ADRENAL CORTICAL ATROPHY IN THE DOG
REPORT OF THREE CASES *

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Pathologic alteration of the adrenal cortex morphologically comparable to adrenal cortical atrophy causing some cases of Addison's disease in man apparently occurs rarely as a spontaneous lesion in domestic animals. Reports of such a pathologic entity in the dog were not found in the available literature. Nelson and Woodard[1] mentioned that they too failed to find reference to this lesion in animals. Nieberle and Cohrs[2] stated that no unequivocal case of the counterpart of Addison's disease has been reported in domestic animals. These authors do not, however, specifically mention adrenal cortical atrophy in domestic animals.

Three cases of adrenal cortical atrophy in the dog were encountered at necropsy. This morphologic description is presented because of the apparent rarity of the entity in dogs and because the observations should be of interest to comparative pathologists.

CLINICAL FEATURES

The dogs were brought to veterinary attention because of varying periods of illness marked by listlessness, anorexia, emesis, and weight loss. No abnormal rectal temperatures were recorded. Hematologic findings in two of the dogs were consistent with extreme hemoconcentration. The blood urea nitrogen of these same dogs was elevated to above 75 mg. per cent. Urinalyses were negative. Unfortunately, no additional laboratory data were determined.

Euthanasia was performed in case of dog A about 25 days after the first reported signs of illness. Dog B died suddenly after an illness of about 1 week's duration. Dog C died about 1 month after the onset of marked clinical disease.

NECROPSY FINDINGS

Dog A, a 3-year-old male, was necropsied immediately following euthanasia. The animal was in a fair state of nourishment and showed evidence of marked dehydration. No gross lesions were observed in the pericardium, heart, lungs, thymus, thyroid gland, pancreas, gall-

* Paper No. 2892, Scientific Journal Series, Minnesota Agricultural Experiment Station. Received for publication, September 2, 1952.

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bladder, spleen, prostate, urinary bladder, testes, and brain. The liver was dark red and of normal consistency. Its cut surface was somewhat mottled. Several small ulcers were present in the oral mucosa. Small hemorrhagic erosions were found in the gastric mucosa at the pylorus. The small intestine was hyperemic. The kidneys were pale and slightly swollen. Their capsules were not adherent.

Because the adrenal glands were not identified at necropsy, the entire dorsal retroperitoneal area was removed and fixed in 10 per cent formalin. Careful gross serial sectioning later revealed remnants of the adrenal glands. They measured 1.4 by 0.6 cm., were dark brown, and appeared to consist of medullary tissue only. The capsules appeared thickened, smooth, and white.

Microscopic examination of sections of myocardium, lung, gallbladder, pancreas, parathyroid gland, prostate, and small intestine revealed no noteworthy changes. Scattered foci of myeloid metaplasia were found in the spleen and in several lymph nodes. Several small foci of lymphocytes were observed in the thyroid gland. Varying degrees of tubular degeneration were noted in the kidneys. There was considerable centrolobular atrophy and congestion in the liver.

Each adrenal gland exhibited about the same degree of parenchymal destruction and stromal collapse. In most areas the cortical parenchyma was entirely gone and the stroma was condensed to a thin layer. An occasional focus contained vacuolated cells which were believed to be remnants of the zona glomerulosa. There were a few larger masses of eosinophilic cortical cells which showed degenerative changes. Only an occasional area was noted where the hyperchromatic cells suggested attempts at regeneration. Fine granules of yellowish brown pigment were found in some portions of the contracted cortex. The remaining cortical parenchyma and the collapsed reticular stroma were rather densely infiltrated with lymphocytes, plasma cells, and other large mononuclear cells. There was no apparent fibrosis of the cortex with the exception of one small area which resembled a granulomatous process. The medullas were intact.

The anterior lobe of the hypophysis was examined microscopically by median sagittal sections stained with hematoxylin and eosin and by the azocarmine technique. When compared with similarly prepared sections of the hypophysis from normal adult dogs, the anterior lobe showed a definite decrease in acidophil cells. This change was accompanied by a decided increase in the chromophobes. Differential cell counts were not made on any of the sections from this or the subsequent cases.

