Journal of Feline Medicine and Surgery (2008) **10**, 9–15
doi:10.1016/j.jfms.2007.06.004

ELSEVIER

jfms

Retrospective analysis of seizures associated with feline infectious peritonitis in cats

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Seizures have been reported frequently in feline infectious peritonitis (FIP) but have not been studied in detail in association with this disease. The purpose of this study was to perform a retrospective analysis of neurological signs in a population of 55 cats with a histopathologically confirmed neurological form of FIP. Seizure patterns were determined and it was attempted to relate occurrence of seizures with age, breed, sex and neuropathological features. Fourteen cats had seizure(s), while 41 cats had no history of seizure(s). Generalised tonic–clonic seizures were seen in nine cats; and complex focal seizures were observed in four patients. The exact type of seizure could not be determined in one cat. Status epilepticus was observed in one patient but seizure clusters were not encountered. Occurrence of seizures was not related to age, sex, breed or intensity of the inflammation in the central nervous system. However, seizures were significantly more frequent in animals with marked extension of the inflammatory lesions to the forebrain ($P = 0.038$). Thus, the occurrence of seizures in FIP indicates extensive brain damage and can, therefore, be considered to be an unfavourable prognostic sign.

Date accepted: 20 June 2007

Feline infectious peritonitis (FIP), a multisystemic disease caused by a macrophage tropic mutant of feline coronavirus (FCoV), is the leading infectious cause of cat death (Vennema et al 1998, Hartmann 2005). FIP associated meningoencephalitis is considered to be the most common inflammatory disorder in the feline neuraxis (Bradshaw et al 2004). The development of FIP is thought to be due to the host immune response (Addie et al 2004). Up to one-third of cats with 'dry' FIP, but also some cats with the effusive form, have neurological abnormalities (Foley et al 1998). In the brains of cats with FIP histopathological findings were meningitis, ependymitis, periventriculitis and chorioiditis of varying severity (Baroni and Heinhold 1995). Perivascular inflammatory infiltrates of lymphocytes, plasma cells, neutrophils and macrophages are usually associated with the cerebrospinal fluid (CSF) pathways, but may extend into the neurophil (Rand et al 1994). Arteritis, phlebitis, secondary oedema fluid exudation and haemorrhage have also been observed. Hydrocephalus has been seen in association with meningitis, choroiditis, accumulation of cellular debris in the ventricles and obstruction of the CSF flow (Summers et al 1995, Muñana 1996).

The route of entry of feline infectious peritonitis virus (FIPV) into the CSF is probably haematogenous through macrophages (Hartmann 2005). It has been shown that inflammatory cells are recruited to the central nervous system (CNS) and contribute to the clinical manifestation of disease through secretion of cytokines (Foley et al 2003, Kipar et al 2005).

Commonly described historical findings in cats with neurological FIP include dementia, inappropriate elimination, behavioural changes (Feldmann 1974, Hoskins 1993) and – most frequently – central vestibular signs (Holzworth 1987). Most clinical studies, review articles and text books on FIP report that seizures occur in this disease, and may even be the only sign in

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1098-612X/08/010009+07 $32.00/0

neurological FIP, although seizure prevalence and characteristics in FIP have not been described. The purpose of this study was to determine the prevalence of seizures in our population of cats with neurological FIP, to describe the seizure type in this disease and to investigate whether the occurrence of seizures in cats with neurological FIP could be related to age, breed, sex and neuropathological features.

Materials and methods

Selection of cases

A retrospective evaluation was made of the clinical records of cats from the neurology service with clinical neurological signs and a diagnosis of CNS infection with FIPV as confirmed in histopathological examination. The histopathological diagnosis was based on the presence of typical lesions, which are highly characteristic for FIP (Summers et al 1995). Unlike other viral infections of the CNS, FIP lesions are primarily related to deposition of FIPV immune complexes in choroid plexus and blood vessels, precipitating a violent pyogranulomatous inflammation in the CSF compartment. Both distribution and nature of the lesions cannot be confused with other viral infections in which parenchymal mononuclear inflammation, neuronal degenerations and gliosis are the main features. All included cats were examined at the Institute of Animal Neurology, University of Bern, Switzerland, during the years 1985–2005. Only records with detailed history, clinical and neurological examination and histopathological diagnosis were selected. Cats were divided into two groups. Group A was composed of cats with a history of seizure(s) and group B included only cats in which no seizure(s) had been reported.

Evaluation of clinical data

Signalment, history, clinical findings and seizure pattern such as frequency, duration and type of seizures were evaluated. Seizures were classified into focal, complex focal and generalised, based on clinical manifestation. Generalised seizures were characterised by bilateral and symmetrical motor activity with or without accompanying autonomous signs, such as salivation, urination, defecation and with or without loss of consciousness. Unilateral or asymmetrical motor signs without impairment of consciousness characterised simple focal seizures. Complex focal seizures were diagnosed when unilateral or asymmetrical motor signs occurred together with impaired consciousness, or when a purely transient and involuntary change in behaviour (characterised by jumping, biting and attacking real or imaginary objects without provocation or running blindly into objects) was observed. Secondary generalisation of simple or complex focal seizures was noted. Status epilepticus was defined as continuous epileptic activity or the presence of two or more separate seizure episodes without a return to consciousness between them for more than 30 min. Clusters of seizures were diagnosed when two or more isolated seizures were observed during a 24-h period with the patient regaining consciousness between them.

