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Inflammatory cerebrospinal fluid analysis in cats: clinical diagnosis and outcome

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The medical records of 62 cats with clinical signs of central nervous system disease and accompanying inflammatory cerebrospinal fluid (CSF) analysis were examined retrospectively to determine if signalment, clinical signs, CSF analysis and ancillary testing could accurately predict the type of central nervous system disease that was present. An inflammatory CSF was defined as one in which a total nucleated cell count was greater than 5 cells/μl or one in which the total nucleated cell count was normal but the nucleated cell differential count was abnormal. Sex, degree of CSF inflammation, neuroanatomical location and systemic signs provided little contributory information to the final diagnosis. In 63% of the cases a presumptive diagnosis could be made based on a combination of clinical signs, clinicopathological data and ancillary diagnostic tests. CSF analysis alone was useful only in the diagnosis of cats with feline infectious peritonitis, *Cryptococcus* species infection, lymphoma and trauma. Overall, despite extensive diagnostic evaluation, a specific diagnosis could not be made in 37% of cats. The prognosis for cats with inflammatory CSF was poor with 77% of cats surviving less than 1 year.

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Inflammation of the central nervous system (CNS) is common in cats of all ages. Despite this, few attempts have been made to evaluate how well cerebrospinal fluid (CSF) analysis, clinical signs and other clinicopathological data correlate with the disease occurring in the CNS. Causes of CNS inflammation in cats include viral, protozoal, fungal, parasitic and bacterial infection, 'immune mediated' and 'idiopathic' disorders of uncertain aetiology (eg, non-suppurative and eosinophilic meningoencephalitis) (Munana 2001). In addition to primary inflammatory CNS disorders, there are diseases that can induce CNS inflammation secondary to tissue damage and necrosis. Such diseases include neoplasia, trauma, intervertebral disc disease, cerebrovascular disease and nutritional disorders (eg, thiamine deficiency). The purpose of this study was to determine if signalment, clinical signs, CSF analysis, additional clinicopathological data and diagnostic imaging could be used to determine the specific aetiology of the CNS disease in cats with inflammatory CSF. Prognosis for the various disease classifications was also evaluated.

Materials and methods

Records were searched at three referral centres in Sydney, NSW from January 1995 to December 2002 for cats that had CSF analysis. CSF collection was performed in 111 cats. Of these, 62 cats could be classified as having inflammatory CNS disease. CSF was collected by percutaneous puncture from either the cerebello-medullary cistern or via lumbar puncture from either the L5–L6 or L4–L5 intervertebral space. CSF analysis included total white cell count and red cell count by haemocytometer, cytomorphology by stained sediment (Idexx Laboratories Pty Ltd) or stained cytocentrifuge smears (The University of Sydney Department of Clinical Pathology) and total protein by evaluation of microprotein on a commercial biochemistry autoanalyser (Olympus AU 400, Idexx Laboratories; Cobas Mira, The

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University of Sydney Department of Clinical Pathology). The reference ranges used by our laboratories for feline CSF were: white cell count ≤5 cells/μl, red cell count 0 cells/μl and total protein <0.3 g/l (Canfield and Martin 1998, Raskin and Myer 2001). An inflammatory CSF analysis was defined as one in which a total nucleated cell count was greater than 5 cells/μl or one in which the total nucleated cell count was normal but the nucleated cell differential count was abnormal (>9% neutrophils, >1% eosinophils) (Canfield and Martin 1998, Raskin and Myer 2001). CSF nucleated cell counts were classified as: 1, normal: ≤5 cells/μl; 2, mildly elevated: 6–50 cells/μl; 3, moderately elevated: 51–1000 cells/μl and 4, markedly elevated: >1000 cells/μl. The nucleated cell population was classified as: 1, suppurative: >50% neutrophils; 2, non-suppurative: >80% mononuclear cells; 3, eosinophilic: >50% eosinophils and 4, mixed: no predominance of any one cell type (Canfield and Martin 1998, Raskin and Myer 2001). CSF total protein was classified as 1, normal: ≤0.3 g/l; 2, mildly elevated: 0.31–10 g/l; 3, moderately elevated: 11–20 g/l and markedly elevated: >20 g/l. CSF erythrocyte counts were classified as: 1, normal: 0 cells/μl; 2, mildly elevated: 1–49 cells/μl; 3, moderately elevated 50–1000 cells/μl and 4, markedly elevated: >1000 cells/μl (Table 1). The contribution of CSF red cell contamination to the total CSF white cell count was corrected for by subtracting 1 nucleated cell for every 100 red cells (Rand et al 1990). Cats with CSF nucleated cell count that was less than or equal to that predicted from blood contamination were excluded from the study.

Haematology was performed on standard automated counters (Celldyn, Idexx Laboratories; Sysmex K-4500, University of Sydney Clinical Pathology). Differential cell counts were performed via stained (Diff Quick) blood smear examination. Biochemistry was performed using standard automated chemistry analysers described previously. Cryptococcal antigen titres were measured by latex agglutination (negative titre = 0), *Toxoplasma gondii* IgG antibody titres were measured by indirect haemagglutination (negative = <1:80), *T gondii* IgM titres were measured by indirect fluorescent antibody (negative < 1:80), feline leukaemia virus (FeLV) antigen was measured by ELISA, feline immunodeficiency virus (FIV) specific antibody was measured by ELISA and feline coronavirus (FeCoV) antibody was measured by indirect fluorescent antibody (negative titre = <1:100).

Owners and referring veterinarians were contacted to determine the outcome after discharge from hospital. The evaluation period ranged from 0.4 months to 7 years. The following parameters were evaluated for each cat: 1, signalment; 2, duration of clinical signs; 3, neurological findings and localisation of lesion; 4, systemic signs; 5, cerebrospinal fluid testing; 6, ancillary testing including results of culture, diagnostic imaging, blood evaluation and 7, outcome.

Results

Sixty-two cases had sufficient data and met the criteria of inflammatory changes within the CNS.

Signalment

There was no age or sex distribution pattern. The majority of cats were domestic shorthair (56%), while there were four Burmese, four Persians and three Ragdolls. The remainder represented many different breeds including Himalayan, Chinchilla, Birman, Burmilla, Siamese, Cornish Rex, Devon Rex, Russian Blue, Oriental Shorthair, Abyssinian and domestic longhair. There were no obvious breed risks associated with the inflammatory changes within the CNS. The age range was 4 months to 17 years, with a median age of 8 years.

