Systemic Vascular Lesions in Feline Infectious Peritonitis

Toshiharu HAYASHI, Naoaki GOTO, Reiji TAKAHASHI
and Kōsaku FUJIWARA

Department of Veterinary Pathology, Faculty of Agriculture,
University of Tokyo, Bunkyo-ku, Tokyo 113

(Received for publication December 14, 1976)

Abstract. Twenty-nine cases of feline infectious peritonitis were studied histopatho-
logically. It was notable that, in most cases examined, there existed degenerative and
proliferative changes in the blood vessels of different parts, especially in endothelial and
medial layers of fine and smaller veins or arteries with perivascular infiltration of neu-
троphils and mononuclear cells. Every case had varied amounts of ascites and/or hydrothorax
with fibrinous precipitates covering the entire surface of peritoneal and thoracic cavities
as well as visceral organs. Metaplasia and hyperplasia with syncytium formation were
remarkable in mesothelial and mesenchymal cells of the peritoneum and pleura. In most
cases multiple necrotic foci with infiltration of neutrophils and mononuclear cells and
hemorrhage were seen in the serosa, omentum and mesenteries, and the same lesions
appeared also in the parenchyma of the liver, spleen and mesenteric lymph nodes of some
cases.

Feline infectious peritonitis (FIP) is characterized by fibrinous inflammation in the
peritoneal cavity resulting in accumulation of a large amount of ascites with some
necrotizing lesions in the liver, spleen and some other organs. Since it was first de-
scribed as a disease entity by Wolfe and Griesemer [25], there have been many re-
ports on natural cases in the United States [2–4, 9, 15, 17, 18, 26], England [10, 11],
Canada [20], Netherlands [18], Germany [21], and also in Japan [12]. A member of
coronaviruses was suspected as the causative agent [22]. Pathogenesis of this disease,
however, especially the mechanism of production of ascites remains unclear.

We experienced autopsies of 29 cases which had been clinically diagnosed as FIP
during a period from 1969 to 1975. This paper describes the results of histopatho-
logical examination of these cases, in special reference to local and systemic vascular
lesions which might be of importance for the accumulation of ascites in FIP.

Materials and Methods

Twenty-nine cases which had been clinically diagnosed as FIP were derived from either Tokyo
University Animal Clinic or some practitioners in Tokyo area during a period from 1969 to 1975.
All cases had ascites and/or hydrothorax varying in amount, some showing fever, anorexia, de-
pression, diarrhea, anemia, dyspnea, blindness, icterus, leukocytosis, or body weight loss (Table 1).

Most cases were autopsied immediately after death, and tissue samples were fixed in buffered
formalin, pH 7.0. Paraffin sections were made by a routine procedure and they were stained with
hematoxylin-eosin (HE) and others if necessary.

Results

1. Gross findings

As shown in Table 1, in most cases
ascites were colored yellow-brown or green-yellow and the volumes varied from about 5 to 1,000 ml. Some cases had rather serous and transparent ascites, while others had albuminous ones containing some precipitates. Rapid coagulation occurred after ascites were exposed to the air. Thoracic fluid seen in several cases was also of the same nature as ascites.

The whole surfaces of the omentum, mesenterium and peritoneal organs were covered with jelly-like or caseous greyish-white fibrinous deposit, sometimes forming miliary or rice grain-sized nodules. The omentum became thick remarkably showing an edematous and turbid appearance. In most cases, mesenteric lymph nodes were slightly enlarged with parenchymatous edema or hyperemia as well as necrotic patches in some cases. In about a half of cases, splenic follicles were clearly visible on the cut surface, and pinhead-sized or mili-