Post-mortem examination of *dog B,* an 18-months-old ovariectomized female, was completed within 4 hours after death. The animal was well developed and showed no evidence of appreciable weight loss. The mesenteric vessels were markedly congested. The left lobe of the thyroid gland was about one half the size of the normal-appearing right lobe. The tonsils were moderately enlarged. The thymus was prominent and was dotted with petechiae. The pericardium, heart, lungs, mediastinal lymph nodes, pancreas, spleen, gallbladder, kidneys, lower urinary tract, and brain presented no significant gross changes. The liver was of normal size. Its cut surface was mottled with dark red and pale areas. The oral mucosa was bright red and contained several small erosions. The gastric mucosa was reddened. A rather deep ulcer, 1 cm. in diameter, was present in the duodenum near the pyloric ring. Several smaller ulcers were present in the adjacent mucosa, one of which contained a firm blood clot, 1 cm. in diameter. A large amount of tarry, partially digested blood was present in the jejunum. Large, soft, black clots filled the colon. The mesenteric lymph nodes were firm and slightly enlarged.

The adrenal glands were found to be small, dark brown masses embedded in the perirenal fat. The left adrenal gland measured 2 cm. long; the right measured 2.5 cm., was flattened, and appeared to consist of capsule only at one pole. On cross section the capsules were thickened and white. The cortices were not distinctly identifiable since the bulk of the glands consisted of normal-appearing, brown, medullary tissue.

No microscopic changes of note were found in sections of myocardium, kidney, pancreas, stomach, spleen, lung, and thyroid gland. The liver showed marked centrolobular atrophy and congestion. The duodenal ulcer was found to be an acute inflammatory process with necrosis which extended to the serosa, where there was focal peritonitis. The mesenteric lymph nodes and the thymus exhibited lymphocytic hyperplasia and areas of hemorrhage.

The normal architecture of the adrenal cortices was so completely destroyed that in many areas only the capsule and a thin layer of condensed stroma remained. An occasional mass of foamy, degenerating parenchymal cells was observed. Several small nodules of cortical cells were situated within the capsule. The collapsed reticular stroma was rather densely infiltrated with lymphocytes and plasma cells. In the juxtamedullary region the stromal collapse was less advanced so that many dilated, blood-filled vascular channels remained. Small amounts of finely granular yellowish brown pigment were present in the outer

portions of the thinned cortex. The microscopic changes were equally severe in both adrenal cortices. No apparent alteration was observed in the medullas.

Sections of the anterior lobe of the hypophysis were prepared and examined as in the previous case. There was a decrease in acidophil cells and an increase in the chromophobes. Changes in the basophils were not determined in the sections examined.

Dog C, a 5-year-old ovariectomized female, showed evidence of weight loss. No gross lesions were observed in the heart, lungs, liver, pancreas, spleen, stomach, and intestines. The cortices of the kidneys were slightly decreased in thickness. The right adrenal gland was not found. The left adrenal gland was only about one quarter the normal size.

Microscopic examination of sections of pancreas and lymph node revealed no changes. Sections of kidney contained scattered lymphocytic foci in the interstitial tissue of the cortex. Only a portion of a cross section of the left adrenal gland was available for study, but the cortical changes were well demonstrated. The changes resembled those observed in dog A. The cortical parenchyma was completely destroyed and the stroma was contracted to a marked degree. Large vacuolated areas containing finely granular brownish pigment were scattered in the outer zone of the collapsed cortex. In several areas the remaining vascular channels were moderately distended with erythrocytes. Lymphocytes were diffusely infiltrated through the cortical remnant. Occasionally these cells were found in dense masses in the subcapsular zone. The medulla showed autolytic changes only. Only one section of the anterior lobe of the hypophysis, stained with hematoxylin and eosin, was available for microscopic examination. Changes similar to those found in dogs A and B were observed.

Comment

The microscopic features of the adrenal cortices in the three dogs are strikingly similar to those of adrenal cortical contraction in man as described by most pathologists.[3] The cortical destruction appears to be considerably more extensive and severe than that produced experimentally in dogs fed the insecticide, 2,2-*bis* (parachlorophenyl)-1, 1-dichloroethane (TDE).[1]

An increase in the number of chromophobes and a reduction in the number of acidophils and basophils in the anterior hypophysis have been reported in cases of Addison's disease in man.[4] Similar changes were observed in the three dogs in regard to the number of chromophobes and acidophils. This finding is in contrast to that of Nicholson[5]

for adrenalectomized dogs. He reported that no difference could be detected between the anterior hypophyses of adrenalectomized and of normal dogs.

At present the etiology of the spontaneous adrenal cortical changes in the dog appears to be as obscure as it is in man.

