Clinical, cerebrospinal fluid, and histological data from twenty-seven cats with primary inflammatory disease of the central nervous system

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Abstract

The purpose of this report is to present the clinical, cerebrospinal fluid (CSF) and histological data from 27 cats with inflammatory disease of the central nervous system (CNS). The cats were part of a study of 61 cats admitted to two university clinics over an eight-year period because of signs of CNS disease. The most frequent diseases were feline infectious peritonitis (FIP) (12/27) and suspected viral disease other than FIP (10/27). Typical CSF findings in cats with FIP were a protein concentration of greater than 2 g/L (200 mg/dL) and a white cell count of over 100 cells/μL, which consisted predominantly of neutrophils. In contrast, the CSF of cats with suspected viral disease had a protein concentration of less than 1 g/L (100 mg/dL) and a total white cell count of less than 50 cells/μL. In general, cats with FIP or suspected viral disease were less than four years of age. Neurological signs were usually multifocal in cats with FIP, but focal in cats with suspected viral disease. The CSF findings were variable in five other inflammatory diseases represented. Two cats with protozoan infection had normal CSF total cell counts but abnormal differential counts. The CSF findings were invaluable in differentiating FIP from other causes of inflammatory CNS disease.

Résumé

Revue des données de l'examen physique, de l'analyse du liquide céphalorachidien et de l'histologie de 27 chats atteints d'une pathologie inflammatoire primaire du système nerveux central

Cette étude de dossiers présente les aspects de l'examen physique, de l'analyse du liquide céphalorachidien et de l'histologie de 27 chats atteints d'une pathologie inflammatoire du système nerveux central. Ces animaux font partie d'une étude de 61 chats admis pour symptômes reliés au système nerveux central sur une période de huit ans à deux centres universitaires. Les maladies les plus fréquentes ont été la péritonite infectieuse féline (PIF) (12/27) et une pathologie soupçonnée d'origine virale autre que le PIF (10/27). Dans les cas de PIF, l'analyse du liquide céphalorachidien (LCR) présentait un taux de protéines de plus de 2 g/L (200 mg/dL) et un compte leucocytaire supérieur à 100 cellules/μL avec une prédominance de neutrophiles. Les autres chats soupçonnés d'une pathologie d'origine virale avaient un taux de protéines inférieur à 1 g/L (100 mg/dL) et un compte leucocytaire de moins de 50 cellules/μL. En général, les chats atteints de PIF ou d'une pathologie soupçonnée d'origine virale étaient âgés de moins de quatre ans. Les signes neurologiques étaient habituellement multifocaux chez les chats présentant du PIF, mais unifocaux pour les animaux soupçonnés d'atteinte virale. Les analyses du LCR étaient variables dans cinq cas présentant d'autres pathologies inflammatoires. Deux chats ayant une infection à protozoaires présentaient des résultats normaux du compte leucocytaire du LCR, mais un différentiel anormal. Les analyses du LCR sont inestimables pour différencier la PIF des autres maladies inflammatoires du système nerveux central.

(Traduit par Dr Thérèse Lanthier)

Can Vet J 1994; 35: 103–110

Introduction

We have previously reported the reference intervals for feline cerebrospinal fluid (CSF) (1–3). There are only a few reports in the veterinary literature of feline neurological disease in which CSF data are also available (4–10). The main objective of our report is to present the clinical, CSF, and pathological data derived from 27 cats with inflammatory central nervous system (CNS) disease, and to identify which clinical, general laboratory, and CSF parameters were the most useful indicators of the final pathological diagnosis. The data derived from 34 cats with noninflammatory CNS disease and the CSF enzyme activities for all cats will be presented in a second report.

Materials and methods

Data

Twenty-seven cats with primary inflammatory CNS disease were selected from a combined retrospective and prospective study of 61 cats that were admitted to the Universities of Guelph and Saskatchewan from 1978 to 1986 because of clinical signs of CNS disease. All cats had both a CSF analysis and a postmortem examination. The following factors were evaluated: age, clinical signs, duration of clinical signs, complete blood count, serum biochemistry, feline leukemia virus (FeLV) status, and serum antibody titers for coronavirus and *Toxoplasma* spp. The CSF parameters that were assessed for each cat, when available, were: total red and white

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This project partially fulfilled the requirements for the Doctor of Veterinary Science degree and was funded by the University of Guelph and the Pet Trust Fund.