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Breed</th>
<th>Age</th>
<th>Sex</th>
<th>Ascites</th>
<th>Hydrothorax</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Volume</td>
<td>Nature</td>
</tr>
<tr>
<td>1</td>
<td>Siamese</td>
<td>1*</td>
<td>♀</td>
<td>++</td>
<td>**</td>
</tr>
<tr>
<td>2</td>
<td>Himalayan</td>
<td>1.5y</td>
<td>♂</td>
<td>+++</td>
<td>amber</td>
</tr>
<tr>
<td>3</td>
<td>Mongrel</td>
<td>6m</td>
<td>♂</td>
<td>+++</td>
<td>dark amber</td>
</tr>
<tr>
<td>4</td>
<td>Persian</td>
<td>4y</td>
<td>♂</td>
<td>+++</td>
<td>amber, viscid</td>
</tr>
<tr>
<td>5</td>
<td>Abyssinian</td>
<td>1.5y</td>
<td>♂</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>6</td>
<td>Siamese</td>
<td>7y</td>
<td>♂</td>
<td>+</td>
<td>bloody, viscid</td>
</tr>
<tr>
<td>7</td>
<td>Mongrel</td>
<td>6y</td>
<td>♂</td>
<td>+</td>
<td>amber, cloudy, viscid</td>
</tr>
<tr>
<td>8</td>
<td>Mongrel</td>
<td>2y</td>
<td>♂</td>
<td>+</td>
<td>yellowish green, cloudy</td>
</tr>
<tr>
<td>9</td>
<td>Mongrel</td>
<td>3m</td>
<td>♂</td>
<td>+</td>
<td>amber, viscid, transparent</td>
</tr>
<tr>
<td>10</td>
<td>Siamese</td>
<td>4m</td>
<td>♂</td>
<td>+</td>
<td>straw colored, viscid, transparent</td>
</tr>
<tr>
<td>11</td>
<td>Siamese</td>
<td>4.5m</td>
<td>♂</td>
<td>+</td>
<td>yellowish green</td>
</tr>
<tr>
<td>12</td>
<td>Siamese</td>
<td>1y</td>
<td>♂</td>
<td>+</td>
<td>yellow</td>
</tr>
<tr>
<td>13</td>
<td>Siamese</td>
<td>4m</td>
<td>♂</td>
<td>+</td>
<td>yellow</td>
</tr>
<tr>
<td>14</td>
<td>Mongrel</td>
<td>4m</td>
<td>♂</td>
<td>+</td>
<td>yellow, viscid</td>
</tr>
<tr>
<td>15</td>
<td>Siamese</td>
<td>6.5m</td>
<td>♂</td>
<td>+</td>
<td>amber, transparent</td>
</tr>
<tr>
<td>16</td>
<td>Siamese</td>
<td>4m</td>
<td>♂</td>
<td>+</td>
<td>yellow, viscid, transparent</td>
</tr>
<tr>
<td>17</td>
<td>Mongrel</td>
<td>4m</td>
<td>♂</td>
<td>+</td>
<td>yellow, transparent</td>
</tr>
<tr>
<td>18</td>
<td>Siamese</td>
<td>4m</td>
<td>♂</td>
<td>+</td>
<td>yellow, transparent</td>
</tr>
<tr>
<td>19</td>
<td>Siamese</td>
<td>4.5m</td>
<td>♂</td>
<td>+</td>
<td>straw colored</td>
</tr>
<tr>
<td>20</td>
<td>Siamese</td>
<td>6y</td>
<td>♂</td>
<td>+</td>
<td>yellow, viscid, transparent</td>
</tr>
<tr>
<td>21</td>
<td>Persian</td>
<td>7m</td>
<td>♂</td>
<td>+</td>
<td>yellowish green</td>
</tr>
<tr>
<td>22</td>
<td>Mongrel</td>
<td>10m</td>
<td>♂</td>
<td>+</td>
<td>viscid, cloudy</td>
</tr>
<tr>
<td>23</td>
<td>Siamese</td>
<td>1y</td>
<td>♂</td>
<td>+</td>
<td>yellow, viscid, transparent</td>
</tr>
<tr>
<td>24</td>
<td>Mongrel</td>
<td>1y</td>
<td>♂</td>
<td>+</td>
<td>yellowish green, transparent</td>
</tr>
</tbody>
</table>

Remarks.
* : Not examined.
** : - ; Negative,
+ : <100 ml.
+++ : 100-300 ml.
+++ : >300 ml.
ary white necrotic patches were disseminated in the spleen of 3 cases (Nos. 4, 9 and 11). A large number of such necrotic patches were seen also on the cut surface of the liver of 7 cases (Nos. 4, 6, 9, 11, 23, 25 and 28), with centrilobular fat degeneration or icterus in some cases. In the pancreas of 3 cases (Nos. 4, 27 and 28) milary grey-white areas were extended diffusely from the surface into the edematous parenchyma having some necrotic foci. In the small intestines of rather chronic cases, fibrous adhesions were recognized either with some other parts of the same organ or with the omentum, peritoneum, liver and spleen. The mucosal surface of the intestines showed edema or hyperemia and sometimes catarrhal inflammation. Two cases (Nos. 15 and 18) were shown to be parasitized with some *Toxocara cati*.

