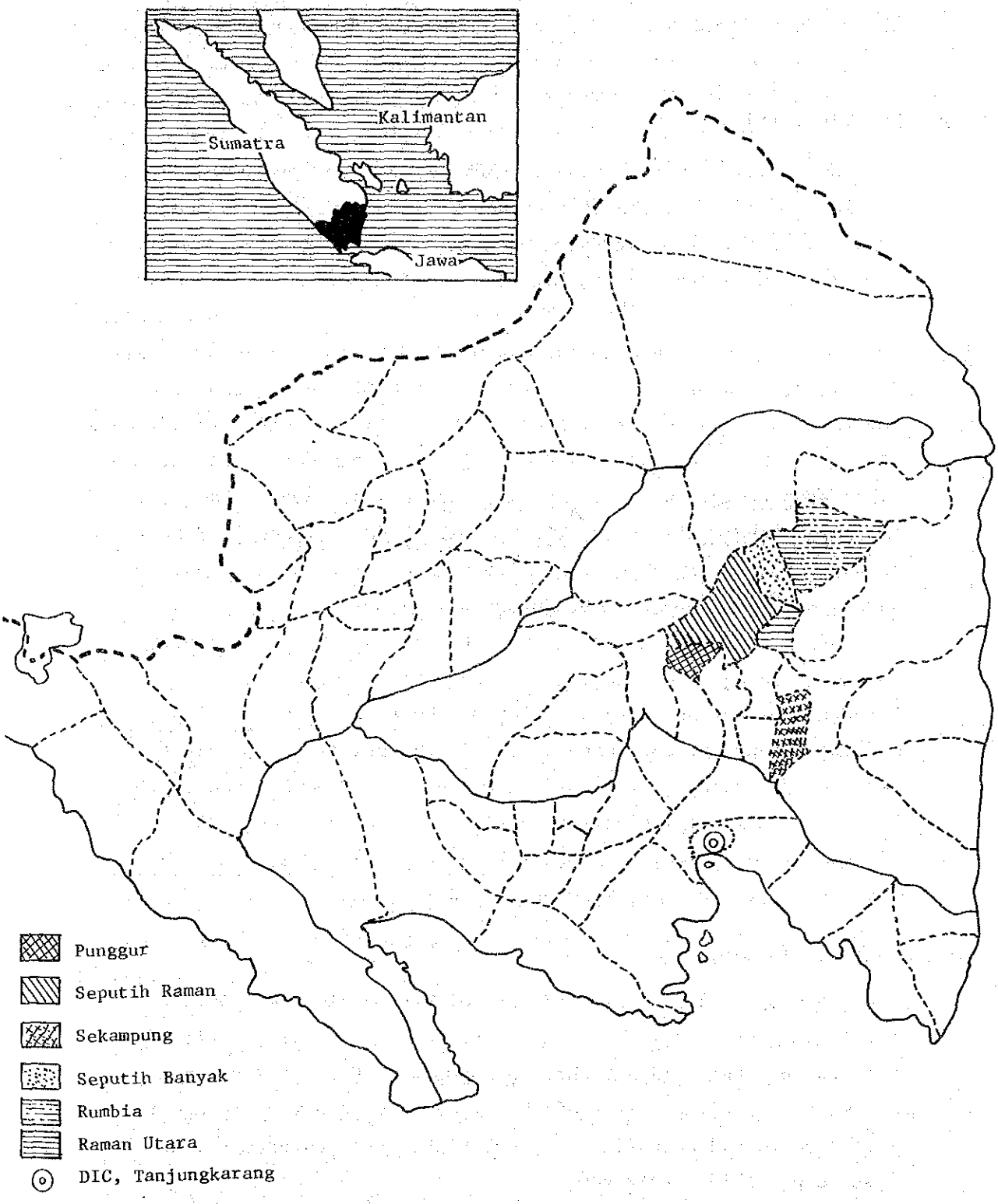
In 1980, outbreak occurred mainly at both subdistricts of Seputih Raman and Punggur, 63 out of 363 heads of Bali cattle which were raised in the subdistrict of Punggur, died or condemned. Namely at Punggur, in a periode from June to August, 50 heads of Bali cattle died or condemned (Case No.1 was obtained.) and about same number of Bali cattle were suspected to die or condemn at Seputih Raman. In a period from September to October, 13 heads of Bali cattle which were raised at 3 neighboring farmers died one after another at Punggur (Case No.2 was obtained.). Moreover in November, 10 heads of Bali cattle died or condemned at one village of Seputih Raman subdistrict (Case No.3 was obtained.), and in December, about same number of Bali cattle died at another 2 villages of same subdistrict (Case Nos. 4 and 5 were obtained.).

In 1982, outbreak occurred only at subdistrict of Punggur, and numbers of diseased cattle were less than that in 1980. Namely during July and August, 14 out of 169 heads of Bali cattle died or condemned at 2 villages of this subdistrict (Case Nos. 6, 7, 8, 9 and 10 were obtained.).

In March, 1983, outbreak of small scale occurred at the same villages around Punggur (Case No. 11 was obtained.). Case No. 12 was obtained in subdistrict of Sekampung near Punggur.

On the other hand, in January, February and November, 1982, Bali cattle raised at Animal Disease Investigation Centre (DIC) Region III, Tanjungkarang, died with the symptom of Rama Dewa (Case Nos.13, 14 and 15). Subdistricts of Punggur and Seputih Raman where

Fig. 1. Subdistricts where Rama Dewa occurred in Lampung Province.



Rama Dewa occurred are not so far, and the staffs and workers of DIC went to the villages frequently for the investigation, so that it was thought that unknown agent might be carried to DIC. According to this reason 3 cases stated above were used in the investigations.

2. Materials and methods.

As it has been described already, 15 cases of autopsied cattle were used for the histopathological studies. As shown in Table 1, they were divided into 3 groups, namely Group I involved the cases obtained in 1980, Group II in 1982 and 1983, and Group III in DIC, respectively.

Complete autopsy examinations were performed on 9 cases (Case Nos. 1, 2, 3, 6, 7, 11, 13, 14 and 15) at DIC, Tanjungkarang, and remaining 6 cases were autopsied at the field by veterinary officers of subdistricts, and sent to DIC. Tissue blocks were routinely taken from tougue, palate, pharynx, oesophagus, rumen, abomasum, small and large intestines, liver, gall bladder, kidney, urinary bladder, spleen, lymph node, salivary gland, larynx, trachea, lung, heart, adrenal gland, and brain and spinal cord.

Of the brain and spinal cord after fixing the tissues with 10% formalin solution, the small tissue blocks were taken from Cerebral hemisphere, Corpus striatum, Thalamus, Midbrain, Pons, Medulla oblongata, and Cerebellum. In the spinal cord, only the portion of neck was taken. These tissue blocks were fixed in 10 percent formalin solution. Paraffin sections were cut at 6 microns and stained with hematoxylin-eosin. PAS, Azan, and one step trichrom stainings were performed in some sections.

3. Clinical findings

Only Bali cattle were affected with this disease. Ongole cattle and sheep were raised at the farmers mixed with diseased animals, but they did not show any symptom. Calf of 2 months old was affected, but most of the diseased cattle were in age from 2 to 7 years and mortality rate was high, and recovery from the disease was seldom observed.

Clinical symptoms of each cases were shown in Table 1, and were as followings. Animals showed high fever of more than 41.0°C. decrease of appetite or anorexia and depression. Reddening of conjunctiva, lacrymation, excreation of serous or purulent discharge from nose, and diarrhea were observed, and diarrheal feces contained blood frequently. Corneal opacity (Fig. 2) was found in some cases. Erosions and shallow ulcers of tongue, pharynx and palate were usually observed, and that of gingive and lip were found in few cases. Skin lesions were rarely found. Subctaneous lymph nodes such as mandible, parotide, prescapural and femoral ones were swollen in various degrees, but some cases showed no enlargement. Slight nervous symptom was observed in 3 cases which manifested corneal opacity.

4. Macroscopic findings.

Group I

Case No. 1: External examinations revealed hyperemia of conjunctive and sclera, mucous discharge of nasal cavity, and attachment of diarrheal feces to the skin near anus. Subcutaneous lymph nodes such as mandible, parotide and prescapular ones enlarged. Erosion and hemorrhage were observed in tongue, pharynx, oesophagus and rumen. Hemorrhage and erosion were also manifested in abomasum, and small and large intestines. In upper respiratory organ mucous membranes of nose, larynx and trachea were congested, and petechial hemorrhages were scattered. Circumscripted pneumonic lesions was found in frontal lobe of right lung. Spleen enlarged and was hemorrhagic. Liver enlarged, mottled and was grayish brown in colour. Kidney and heart showed cloudy swelling. Mesenteric and hepatic lymph nodes enlarged and congested. Congestion was observed in brain.

Case No. 2: Changes of upper alimentary organ were prominent (Figs. 3 and 4) and that of low alimentary organ were somewhat slighter than Case No. 1, but congestion and petechial hemorrhages were seen in large extent. Nose, pharynx and trachea were congested severely. Spleen enlarged and was hemorrhagic. Liver and Kidney showed mottled appearance and cloudy swelling. There was no pneumonic lesions in both lungs, and subendocardial hemorrhages were

Table 1. Field cases of Rama Dewa

Group No.	Case No. of autopsied cattle	Sex Male (M) or Female(F)	Age (Month)	Died (D) or slaughtered (S)	Date of autopsy
I	1	F	30	D	23, Aug., '80
	2	M	6	D	6, Oct., '80
	3	M	7	D	18, Nov., '80
	4	F	48	S	17, Dec., '80
	5	F	30	S	18, Dec., '80
II	6	F	24	S	7, Aug., '82
	7	F	24	D	7, Aug., '82
	8	F	2	D	11, Aug., '82
	9	F	adult	S	14, Aug., '82
	10	F	adult	S	12, Aug., '82
	11	F	36	D	18, March, '83
	12	M	adult	D	2, Oct., '83
III	13	F	24	D	22, Jan., '82
	14	F	30	D	1, Feb., '82
	15	M	adult	D	10, Nov., '82

Note: * Name of subdistricts where field cases were obtained.