Duration of clinical signs

Thirteen cats (21%) presented with acute clinical signs of less than 2 weeks' duration. Forty-nine cats (79%) presented with clinical signs longer than 2 weeks duration.

Neurological findings and localisation of lesion

Fifty-two cats (83.9%) presented with gait abnormalities that included ataxia, hypermetria, paresis/paralysis or unilateral lameness. Fifteen cats (24.2%) presented with seizures, partial or generalised. Twelve cats (19.4%) exhibited nystagmus, 12 cats (19.4%) had a head tilt, 11 cats (17.7%) had other cranial nerve deficits (facial nerve or trigeminal nerve paralysis), eight cats (13%) had reduced mentation and eight (13%) had abnormal postural reactions. Other signs encountered included circling, spinal pain, anisocoria and behavioural abnormalities. In 23 cats (37%) the presentation suggested multifocal CNS disease while in 39 cats (63%) the lesions appeared focal (cerebral cortex in nine cats, cerebellum in two cats, central vestibular in five

Table 1. Cerebrospinal fluid abnormalities in cats with inflammatory CSF

	Protein (g/l)				White cells (cells/μl)[a]							Red cells (cells/μl)				Number of cats
	Normal <0.3	Mild 0.31–10	Mod 11–20	Marked >20	Normal ≤5	Mild 6–50	Mod 51–1000	Marked >1000	Mononuclear	Suppurative	Mixed	Normal 0	Mild 1–49	Mod 50–1000	Marked >1000	
FIP	2	4	1	2	0	2	3	5	3	6	2	3	3	0	3	11
Cryptococcus	2	0	0	1	0	2	1	1	2	2	0	1	1	2	0	4
Toxoplasma	0	1	0	0	0	2	0	0	1	1	0	0	1	1	0	2
Other meningoencephalitis	2	1	1	0	0	2	2	1	4	0	1	1	1	3	0	5
Thiamine deficiency	1	1	0	0	1	1	0	0	0	2	0	0	1	1	0	2
Lymphoma	1	4	0	0	0	4	1	1	6	0	0	0	3	2	1	6
Other neoplasia	0	6	0	0	0	7	1	0	2	0	6	0	1	4	3	8
Trauma	0	3	0	0	0	1	2	0	0	2	1	0	0	0	3	3
Intervertebral disc disease	0	1	0	0	0	1	0	0	0	0	1	0	0	1	0	1
Spinal cord granuloma	0	1	0	0	0	1	1	0	0	0	2	0	1	1	0	2
Undiagnosed	4	12	0	0	3	12	3	0	3	8	6	1	7	7	2	18
Number of cats	12	34	2	3	5	34	14	14	21	22	18	6	19	22	12	

Mod = moderate, FIP = feline infectious peritonitis.
[a] Eosinophilic category not shown (one cat).

cats, brainstem in six cats and spinal cord in 17 cats).

Systemic signs

Ten cats had ocular abnormalities including uveitis, keratinic precipitates, fundic abnormalities and panophthalmitis, while nine cats had inappetence, and nine cats had pyrexia.

Cerebrospinal fluid testing

Five cats had 0–5 nucleated cells/μl, 34 cats had 6–50 nucleated cells/μl, 14 cats had 51–1000 nucleated cells/μl and eight cats had >1000 cells/μl (Table 1). One cat did not have a nucleated cell count performed because of insufficient sample. Twenty-two cats (35%) had a suppurative CSF, 21 cats (34%) had a mononuclear pleocytosis, 18 cats (29%) had mixed inflammation and one cat had an eosinophilic inflammation. Thirty-nine cats (63%) had an elevated total protein. Thirty-four cats (55%) had a total protein of 0.31–10 g/l, two cats had a total protein of 11–20 g/l and three cats had a total protein of >20 g/l. Eleven cats (17.7%) did not have a total protein measured because of insufficient sample. Twelve cats had more than 1000 red cells/μl. Three cats had CSF characteristics indicative of haemorrhage characterised by activated macrophages and large numbers of haemosiderophages with erythrophagocytosis. In the remaining cases the increased CSF erythrocyte count was consistent with blood contamination during collection.

Ancillary test procedures

The most common ancillary testing procedures were haematology in 47 cats (76%), biochemistry in 46 cats (74%) and cryptococcal antigen titres in 26 cats (42%). Fifteen cats (24.2%) had a myelogram performed, 14 cats (22.5%) had *T gondii* IgG antibody titres measured and 10 cats (16.1%) had FeLV antigen tested. FIV antibody, thoracic radiographs, skull radiographs and abdominal ultrasound were performed in nine cats each. Surgical biopsy was performed in five cats. FeCoV antibody titre measurement and computed tomography (CT) scan were performed in four cats each. Urinalysis and fine needle aspirate biopsy were performed in four cats each. Blood pressure, *T gondii* IgM antibody titre, magnetic resonance imaging (MRI), thyroid hormone concentration and cholinesterase concentration were performed in two cats each. Ammonia tolerance testing, blood lead concentrations and bone marrow aspiration were performed in one cat each. The most frequent abnormalities seen were increased serum globulins in 16 cats (26%), anaemia in nine cats (14.5%), an abnormal myelogram in 11 cats (18%) and a peripheral neutrophilia in five cats (8%).

Outcome

Thirty-five cats (53.2%) survived less than 1 month after presentation. Three died or were euthanased immediately after cerebrospinal fluid collection. Of these, one died due to uncontrollable seizures and two were euthanased because of persistent apnoea. Ten cats (16.1%) survived 1–6 months, three cats (4.8%) survived 7–12 months and 12 cats (19.4%) survived greater than 12 months and were still alive at the time of writing. Of the cats that died or were euthanased, 18 (37%) had a post mortem performed, 30 cats (62%) did not. Two cats were lost to follow-up.

Classification

Based on the results, clinical information, clinical pathology and ancillary testing procedures the following classifications of disease could be made: 1, feline infectious peritonitis (FIP); 2, *Cryptococcus* species infection; 3, *Toxoplasma* species infection; 4, other meningoencephalitis; 5, thiamine deficiency; 6, lymphoma; 7, other neoplasia; 8, trauma; 9, intervertebral disc disease; 10, spinal cord granuloma and 11, undiagnosed (Table 2).