Table 1. Reference intervals for feline cerebrospinal fluid (1,2)

Cell counts and cytology		Low	High
Erythrocytes	/μL	0	30
Total white cells	/μL	0	2
Neutrophils	%	0	9
Lymphocytes	%	0	27
Monocytes	%	69	100
Macrophages	%	0	3
Eosinophils	%	0	0
Biochemistry			
Total protein	g/L	0.06	0.36
CK[a]	U/L	2	236
LDH[b]	U/L	0	24
AST[c]	U/L	0	34

[a]Creatinine kinase
[b]Lactate dehydrogenase
[c]Aspartate transferase

cell counts, differential cell count, total protein, Pándy's test score, and the activities of lactate dehydrogenase (LDH), aspartate transferase (AST), and creatine kinase (CK). Values were compared with reference intervals previously established by the authors for feline CSF (Table 1) (1–3). The CSF samples were collected by percutaneous puncture of the cerebellomedullary cistern, and the methods used in the CSF analysis were similar in the two institutions. The CSF red and white cell counts were determined using a hemocytometer. The CSF total protein was measured using the ponceau S method (11). Serum and CSF enzyme activities were measured with automated chemistry analyzers. Serum antibody titers for coronavirus were determined using a variety of enzyme-linked immunosorbent assays (ELISA). Antibodies to *Toxoplasma* spp. were measured with an indirect hemagglutination test (Toxo-IHA Test, International Scientific of Canada, P.O. Box 6220, Ottawa). Feline leukemia virus (FeLV) antigen in serum was detected by an ELISA assay (Leukassay F, Pitman-Moore Inc, Washington Crossing, New Jersey, USA).

Cytology

Sedimentation and/or cytocentrifuge techniques were used in the preparation of CSF cytological specimens. The sedimentation method was a modification of the technique reported by Sörnäs (1,12), in which a 0.5 mL aliquot of CSF was allowed to stand for 30 min before the supernatant was aspirated. Cells were classified as neutrophils, eosinophils, lymphocytes, monocytoids, or macrophages. Monocytoids were defined as mononuclear cells, excluding lymphocytes and macrophages. Only mononuclear cells with phagocytosed material within the cytoplasm were classed as macrophages. As a CSF red cell count was not available for all cats, an estimate of CSF contamination by blood was made from the cytological preparations as follows: the total number of red cells in four high power (400 X) fields (one field in each of the four quadrants of the slide) were summed and divided by 4 (13). The estimated red cell concentration was found to correlate significantly (r=0.7) with the measured red cell count for those cats in which both values were available (data not shown).

Table 2. Frequency of disease in 27 cats with primary inflammatory disease of the central nervous system

	Number	Percent
Feline infectious peritonitis	11	41
Suspected viral	10	37
Protozoal	2	7
Miscellaneous inflammatory diseases	4	15
Bacterial emboli	1	
Choroid plexitis	1	
Eosinophilic meningoencephalitis	1	
Chronic meningoencephalitis	1	

Adjustment for blood contamination

In our study of CSF in normal cats, blood contamination (>30 RBC/μL) was shown to affect some CSF parameters, such as total and differential white cell counts (1–3). In the present study, an attempt was made to account for the effect of blood contamination. Before determining whether a value was outside the reference intervals for feline CSF, the maximum expected change in the parameter, based on the CSF red cell count, was calculated as follows: for total white cell count, the maximum expected increase was estimated using the previously observed maximum rate of one additional white cell/μL per 100 red cells/μL (1). For CSF protein, the increase was estimated to be 0.01 g/L per 1200 red cells/μL (2,14). The results of the differential cell counts were compared with the reference intervals established for the corresponding level of blood contamination (1,3). When the level of blood contamination was higher than the maximum seen in the reference study (3,700 red cells/μL), no assessment of the normalcy of the differential count was made. When parameters were higher than normal and greater than the expected increase due to blood contamination, the increase was attributed to CNS disease (1,15). This adjustment for blood contamination is based on the assumption that all red blood cells in the CSF were the result of contamination, and not disease. In cats in which the CSF red blood cell concentration had not been recorded, the estimated CSF red cell concentration obtained from the cytological preparation was used to indicate the degree of blood contamination.