Main clinical symptoms	Remarks
high fever of 41.7°C, anorexia, depression, erosion of mouth cavity, bloody diarrhea, swelling of lymph nodes	Punggur *
high fever of 41°C, anorexia, erosion of mouth cavity, diarrhea, marked swelling of prescapular lymph node	Punggur
high fever of 41.0°C, anorexia, swelling of prescapular lymph node	Seputih Raman *
	Seputin Raman
erosion of mouth cavity	Seputih Raman
high fever of 41.7°C, anorexia, reddening of conjunctiva, lacrymation, corneal opacity, bloody diarrhea	Punggur
high fever, anorexia, diarrhea, swelling of lymph node	Punggur
high fever, anorexia, diarrhea, swelling of lymph node	Punggur
high fever, anorexia, diarrhea, swelling of lymph node	Punggur
high fever, anorexia, diarrhea	Punggur
high fever of more than 41°C, anorexia, reddening of conjunctiva, lacrymation, corneal opacity, nervous symptom	Punggur used for transmission experiment
marked corneal opacity, mucopurulent nasal discharge, erosion of mouth cavity, bloody diarrhea, nervous symptom	Sekampung*
high fever, anorexia, depression, severe diarrhea	DIC, Tanjungkarang
high fever, anorexia, depression, severe diarrhea, erosion of mouth cavity	DIC, Tanjungkarang
high fever of more than 41°C, anorexia, lacrymation, marked corneal opacity, erosion of mouth cavity, slight nervous symptom	DIC, Tanjungkarang used for transmission experiment

present in left ventricle of heart. Cerebro-spinal fluid increased slightly.

Case No. 3: Subctaneous lymph nodes were highly swollen, but changes of upper alimentary organ were slight, showing narrow-ranged hemorrhage and erosion in tongue and pharynx. Changes of intestines were slight. There was congestion of trachea, congestion and edema of lung, and epicardial hemorrhage of heart. No macroscopic changes were present in brain.

Case Nos. 4 and 5 were the cases slaughtered and dissected at the field, and tissue specimens were sent to DIC, so that details of autopsy findings were unclear, but No. 5 had a lesions of showing hemorrhage, edema and erosions in palate, tongue and pharynx, but No.4 did not show such changes.

Group II.

Case No. 6 was slaughtered on 2 days after appearance of symptom and dissected at DIC. Subctaneous lymph nodes enlarged, and congested. There was slight erosion in the dorsal surface of the tongue and gingiva, but no changes were present in oesophagus and rumen. Abomasum was congested and involved petechial hemorrhages and small erosive lesions. Congestion and Petechial hemorrhages were noted in large intestine. Nose, larynx and trachea were congested, and no pneumonic changes were observed in lung. Spleen enlarged slightly, and mesenteric and hepatic lymph nodes were swollen. Liver and kidney showed mottled appearance and cloudy swelling in cutting the surface. No changes were present in heart and brain macroscopically.

Case No. 11 died on 2 days after appearance of symptom, and was dissected at DIC. Subctaneous lymph nodes enlarged and congested. There was erosions and shallow ulcers in lip, tongue, hard palate, pharynx and oesophagus. Congestion and erosion were present in abomasum, but changes were slight in intestines. Nose, pharynx and trachea were congested, and showed petechial hemorrhages in mucous membrane. Spleen enlarged slightly. There was marked congestion and hemorrhage in urinary bladder. Slight cloudy swelling were observed in liver and kidney. Cerebrospinal fluid increased.

In Case No. 12, only head and foot were brought to DIC, and dissected. There was marked corneal opacity, and necrotized mass of epithelium and erosions were present in tongue (Fig. 5), platate and lip. Foot also showed ulcerations.

Group III.

In Case No. 13, period from the onset of symptom to death were 7 days. High fever of more than 41° C continued and died. Subctaneous lymph nodes enlarged, but no changes were not present in upper alimentary organ. Prominent changes were observed in low alimentary organ, namely marked congestion and hemorrhage of abomasum, and small and large intestines were noted. Mesenteric lymph nodes were swollen and congested, but spleen was normal in size. Liver and kidney showed cloudy swelling. Brain was normal in nacked eye.

Case No. 14 was raised in the same stable of No. 13. On 10 days after death of No. 13, the animal showed symptom, and died taking the 7 days of disease course. In this case, changes of upper alimentary organ such as erosions of tongue, pharynx and palate were observed. The changes of low alimentary organ were also marked. Spleen enlarged, and cloudy swelling of liver and kidneys were manifested.

Case No. 15 got sick suddenly, showing high fever of 41.5°C, anorexia, lacrymation and marked corneal opacity, and died on 3 days after onset of symptom. In autopsy, the changes of upper alimentary organ were marked, and shallow ulcers were present in gingiva, palate, tongue, pharynx, oesophagus and rumen. Low alimentary organ showed also marked changes such as hemorrhage and erosion of abomasum and small intestine. Changes of large intestine were slight. Subctaneous lymph nodes enlarged. Spleen also enlarged and showed hemorrhage. Hemorrhage of urinary bladder was prominent. Cerebrospinal fluid somewhat increased.

4. Microscopic findings

Group I.

Extensive epithelial lesions of oral mucosa were observed in Case Nos. 1, 2 and 5 (Fig. 6). Histological studies revealed degenerative changes such as vacuolar degeneration and intense eosin staining of cytoplasm, and pycnosis of nuclei in epithelial cells of Stratum spinosum. Other areas of epithelium showed diffus coagulation necrosis extending from the surface to the basal layer. In some areas, liquefactive necrosis with infiltration of neutrophiles and mass of contaminated bacteria were present. Especially in Case No. 5, liquefactive necrosis and edema were marked.

On the other hand, noticeable changes were observed in Stratum germinativum. Numerous distinct lacunae of round or irregular in its shape, containing degenerative or necrotic epithelial cells and few lymphoid cells were observed within germinative layer, and necrotic changes were present extending from this layer to the epithelial surface (Fig. 7). In some areas, necrotic mass of epithelium was desquamated making erosive lesions.

A large numbers of lymphoid cells and histiocytes were present in lamina propria, and proliferation of the cells was severe in the areas of showing prominent epithelial changes (Fig. 6). The character of lymphoid cells was essentially and predominantly composed of lymphoblasts and lymphocytes. The nuclei of the lymphoblasts were most often round or oval with course chromatin and one or two nucleioli. Cytoplasm was wide and stained with eosin (Fig. 15). Lymphocytes were characterized by deeply basophilic nuclei and narrow cytoplasm. Neutrophiles and eosinophiles infiltrated mixing with lymphoid cells, although their number was varied. Plasma cells were few in number. Hemorrhages were accompanied at the edematous and hyperemic lamina propria, but the thrombi was seldom seen.

The lymphoid-cell proliferation was extended to the mucous gland and muscularis, exhibiting degeneration and desquamation of gland epithelium and hyaline degeneration of striated muscle. The lymphoid-cell proliferation occurred perivasculary and in the vascular wall, causing lymphoid vasculitis in small-sized arteries,

arterioles and related vein (Nos. 1 & 2).

Almost same lesions as in oral mucosa were found in the oesophagus. Microscopic examinations indicated that coagulation necrosis and detachment of such necrotized mass produced erosive lesions (Fig. 12). The formation of distinct lacunae as descrived in oral mucosa were detected, and the kind of extension from germinative layer to the surface were also observed in this area. Perivascular and diffus lymphoid-cell proliferation was found in lamina propria. Hemorrhage and edema were oftenly seen in erosive lesions (Fig. 12).

As the pathological changes of glandular mucosa of both cases (Nos. 1 and 2) epithelial degeneration and erosions were observed in low alimentary organ such as abomasum, and small and large intestines. Marked lymphoid-cell proliferation, hemorrhage, edema and sometimes increase of connective tissues were found. Perivascular lymphoid-cell proliferation and lymphoid vasculitis were present (Fig. 29), but necrotizing vasculitis (Fig. 28) was rarely found in small-sized arteries in submucosa of large intestine. Such blood vessels contained lymphoid cells in their lumens.

In upper respiratory organs of both cases, trachea showd degeneration and desquamation of epithelium, hemorrhage, edema and perivascular and diffus proliferation of lymphoid cells. Polymorphonuclear leukocytes were few in number. The changes of larynx were rather slight than that of trachea.

Lymph nodes denoted the characteristic changes as followings in all cases. The destruction and decrease of mature lymphocytes were noted in the follicles and perifollicular areas. Prominent findings were proliferation of lymphoid cells, occupying paracortical areas and follicles (Fig. 14). Among lymphoid cells, lymphoblasts were main component, but lymphocytes also proliferated (Fig. 15) and mitotic figures could be detected. Hyperplasia of reticular cells and increase of macrophages were constant findings. Plasma cells, neutrophiles and eosinophiles were also found, although their numbers varied from case to case.

Characteristic changes of the spleen were nearly sama as that of lymph nodes. Destruction and decreased number of lymphocyte were

observed in the follicles, and proliferation of lymphoid cells were detected in the follicles (Fig. 17) as well as in the red pulp. In the cases in which the changes were moderate or slight, lymphoid cells proliferated principally in the spaces around the arteries of follicle and red pulp. Reticular cells showed hyperplasia.

Histological examinations of liver revealed characteristic changes in all cases. The lymphoid-cell proliferation and increase of reticular cells were marked in Glisson's sheath, especially in periportal and pericholangeal tissues (Figs. 18 & 19). Infiltrates invaded the vascular wall and extended under the endothelium of portal vein and hepatic arteries and arterioles showing lymphoid vasculitis. There was degeneration and detachment of epithelium of biliary duct, and marked pericholangeal lymphoid-cell proliferation. Newly formed bile ducts, capillaries and fibroblasts increased in Glisson's sheath, so that finger-like projections of the tissues extended into the lobules. In the lobules, infiltration of lymphoid cells, activation of endothelial cells, increase of macrophages (No. 2), and fatty changes of liver cells(Nos. 1 & 2) were observed.

In the kidneys of all cases, proliferation or infiltration of lymphoid cells were present, although the extent varied from case to case. In Case Nos. 2, 4 & 5, the lymphoid cells distributed in perivascular, periglomerular and interstitial tissues in renal cortex (Fig. 20). In the lungs of 3 cases (Nos. 1, 2 & 4), lymphoid-cell proliferation was manifested diffusly in alveolar septum (Fig. 21) and perivascular and peribronchial areas. Lymphoid vasculitis of being same as that in liver was found in the small-sized arteries and vein. Slight changes such as nodular aggregation of lymphoid cells were detected in interstitial tissues of myocardium of 3 cases (Nos. 1, 2 & 4).