FIP

Eleven cats (17%) were diagnosed with FIP. A presumptive diagnosis of FIP was made based on age, a suppurative or mixed inflammatory CSF, poor response to treatment, elevated serum or body cavity effusion FeCoV antibody titre or a reduced albumin:globulin ratio (<0.5) of serum or body cavity effusions. The diagnosis was confirmed at necropsy in eight cats and on surgical biopsy in one other (83% of the cases). The remaining two cats had a diagnosis of FIP made based on ancillary diagnostic tests (serum and body cavity effusion albumin:globulin ratio <0.5, and elevated FeCoV antibody titre in serum and body cavity effusions). The ages ranged from 4 months to 11 years of age with a median of 12 months. Breed distribution did not differ from the total population of cats

Table 2. Disease classification and outcome

Disease	Number of cats (clinical/post mortem diagnosis)	Mortality				Still alive	Unknown
		0–1 months	1–6 months	6–12 months	>12 months		
FIP	11/8	11	0	0	0	0	–
Cryptococcus	4/1	1	0	1	0	2	–
Toxoplasmosis	2/2	1	1	0	0	0	–
Other meningoencephalitis	5/2	2	0	0	0	3	–
Thiamine deficiency	2/1	1	0	0	0	1	–
Neoplasia							
Lymphoma	6/0	3	2	0	0	0	1
Other	8/2	4	1	2	0	1	–
Trauma	3/0	0	0	0	0	3	–
Intervertebral disc disease (IVDD)	1/0	0	0	0	0	1	–
Spinal cord granuloma	2/0	0	1	0	0	1	–
Undiagnosed	18/2	11	5	0	1	0	1

studied. The most common neurological signs were gait abnormalities in seven cats and reduced mentation in three cats. Systemic signs included pyrexia in six cats, lethargy in three cats, ocular abnormalities in six cats and weight loss in four cats. Four cats had an abdominal effusion and one cat a pleural effusion. Ten cats had clinical signs consistent with multifocal lesions of the central nervous system. These were referrable to a combination of cerebral cortex and brainstem (often central vestibular) signs. One cat had cerebellar signs only.

CSF analysis was characterised as suppurative in seven cats, mixed in one and mononuclear in three. Five cats had a marked elevation in the CSF white cell count (>1000 cells/μl), three cats had moderate elevations (51–1000 cells/μl), two cats had mild elevations (6–50 cells/μl) and one cat had insufficient sample for a white cell count. Seven cats had increased CSF protein concentrations. Four cats had a mild elevation (0.31–10 g/l), one cat had a moderate elevation (11–20 g/l) and two cats had marked elevations (>20 g/l). Two cats had normal CSF protein concentrations and two cats did not have CSF protein concentrations measured because of insufficient sample. All 11 cats had haematology and serum biochemistry preformed. Six cats had a mild, non-regenerative anaemia and three cats had a mild-moderate mature neutrophilia. No cat was lymphopenic. Ten cats had elevated serum globulin concentrations, two had elevated alanine aminotransferase (ALT) and one cat had an elevated bilirubin concentration. Of the two cats without a histopathological diagnosis, one had a serum FeCoV antibody titre of 1:800 and an FeCoV antibody titre in ascitic fluid of 1:800. This cat had an albumin:globulin ratio of 0.4 in the ascitic fluid and a CSF albumin:globulin ratio of 0.39. The second cat had a serum FeCoV antibody titre of 1:1600 and serum albumin:globulin ratio of 0.3. No cat survived longer than 10 days after presentation. One cat has a seizure and died immediately after CSF collection.

Cryptococcus

Four cats were diagnosed with central nervous system cryptococcosis based on a positive serum antigen titre and/or the presence of cryptococcal organisms in the CSF. All were spayed female. The age range was 2–11 years with median age of 9 years. Three cats were domestic shorthair and one was a Burmese. All four cats had gait abnormalities. Other clinical signs observed were reduced mentation, spinal pain, seizures, circling, reduced postural reactions and nystagmus. Systemic signs observed included bradycardia, inappetence and ocular abnormalities. The location of the neurological signs was variable, encompassing all central regions except the cerebellum.

Two cats had suppurative CSF and two had mononuclear CSF. Three cats had large numbers of cryptococcal organisms in the CSF. One had no organisms detected in the CSF and was initially diagnosed with CNS toxoplasmosis (based on a moderate mixed inflammatory CSF,

T gondii IgG of 1:60 and IgM of >1:160). At necropsy 8 months later, a large cryptococcal granuloma was detected in the cerebrum. This cat had a premortem cryptococcal antigen titre of 1:32.

CSF nucleated cell numbers ranged from 15 to 2098 cells/μl. The protein concentration was normal in two cats, markedly elevated in one cat (26 g/l) and not measured in the other. Three cats had positive serum cryptococcal antigen titres, although in one cat it was very low (1:32). An antigen titre was not measured in one case. Two cats had elevated serum globulins and one cat had a moderate mature neutrophilia. Two cats recovered and survived more than 5 years after diagnosis. One cat was euthanased 4 days after presentation and did not undergo a post mortem examination.

Toxoplasma

Two cats were diagnosed with CNS toxoplasmosis. A presumptive diagnosis was made based on a mild suppurative-mixed CSF inflammation, normal-mildly increased CSF protein levels and positive IgG antibody titre. Both cats had the clinical diagnosis confirmed at necropsy (*T gondii* tachyzoites observed in the CNS). One of the cats was diagnosed with suppurative meningitis of unknown cause. It was euthanased 3 months after presentation and necropsy revealed CNS toxoplasmosis. Both cats presented with gait abnormalities, head tilt and anisocoria. One cat had mild neutrophilic CSF inflammation with a mildly elevated protein (0.58 g/l) and did not have *T gondii* antibody titres measured. The other cat had mild mononuclear CSF inflammation. The CSF protein levels were not measured in this cat because of insufficient sample. One had a positive *T gondii* IgG antibody titre (1:1024). Both cats presented with a chronic course of disease and multifocal neurological signs. Both cats had elevated serum globulins and abnormal thoracic radiographs (a single nodular opacity present in both). Survival times were 2 and 12 weeks.

Other meningoencephalitis

Five cats were diagnosed with meningoencephalitis of unknown cause. They were subclassified, according to response to treatment, into non-suppurative meningoencephalitis and steroid responsive meningoencephalitis.