Histology

All histological preparations of the CNS were reviewed by the authors. Lesions were classified as being consistent with feline infectious peritonitis (FIP), if meningitis was present together with histological evidence of pyogranulomatous inflammation in other viscera (16,17). Lesions were classified as consistent with a viral etiology other than FIP, when a predominantly mononuclear meningitis was present but visceral lesions consistent with FIP were absent, or perivascular cuffing was the only histological lesion (18,19). In the text, focal is used when the lesion was confined to a small area, diffuse when the lesion was evident throughout an anatomical region of the brain or spinal cord, zonal for marked involvement of a large area but not the entire anatomical region, and multifocal when more than one focal lesion was evident.

Table 3. Cerebrospinal fluid parameters in 27 cats with inflammatory disease of the central nervous system

Cat	Protein	Pándy's test	RBC	WBC	Total	% Neut	% Lym	% Mono	% Mac	% Eos	Vol	Prep
Feline infectious peritonitis												
9	4.40[a]	4	0	243[a]	15000	88[a]	3	10	0	0	0	S
12	3.60[a]	4	—	—	5600	71[a]	11	18	0	0	0	S
19	6.60[a]	4	—	—	—	—	—	—	—	—	—	
29	—	1	48	4[a]	1	0	0	100	0	0	0	C
44	4.20[a]	4	—	472[a]	14000	98[a]	1	2	0	0	0	S
46	3.50[a]	4	—	8[a]	3000	81[a]	10	9	0	0	0	S
47	—	—	—	—	100	91[a]	0	9	0	0	0	S
52	2.80[a]	—	26130	623[a]	—	—	—	—	—	—	—	
58	0.84[a]	—	60	1	—	—	—	—	—	—	—	
63	—	—	1325	2500[a]	—	—	—	—	—	—	—	
65	3.50[a]	—	3	227[a]	24000	81[a]	5	14	0	0	0	S
Mean	3.68	4	4594	510	10283	85	5	10	0	0	0	
S.d.	1.6	1	10563	836	8977	9	5	5	0	0	0	
Suspected viral												
2	0.62[a]	0	24	34[a]	144	8	17	72	0	0	0	C
7	0.07	0	13	8[a]	33	0	30	70	0	0	0	C
15	0.11	0	2	0	17	0	6	82	6	0	1	C
33	0.12	0	1	0	33	0	12	85	0	0	1	C
34	0.72[a]	0	560	1	96	17[b]	43[a]	40	0	0	0	S
35	0.21	0	30	2	11	27[a]	18	46	0	0	0	S
37	—	—	—	—	—	75[a]	10	15	0	0	0	S
64	0.14	—	2	0	—	—	—	—	—	—	—	
66	0.31	—	3	0	—	—	—	—	—	—	—	
69	0.26	—	2	4[a]	—	—	—	—	—	—	—	
Mean	0.28	0	71	5	54	40	24	34	0	0	0	
S.d.	0.23	0	184	11	60	31	17	16	0	0	0	
Protozoal												
6	0.69[a]	1	1000	1	335	23[b]	3	71	2	0	95[a]	S
22	—	1	123	2	226	0	85[a]	14	0	0	0	S
Other inflammatory disease												
5	0.94[a]	4	1400	900[a]	12000	55[b]	15	30	1	0	2	S
30	0.18	0	2	1	25	0	56[a]	44	0	0	0	S
32	—	1	—	0	112	1	39[a]	46	2	9[a]	4	S
53	0.12	—	161	4[b]	770	28[a]	40[a]	33	0	0	0	S

All parameters shown as raw data without adjustment for blood contamination. Percentage of cells may add to less than 100% as not all categories of cells are shown. Percent values were rounded to the nearest whole number, so in some cases they add to 101%

— = value not available

[a] = parameter higher than reference value

[b] = parameter higher than normal but compatible with degree of blood contamination

Abbreviations:
RBC or WBC = red blood cell or white blood cell count using hemocytometer
Total = total number of white blood cells on cytological slide preparation
Prep = type of cytological slide preparation, S=sedimentation, C=cytocentrifuge
Sedimentation results are shown in those cats with both sedimentation and cytocentrifuge data
% neut, % lym, % mono, % mac, % eos = percentage of neutrophils, lymphocytes, monocytoids, macrophages, or eosinophils, respectively, in cytological preparation
Vol = absolute number of voluminous cells in cytological preparation

Clinical signs

Focal is used when neurological signs were localized to one part of the CNS, even if the lesion appeared diffuse in that area. Multifocal or diffuse is used when neurological signs indicated two or more separate lesions. Lethargy is used to describe systemic clinical signs characterized by reduced activity, while depression is used to describe neurological signs consistent with reduced mentation associated with CNS disease.