In the brain of one case (No. 2), there was generalized hyperemia and perivascular hemorrhage. The wall of vein as well as small-sized arteries showed edematous swelling containing lymphoid cells in the lumen. Only congestion was found in brain of the other cases.

Group II.

Characteristic changes of upper alimentary organ described already in Group I, were also observed in Case Nos. 6, 11 and 12 of Group II. Oral legions of Case Nos. 11 and 12 were especially marked, indicating formation of lacunae in Stratum germinativum, extensive coagulation necrosis of epithelium in Stratum spinosum, and prominent lymphoid-cell proliferation in lamina propria. Lymphoid vasculitis was present in small-sized arteries (Fig. 26) and vein.

In case No. 6, hemorrhage and lymphoid-cell proliferation were noted in the submucosa of large intestine. Conspicuous was the proliferation of the lymphoid cells detected in lymph nodes, spleen, liver and kidney in all cases examined. Perivascular lymphoid-cell proliferation and lymphoid vasculitis were observed in liver and lymph nodes. There was necrosis of cardiac muscle cells, hyperplasia of interstitial connective tissues and aggregation of lymphoid cells mixing with myogeneic giant cells in the myocardium of Case No. 7 (Figs. 22 & 23). Marked congestion, hemorrhage, diffus and perivascular lymphoid-cell proliferation, and lymphoid vasculitis in small-sized arteries, arterioles and vein in urinary bladder of case No. 11, and proliferation of lymphoid cells and vascular lymphoid-cell infiltration in adrenal gland of Case No. 1 were detected, respectively. Trachea and larynx showed congestion and lymphoid-cell infiltration in lamina propria of case Nos. 6 and 11. In the lung slight lymphocytic infiltration was present.

The histological examinations of brain and spinal cord revealed following changes in 3 cases, Nos. 6, 11 and 12. Characteristics were the vascular lymphoid-cell infiltration of distributing diffusly in Cerebral hemisphere, Corpus striatum, Thalamus and Midbrain (Figs. 32 & 33). Hyperplasia of glia cells was negligible. Eye of showing marked corneal opacity was examined in Case No. 12, and lymphoid-cell infiltration was observed.

Group III.

Characteristic changes of upper alimentary organ were observed in 2 out of 3 cases, especially marked in Case No. 15. Extensive coagulation necrosis, liquefactive necrosis caused by mixed infection

of bacteria, and numerous distinct lacunae of showing irregular form in Stratum germinativum were found in oral and oesophageal mucosa. The lacunae contained degenerated epithelium and a few lymphoid cells, and were in contact with tissues where marked proliferation of lymphoid cells was manifested (Figs. 8 & 9). Lymphoid vasculitis was present, but it occurred in small-sized arteries, arterioles and related veins, and not in middle-sized muscular arteries.

The changes of the low alimentary organs were marked in Case Nos. 13 and 14. Congestion, hemorrhage and proliferation of lymphoid cells were observed in abomasum, and small and large intestines. Livers of all cases showed prominent changes such as proliferation of lymphoid cells, hyperplasia of reticular cells, increase of bile duct and granulation tissue, and vasculitis of portal vein. Hemorrhage and focal necrosis was seen in the hepatic lobules of No. 15. In lymph nodes and spleen, there was proliferation of lymphoid cells in all cases. Although enlargement was not observed macroscopically in lymph node of No. 15, microscopic observations revealed marked lymphoid-cell proliferation, and hyperplasia of reticular cells in parenchym. Lymphoid vasculitis were found in small-sized arteries in capsule. Hemorrhage and necrosis of follicle were manifested in the spleen of No. 15. Lymphoid-cell proliferation was also observed in the kidney of all cases, but was prominent in No. 15. There was also vasculitis of small-sized arteries in this case. Lungs involved vasculitis in vein of No. 14, and hemorrhage, lymphoid-cell proliferation and vasculitis of small-sized arteries in No. 15, respectively. Degeneration and desquamation of epithelial cells, hemorrhage and lymphoid-cell proliferation in lamina propria were observed in trachea of all cases.

Urinary bladder, adrenal gland, testis and epididymis were examined on Case No. 15. Degeneration and necrosis of transitional epithelium, hemorrhage, marked proliferation of lymphoid cells, and lymphoid vasculitis of vein in urinary bladder and adrenal gland, and interstitial lymphoid-cell proliferation and lymphoid-necrotizing vasculitis were observed (Fig. 31), respectively.

Case No. 15 also showed encephalitic changes of being same as that in Case Nos. 11 and 12 of Group III.

5. Summary of the findings of field cases

Fifteen field cases were divided into 3 groups according to the year and place of occurrence, and examined macro-and microscopically. The most common and prominent findings were the proliferation of lymphoid cells throughout the body, although severity was different according to the organ. This change was observed in all cases through the groups.

Necrotic changes of stratified squamous epithelium of oral and oesophageal mucosa were also characteristic of this disease, and were detected in the cases of each groups. Perivascular lymphoid-cell infiltration and lymphoid vasculitis were found mostly in liver, upper-and low-alimentary organs, lymph node, lung, kidney, urinary bladder, adrenal gland and epididymis, however, necrotizing vasculitis was rare, and found only in tongue of case No. 12, large intestine of No. 1 and epididymis of No. 15. Moreover, vasculitis was present in arterioles, small sized arteries, and related vein, and not in medium-sized muscular arteries.

Encephalitic changes were slight or moderate, and detected in 3 out of 7 cases in Group II, and 1 out of 3 cases in Group III, but not in cases of Group I.

INVESTIGATIONS ON EXPERIMENTAL CASES

1. Materials and methods

At first two field cases, No. 6 and other one case which occurred in the same village and time, and showed same clinical symptom as No. 6, were used in the inoculation experiments. Each 5 ml of defibrinated whole blood collected from diseased animals stated above, were inoculated subctaneously into each one healthy Bali cattle of being female and one year and 7 months old, respectively. Animals were observed clinically in 35 days after inoculation, but no clinical symptoms were observed.

Then, another two field cases, Nos. 15 and 11 were used in experimental inoculation test using a large amount of blood. Namely in the first experiment, each 50 ml of whole blood of Case No. 15 were mixed with the anticoagulant of 10% sodium citrate

solution with the ratio of 9:1, and inoculated into 2 healthy Bali cattle intravenously (Cattle A) and subctaneously (Cattle B). Cattle A got sick on 6 days after inoculation, and died on 3 days after appearance of symptom, but cattle B did not show any symptom within 100 days after inoculation.

Then, each 100 ml of citrated whole blood of cattle A were inoculated intravenously into one Bali cattle (Cattle C) and one Ongole cattle, respectively. As the results, only cattle C got sick on 18 days after inoculation and died 5 days after onset of symptom. On the 2nd day after the appearance of symptom, 100 ml of citrated whole blood of Cattle C were inoculated intravenously into one Bali cattle (Cattle D), but the animal did not show any symptom during 100 days after inoculation.

Then, new trials were made as followings. 100 ml of whole blood taken from field case No. 11, and added with 10% sodium citrate solution in the same method stated above. This citrated whole blood were inoculated intravenously into Bali cattle (Cattle E). The symptom of animal were same as original case on 18 days after inoculation, and was slaughtered on 2 days after appearance of symptom. Just before slaughter, 200 ml of citrated whole blood of cattle E were inoculated intravenously into one healthy Bali cattle (Cattle F), but this cattle did not show any symptoms. The results of inoculation experiments were shown in Table 2.

In order to provoke the disease using Dexamethason (DM), experiments were carried out. Cattle B and F which were inoculated intravenously with citrated whole blood of diseased Cattle-C and -E, respectively which had been described already. Both animals did not show any symptom within 140 and 110 days after inoculation, respectively. DM was injected 7 times intravenously one time per day. The dosage of one time was 0. 1 mg per kg of body weight, namely 25 ml and 20 ml of DM solution were injected intravenously into cattle B and F. Both cattle were slaughtered on 3 days after last injection, and were examined histopathologically.

Method of histopathology was same as that of field cases.

Table 2. Cases used in inoculation experiments of Rama Dewa

No. of cattle inoculated	Sex Female(F)	Age (Month)	Materials used for inoculation	Route of inoculation	Dosage of inoculum (ml)	Incubation (days)	Disease course (days)	Days from inoculation to collection of serum	Main clinical symptoms: Fever	Anorexia	Lacrymation and nasal discharge	Erosion of mouth cavity	Diarrhea	Characteristic pathological changes	Remarks
A	F	12	Citrated whole blood of field case No.15	iv	50	5	3	6	+++	+++	++	+++	+++	+++	Case No. 16 in autopsied cattle
B	F	14	" "	sc	50			6	-	-	-	-	-	-	used for DM experiment
C	F	12	Citrated whole blood of cattle A	iv	100	12	5	17	+++	+++	++	++	+	+++	Case No. 17 in autopsied cattle
E	F	12	Citrated whole blood of field case No.11	iv	100	12	6	18	+++	+++	++	++	+	++	Case No. 18 in autopsied cattle
F	F	10	Citrated whole blood of cattle E	iv	200			22	-	-	-	-	-	-	used for DM experiment

2. Clinical signs and macroscopic findings

In 3 cases (Nos. 16, 17 and 18) of Bali cattle which were positive in inoculation test, clinical signs such as high fever, anorexia, lacrymation, nasal discharge and erosion of mouth cavity were observed, but diarrhea was severe in only one case (Table 2). In macroscopic findings, necrotic and erosive lesions were present in such upper alimentary organ as hard palate (Nos. 16 & 18), tongue, pharynx (Nos. 16, 17 & 18) and oesophagus (Nos. 16 & 18). Hemorrhage was found in some parts. The changes of rumen were slight.

In Case No. 16, abomasum and intestine were diffusly congested, and showed hemorrhage and erosion in some parts. The changes of large intestine were prominent. Changes of low alimentary organ were slight in Nos. 16 and 17. Mucous membranes of nose, larynx and trachea were highly congested and edematous in all cases.

Subctaneous lymph nodes showed congestion and swelling although their degrees somewhat varied. Spleen enlarged, and hemorrhage were present in the parenchym (Nos. 16 & 17). Liver enlarged slightly, mottled and was greyish brown in colour. Macroscopically, changes of kidney were not characteristic, but marked hemorrhage was present in urinary bladder. Lungs of all cases showed normal appearance in nacked eye.