Non-suppurative meningoencephalitis

Three cats were diagnosed with non-suppurative meningoencephalitis on the basis of a mononuclear pleocytosis in the CSF or histopathology indicating a perivascular mononuclear infiltrate. One cat recovered from the disease over 10 days and was clinically normal 2 years later. The other two cats were euthanased within 4 weeks because of persistent neurological signs. Both cats received a post mortem examination that revealed a mononuclear perivascular cellular infiltrate in the cerebral cortex of one cat and in the cerebral cortex and brainstem of the other cat. The ages of these cats ranged from 3 to 8 years with a median age of 5 years. The main neurological signs were seizures in two, and one each of nystagmus, gait abnormalities, behavioural changes and reduced mentation. Extra-neural signs included one each of weight loss, inappetence, pyrexia and ocular abnormalities. All three cats presented with chronic signs of illness. Two cats had multifocal signs and one had focal cerebral signs. All three cats had mononuclear CSF inflammation with mild-moderate elevations in the CSF leukocyte counts. CSF protein concentrations were normal in two cats and not measured in one because of insufficient sample. No cat had serological testing for infectious diseases.

Steroid responsive meningoencephalitis

Two cats had a marked inflammatory pleocytosis on CSF analysis but made a complete recovery with corticosteroid therapy and were diagnosed as a steroid responsive meningoencephalitis. One cat was a 5-year-old female spayed Ragdoll, the other a 12-year-old male castrate Russian Blue. The Ragdoll presented with acute signs of seizures, circling, proprioceptive deficits and ataxia. The CSF nucleated cell count was markedly elevated (2960 cells/μl) with a mixed inflammatory pattern and moderate blood contamination (90 cells/μl). The CSF protein concentration was mildly elevated (4.3 g/l). FIP was the initial clinical diagnosis and prednisolone (Macrolone; Mavab) was prescribed. The cat made a dramatic improvement within 2 days of prednisolone therapy and was gradually weaned off prednisolone over 6 months. Twelve months later the cat was clinically normal. The Russian Blue presented with a chronic history of hindlimb paresis with localisation of signs to the thoracolumbar spinal cord. The nucleated cell count was moderately elevated (623.7 cells/μl)

with moderate blood contamination (53 cells/μl). The CSF protein concentration was moderately elevated (11.6 g/l). The inflammation was mononuclear. Complete blood count, biochemical profile and spinal radiographs did not reveal any abnormalities. The cat was treated with prednisolone (Macrolone; Mavlab) and gradually improved over the following 6 days. This cat was also gradually weaned off prednisolone over 6 months and was clinically normal 2 years later.

Thiamine deficiency

Two cats were diagnosed with thiamine deficiency on the basis of a clinical history of a thiamine deficient diet, response to thiamine supplementation and a mild suppurative inflammatory CSF. A 6-year-old female spayed Himalayan had an acute onset of ataxia, behavioural abnormalities, absent menace and absence of physiological nystagmus. There was a mildly elevated CSF nucleated cell count (10.1 cells/μl) that was suppurative. The cat had been fed a diet of sulphur dioxide preserved kangaroo meat exclusively for 8 weeks. Treatment included thiamine (thiamine hydrochloride; Natural Health Products), 20 mg PO q12 h and a diet change to a balanced cat food. The cat showed a dramatic improvement within 24 h of therapy and was clinically normal at long term follow-up 2 years later. The other was a 9-year-old male castrated domestic shorthair that also had with an acute onset of ataxia, depression, nystagmus, postural reaction deficits and weight loss. The CSF leukocyte count was normal (2 cells/μl) but consisted entirely of non-degenerate neutrophils. The cat was treated with clindamycin (Antirobe; Pharmacia and Upjohn) as blood tests revealed elevated bilirubin, alanine aminotransferase (ALT), alkaline phosphatase (ALP), and a *Toxoplasma* species IgG of >2560. The cat was euthanased 3 days later because of marked deterioration. Necropsy revealed changes consistent with thiamine deficiency and hepatic lipidosis.

Neoplasia

Fourteen cats (22.6%) with inflammatory cerebrospinal fluid were diagnosed with neoplasia. Of these 14 cats, six (43%) had a confirmed diagnosis of lymphoma based on a mononuclear inflammatory CSF with abnormal lymphocytes, or when needle aspiration/biopsy of extracranial sites confirmed malignant lymphoid cells. Eight (57%) were diagnosed with other neoplasms on the basis of chronic progressive clinical signs, mild mixed inflammatory CSF and ancillary laboratory tests (radiographs, myelogram, MRI, CT, surgery and biopsy, Table 3). Only three of these cases had a confirmed histopathological diagnosis of the type of neoplasia.

Lymphoma

Six cats were diagnosed with CNS lymphoma. The ages ranged from 4 to 17 years with a median of 13.5 years. The main clinical signs were gait abnormalities in six cats, reduced conscious proprioception in three cats and a head tilt in two cats. One cat had multiple cranial nerve deficits (nystagmus, anisocoria and facial nerve paralysis). Systemic signs included anorexia in one cat and ocular abnormalities in one cat. Five cats had a chronic course of disease while one was presented with acute clinical signs. In three cats the clinical signs localised the disease to the spinal cord. Two cats had multifocal disease and one cat focal cerebral disease. All six cats had mononuclear inflammation and in five cats, large atypical lymphoid cells were observed in the CSF. In the sixth cat, lymphoma was diagnosed via fine needle aspirate biopsy of the kidneys. Four cats had mildly elevated CSF leukocyte counts (6–50 cells/μl). The remaining two cats had moderate (218 cells/μl) and marked (1144 cells/μl) CNS inflammation. One cat had a normal CSF protein level and four had mild elevations (0.31–10 g/l). In one cat the protein was not measured due to insufficient sample. The most common abnormal ancillary tests were non-regenerative anaemia in three and leukocyte abnormalities (varying combinations of eosinophilia, neutrophilia, monocytosis and abnormal peripheral lymphocytes) in three. One cat had abnormal thoracic and abdominal radiographs (sternal lymphadenopathy and hepatomegaly), one had an abnormal myelogram (spinal cord compression), one had lymphoma revealed by fine needle aspirate biopsy of the kidneys and one had a computed tomography scan which revealed a mass in the frontal lobe. The cat with hepatomegaly had lymphoma confirmed by transabdominal hepatic fine needle aspirate biopsy. The cat with the abnormal myelogram had lymphoma of the spinal cord confirmed by surgical fine needle aspirate biopsy. Three cats survived less than 1 month, two survived 1–6 months and one cat was lost to follow-up. No cat