Statistical methods

In most cases, only frequencies are reported. Statistical analysis was often inappropriate due to missing data, small sample size, and the presence of blood contamination in 60% of samples. Pearson's product-moment correlation coefficient was used to determine the correlation coefficient between the measured CSF red blood cell concentration and the red blood cell concentration estimated from the cytological preparation (20).

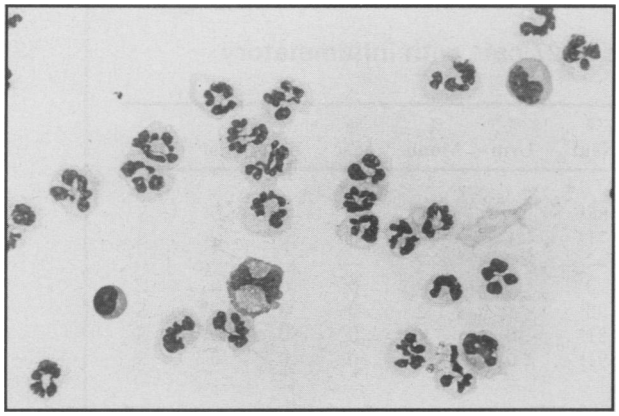

Figure 1. The cerobrospinal fluid of a cat with feline infectious peritonitis (cat 44). A typical neutrophilic pleocytosis is present (cytocentrifuge, Wright's stain, 400×).

Results

Based on the histological findings, the frequency of diseases in the 27 cats is shown in Table 2. The most common diseases were FIP (11/27, 41%) and suspected viral disease (10/27, 37%).

Feline infectious peritonitis

Clinical data

The majority of cats (8/11) with the CNS form of FIP were less than four years of age (median two years), and had clinical signs for greater than three weeks. Anorexia (n=9), weight loss (n=8), lethargy (n=6), and ocular lesions (n=7) were the most common systemic signs. Depression (n=8), ataxia (n=7), head tremor (n=5), and seizures (n=3) were the most frequently observed neurological signs. Neurological signs were most often consistent with multifocal lesions (n=8) and were referable to the cerebellum (n=6), brainstem (n=6), thalamocortex (n=4), and spinal cord (n=4).

Clinical pathology

The hematology and serum biochemistry test results did not suggest any consistent abnormality (data not shown) (3). Of the six cats for which serum globulin concentrations were available, only one had hyperglobulinemia.

Only two of six cats tested for coronavirus antibody titer had titers of 1:400 or more, and two of eight cats tested for FeLV were positive.

The CSF total protein concentration was increased in all eight cats in which it was measured (Table 3). The mean CSF total protein was 3.68 g/L, and only one cat had less than 2 g/L. The Pándy's test score was abnormal in all six cats tested. The CSF total white cell count was increased in seven of eight cats (mean 510 cells/μL, range 1 to 2,500 cells/μL). Neutrophils were the predominant cell type in the CSF cytology and comprised over 80% of the cells in five of six cats with sedimentation preparations available (Figure 1).

Histology

Of the 10 cats in which the CNS was examined histologically, all had meningitis, eight had perivascular cuffing, six had degenerative changes which consisted predominantly of necrosis, and five had choroid plexitis. Lesions were considered diffuse in six cats and multifocal in four. The following areas of the brain were involved in decreasing order of frequency: cerebellum (n=7), thalamocortex (n=7), brain stem (n=6), and spinal cord (n=4). Lymphocytes and/or macrophages were found in the lesions of 10 cats, and neutrophils were present in eight cats. Pyogranulomatous inflammation was present in other viscera of all 11 cats.

Suspected viral disease

Clinical data

The majority of cats (8/10) were two years of age or less (median 1.5 years) and had clinical signs for less than five weeks. Anorexia (n=3) and lethargy (n=3) were the most frequent systemic signs. Seizures (n=5) and head tremor (n=3) were the most commonly reported neurological signs. Neurological signs were most often suggestive of a focal lesion (n=7) and were referable to the thalamocortex (n=5).

Clinical pathology

Four cats had increased white cell counts in the peripheral blood, but consistent abnormalities were not present in serum biochemistry test results (data not shown). All three cats in which coronavirus antibody titers were measured had titers of 1:100 or less, and one of the six cats tested for FeLV was positive.