3. Microscopic findings

The changes of stratified squamous epithelium of oral mucosa were almost same as seen in field cases, Nos. 11 and 15 which were original cases in inoculation experiments. In all experimental cases, there was prominent coagulation necrosis and erosive lesions of showing detachment of necrotic epithelum and liquefaction with infiltrated neutrophiles in some parts. In contact with such necrotic lesions, a lot of lacunae containing lymphoid cells and degenerated epithelium were present, and most of the cells of Stratum germinativum disappeared and remainings showed degenerative changes such as pycnosis and karyorrhexis (Fig. 10). Lymphoid cells proliferated markedly in lamina propria and lymphoid vasculitis were observed in small-sized arteries, arterioles and related veins.

Such epithelial changes as seen in oral mucosa were also present in oesophagus. A lot of lacunae found in Stratum germinativum, degeneration and decrease of basal cells, and necrotic changes of extending from Stratum germinativum to epithelial surface were noted (Fig. 11). Marked infiltration of lymphoid cells was also observed in lamina propria of oesophagus.

In one case in which changes of low alimentary organ were prominent, lymphoid cells proliferated diffusly in mucosa propria and submucosa, especially in that of large intestine. They infiltrated perivasculary, and lymphoid vasculitis was seen.

In tracheae of all cases, degeneration and desquamation of epithelium and congestion were observed, and lymphoid cells infiltrated in perivascular spaces and interglandular tissues. Pharynx showed same changes.

Changes of the liver were prominent in all cases. Lymphoid-cell proliferation, hyperplasia of reticular cells, increase of bile ducts and connective tissues were observed in Glisson's sheath. Lymphoid vasculitis were noted in portal vein (Fig. 27), and observation of hepatic lobules revealed cirrhotic change in No. 16 and focal necrosis in No. 17, respectively. In the kidneys, aggregation of lymphoid cells was slight, and no lymphoid vasculitis were present.

The proliferation of lymphoid cells was not so prominent in lymph nodes of all cases, but lymphoid vasculitis was noted in No. 18. Spleens of Nos. 17 and 18 showed atrophy and necrobiosis of follicle, and lymphoid cells proliferated in follicle and red pulp. Reticular cells also hyperplasied.

Although necked-eye appearance of lung was about normal, but microscopic examination indicated proliferation of lymphoid cells in alveolar septum, perivascular and peribronchial spaces. Lymphoid-cell accumulation and vasculitis were observed in vein and small-sized arteries.

In urinary bladder of Nos. 16 and 18, epithelial changes such as formation of lacunae and degeneration and desquamation of transitional epithelium (Fig. 13) and marked lymphoid-cell proliferation, hemorrhage and edema in lamina propria were observed. Vascular lymphoid-cell accumulation was also present

(Fig. 24). Adrenal glands denoted lymphoid-cell proliferation and vascular lymphoid-cell aggregation in medulla (Fig. 25).

Encephalitic changes such as vascular lymphoid-cell infiltration and slight increase of glia cells were observed in all cases, but the changes were variable from slight to moderate. In No. 18 of showing moderate degree, vascular changes were distributed in Cerebral hemisphere, Corpus striatum, Thalamus and Midbrain. Meningitis was also present.

Regarding to the cases of provocation experiment using Dexamethason, no changes of characteristic of this disease were detected in microscopic specimens.

4. Other findings

Intranuclear and cytoplasmic inclusion bodies were examined on epithelial cells of oral mucosa and oesophagus using hematoxylin-eosin staining, but they were not detected at all. Moreover, in the stamp-smears of spleen and lymph node stained with May. Greenwald Giemsa or Machiavello stainings, detection of Rickettsia-like bodies was tried, but confirmation of the bodies was not succeeded.

5. Summary of the findings of experimental cases

In 3 experimental cases, marked proliferation and infiltration of lymphoid cells were recognized throughout the body. Epithelial necrosis and vascular lymphoid-cell infiltration and lymphoid vasculitis were also present in all cases. Encephalitic changes of brain were also observed in all cases, but were not prominent. Above stated microscopic findings were same as that seen in original cases used in inoculation experiments.

Experiments to provoke the disease by use of Dexamethason did not show the positive results.

DISCUSSION

Fifteen field cases and 3 experimental cases of Rama Dewa were investigated histopathologically and some discussions were made as followings.

Characteristic histological changes of this disease were lymphoid-cell proliferation observed in lymphoreticular tissue, perivascular tissue, vascular wall, and subepithelial tissues. Such changes were exhibited as (a) marked proliferation of lymphoid cells distributing throughout the body, (b) vascular lymphoid-cell infiltration and lymphoid vasculitis, (c) necrosis of stratified squamous and transitional epithelium which may relate with lymphoid-cell proliferation.

In the known diseases which provide three histological elements stated above, malignant catarrhal fever (MCF) is the most important. Mattam (1923) stated in his report that Snotsiekte in cattle is an acute specific infectious disease characterized by a general hyperplasia of lymphoid tissue throughout the body, less frequently by inflammation, erosion and necrosis of various mucosa. Snotsiekte is another name of MCF. With regard to African form of MCF, Hunt & Billpus (1979) reported that morphological features of African MCF were same as that of North American form by Liggitt et al. (1978) and that of European form by Selman et al. (1974), and primary feature of the infection was generalized lymphocytic proliferation of most organs and tissues.

Of the histologic changes of MCF, Liggitt et al. (1978) pointed out 3 major components: (1) lymphoid hyperplasia, (2) epithelial lesions, and (3) vascular lesions. They (1980 a, 1980 b) suggested that both epithelial and vascular lesions have a intimate relation with lymphoid hyperplasia, and MCF may be cell-mediated immunopathologic desease. Selman et al. (1974) stated that the most striking histopathological features were infiltration of all tissues and organs with mononuclear cells and presence of severe vascultitis. Furthermore, there was reports on MCF of Asian countries, indicating histological findings stated above [Fujimoto et al. (1958), Ohshima et al. (1977), Vanselaw (1980) and Sobari et al. (1983)].

Vascular lessions have played an important role in histologic differential diagnosis of MCF. Fibrinoid-necrotizing vasculitis and lymphoid vasculitis which are present in small- and middle-sized arteries have been emphasized by Pierson et al. (1973, 1974), Selman et al. (1974), Rweyemamu et al. (1976), Ohshima et al. (1977) and Liggitt & DeMartini (1980a) in cattle, Denholm and Westbury

(1982) in farmed rusa deer, Wyand et al. (1972) in white-tailed deer, Ruth et al. (1977) in bison, and Boever & Kurka (1974) in greater kudu.

In the present cases, systemic vascular lesions with perivascular lymphoid-cell infiltration and lymphoid vasculitis were observed in all cases, but necrotizing vasculitis was very few. Moreover, such vascular lesions were present in small-sized arteries, arterioles and related vein, and not in middle-sized muscular arteries in the present cases.

Epithelial lesions have also been considered as characteristic of MCF. Liggitt and DeMartini (1980b) reported that multifocal, degenerative and necrotic epithelial lesions were associated with lymphoid cells. They emphasized that a characteristic changes in epithelial layer were distinct lacunae comprized of centrally located degenerated or necrotic epithelial cells, often accompanied by lymphoid cells.

In epithelial lesions of the present cases, we also recognized the lacunae of being same as that mentioned by above-stated authors in stratified squamous epithelium of oral and oesophagoal mucosa and transitional epithelium of urinary bladder.

Non-suppurative encephalitis has been considered as an important findings of MCF. In the present cases, such changes as vascular lymphoid-cell infiltration and slight increase of glia cells were observed in 4 out of 15 field cases and 3 experimental cases. As already been described by Jubb & Kennedy (1970), Ohshima et al. (1977) and Liggitt et al. (1978), the encephalitic changes were recognized as systemic lesions associated with the vascular cell proliferation occurring throughout the body. In the early period of present studies [Prabowo et al. (1982)], Rama Dewa had been discriminated from MCF by them, because of the low frequency of encephalitic lesions. But at present period, this disease could be grouped under the same category of MCF from the reasons stated already.

Although our inoculation experiments were small in scale, we succeeded to transmitt the disease using large volume of blood and intravenous root. This method has usually been used in the transmission of MCF [Pierson et al. (1974, 1978, 1979), Zimmer et al. (1981) and Castro et al. (1982)]. The histological changes of 3

experimental cases accorded to that of original cases which were nearly indentical with MCF histologically.

MCF was classified clinically into 4 forms of the disease, the peracute, the intestinal, the head and eye, and the mild form by Götze (1930), and this classification has still been used at present [Callis et al. (1982)]. On the other hand, 2 forms of the conditions are recognized in MCF. Namely (1) Wildbeest-derived MCF also known as African form of MCF and (2) sheep-associated MCF, European or North American MCF. The two forms of MCF are indistinguisable clinico-pathologically [Pierson et al. (1978)], but the agent and pathogenesis of African form of MCF has been clarified by Plowright et al. (1960, 1963), Plowright (1968), Rweyemamu et al. (1974, 1976), Reid et al. (1975) and Mushi et al. (1981).

With regard to sheep-associated MCF, Götze and Liess (1929) could infect to 16 out of 50 cattle kept in intimate contact with apparently healthy sheep. They thought that sheep might be reservoirs of MCF virus. But, at present time this theory has not yet been confirmed. According to Mushi and Purangirwa (1981), no causative agent has been isolated from either carrier sheep or sick cattle in European form of MCF. They proposed that only African form of MCF should be referred to as "true MCF".

On this occassion, we propose the name of Malignant Catarrhal Fever Complex (MCFC), and Rama Dewa should be included in this disease complex as Rama Dewa form. At present time, Rama Dewa form is found in only Bali cattle. Recently, the disease of Bali cattle of being same as Rama Dewa occurred in Province of Bengkulu and Sumatra Selatan, Island of Sumatra [Prabowo et al. (1982, 1984)], and they are felling the necessity of new name of the disease from the point of diagnostic services in the field.

Of the differential diagnosis of the present disease, most important is the "Seputih Raman disease" the first enzootic of Rama Dewa. We compared with the experimental cases of the disease reported by Soeharsono & Darmadi (1976). Pathological findings of both diseases resembled in some points, however, so far as the results of transmission experiments concern, the Rama Dewa seemed to be different from "Seputih Raman disease".

Jembrana disease which occurred in Island of Bali is also important to be differentiated. It has affected only Bali cattle, and its clinical symptoms and pathological changes resembled to Rama Dewa [Temadja & Soeharsono (1980)]. The causal agent of Jembrana disease is now believed to be a Rickettsia [Budiarso & Hardjosworo (1977), Hardjosworo (1979), The Merck Vet. Manual (1979)]. Detailed histological comparison should be carried out after complete clarification of etiological agent.