Table 3. Clinical signs and significant ancillary tests

Cat	Signalment	Clinical signs	Abnormal ancillary tests	Post mortem results
FIP				
8	6 mo, MC Burmilla	Dullness, lethargy, ataxia, URTI, pyrexia, absent visual placing, weight loss	Hyperglobulinaemia, anaemia Elevated serum ALT Serum FeCoV 1:800, 7B FIP 1:160, FeCoV (ascites) 1:800 Albumin:globulin ratio (ascites) 0.4	–
17	1 yo, FS DSH	Ataxia, pyrexia, bilateral anterior uveitis and keratinic precipitates	Hyperglobulinaemia Serum FeCoV 1:1600 Serum albumin:globulin ratio 0.3	–
20	6 mo, FE Ragdoll	Photophobia, opisthotonos pyrexia	Hyperglobulinaemia, anaemia, neutrophilia	Pyogranulomatous inflammation of leptomeninges, choroid plexus, ependyma and surrounding vessels. Reactive mesenteric lymph nodes. Peritonitis
21	6 mo, FS DLH	Ataxia, pyrexia, lethargy, weight loss, bilateral anterior uveitis	Hyperglobulinaemia, anaemia Abdominal ultrasound – enlarged, nodular liver, mesenteric lymphadenopathy Surgical liver biopsy – pyogranulomatous inflammation of liver (surrounding vessels) and mesenteric lymph nodes FeCoV 1:1100	–
40	2 yo, MC Burmese	Dullness, lethargy, ataxia, pyrexia, bilateral blepharospasm, panophthalmitis	Hyperglobulinaemia Thoracic radiographs – sternal lymphadenopathy Abdominal ultrasound – mesenteric lymphadenopathy and splenic nodules FNA – pyogranulomatous inflammation	Pyogranulomatous inflammation, necrosis and vasculitis of spleen, liver, lymph nodes, lungs, brain and eyes
41	1 yo FE Burmese	Weakness, ataxia, hypermetria, weight loss, colitis	Hyperglobulinaemia	Pyogranulomatous meningoencephalitis, mild hydrocephalus
42	4 mo ME DSH	Ataxia, reduced conscious proprioception, postural reaction deficits, anisocoria, hyperreflexia, opisthotonos	Hyperglobulinaemia Abdominal ultrasound – hepatomegaly, mesenteric lymphadenopathy	Pyogranulomatous inflammation of brain, kidneys, eyes and mesenteric lymph nodes

Table 3. *Continued*

Cat	Signalment	Clinical signs	Abnormal ancillary tests	Post mortem results
43	11 yo MC Burmese	Seizures, nystagmus, head tilt	Hyperglobulinaemia, anaemia	Pyogranulomatous inflammation and vasculitis of meninges and mesenteric lymph nodes
44	5 mo ME Cornish Rex	Dullness, lethargy, ascites, icterus	Anaemia, neutrophilia Ascites	Pyogranulomatous inflammation of brain, liver and mesenteric lymph nodes
45	4 yo FE Birman	Weight loss, pyrexia, URTI	Hyperglobulinaemia, elevated ALT, ALP, bilirubin	Pyogranulomatous inflammation of brain, liver, intestinal wall, mesenteric lymph nodes. High protein ascites and pleural effusion
50	3 yo FS DSH	Quadraparesis, opisthotonos, anorexia, muscle wasting, retinal detachment	Hyperglobulinaemia, neutrophilia, anaemia	Pyogranulomatous inflammation of brain and mesenteric lymph nodes
Cryptococcus				
18	11 yo FS DSH	Dullness, spinal pain, paraparesis	No abnormalities	Recovered
39	7 yo FS DSH	Seizures, circling, ataxia	LCAT 1:32	Cerebral cryptococcal granuloma
52	11 yo FS DSH	Ataxia, nystagmus, postural reaction deficits	Hyperglobulinaemia LCAT 1:2048 CSF culture − *Cryptococcus neoformans* var. *gattii*	Recovered
54	2 yo FS Burmese	Tetraparesis, anorexia, blind with dilated pupils and absent PLR, oedematous optic discs, bradycardia	Hyperproteinaemia, neutrophilia Serum LCAT 1:16384, CSF LCAT 1:16384 CSF culture − *Cryptococcus neoformans* var. *neoformans*	−
Toxoplasma				
35	4 yo MC OSH	Ataxia, weakness, head tilt, anisocoria, diarrhoea	Hyperglobulinaemia, proteinuria, haematuria Toxoplasma IgG 1:1024 Thoracic radiographs − 1 cm focal pulmonary opacity, diffuse interstitial lung pattern	Inflammation, necrosis and Toxoplasma tachyzoites in brain and pulmonary parenchyma. Chorioretinitis
47	7 yo MC DSH	Anorexia, circling, head tilt, nystagmus, anisocoria	Hyperglobulinaemia, non-regenerative anaemia Thoracic radiographs − 3 mm nodule	Inflammation, necrosis, Toxoplasma tachyzoites in brain

(*continued on next page*)

Table 3. *Continued*

Cat	Signalment	Clinical signs	Abnormal ancillary tests	Post mortem results
Other ME				
33	3 yo FS Cornish Rex	Seizures, nystagmus, weight loss	Lymphopaenia, neutropenia, toxic neutrophils	Perivascular mononuclear infiltrate in cerebral cortex and brain stem
36	5 yo MC Himalayan	Dullness, lethargy, paraparesis, muscle tremors, behavioural changes, unilateral corneal ulcer	Elevated albumin, elevated HCT, neutrophilia, lymphocytosis	Recovered
55	8 yo FS DSH	Seizures, anorexia, pyrexia	Hyperglobulinaemia, neutropenia, anaemia	Perivascular mononuclear infiltrate in cerebral cortex
7	12 yo MC Russian Blue	Paraparesis	No abnormalities	Recovered
16	5 yo FS Ragdoll	Seizures, ataxia, proprioceptive deficits	No abnormalities	Recovered
Thiamine def				
3	6 yo FS Himalayan	Ataxia, absent physiological nystagmus, absent menace reflex, abnormal behaviour	No abnormalities	Recovered
25	9 yo MC DSH	Lethargy, ataxia, nystagmus weakness, abnormal postural reactions, weight loss	Elevated serum bilirubin, ALP and ALT	Necrosis of the periventricular grey matter, lateral geniculate nuclei, oculomotor nuclei, caudal colliculi and vestibular nuclei in the brain. Hepatic lipidosis
Lymphoma				
5	17 yo FS DSH	Ataxia, head tilt, paraparesis, spinal pain, proprioceptive deficits	Eosinophilia	–
6	15 yo FS DSH	Right hindlimb paresis, anorexia	Hypoproteinaemia, anaemia	–
27	6 yo FE DSH	Hindlimb paresis, hyperreflexia, proprioceptive deficits	Anaemia, neutrophilia, atypical lymphoid cells Radiographs – sternal lymphadenopathy, hepatomegaly FNA – lymphoma	–