The CSF total protein was increased in only two of nine cats in which it was measured, with a mean concentration of 0.28 g/L (range 0.07 g/L to 0.72 g/L, Table 3). The Pándy's test score was normal in all six cats tested. The total white cell count in the CSF was increased in three of the nine cats in which it was measured, but the mean was only 5 cells/μL. The percentage of neutrophils was increased in two of three cats for which sedimentation preparations were available, but in none of the six cats for which cytocentrifuge preparations were available. The percentage of lymphocytes was increased in one sediment preparation and in none of the cytocentrifuge preparations.

Histology

Nine cats had meningitis ranging from mild to severe, and one cat had only perivascular cuffing. Choroid plexitis was uncommon (n=1). In five cats, there was histological evidence of degeneration. Lesions were most often diffuse (n=4) or multifocal (n=3). The following areas of the CNS were involved: thalamocortex (n=8), cerebellum (n=2), spinal cord (n=1). Lymphocytes were evident in the lesions of all cats (n=10), while neutrophils (n=2) and macrophages (n=5) were less frequent.

Protozoan infection

Clinical data

Two cats had *Toxoplasma*-like organisms evident in histological sections of the CNS. Both cats were aged (11 and 13 years). Duration of clinical signs prior to presentation was less than five weeks. In one cat, neurological signs were referable to the thalamocortex and brain stem, and suggested a space occupying lesion. The other cat had clinical evidence of a focal spinal cord lesion.

Clinical pathology

One cat was leukopenic and anemic due to myelodysplasia, and the other was hypoglobulinemic. Both cats

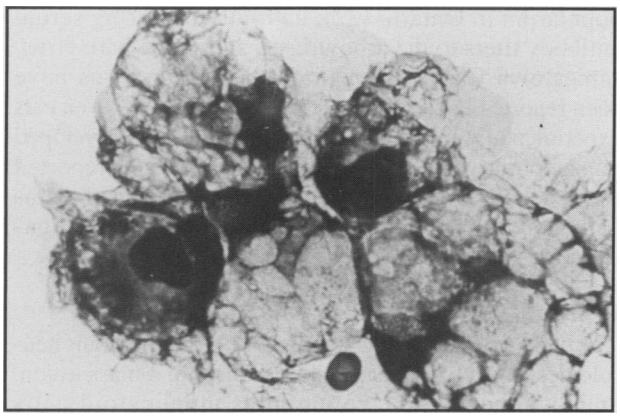

Figure 2. Cerebrospinal fluid from cat 6 with a protozoan infection. Large numbers of voluminous monocytoid cells were present although total white cell count was normal (sedimentation, Wright's stain, 1000×).

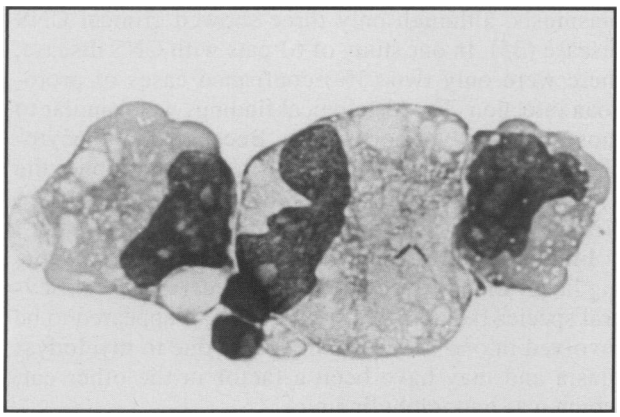

Figure 3. Cerebrospinal fluid of cat 5 with *Pseudomonas* spp. emboli from endocarditis. Marked pleocytosis and intracellular bacteria are evident (cytocentrifuge, Wright's stain, 1000×).

were negative for FeLV. Antibody titers to *Toxoplasma* spp. were not measured in either cat. The CSF total protein in the one cat in which it was measured was twice the normal value and Pándy's test score was abnormal in both cats (Table 3). In both cats, the CSF white cell count was normal but the differential count was abnormal. The percentage of neutrophils was increased in one cat and the percentage of lymphocytes was increased in the other. In cat 6, there were large numbers of voluminous monocytoid cells with "foamy" cytoplasm (Figure 2) and increased numbers of macrophages.