Infectious bovine rhinotracheitis (IBR) [Curtis (1966), Van Dreumell (1974)] and bovine diarrhea - mucosal disease [Ramsey (1953, 1956), Inui et al. (1978)] are the important diseases in the differential diagnosis from Rama Dewa. Marked lymphoid-cell proliferation of distributing throughout the body makes it easy to differentiate Rama Dewa from both disease. Serum neutralizing antibody test against IBR virus was performed on the serum of present experimental cases and some of field cases of Rama Dewa by the Staffs of Division of Exotic Disease, National Institute of Animal Health, Japan (1983). As the results this antibody was not detected. Rama Dewa simulates Rinderpest in many respects [Khater (1983)]. But, Rinderpest is epidemic in occurrence, and infects not only Bali cattle but also other races of cattle and sheep. Rama Dewa affected only Bali cattle.

SUMMARY

Fifteen field cases and 3 experimental cases of Rama Dewa were investigated histopathologically. Characteristic histological changes of this disease were lymphoid-cell proliferation in lympho-reticular tissue, perivascular tissue, vascular wall, and subepithelial tissues. Such changes were exhibited as (a) marked proliferation of lymphoid cells distributing throughout the body, (b) vascular lymphoid-cell infiltration and vasculitis, (c) necrosis of stratified squamous and trasitional epithelium which may relate with proliferated lymphoid cells.

Attempt to compare Rama Dewa with malignant catarrhal fever which provids 3 histological components stated above, were carried out. As the results authors proposed the name of "malignant catarrhal fever complex", and Rama Dewa should be included in this disease complex.

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EXPLANATION OF PLATES

PLATE I

Fig. 2. Corneal opacity, reddening of conjunctiva and nasal mucosa, and lacrimation. After 3 days this cattle died. Case No. 11.

Fig. 3. Shallow erosions (arrows) of hard palate. Case No. 2.

PLATE II

Fig. 4. Erosions (arrows) of tongue and reddening of trachea. Case No.2.

Fig. 5. Tongue covered with necrotized mass of epithelium (arrows). Erosions were also present. Case No. 12.

PLATE III

Fig. 6. Necrosis of stratified squamous epithelium and marked proliferation of lymphoid cells in epithelial layer and lamina propria of tongue. Case No. 2, HE staining, x 100.

Fig. 7. Tongue. Numerous round lacunae (arrows) containing lymphoid cells or degenerated epithelial cells in Stratum germinativum, and necrosis of epithelium in Stratum spinosum. Marked proliferation of lymphoid cells is observed beneath Stratum germinativum. Case No. 2, HE staining, x 400.

Fig. 8. Tongue. Lacunae (arrows) of irregular in form presenting in Stratum germinativum. Vacuolar degeneration and intense eosin staining of cytoplasma and pycnosis of epithelial cells in Stratum spinosum. Case No. 15, HE staining, x 200.

Fig. 9. Tongue. Large magnification of Fig. 7, Lacunae (arrows) contain lymphoid cells and degenerated epithelial cells.

PLATE IV

Fig. 10. Tongue. Extensive coagulation necrosis of stratified squamous epithelium and marked formation of lacunae containing lymphoid cells. Prominent lymphoid-cell proliferation is observed in lamina propria. Case No. 18 HE staining, x 100.

Fig. 11. Cesophagus, Lacunae of being round and irregular in form in Stratum germinativum (arrows), and pycnosis of nuclei of epithelial cells in Stratum spinosum. Case No. 17, HE staining, x 400.

Fig. 12. Erosive lesion in oesophagus. Infiltrated lymphoid cells and edema of muscle layer (arrows) are present.
Case No. 2, HE staining, x 100.

Fig. 13. Lacunae (arrows) and lymphoid cells in transitional epithelium of urinary bladder. Case No. 15, HE staining, x 400.

PLATE V

Fig. 14. Marked proliferation of lymphoid cells in prescapular lymph node. Case No. 2, HE staining, x 200.

Fig. 15. Large magnification of Fig. 14. Lymphoid cells consisted of lymphoblasts (arrows) and lymphocytes. HE staining, x 1,000.

Fig. 16. Enlarged hepatic lymph node. Follicles disappear. Tissue of right side (arrow) is liver. Case No. 2, HE staining, x 40.

Fig. 17. Lymphoid cells profiferate around central artery of spleen. Case No. 1, HE staining, x 200.

PLATE VI

Fig. 18. Liver. Marked proliferation of lymphoid cells, hyperplasia of reticular cells and increase of bile duct in Glisson's sheath. Case No. 1, HE staining, x 100.

Fig. 19. Higher magnification of Fig. 17. HE staining, x 200.

Fig. 20. Kidney. Marked proliferation of lymphoid cells in perivascular, periglomerular and interstitial tissues. Case No. 4, HE staining, x 200.

Fig. 21 Lymphoid cell proliferation in alveolar septum of lung. Capillaries contain lymphoid cells. Case No. 1, HE staining, x 400.

PLATE VII

Fig. 22. Heart. Necrosis of cardiac muscle and aggregation of lymphoid cells with myogenic giant cells. Case No. 7, HE staining, x 100.

Fig. 23. Large magnification of Fig. 22. Arrows show myogenic giant cells. HE staining, x 400.

Fig. 24. Urinary bladder. Perivascular and diffus proliferation of lymphoid cells in lamina propria. Case No. 18, HE staining, x 400.

Fig. 25. Adrenal gland. Vascular lymphoid-cell infiltration observed in vein of medulla. Case No. 16, HE staining, x 400.

PLATE VIII

Fig. 26. Tongue. Lymphoid vasculitis showing infiltration of neutrophils and lymphoid cells in small-sized artery. Case No. 12, HE staining, x 400.

Fig. 27. Liver. Lymphoid vasculitis observed in portal vein. Case No. 11, HE staining, x 400.

Fig. 28. Large intestine. Necrotizing vasculitis in small-sized artery. Lymphoid cells infiltrate in and around blood versel. Case No. 1, HE staining, x 200.

Fig. 29. Large intestine. Lymphoid vasculitis showing perivascular and subintimal lymphoid-cell infiltration in small-sized artery. Case No. 1, HE staining, x 200.

PLATE IX

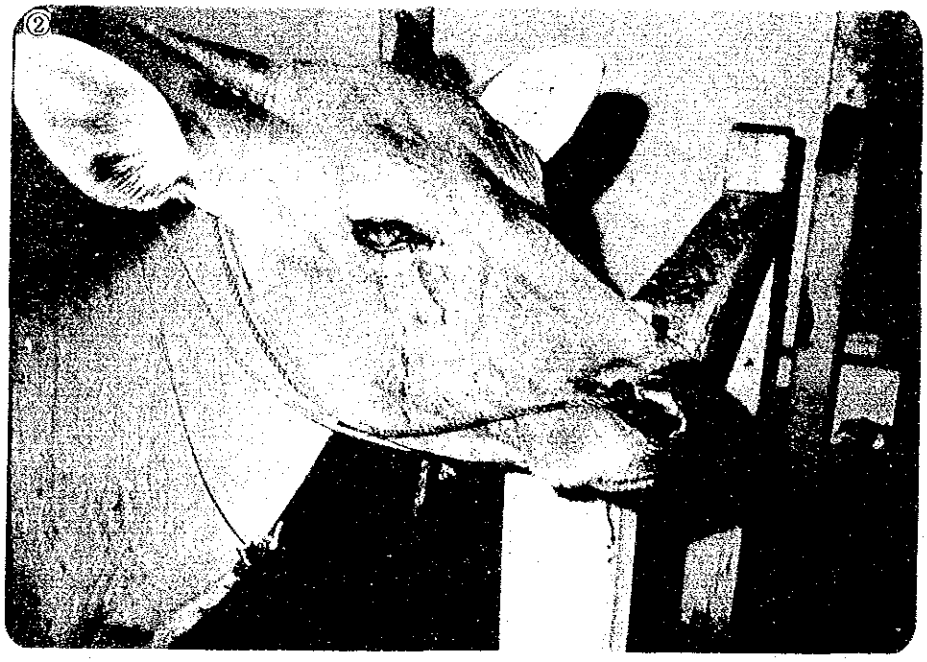
Fig. 30. Lung. Lymphoid vasculitis observed in vein. Case No. 8, HE staining, x 250.

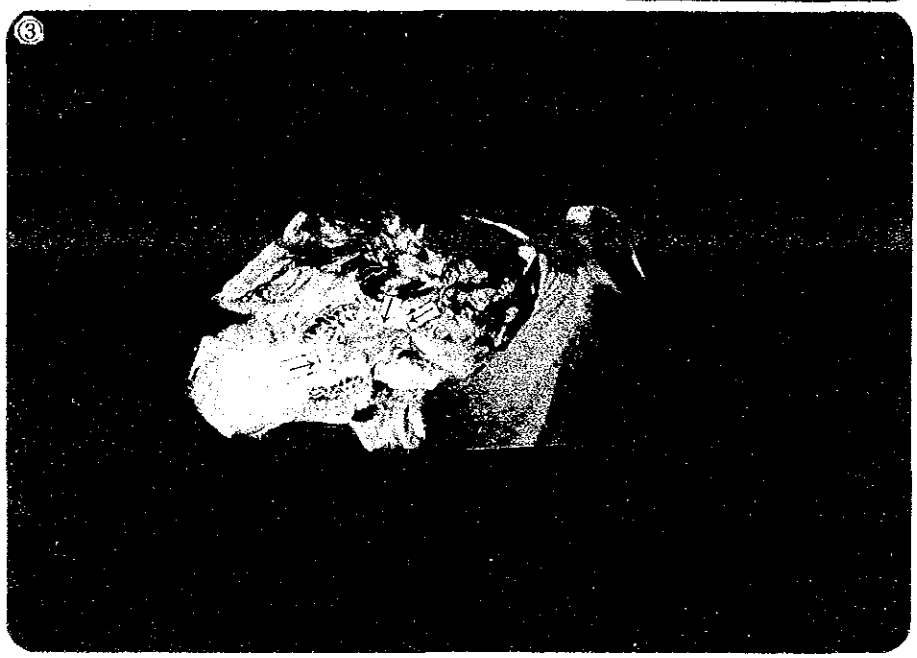
Fig. 31. Epididymis. Lymphoid-necrotizing vasculitis in small-sized artery. Case No. 15, HE staining, x 400.