Table 3. *Continued*

Cat	Signalment	Clinical signs	Abnormal ancillary tests	Post mortem results
32	12 yo FS DSH	Paraparesis	Myelogram − extramedullary spinal cord compression at T1−T2 Surgery − swollen spinal cord FNA − lymphoma	Lost to follow-up
37	16 yo MC DSH	Depression, circling, blindness, tetraparesis, behavioural changes	Anaemia, neutrophilia, lymphopenia, monocytosis CT scan − mass in frontal lobe	−
48	4 yo MC DSH	Facial nerve paralysis, head tilt, anisocoria, tetraparesis, nystagmus, proprioceptive deficits, exophthalmos and retinal inflammation	Elevated BUN, ALP, ALT Abdominal ultrasound FNA kidneys − lymphoma	−
Other neoplasia				
12	10 yo FS Chinchilla	Hindlimb paresis, hyperreflexia, proprioceptive deficits	Hyperglobulinaemia, neutrophilia, monocytosis Myelogram − extramedullary, intradural spinal mass Surgery − osteosarcoma	Improved
30	10 yo MC Burmilla	Paresis to left forelimb	Myelogram − 2 caudal cervical intradural, extramedullary masses	Undifferentiated nerve root neoplasia
38	11 yo FS DSH	Hindlimb paralysis	Myelogram − intradural, intramedullary mass	−
53	14 yo FS DSH	Dullness, ataxia, head tilt, circling, enophthalmos, reduced PLR's, small kidneys	Skull radiographs − bilateral increased radio density in tympanic bullae MRI − mass at cerebello-pontine angle, mass effect on brainstem, invasion of petrous temporal bone	−
56	8 yo MC DSH	Dull, recumbent, nystagmus, absent gag reflex, tetraparesis, nasal discharge	No abnormalities	Meningioma
57	9 yo MC Chinchilla	Hindlimb paresis	Myelogram − intramedullary mass	−
59	8 yo MC DSH	Right sided torticollus, rolling, hemiparesis, anisocoria, head tilt, reduced facial sensation Nasal discharge	Elevated AST, ALT, CK CT − soft tissue density in ethmoid turbinates invading cribiform plate and brain	−

(*continued on next page*)

Table 3. *Continued*

Cat	Signalment	Clinical signs	Abnormal ancillary tests	Post mortem results
		Heart murmur	Nasal biopsies − carcinoma	
60	9 yo FE Siamese	Hindlimb paresis, spinal pain, nystagmus	Myelogram − intramedullary mass	−
Trauma				
22	8 yo MC Abyssinian	Hindlimb paresis	Myelogram − intramedullary swelling L4−L5	Recovered
29	7 yo FS DSH	Hindlimb paresis, hyperreflexia, spinal pain, flaccid tail	Myelogram − loss of spinal cord opacity T12−L1	Recovered
34	8 yo MC DSH	Hindlimb paresis, flaccid tail, urinary and faecal incontinence	Myelogram − under opacification of spinal cord at L5−L6	Recovered
IVDD				
13	12 yo MC DSH	Hindlimb paresis, hyperreflexia, spinal pain	Myelogram − spinal cord compression T13−L1	Recovered
Spinal granuloma				
31	8 yo FS DSH	Hindlimb paresis, thoracolumbar pain	Myelogram − extradural compressive myelopathy Surgery − granuloma, unknown cause	−
62	8 yo MC DSH	Hindlimb paresis, thoracolumbar pain	Myelogram − extradural compression T13−L1 Surgery − granulomatous lesion, unknown cause	Recovered

MC = male castrate, ME = male entire, FS = female spayed, FE = female entire.
mo = months old, yo = year old.
DSH = domestic shorthair, DLH = domestic longhair, OSH = oriental shorthair, URTI = upper respiratory tract infection, PLR = pupillary light reflex, HCT = haematocrit, TPP = total protein, BUN = blood urea nitrogen, FNA = fine needle aspirate, LCAT = latex cryptococcal antigen titre, CT = computed tomography, MRI = magnetic resonance imaging, Other ME = other meningoencephalitis (non-suppurative meningoencephalitis and steroid responsive meningoencephalitis), IVDD = intervertebral disc disease, def = deficiency, − = post mortem not undertaken.

was recorded to have survived more than 3 months after presentation. No cat had a necropsy performed.

Other neoplasia

Of these eight cats, one cat was still alive at the time of the study (11 months after presentation), six were euthanased and one died. The ages ranged from 8 to 14 years with a median age of 9.5 years. The main clinical signs were gait abnormalities in seven, reduced proprioception in three and other cranial nerve deficits (facial nerve paresis, reduced pupillary light response, absent gag reflex) in three. A variety of systemic signs were observed (upper respiratory tract infection, heart murmur, pale mucous membranes). Seven cats had a chronic course of disease while one cat presented acutely. The clinical signs localised the disease to the spinal cord in four cats, brainstem in two cats, and central vestibular system in one cat. In one cat the lesions appeared multifocal (vestibular and spinal). In six cats the inflammation in the CSF was mixed and in two it was mononuclear. In one cat the CSF nucleated cell count was normal (2 cells/μl), in six cats it was mildly elevated (6–50 cells/μl) and in one cat it was moderately elevated (82 cells/μl). Six cats had mildly elevated CSF protein concentrations (0.31–10 g/l). In two cats the CSF protein was not measured because of insufficient sample. Five cats had abnormalities on myelogram (widening of spinal cord, intradural mass lesions). One cat had abnormalities on skull radiographs (bilateral radio density in tympanic bullae). One cat had abnormal CT scan (soft tissue density in ethmoid turbinates invading cribiform plate and rostral brain). One cat had an abnormal magnetic resonance image (mass in cerebello-pontine angle, invading petrous temporal bone). Other abnormalities seen were one each of leukocytosis, elevated globulins, elevated liver enzymes, elevated creatinine kinase and an abnormal biopsy (nasal carcinoma). Four cats survived less than 1 month, one survived 2 months and two cats survived 6–12 months. One cat was still alive at the time of the study (11 months later). This cat was a 10-year-old female, spayed Chinchilla with neurological signs of a thoracolumbar spinal lesion. Myelogram revealed an extramedullary-intradural lesion. Surgical removal was performed which revealed an osteosarcoma of the articular facets. The cat was well but mildly paretic on its hindlimbs 11 months after surgery. Two cats had a post mortem examination that revealed a cerebral meningioma in one cat and an undifferentiated nerve root tumour in the other.