Histology

Multiple protozoan cysts and moderate to severe inflammation and degeneration were present in the thalamocortex, midbrain, and medulla of cat 6 and in the spinal cord of cat 22. Macrophages and lymphocytes were present in the lesions of both cats, but neutrophils were less frequent. Histological lesions consistent with toxoplasmosis were not present in any other body organ examined.

Other inflammatory diseases of the central nervous system

Bacterial

In cat 5, bacterial emboli and infarction of the CNS were associated with *Pseudomonas* spp. infection of an orthopedic surgical wound and secondary endocarditis. Neurological signs of forelimb extension and rigidity suggested meningeal irritation. The CSF white cell count was markedly elevated (900 cells/μL) with a predominance of neutrophils and monocytoid cells (Table 3). The CSF total protein was increased but was less than 1 g/L. Bacteria were evident and found predominantly within monocytoid cells of the CSF (Figure 3). The main histological finding was thalamocortical necrosis, which appeared to be secondary to infarction from bacterial emboli. In contrast to the CSF, parenchymal inflammatory changes were mild and consisted of a neutrophilic infiltration. The majority of the other body organs also had lesions of acute thrombotic infarction, and *Pseudomonas* spp. were isolated from all organs that were cultured.

Choroid plexitis

Focal lymphocytic choroid plexitis of unknown etiology was present in one cat (cat 30). No clinical systemic or neurological signs were observed by humane shelter staff prior to euthanasia. The CSF white cell count, total protein, and Pándy's test score were all normal, but the percentage of lymphocytes present in both cytological preparations was increased.

Eosinophilic meningoencephalitis

Eosinophilic meningoencephalitis of unknown etiology was present in one cat (cat 32). Neurological signs were referable to a thalamocortical lesion. The total white cell count in the CSF was normal, but the percentages of lymphocytes and eosinophils were increased in the sedimentation preparation. Total protein in the CSF was not measured, but the Pándy's test score was abnormal. Histologically, there was both inflammation and degeneration involving the thalamocortex and the meninges. A mixed inflammatory cell population was present and which appeared to be predominantly eosinophils.

Chronic meningoencephalitis of unknown etiology

One cat (cat 53) had degenerative and inflammatory lesions suggestive of protozoan disease, although no organisms were observed. Neurological signs were referable to the cerebellum. The CSF differential white cell count was abnormal with an increased percentage of neutrophils and lymphocytes, although the total white cell count was normal for the degree of blood contamination present. The CSF total protein was normal. Histologically, severe inflammation and moderate degeneration were present. Macrophages and lymphocytes were the predominant inflammatory cells.

Clinical, neurological, and histological data for individual cats are tabulated in the thesis (3).

Discussion

Feline infectious peritonitis

Based on this study, FIP is the most likely cause of CNS disease when a cat less than four years of age has progressive clinical signs of five or more weeks duration; multifocal neurological signs referable to the thalamocortex, brain stem, cerebellum, and/or spinal cord are present; and the CSF has a protein concentration of greater than 2 g/L, a white cell count of greater than

100 cells/μL, and more than 70% neutrophils in the differential cell count. A CSF protein concentration of 2 g/L or more was a distinctive differentiating feature of FIP, as no other inflammatory disease group had a mean CSF protein concentration greater than 1 g/L. The CSF findings of neutrophilic pleocytosis and increased protein concentration were similar to those previously reported (6,7).

Neurological involvement is stated to be more prevalent in the noneffusive form of FIP compared to the classic effusive form (6,21). This was also a finding in our study, as only two out of 10 cats had evidence of effusion into the body cavities. Although the presence of a highly proteinaceous pleural or peritoneal fluid points to the diagnosis of FIP, its absence does not exclude a diagnosis of FIP when neurological signs predominate.

Antibody titer to coronavirus was not a reliable test for indicating infection with coronavirus in cats with the neurological form of FIP. In only two of six cats tested was the titer greater or equal to 1:400. This is in contrast to previous reports, that most cats clinically ill with FIP have titers of 1:400 or greater (22,23). Low titers (<1:400) are reported to occur in older cats with noneffusive disease (24). Serum hyperglobulinaemia was infrequent in our study (1/6) compared to that reported previously for cats with clinical CNS involvement (7,8).