Fig. 32. Brain. Vascular lymphoid-cell infiltration in Cerebral hemisphere. Case No. 11, HE staining, x 100.

Fig. 33. Brain. Higher magnification of Fig. 32.

PLATE I





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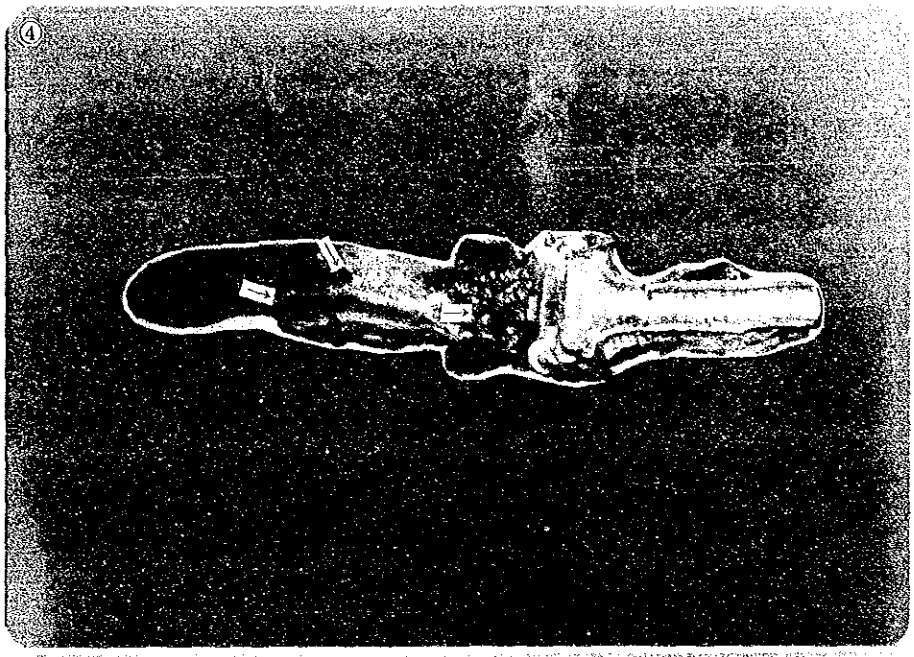