SUMMARY

Three cases of spontaneous adrenal cortical atrophy or contraction in the dog are reported. A morphologic resemblance of the lesion to the adrenal changes in some cases of Addison's disease in man is evident. Likewise, the cellular changes in the anterior hypophysis are similar to those which have been described in Addison's disease. This is believed to be the first report of this adrenal lesion in the dog.

I wish to express my gratitude to Dr. Carl F. Schlotthauer, Rochester, Minnesota, for providing me with the data and microscopic preparations from dog C. In addition I wish to thank Dr. J. R. Dawson, Jr., University of Minnesota, Minneapolis, Minn., for his suggestions in the preparation of the manuscript.

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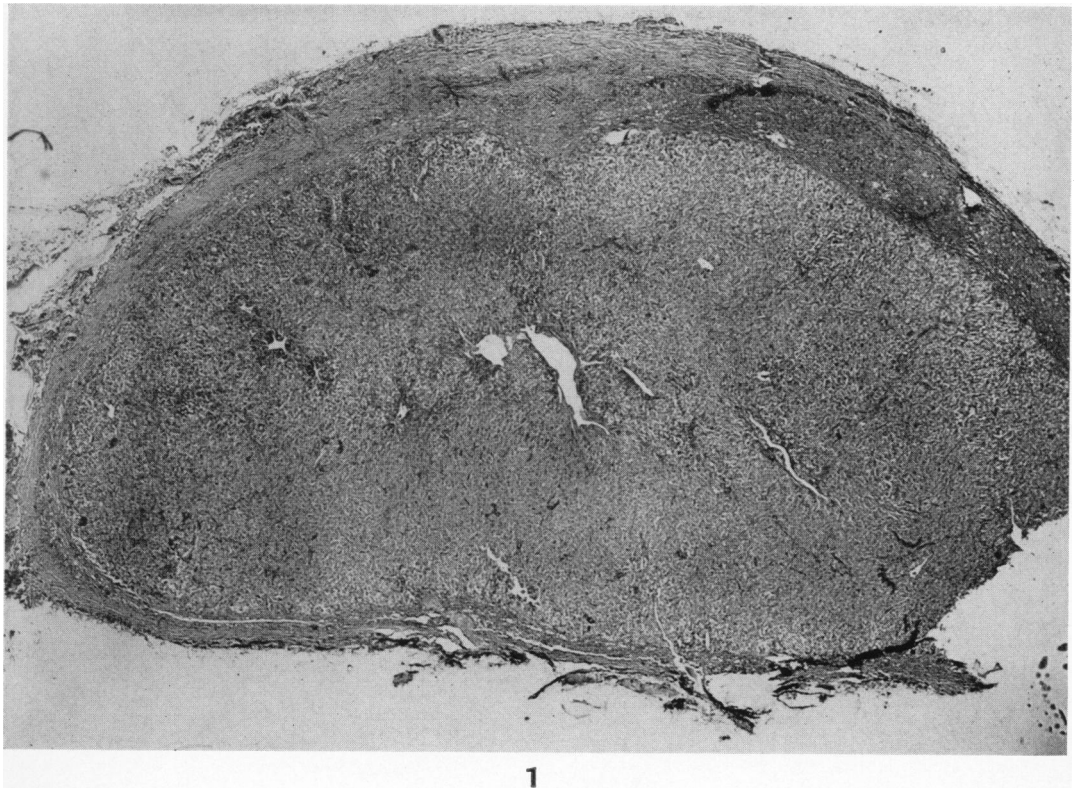
[*Illustrations follow*]

LEGENDS FOR FIGURES

FIG. 1. Dog A. Cross section of an adrenal gland showing extreme cortical destruction and intact medulla. Hematoxylin and eosin stain. × 8.5.

FIG. 2. Dog. A. View of the collapsed adrenal cortex showing lymphocytic infiltration and foamy areas containing the granular material. The normal medulla is below. Hematoxylin and eosin stain. × 112.

FIG. 3. Dog B. Cross section of the left adrenal gland showing the extent of cortical damage. Hematoxylin and eosin stain. × 8.5.