The kidney was found to be involved only in 2 cases (Nos. 2 and 19) showing superficial necrotic lesions as well as rice grain-sized necrotic patches distributed in the deep parenchyma. No visible lesions were observed in the adrenals of any cases. On the serous surface of the urinary bladder hyperemia and small hemorrhagic patches were present with precipitation of fibrinous exudate, and edematous swelling was seen also in the mucosa of some cases.

In 7 cases (Nos. 3, 15, 17, 20, 22, 23 and 29) having a large amount of thoracic fluid, fibrous adhesion with subpleural edema and hemorrhage occurred between some pulmonary lobes and the thoracic wall. In 11 cases (Nos. 2, 4, 6, 9–11, 20, 22, 23, 25 and 26), a limited area of atelectasis, edema or pneumonia were recognized. Fibrinous precipitates having sometimes a thickness of about 3 to 5 mm were seen also on the surface of the heart of 2 cases (Nos. 9 and 15). Pericardial fluid was transparent but increased in amount containing fibrin precipitates sometimes showing green-yellow color. Turbid swelling was common in the myocardium with pericarditis in some cases.

In one case cerebrospinal fluid was increased slightly in amount with turbid appearance of the meninges. Cases No. 19 and No. 29 had blindness due to granulomatous retinitis and uveitis, as described later.

2. Histopathology

The outlines of histopathologic changes, in special reference to vascular changes are presented in Figs. 1 and 2.

Omentum and mesenterium: The whole surface of the membranes was covered with thick layers of eosinophilic and fibrin stain-positive materials. Severe degeneration, metaplasia and hyperplasia of mesothelial cells with frequent syncytium formation were seen, and many necrotic foci containing a large number of nuclear debris were present (Figs. 3 and 4).

In many cases, the lesions were diffusely infiltrated with neutrophils, macrophages and plasma cells, all of which underwent severe degeneration. In some cases there were hemorrhage or disseminated granulomatous lesions with fibrosis.

It was notable in most cases that inflammatory changes were present in smaller veins and arteries as well as venules and arterioles. In involved vessels, endothelial cells showed edematous swelling and vacuolation and some of them were desquamated falling into the lumen. A large number of mononuclear cells and neutrophils were accumulated within the lumen, wall and perivascular areas, and sometimes diffuse or nodular proliferation of spindle-shaped endothelial cells were seen resulting in narrowed cavities. The swollen and rarefied media of affected veins had pyknotic nuclei
with perivascular infiltration of lymphocytes and plasma cells, and such changes were sometimes extended to adjacent tissues (Figs. 5, 6 and 7). Considerable narrowing of the cavity was also seen in arteries having rarefied and vacuolated media with segmentation of the elastica interna and externa. In some cases, medial and endothelial proliferation were prominent accompanying adventitial fibrosis with only a very small number of inflammatory cells.

Digestive tract: In almost all cases, the serosa showed coagulation necrosis with a large amount of fibrin deposit and nuclear debris were scattered with infiltration of neutrophils, macrophages, lymphocytes and plasma cells. Cuboid and columnar metaplasia of mesenchymal cells with syncytium
formation were common even in cases having no prominent fibrin deposit (Fig. 8). In many cases, the muscle layers were also involved with severe degeneration in peripheral nerve ganglions.

Catarrhal and necrotizing enteritis was seen in the small intestines of some cases, showing edematous submucosa and tunica propria with hypertrophy of lymphoid tissues and mononuclear cell infiltration. The walls of venules and small veins tending to be necrotized were infiltrated by neutrophils and mononuclear cells. Sometimes the vascular lumen was filled up with the same kinds of cells. There were degeneration and desquamation of endothelial cells, rarefaction of medial cells and hemorrhage, sometimes with thrombosis. In most cases, adventitial and perivascular areas were also infiltrated predominantly with mononuclear cells tending to be necrotized (Fig. 9). Even in cases having less remarkable infiltration, were recognized rarefaction of tissues around small vessels, edematous swelling of the media and desquamation of endothelial cells.