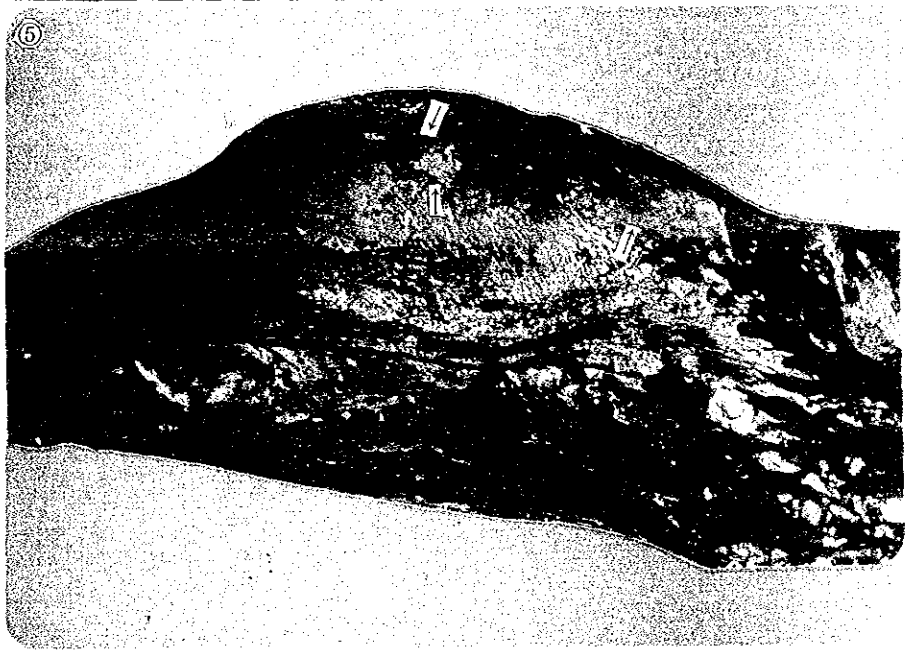


PLATE Ⅲ

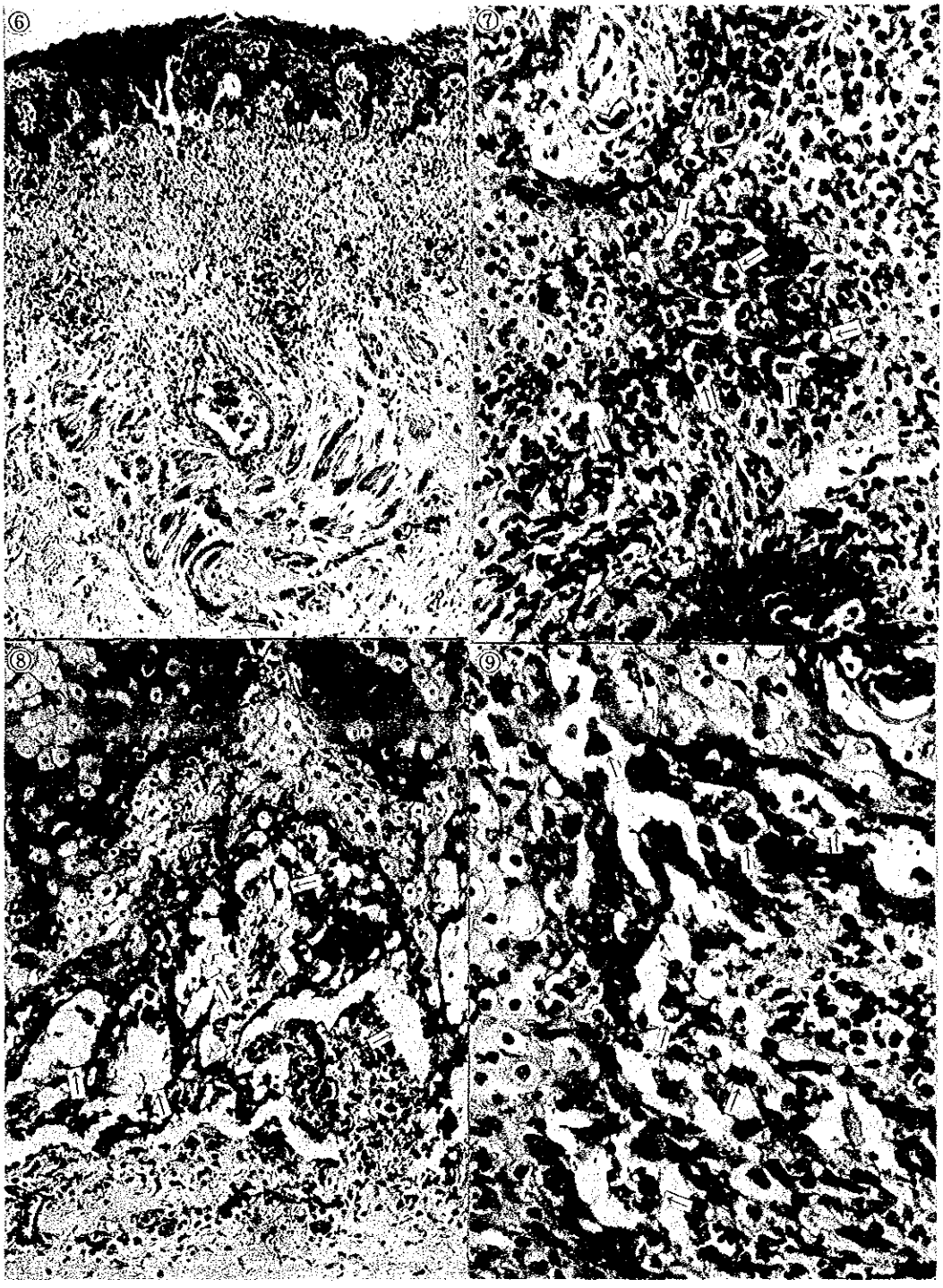


PLATE IV

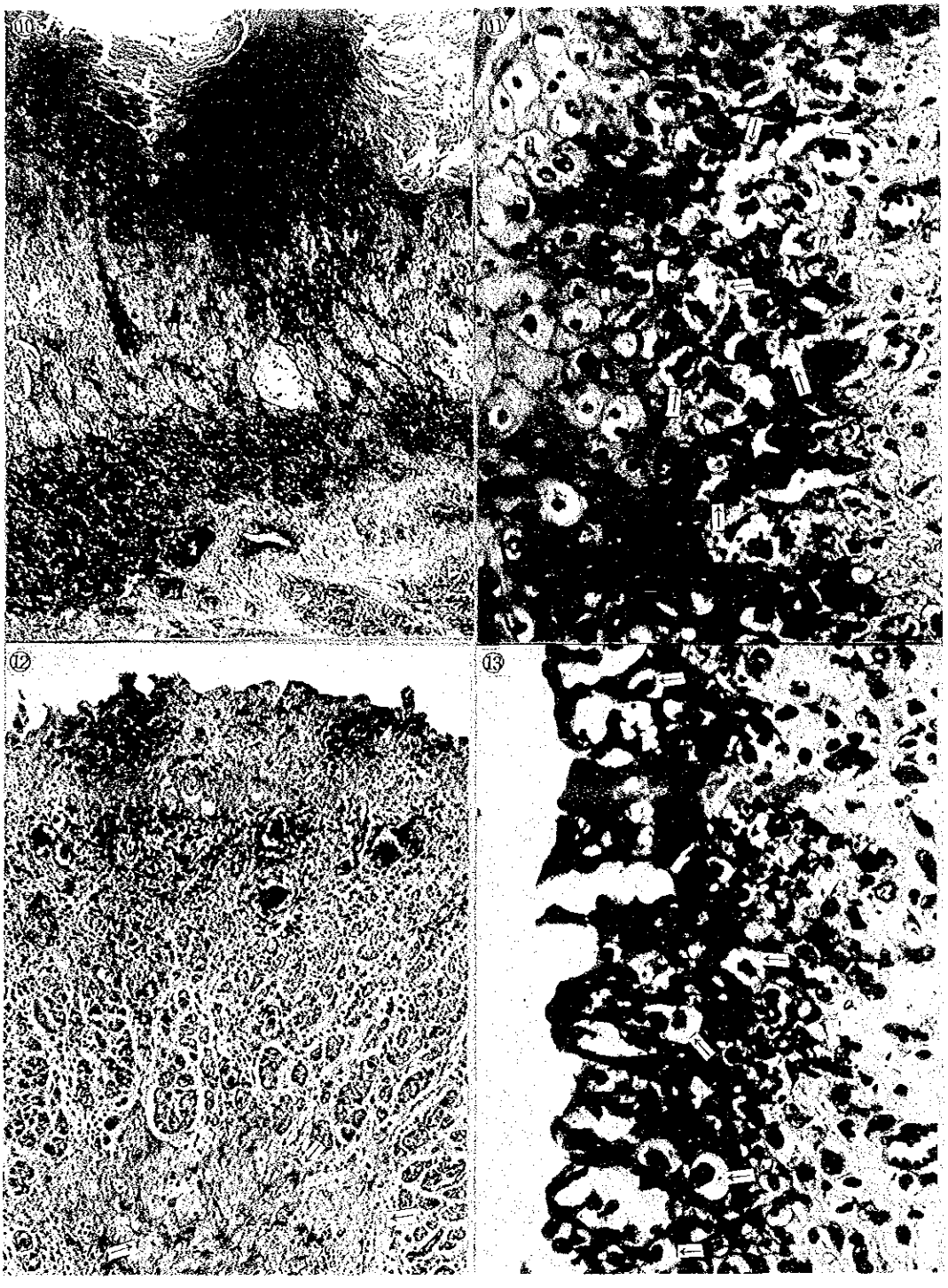


PLATE V

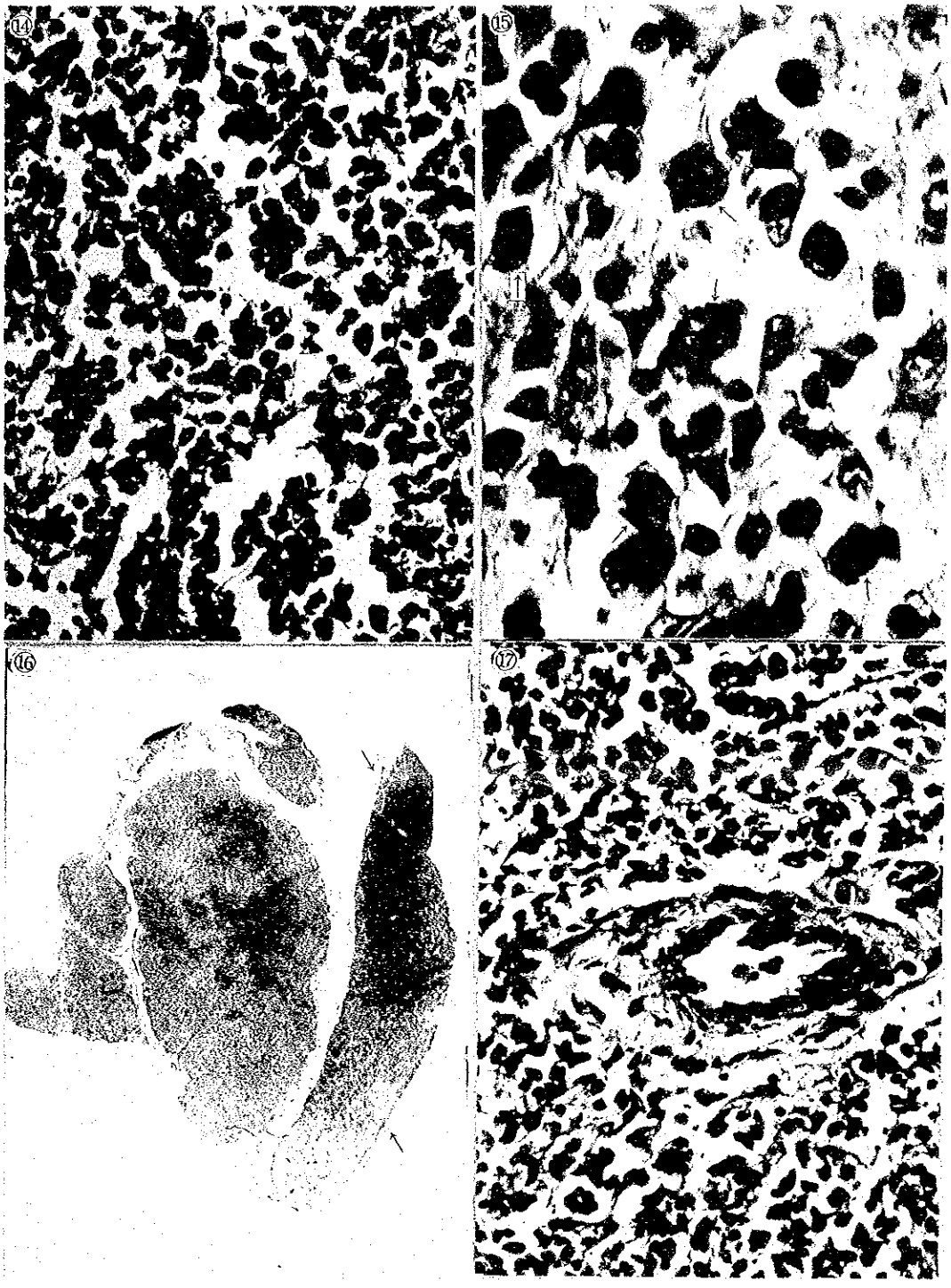


PLATE VI

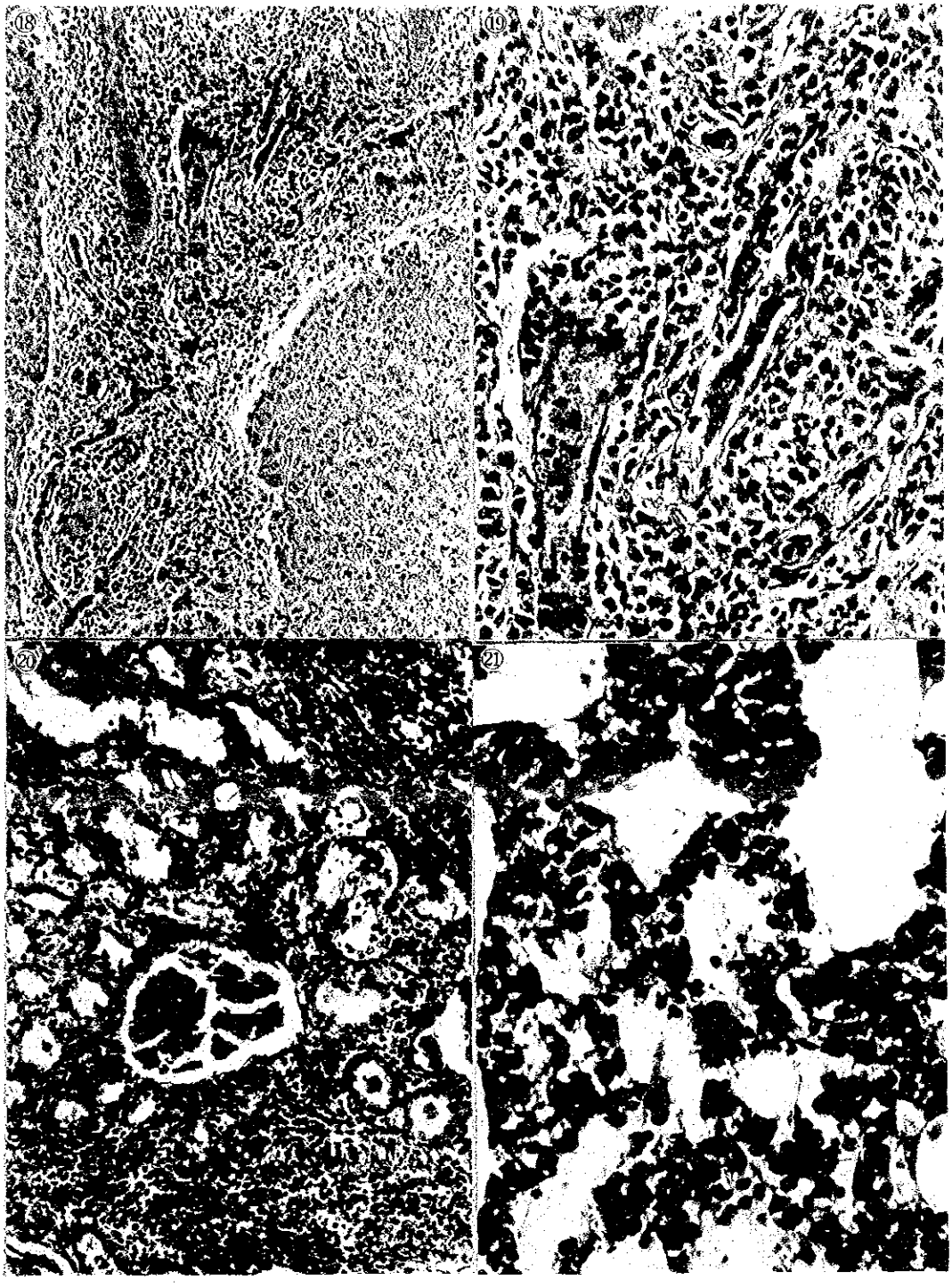