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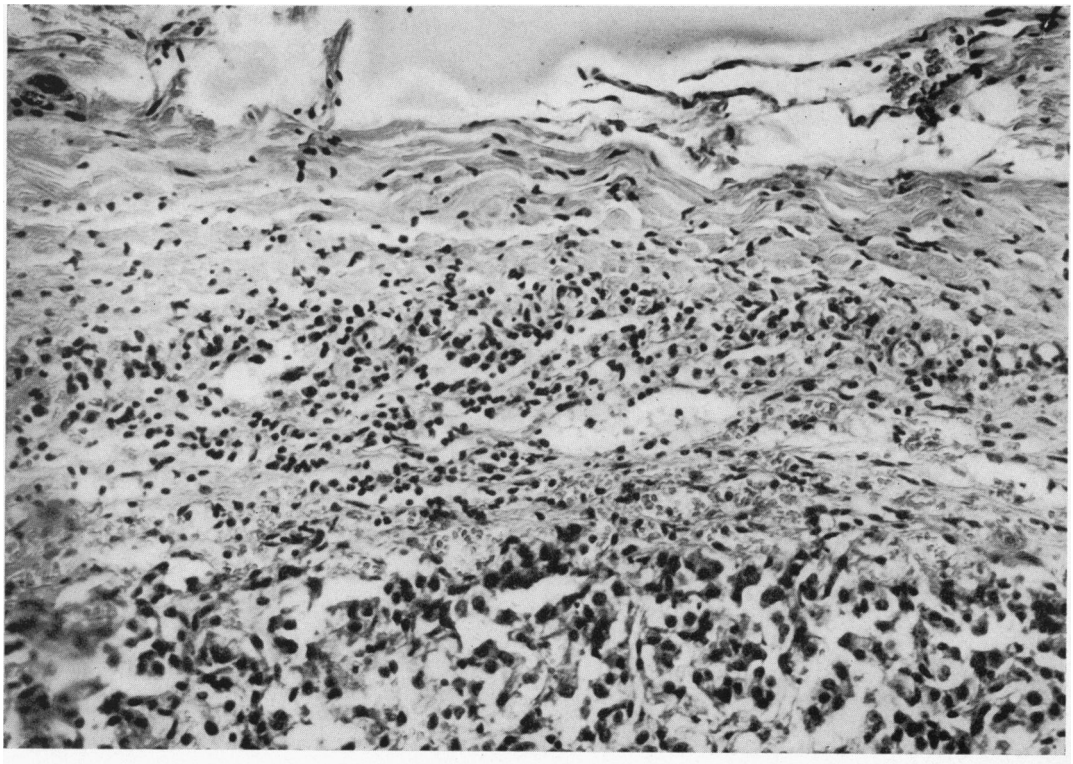

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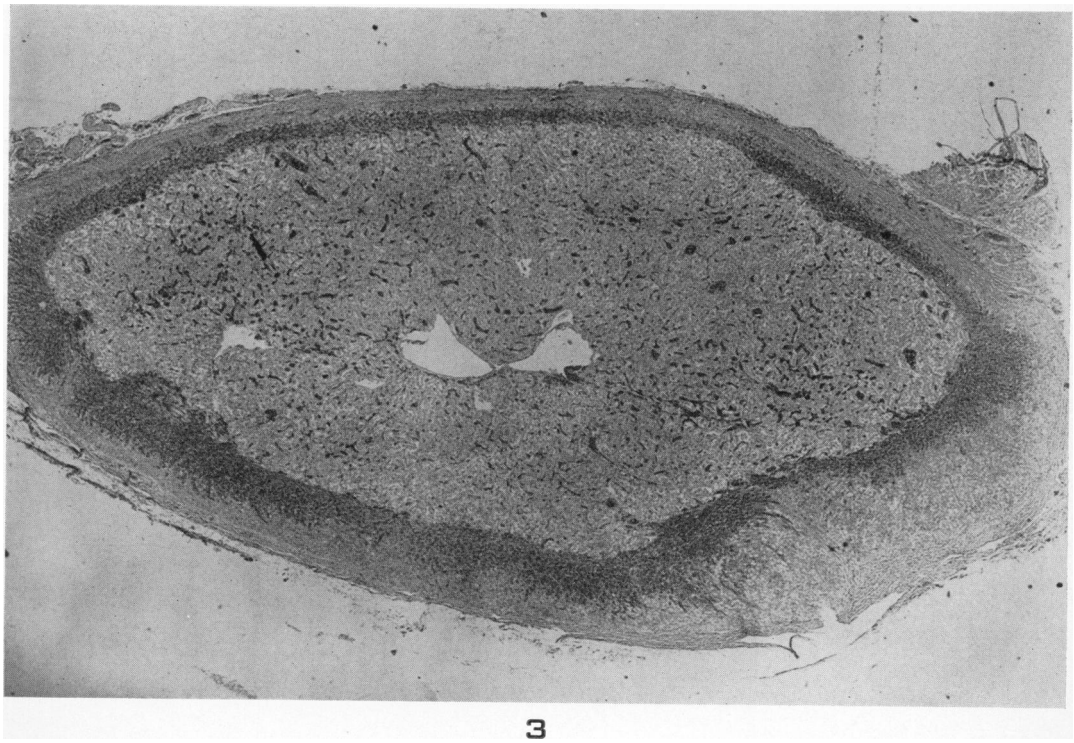