Mesenteric lymph nodes: Massive fibrin deposits with some infiltration of lymphocytes and plasma cells were observed on the capsule in a few cases examined. Such cellular infiltration was also seen around venules and small veins of the capsule. Reticulum cells of the medullary cord were strongly hyperplastic, and the sinuses were filled with neutrophils, macrophages, lymphocytes and plasma cells. In the medulla, there were numerous foci of coagulation necrosis sometimes showing eosinophilic caseous appearance without marked inflammation on the capsule. The trabecular arterioles and small arteries of a few cases showed a fibrous thickening at the adventitia and swelling of the media with narrowing of the cavity. In some other cases lymphatic vessels were markedly dilated. In several cases some proliferation of fibroblasts resulted in enlargement of the affected nodules.

Spleen: Fibrin precipitation was remarkable on the entire capsular surface with severe infiltration of neutrophils, lymphocytes, plasma cells as well as debris of these cellular elements. Notable was a considerable proliferation of mesothelial and other mesenchymal cells showing conspicuous cuboid or columnar metaplasia, and syncytium formation. Such changes were most remarkable in cases having no fibrin precipitation on the capsular surface (Fig. 10). Trabecular connective tissues were dissociated around arteries having degenerative and desquamative endothelial cells. Continuation between subcapsular and parenchymatous lesions was apparent only in a few cases. Neutrophils, macrophages, lymphocytes and plasma cells were abundant with some proliferation of fibroblasts in the medullary sinuses. Reticuloendothelial cells seemed to be activated in the medullary cord. Coagulation necrosis were produced focally in the red and white pulps accompanying hemorrhage and fibrin accumulation. Germinal centers were enlarged in some cases but atrophic in others.

Liver: A thick pseudomembrane of fibrin precipitation was present on the entire surface of the liver with diffuse cell infiltration (Fig. 11). Some layers of such fibrin deposits were already replaced by either immature granulation tissues or fibrous connective tissues. Metaplasia of mesothelial cells and syncytium formation were commonly seen.

Most cases showed dissociation of hepatic plates and centrilobular fat degeneration, while a few cases had focal necrosis near the central vein containing numerous cell debris. Activation of Kupffer cells were ap-
parent with some accumulation of neutrophils, plasma cells and nodular proliferation of reticuloendothelial cells within the sinusoid. Glisson’s capsule had remarkably dilated lymphatic vessels with infiltration of large mononuclear cells, lymphocytes and plasma cells. The same kinds of cells were also accumulated within the wall and lumen of interlobular arteries or veins, and fibrinous thrombi in the interlobular veins were recognized in some cases.

Pancreas: Although a few cases had degenerative and necrotic foci in the parenchyma as well as in the interstitial tissue, superficial fibrin precipitation was not so severe even in cases having a large amount of ascites. The foci of coagulative necrosis containing numerous nuclear debris were seen around interstitial small veins and they were infiltrated with lymphocytes and plasma cells. In case No. 16 a considerable number of neutrophils were present within or around the pancreatic duct.

Kidney: The surface lesions were almost similar to those described with other organs, but organization process seemed to be more advanced in the kidney. Under the capsule where small arteries and veins were affected, the cortical parenchyma was also involved, and necrotic foci with central accumulation of cellular debris were disseminated in the interstitium. Granulomatous proliferation occurred with infiltration of lymphocytes and plasma cells in predominance. Some branches of interlobular arteries and veins were found to have degenerative and desquamative endothelial cells as well as perivascular inflammation. The media was thickened but rarefied showing fragmentation of the internal and external elastic fibers. Edematous changes were seen around the arcuated arteries, and thrombosis was encountered in some subcapsular veins.

Adrenal: In only one case (No. 9) some small foci of caseous necrosis were detected in the adrenal cortex without no significant cellular response.