Suspected viral disease

Based on our study, suspected viral disease other than FIP is the most likely cause of CNS disease when a cat two years of age or younger presents with progressive clinical signs of less than five weeks duration; focal neurlogical signs referable to the thalamocortex, brain stem, or cerebellum are present; and the CSF protein concentration is less than 1 g/L, the total cell count is less than 50 cells/μL, and the differential cell count is normal or has an increased percentage of neutrophils or lymphocytes.

The age range in this group was similar to that of FIP, but the mean duration of clinical signs prior to presentation was shorter, although still characteristic of a chronic progressive disease. In two reports of cats with meningoencephalitis or encephalitis, in which a viral origin other than FIP was suspected, the course of illness was usually prolonged (mean 7–10 weeks), and many of the cats were less than three years of age (18,19). The lesions reported were similar to those observed in our study and consisted of mononuclear perivascular cuffs, lymphocytic-histiocytic meningeal inflammation, and microgliosis. A number of cats were reported to have spinal cord involvement, but this was not present in our study.

A variety of viruses, including parvovirus (25), calicivirus (26), herpesvirus (27), Newcastle disease virus (28), Near Eastern equine encephalomyelitis virus (29,30), and Powassan virus (31), have been reported to be associated with encephalitis in cats, following natural or experimental infection. Although insufficient information is available to determine which virus/viruses were involved, an arbovirus would be a possible candidate, because six different arboviruses known to cause encephalitis in man have been isolated in Ontario (32); Powassan, snowshoe hare, and St. Louis encephalitis viruses are considered endemic in the wild mammal population in Ontario (32); naturally occurring serum antibody titers to the arboviruses, snowshoe hare virus, Jamestown Canyon virus, and Powassan virus have been reported in cats in Canada (31,33); two of seven cats experimentally inoculated with Powassan virus developed nonsuppurative encephalitis (31); and two cats reported with naturally occurring titers to Jamestown Canyon virus had anorexia and depression, and one developed nonsuppurative meningitis and uveitis (31).

Protozoan infection

The two cats with protozoan infection were old, their neurological signs were focal and suggestive of a mass lesion, and lymphocytes or voluminous monocytoid cells predominated in the CSF.

Clinical CNS disease is reported to be one of the less frequent signs associated with *Toxoplasma* infection in cats (34,35). In one study, the organism was isolated from the brain of seven out of 16 cats with toxoplasmosis, although only three showed clinical CNS disease (35). In our study of 61 cats with CNS disease, there were only two (5%) confirmed cases of protozoan infection. The histological findings were similar to those previously reported (35). Because immunocytochemical staining of the organisms was not done, the cysts cannot be differentiated with certainty from *Neospora* spp.

Immunosuppression is reported to be a predisposing factor in the development of clinical disease in several species including cats (34,36,37); it appeared to be involved in one cat with leukopenia due to myelodysplasia and may have been a factor in the other cat, which was hypoglobulinemic.

Published reports indicate that toxoplasmosis causes increases in CSF protein and the percentage of neutrophils, similar to those seen in FIP (7,38). However, in our study, only mild increases in CSF protein were present, and the CSF white cell count and the percentage of neutrophils were not increased when the degree of blood contamination was taken into consideration. The lesions were confined to the brain parenchyma or the lumbar spinal cord, which may account for the disparity between the histological and CSF findings. The differential cell count was abnormal, although not typical for FIP, with increased lymphocytes, macrophages, or voluminous monocytoid cells. Voluminous monocytoid cells, characterized by a very low nuclear/cytoplasmic ratio and voluminous, often foamy, cytoplasm, are rare in the CSF of normal cats. Abnormally high numbers of these cells were present in one cat with protozoan infection and in another with bacterial infection, both of which had marked CNS necrosis.

Other inflammatory diseases

Bacterial

Bacterial infection of the CNS was a rare finding in this study, as it was in the dog (39). Although the diagnosis was evident from the intracellular bacteria and marked inflammatory response in the CSF, bacteria were relatively sparse and only detected by careful microscopic examination. Based on the long duration of the clinical signs and the high cell count, FIP would be a major consideration if bacteria were not seen. However, the protein concentration and differential cell count (<70%

neutrophils and increased voluminous monocytoid cells) suggested a disease process other than FIP. The severe inflammatory process in the CSF was unexpected in view of the necrosis and absence of marked inflammation on histology.