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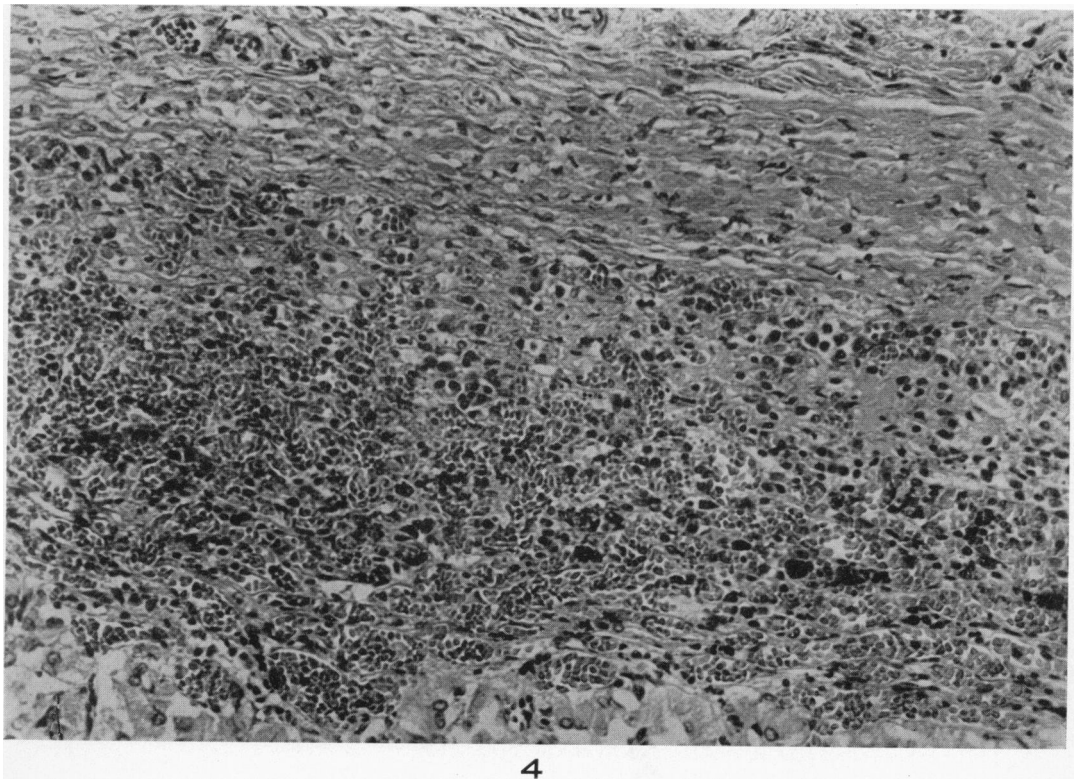
FIG. 4. Dog B. The entire thickness of the remaining adrenal cortex. Of note are the blood filled channels. The intact medulla is the narrow strip below. Hematoxylin and eosin stain. × 112.

FIG. 5. Dog C. Incomplete cross section of the left adrenal gland showing cortical collapse. Hematoxylin and eosin stain. × 12.

FIG. 6. Dog C. Section through the entire thickness of the collapsed cortex. Resemblance to Figure 2 may be noted. The medulla is at the right. Hematoxylin and eosin stain. × 112.