Urinary bladder: Fibrin deposit on the serosa was not so severe but a considerable number of neutrophils, lymphocytes and plasma cells covered mesothelial cells undergoing hyperplasia, metaplasia and syncytium formation. Sometimes small granulomatous lesions were seen with severe fibrosis and perivascular infiltration of lymphocytes and plasma cells. In the underlying muscle layers there was necrosis with diffuse cell infiltration. Though severe phlebitis was not recognized, there existed some proliferation of venous endothelial cells resulting in narrowing of the lumen (Fig. 12). Small arteries of the ligaments exhibited fibrous proliferation of the adventitia and vacuolation of endothelial cells. In the mucosa of some cases, edema and cell infiltration, though not severe, were present. Transitional epithelial cells were proliferating and some were found to degenerate and fall into the cavity.

Genital organs: Fibrinous inflammation was seen in the uterine serosa of only one case (No. 10).

Lungs: Catarrhal lobular inflammation with edema and hemorrhage were noticed in several cases (Nos. 2, 4, 6, 9–11, 20, 22, 23, 25 and 26). Two cases (Nos. 3 and 17) were shown to have severe hydrothorax with a large amount of fibrin deposit covering the pleural surface. Some necrotic foci with numerous nuclear debris, metaplasia of mesothelial cells and nodular hyperplasia of mononuclear cells were observed. Sometimes organization of fibrin deposit occurred on the pleura. Even in other cases having no such pleural lesions, foci of coagulation necrosis were produced with accumulation of neutrophils, macrophages,
lymphocytes and plasma cells.

In venules and small veins of the lungs also, endothelial cells underwent severe degeneration and necrosis with infiltration of neutrophils, macrophages and lymphocytes, and occasionally with thrombosis (Fig. 13).

Heart: A pseudomembrane containing a number of nuclear debris was formed also on the pericardium of 3 cases (Nos. 5, 21 and 29), and fibrosis and lymphocyte infiltration were associated with similar lesions of two cases (Nos. 9 and 15).

Brain: In two cases (Nos. 19 and 29), a considerable number of plasma cells and various kinds of other inflammatory cells appeared in the meninges, choroid plexus (Fig. 14) and subependymal areas. Middle sized veins of the meninges and parenchyma were dilated and they had cuffing of lymphocytes and plasma cells. Occasionally, there were small foci of softening, and spongy degeneration in the subependymal parenchyma with degeneration of some neurons.

One case (No. 29) was shown to be parasitized with Toxoplasma gondii.

Eye: In the uvea of No. 29 eosinophilic serous exudate and hemorrhage were seen. A considerable number of lymphocytes and plasma cells as well as some neutrophils and macrophages were present in the uvea and around smaller vessels of both eyes from two cases (Nos. 19 and 29). Endothelial proliferation occurred in smaller arteries. Cellular infiltration was also seen in parts of the retina, sclera and cornea as well as optic nerves and neighboring muscular tissues.

Other organs: In the thyroids, parathyroids and salivary glands examined, no lesions were detected.

Discussion

In accordance with those reported by other workers, the incidences of natural FIP cases seem to be higher in animals inbred than mongrel, male than female, and younger than older. Although the disease was reported to be prevalent in autumn and winter [17], our cases were experienced all around the year.

While antibiotic treatment was reported to be successful [8], no significant effects of various antibiotics were observed with the present cases. Etiological examinations of ascites [9–14, 18, 20, 21, 25, 27] from most cases examined, which was characteristic of FIP, revealed no significant bacteria, mycoplasma or protozoae.

It was reported that the disease can be reproduced by inoculation of unfiltrated or filtrated ascites from FIP infected animals into germfree or conventional cats [2, 4, 12–14, 16, 21, 25, 27]. Blood, urine and liver emulsions from diseased animals were also found to be infective for cats when inoculated perorally, intraperitoneally, subcutaneously and intravenously [8, 14, 26], and we also could transmit the disease to kittens by intraperitoneal or intravenous inoculation with ascites samples from diseased animals. However, the disease failed to be transmitted to embryonated eggs, rats, mice, guinea-pigs and hamsters [8, 12, 25]. Electron microscopy of peritoneal cells from affected animals revealed coronavirus-like particles [8, 22–24, 27]. While the viral growth on autochthonous peritoneal cell culture was reported recently, but isolation of the causative virus on established cell lines remains unsuccessful [16]. Despite of these results strongly suggesting the viral etiology of FIP, little knowledge has been provided about target cells or organs and the mechanism for production of a large amount of ascites or thoracic fluid characteristic of FIP.