Choroid plexitis

The etiology of the choroid plexitis was unknown, although the lymphocytic infiltrate was compatible with an immune response, possibly due to a viral infection or immune-mediated disease. Immune complex deposition occurs in the choroid plexus of humans with systemic lupus erythematosus, and in rabbits with experimentally induced acute immune complex disease (40). Choroid plexitis is also a common lesion in FIP (41), although other lesions typical of FIP were absent in this cat.

Eosinophilic meningoencephalitis

Our clinical and CSF findings are similar to those previously reported in one cat (42). The cat in our study did not show evidence of facial pawing, but it did have a nasal discharge. In the absence of blood contamination, significant numbers of eosinophils in the CSF are suggestive of parasitic CNS disease (e.g., *Dirofilaria immitis*, *Cuterebra* spp., *Toxoplasma* spp. (5,43,44), or eosinophilic meningoencephalitis (42). In man (45,46) and dogs (44,47), many etiologies have been associated with eosinophilic pleocytosis, including CNS parasitic infection and CNS neoplasia. Blood contamination of the CSF increases the percentage of eosinophils and must be considered before ascribing the increase in eosinophils to disease (1).

In conclusion, in our study, more than 75% of all inflammatory CNS disease observed was attributed to viral agents. Half of these cases were due to FIP, but in approximately 50%, the causative virus was not identified. Further investigation is needed to isolate the viral agent/s involved, as it is a significant cause of CNS disease in cats. In the remaining 25% of cats with non-viral inflammatory CNS disease, a diverse range of etiologies were represented. In most cats with FIP, the CSF findings were distinctive and diagnostic. In all other causes of CNS inflammatory disease, excepting bacterial infection, the CSF findings were nonspecific. In these cats, by combining the CSF analytical results with the clinical data, a better diagnostic assessment was possible.

Acknowledgments

We thank Dr. Brian Allan, Mr. Denis Thomson, and Ms. Anne Valliant for assistance with statistical analysis.

CVJ

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Answers to Quiz Corner/Les réponses du Test Éclair

1. b — There are many clinical manifestations of bovine virus diarrhea, but most infections are inapparent.
   b — Il existe plusieurs manifestations cliniques de la diarrhée virale bovine, mais la plupart des infections sont inapparentes.
2. e — Especially if bred for the first time after eight months of age.
   e — Surtout si les animaux sont accouplés pour la première fois après l'âge de huit mois.
3. d — In a low-incidence population, a test with a high positive predictive value is the best choice. Tests of high specificity have a high positive predictive value.
   d — Dans une population à faible incidence, un test avec une valeur prédictive positive élevée constitue le meilleur choix. Les tests de spécificité élevée possèdent une valeur prédictive positive élevée.
4. c — Ventricular septal defect is relatively common in cats. The location of this murmur is consistent with a ventricular septal defect. The radiographs confirm the volume overload state.
   c — La communication interventriculaire est relativement fréquente chez les chats. La localisation de ce souffle est compatible avec une communication interventriculaire. Les radiographies confirment l'état de surcharge volumique.
5. e — Over 85% have stage-I or more severe periodontal disease.
   e — Plus de 85 % des animaux souffrent de paradontolyse de stade 1 ou plus grave.
6. a — Pleuropneumonia or lung abscesses are found in approximately two-thirds of patients with pleural effusion.
   a — Une pleuropneumonie ou des abcès pulmonaires se rencontrent chez environ les deux tiers des animaux avec un épanchement pleural.
7. b
8. d — Exudative epidermitis, also known as greasy pig disease, is a common bacterial skin disease of piglets 5–35 days of age. A toxin produced by Staphylococcus hyicus causes marked exudation on the epidermis. The toxin can also damage the kidneys.
   d — L'épidermite exsudative, également connue sous le nom d'épidermite séborrhéique du porc (greasy pig disease) est une infection cutanée bactérienne fréquente chez les porcelets âgés de 5 à 35 jours. Une toxine produite par Staphylococcus hyicus cause une exsudation importante de l'épiderme. La toxine peut également endommager les reins.
9. b — Evaluation of the eating patterns of acetonemic cows is helpful in making a diagnosis. Affected cows usually refuse grain first and then silage. These cows usually continue to eat hay.
   b — Une évaluation de la façon dont les vaches acétonémiques se nourrissent est utile pour poser un diagnostic. Habituellement, les vaches affectées refusent d'abord les grains puis ensuite l'ensilage. Habituellement, ces animaux continuent à manger du foin.
10. c