Trauma

Three cats were diagnosed with CNS trauma based on history, myelogram and a marked haemorrhagic CSF. All had acute onset of disease (24–48 h). One cat was 7 years of age and two were 8 years of age. The main clinical signs were gait abnormalities in two cats, and one each of increased reflexes, spinal pain, reduced conscious proprioception, urinary and faecal incontinence. No systemic clinical signs were seen. In all three cats the neuroanatomical location was the spinal cord (thoracolumbar in two cats and lumbosacral in one cat). The CSF inflammation was mixed in two cats and suppurative in one cat. All cats had a mild elevation in CSF protein concentration. Myelography was performed in all three cats. This revealed intramedullary swelling in one, loss of spinal cord opacity in another and under opacification with compression of the spinal cord in the third cat. CSF haemorrhage was marked in all three cats with evidence of erythrophagia and xanthochromia, however the total nucleated cell counts were higher than could be accounted for with blood contamination alone. All three cats made a full recovery.

Intervertebral disc disease

One cat was diagnosed with intervertebral disc disease. It was a 12-year-old male castrate domestic shorthair with chronic signs of hindlimb paresis localised to the thoracolumbar spinal cord. The cat had a mild mixed inflammatory CSF (19.8 cells/μl) with a mildly elevated total protein (2.34 g/l). Myelogram revealed spinal cord compression. Hemilaminectomy was performed and the cat recovered.

Spinal cord granuloma

Two cats were diagnosed with a spinal cord granuloma of unknown cause. Both cats had a chronic history of hindlimb paresis and the lesions were localised to the spinal cord in both cats. One cat had a mild mixed inflammatory CSF (16 cells/μl). The other cat had a moderate suppurative inflammatory CSF (167 cells/μl).

The first cat did not have CSF protein measured while the second had a mild elevation in CSF protein (1.43 g/l). Myelography revealed extradural spinal cord compression in both cats. Surgery and biopsy in both cats revealed granulomatous spinal cord lesions but no underlying cause was found. The first cat was euthanased 4 months later because of a recurrence of clinical signs. A post mortem examination was not performed. The second cat recovered and was still alive 6 years later.

Undiagnosed

Eighteen cats (29%) remained without a diagnosis often despite extensive clinicopathological testing (Table 3). Of these, one cat made a complete recovery and was euthanased 3 years later for renal failure. One cat was lost to follow-up. The remaining 16 cats died or were euthanased. The age range was 4 months−15 years with a median age of 12 years. The predominant neurological signs were gait abnormalities (13 cats), seizures (nine cats), reduced proprioception (six cats), head tilt (four cats), postural reaction deficits (three cats) and other cranial nerve deficits (facial nerve, trigeminal nerve paralysis) (three cats). The main systemic signs were inappetence, weight loss and pyrexia in two cats each. Sixteen cats had a chronic course of disease and two cats had acute clinical signs. The neuroanatomical location was multifocal in eight cats, cerebral in four cats, brainstem in five cats, central vestibular in two cats and spinal cord in three cats. The type of inflammation was mixed in six cats, suppurative in eight, mononuclear in three and eosinophilic in one cat. Three cats had a normal CSF white cell count, 12 had mild elevations in the CSF nucleated cell counts (6−50 cells/μl) while three cats had moderate elevations (51−1000 cells/μl). The CSF protein concentration was normal in four cats and mildly elevated in 12 cats (0.31−10 g/l). In two cats the CSF protein concentration was not measured due to insufficient sample. Three cats had blood leukocyte abnormalities (neutropenia, lymphopenia and monocytosis). Three cats had elevated globulins, two had azotaemia, one cat had elevated T4 and one had elevated liver enzymes. Eleven cats survived less than 1 month, five cats 1−6 months, one cat was lost to follow-up and one cat recovered. Two cats underwent a post mortem examination, in which a cause for chronic partial seizures, behavioural changes and hemiparesis in one cat and seizures with cranial nerve deficits in the other cat was not found.

Discussion

The purpose of this study was to establish if an accurate diagnosis in cats with inflammatory CSF could be established by clinical signs, CSF analysis and ancillary diagnostic tests. A previous study (Rand et al 1994b) failed to demonstrate any significant difference in CSF parameters between cats diagnosed with primary inflammatory, degenerative, or neoplastic disease of the CNS. This may reflect the presence of secondary inflammation and degeneration associated with many CNS disorders.

Categorising CSF as 'inflammatory' depends on a number of factors such as the method of collection (particularly peripheral blood contamination), laboratory reference ranges, loss of cells in CSF due to delays between sample collection and analysis and experience of the technician interpreting the sample. To control these variables, all the reference ranges chosen were those used by the laboratories involved in processing the sample. All samples were evaluated by an experienced veterinary pathologist within 1 h of collection.

Peripheral blood contamination is a common confounding problem in CSF collection despite good technique. Normal CSF should not contain any erythrocytes, however up to 30 red blood cells (RBCs/μl) is considered 'normal' contamination as it does not have a significant effect on the CSF white cell count (Rand et al 1990). Correcting for peripheral blood contamination using the ratio of red cells to white cells in the peripheral blood may not be an accurate indicator of the effects of blood contamination on CSF fluid (Wilson and Stevens 1977). Rand et al (1990) suggested a formula for the correction of CSF white blood cell counts based on the CSF red blood cell number. This study also showed that the formula was likely to overestimate the degree that blood contamination elevated CSF white cell counts in approximately 50% of cases. The maximum expected increase in white cells is calculated based on the assumption that there is one additional white cell/μl of CSF per 100 red cells/μl of CSF. In this study, cases were excluded if blood contamination was significant enough to explain the elevated white cell count. Thus, all cats with true CNS inflammation would

be included but is possible that some cats with true inflammation were excluded.