Although systemic vasculitis was de-
scribed before FIP was noticed as a disease entity [7] and some vascular lesions were mentioned in most reports on FIP, little attention has been paid to detailed characteristic of the lesions. Through the present studies with natural cases of FIP, our attention was focused on degenerative and inflammatory changes in fine and smaller vessels 10 to 200 μm in diameter that were detectable in almost all cases examined. Such vascular lesions were widely distributed not only in the pleural and peritoneal serosa, omentum and mesenterium but also in interstitial connective tissues of parenchymatous organs. The vascular alterations which were of degeneration, metaplasia and proliferation, might have resulted from the growth of FIP agent in vascular endothelium. Mesothelial cells covering the peritoneal and thoracic cavities were also severely affected. These findings are of great interest in considering that the nature of a large amount of peritoneal or thoracic exudate is similar to that of blood plasma.

However, there have been no reports that described the presence of virus-like particles in vascular endothelial cells. While they were detected in macrophages in the peritoneal cavity, spleen and liver as well as mesothelial cells of experimentally infected cats. Our studies are being focused on the detection of virus-like particles within vascular endothelial cells while not yet successful. Degeneration and syncytium formation of peritoneal and thoracic macrophages were common in most of the present cases, suggesting viral growth within such kind of cells which might be target cells for FIP agent [22], as in case of mouse hepatitis virus, another member of coronaviruses.

Another hypothetical pathogenesis of the vascular changes in FIP might be that they were produced by some immunologic process following the infection.

Ascites and thoracic exudate in FIP are known to contain a high level of serum immunoglobulin-fractions, especially IgG as well as many kinds of cellular elements including neutrophils, macrophages, lymphocytes, plasma cells and mesothelial cells [7, 8, 17, 18]. Some of our cases examined gave also similar results. To see immunological aspects of FIP, some serological procedures for FIP should be devised and developed. Although clinical signs of FIP were characterized by massive ascites, fever, anorexia and weight loss, almost all of the present cases were found to have such signs and some of them had lesions in different organs, such as pancreatitis, meningoencephalitis and ophthalmitis. Some workers reported non-typical cases of FIP that have no peritoneal or pleural effusion but some lesions in the eye, lungs, lymph nodes, kidneys and central nervous system, as described already [1, 5, 6, 14, 19]. And vascular lesions, though briefly, were described about these affected organs, and samples taken from these organs were shown to be able to produce FIP after inoculation into susceptible animals [14]. Pathogenesis in such non-typical cases as well as clinical variation of FIP awaits further experimental studies.

References


Explanation of Figures

Fig. 3. A part of granuloma with a syncytium formation in the mesentery. Case No. 17. \( \times 930 \).

Fig. 4. Necrotic foci with accumulation of many inflammatory cells and cell debris in the omentum. Case No. 24. \( \times 310 \).

Fig. 5. Phlebitis in a small vein of the omentum showing swelling of endothelium, degeneration of media and inflammatory reactions in adventitia and surrounding tissues. Case No. 25. \( \times 620 \).

Fig. 6. Arteriolitis in the omentum. Case No. 25. \( \times 620 \).

Fig. 7. Prominent perivascular infiltration of lymphocytes in the omentum. Case No. 9. \( \times 620 \).

Fig. 8. Proliferation of spindle-shaped cells on the intestinal serosa. Case No. 17. \( \times 310 \).

Fig. 9. Phlebitis in a small vein of the serosa of the intestine showing severe infiltration of neutrophils, mononuclear cells and lymphocytes with proliferation of fibroblasts. Case No. 27. \( \times 310 \).

Fig. 10. Metaplasia, hyperplasia and desquamation of mesothelial cells on the capsule of the spleen. Case No. 15. \( \times 620 \).

Fig. 11. Diffuse deposit of fibrin with infiltration of lymphocytes and plasma cells on the capsule of the liver. Case No. 2. \( \times 310 \).

Fig. 12. Proliferation of spindle-shaped endothelial cells narrowing the lumen in a small venule of the ligaments of the urinary bladder. Case No. 11. \( \times 930 \).

Fig. 13. Phlebitis in a branch of the pulmonary vein. Degeneration of endothelial and medial cells and perivascular inflammation. Case No. 22. \( \times 620 \).

Fig. 14. Perivascular accumulation of plasma cells around a vessel of the choroid plexus. Case No. 19. \( \times 620 \).