PLATE VII

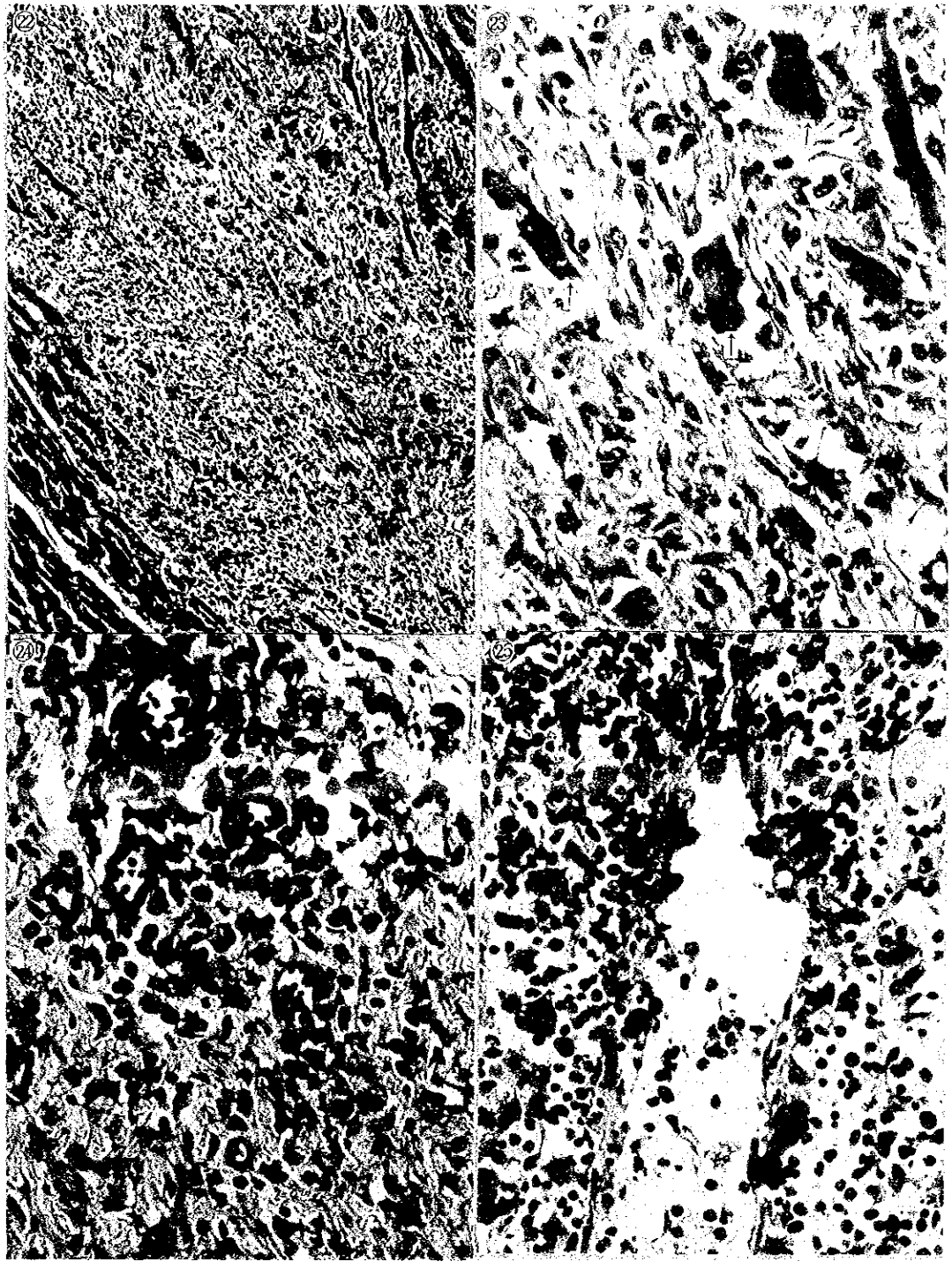


PLATE VIII

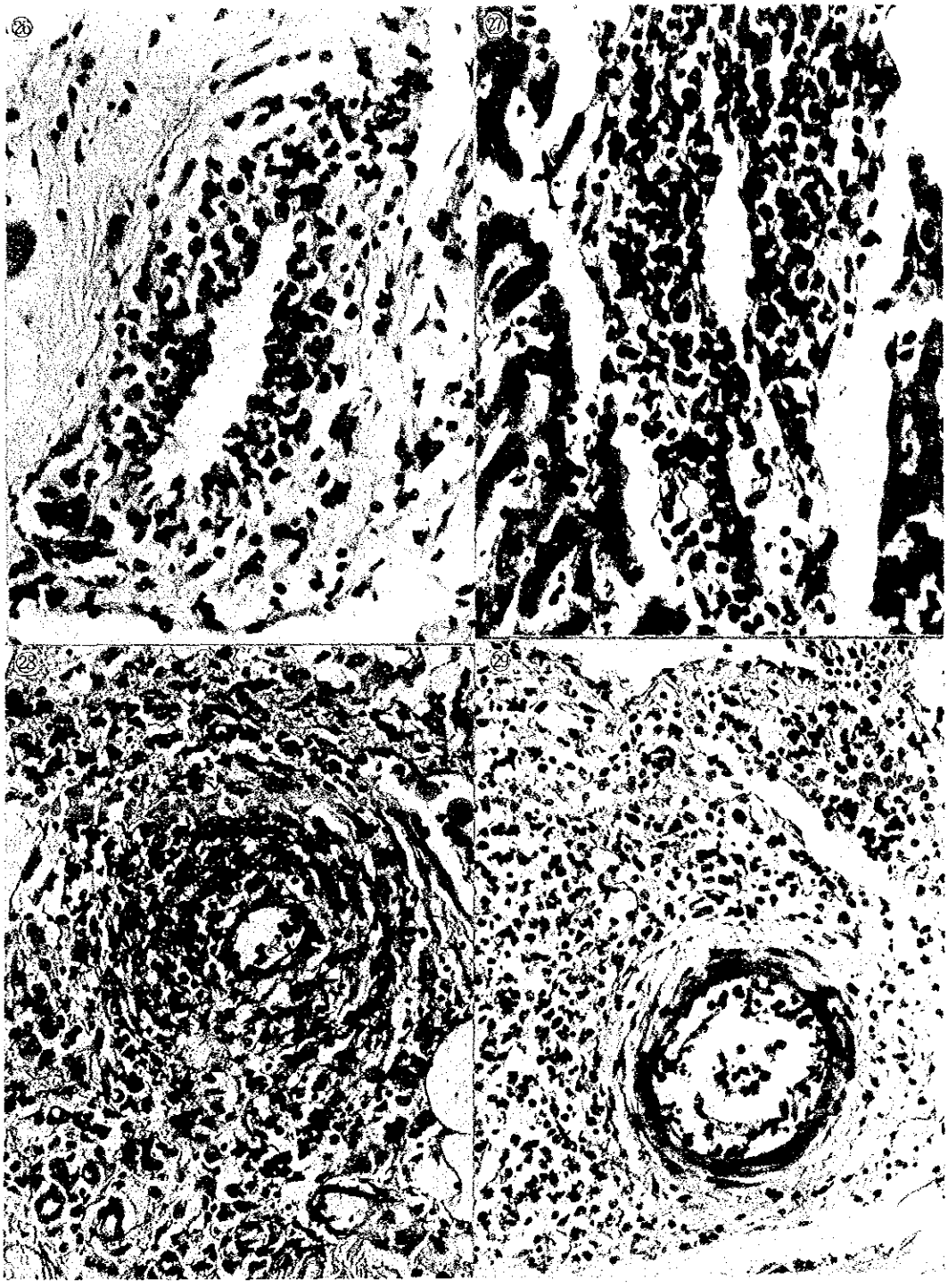
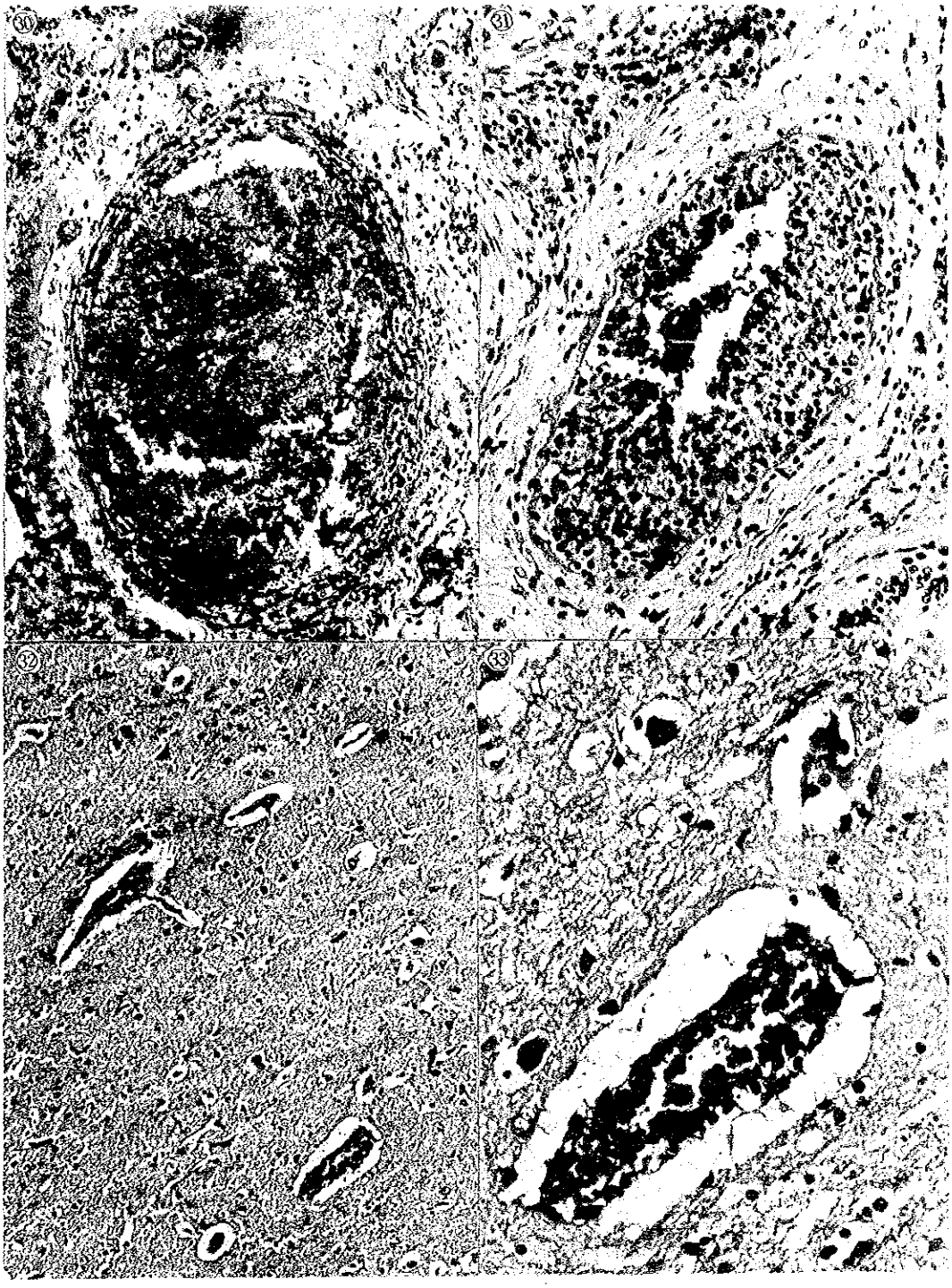


PLATE IX



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