Location of CSF collection may alter protein concentration and cell counts. Lumbar CSF white cell counts can be less and protein concentrations higher than that of cisternally collected CSF in dogs. The reason for the reduced white cell counts in the lumbar CSF is not known. Possible causes include cell lysis, less cells entering the lumbar CSF or migration of white cells from lumbar CSF to blood (Bailey and Higgins 1985). Increased protein concentration in the lumbar CSF may be caused by sluggish spinal CSF circulation resulting in protein accumulation. Similar differences have been observed in feline CSF protein levels (Hochwold et al 1969). Changes in white blood cell counts between lumbar and cisternal punctures have not been compared in cats. CSF was collected from both sites as this study encompassed cases from three different referral hospitals and the method of collection was chosen according to the preference of the veterinarian involved. Often, the area of collection was not specified in the data. Differences may have occurred but this could not be accounted for in the present study.

FIP was the most common cause of inflammatory CSF and accounted for 18% of the cases. This is similar to a previous study (Rand et al 1994a, 1994b) where 18% of cats with primary inflammatory and primary non-inflammatory CNS disease were diagnosed with FIP. FIP is a difficult disease to diagnosis antemortem. The 'gold standard' for diagnosis is histopathology which reveals a perivascular proliferation of macrophages, lymphocytes plasma cells and neutrophils with a central area of necrosis (Hartmann et al 2003). It has been shown that evaluation of albumin:globulin ratio (<0.5) and anti-coronavirus antibody in body cavity effusions (any titre) had a good positive predictive value for the diagnosis of FIP. In addition, the highest serum anti-coronavirus antibody titre (1:1600) also had a good positive predictive value however, any serum antibody titre less than this was not valuable (Hartmann et al 2003). While nine cases of FIP in this study were diagnosed via histopathology, two cases were diagnosed via the clinical and serological criteria.

The next most common disease category in our study was neoplasia, with both confirmed lymphoma and undetermined forms accounting for 22.6% of the cases. In contrast, Rand et al (1994a, 1994b) found that 16% of cases (combined primary inflammatory and non-inflammatory CNS disease) were diagnosed with non-suppurative meningoencephalitis of suspected viral origin. Quesnel et al (1997) found 47% of cats presented for seizure disorders to have non-suppurative meningoencephalitis of possible viral cause. Our study found that only three cats had findings that were supportive of non-FIP viral infection. The diagnosis is suggested by mononuclear meningitis without visceral lesions of FIP and perivascular cuffing as the only histologic lesion (Lundgren 1992). It has been postulated that certain viruses, eg, parvovirus, feline herpesvirus, calicivirus, FIV and FeLV could all cause these histopathological lesions. The arboviruses have received special interest in Canada. There are six strains of arbovirus known to cause encephalitis in humans in Canada (Artsob and Spencer 1979). Some of these are also known to be endemic in the wild animal population and experimental infections in cats (Powassan virus) have caused non-suppurative meningoencephalitis (Keane et al 1987, Rand et al 1994a). There are more than 70 strains of arbovirus in Australia, only two of which are known to cause encephalitis in humans (Murray River and Kunjiin virus) (Boughton 1994). The cause of feline non-suppurative meningoencephalitis is not known but the apparent variation in the diagnosis of this condition may reflect differences in causative agent(s) seen in the two geographical locations.

The median age for cats in this study was 8 years and was reflected in all disease categories except FIP, which was markedly lower (median age 1 year). Overall, the most common systemic clinical signs were weight loss (18%) and ocular abnormalities (16%), apart from FIP where pyrexia was the most common clinical finding (75%). In all groups, the majority of cats had gait abnormalities as the major neurological sign (84%).

Neuroanatomical location did not help in differentiating between disease categories as many cats were diagnosed with multifocal/diffuse disease (37%). Exceptions to this were cats with lymphoma, other neoplasia, spinal cord granuloma and trauma in which the spinal cord was the most frequent neuroanatomical location.

CSF protein concentration may provide some help in categorising disease groups. The only cats with moderate or marked CSF protein elevations (>10 g/l) were cats with FIP (23%), one cat with cryptococcus (26.0 g/l) and one case of other meningoencephalitis (steroid responsive meningitis) (11.6 g/l). Cats with FIP had, by far,

the highest CSF protein levels (53 and 62 g/l). Hence, markedly elevated protein concentrations should increase the index of suspicion for FIP. The lack of protein measurements in 18% of the cases as a consequence of insufficient sample, may have skewed the interpretation.

The degree of CNS inflammation was inconsistent within groups. Severe inflammatory CSF was observed more commonly in FIP cases (38%). Other categories had variable degrees of inflammation.

The type of inflammatory pattern was helpful only in cats with lymphoma as all had a mononuclear inflammation. Mixed inflammation of the CSF was the most common type of inflammatory pattern observed (48.4%) and when observed did little in aiding the diagnosis. Interestingly, both cats with thiamine deficiency had a mild suppurative inflammation. To the author's knowledge, there are no reports in veterinary or human literature on the CSF findings with thiamine deficiency. As CSF collection is a diagnostic test, it is not required if the diagnosis is known. A diagnosis of thiamine deficiency can often be suspected from dietary history and clinical signs. Thiamine deficiency results in cerebrocortical necrosis (Read and Harrington 1986, Steel 1997) that could result in a mild suppurative CSF infiltrate.

Seventy-seven percent of cats survived less than 1 year. Three cats died or were euthanased immediately after CSF collection. This represents almost 5% of cases and serves to illustrate that cats with central nervous system disease can be poor anaesthetic risks and that CSF collection can be associated with significant morbidity and mortality. Cats diagnosed with cryptococcus had a better prognosis (50% still alive more than 5 years after presentation), as did those diagnosed with trauma and steroid responsive meningitis (100% still alive more than 12 months after presentation). Survival times were variable in cats with toxoplasmosis, non-suppurative meningitis and thiamine deficiency (Table 2).

This study revealed that CSF analysis alone was helpful only in CNS FIP, cryptococcus, lymphoma and trauma. In other conditions a presumptive diagnosis could be made using a combination of antemortem findings (age, CSF cellular and protein levels, serum and effusion antibody titres, biopsy and outcome). Neuroanatomical location provided some indication of the diagnosis in cats with lymphoma, other neoplasia, spinal cord granuloma or trauma (spinal cord). The degree of CSF inflammation, CSF protein concentration and systemic signs provided little contributory information to the final diagnosis other than in cats with FIP. Despite often extensive diagnostic evaluation, 37% of cats was left without a premortem aetiological diagnosis. Future prospective studies with thorough post mortem examination are warranted as post mortem evaluation was only performed in this retrospective study in 18 of the cases. The increased use of advanced imaging (CT/MRI), CSF antibody, molecular biological methods, and CNS biopsy may aid in the diagnosis of CNS disease in the living patient.

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