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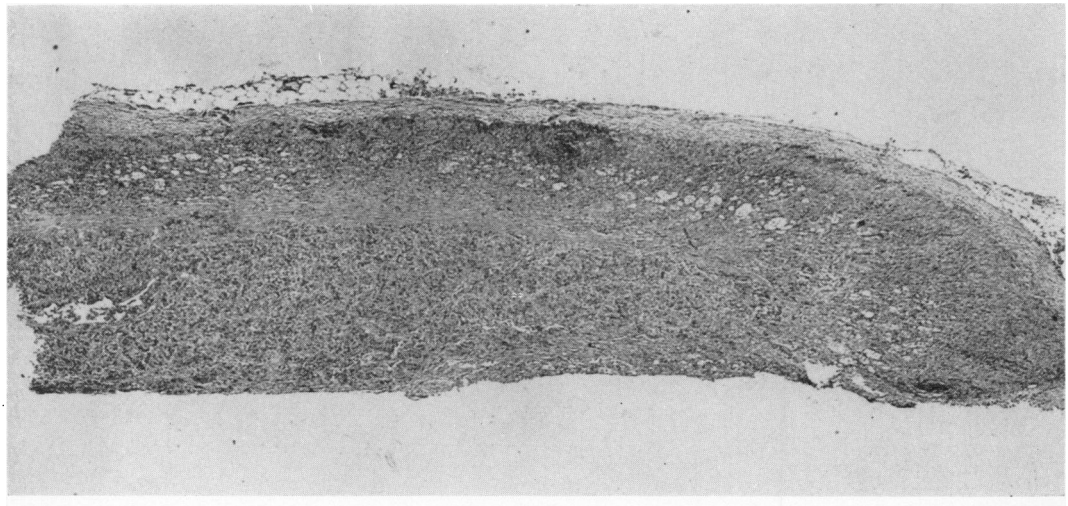

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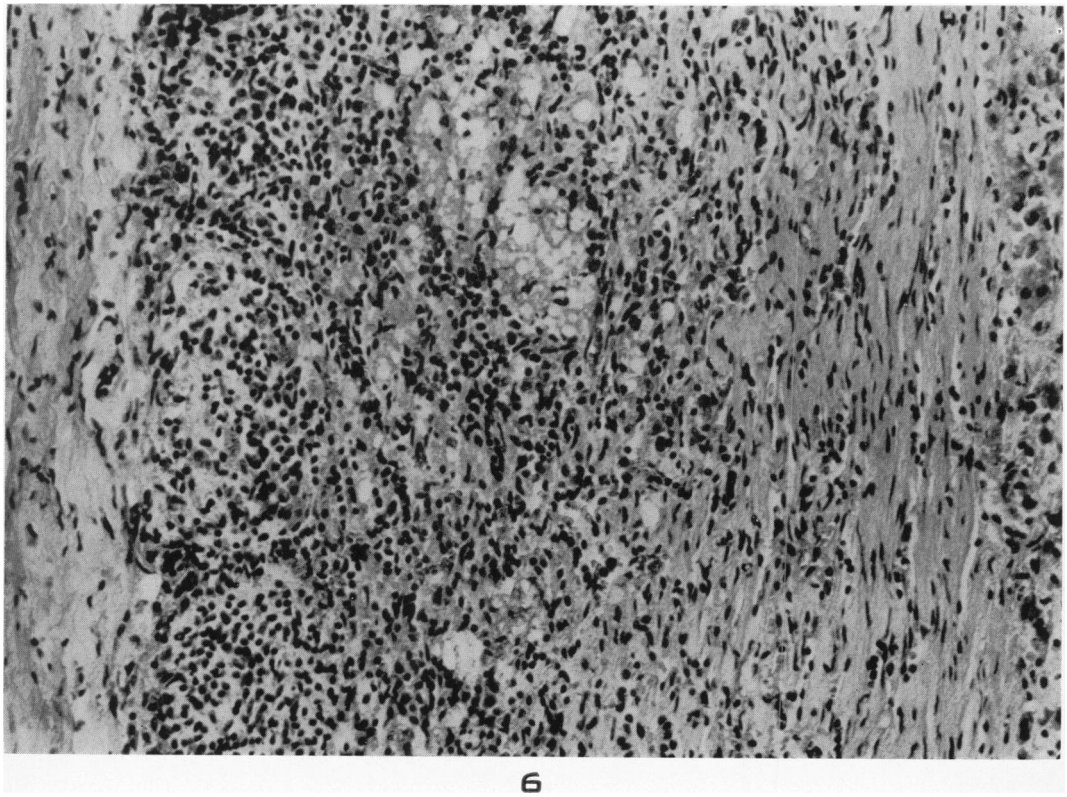

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