The results of the neurological examination were reviewed. Particular note was made of neuroanatomical localisation. A single neuroanatomical lesion was assumed when all the neurological deficits could be explained by one lesion in the CNS (eg, forebrain, brainstem, cerebellum, and spinal cord). A multifocal localisation was recorded, when two or more of the intracranial compartments (forebrain, brainstem and cerebellum) were affected, or when an intracranial lesion was combined with signs suggestive of spinal cord involvement such as decreased segmental spinal reflexes.

Semi-quantitative neuropathological assessment

The histological slides of 28 cats including all cats of group A and 14 randomly selected cats from group B were subjected to a blind re-evaluation. The following features were assessed: intensity of inflammation in the posterior brain compartment, enlargement of the ventricles, extent of inflammation in the CSF compartment in the forebrain, and extent of parenchymal involvement in the forebrain (encephalitis, brain oedema, and encephalomalacia). All parameters were graded with scores of 0 for none, 1 for mild to medium and 2 for severe variances. Breed, sex and the frequency of the respective scores were compared between groups A and B using cross-tabulation. Due to low frequencies, score categories 1 (mild/medium) and 2 (severe) had to be combined and compared to category 0 (no lesions). For statistical analysis, the Fisher's exact test (FET) was used. A Kruskal–Wallis analysis of variance (ANOVA) on ranks was used to compare the age between groups.

Results

History and clinical findings

The medical records of 78 cats with the neurological form of FIP were found. However, 13 of these cats were excluded because there was no neuropathological confirmation of the condition, and a further 10 cats were excluded because the clinical information was incomplete. Fifty-five cats with neurological FIP confirmed in histopathological studies were further evaluated. Out of these 55 cats, 14 (25%) had a history of seizure(s) and comprised group A. Forty-one cats (75%) had no history of seizure(s) and were included in group B. The median time between the onset of clinical signs and the first presentation to a veterinarian was 26 days (range 3–90 days).

Group A

The median age of the cats was 12 months (range 3–96 months). Both sexes were almost equally represented and included seven intact and one neutered female, as well as five intact and one castrated male. Domestic shorthaired cats were the most frequently encountered (9/14; 64%), followed by Birman cats (4/14; 29%) and one Devon Rex (1/14; 7%). In three cats, a seizure was the first sign noted by the owner, whereas in 11 cats other neurological deficits were noted, including gait abnormalities, such as ataxia and paresis (six cats) and depression (five cats), before the occurrence of the first seizure.

Generalised tonic–clonic seizures were seen in nine cats (64%), complex focal seizures were observed in four patients (28%). The distinction between the generalised and complex focal seizures was impossible to determine in one cat. Generalised seizures were mainly characterised by tonic–clonic seizures in lateral recumbency with complete loss of consciousness. Complex focal seizures were mainly noted as a combination of motor activity (usually twitching of the facial muscles) and behaviour changes (usually aggression or inappropriate response to imaginary objects). Both generalised and complex focal seizures were of short duration (30 s to several minutes; usually under 3 min). Status epilepticus was observed in one patient and was the cause of death in this cat. Clusters of seizures were not observed in our cat population.

Neurological examination revealed neurological deficits, which pointed to a single lesion localisation in the CNS in 12/14 epileptic cats (forebrain 7/14 and brainstem 5/14). The most frequent signs of forebrain involvement were abnormal mental status, behaviour changes (usually disorientation or severe agitation) and abnormal aggression as well as postural reactions. The central vestibular signs, such as head tilt and/or nystagmus, were noticed in 4/5 cats with brainstem lesions. In two cats, a multifocal process in the CNS was suspected as a combination of forebrain and spinal cord or brainstem and spinal cord signs.

The most frequent clinical finding in cats in group A was a poor body condition, characterised by weight loss and/or muscle atrophy (8/14) followed by abnormal rectal body temperature (6/14; with hypothermia 5/14 and hyperthermia 1/14), tachycardia 3/14, diarrhoea 1/14 and pleural effusion 1/14. Body condition scores could not be determined, because of the imprecise description of the patients in this respect. Clinical suspicion of FIP infection in group A (14) was based on finding neurological deficits together with extra-neural signs and laboratory results, which included high coronavirus titre (4/4 assessed), hyperglobulinaemia (3/3 assessed), pleocytosis on CSF examination (1/1 assessed) and magnetic resonance imaging (1/1 assessed) by which normal findings were not reported. Palliative therapy was attempted in 7/14 cats using a combination of corticosteroids or non-steroidal anti-inflammatory drugs and antibiotics. Phenobarbital was administered to one of the patients at a dose of 2 mg/kg body weight twice per day. No significant clinical improvement was noticed in any of the treated cats. The owners of 13/14 cats elected euthanasia after the therapy was considered to be unsuccessful or immediately after the initial clinical evaluation because of severe clinical signs. One cat died during hospitalisation as a result of status epilepticus and respiratory and cardiac arrest.

Group B

The mean median age of cats in this group was 12 months (range 4–264 months). In two cases the age was unknown. Both sexes were represented with 12 intact and two neutered females as well as 16 intact and 10 castrated males. The sex was unknown in one case. As in group A, domestic shorthair cats were the most frequently represented breed (26/41; 63%) followed by Birman cats (8/41; 19%), Persian (3/41; 7%), Siamese (2/41; 5%), one Ragdoll and one Devon Rex cat (1/41; 3%). The history of onset of the neurological problems varied from acute (under 24 h) to chronic (up to 2 months) and the disease was

reported to be progressive in all patients. The most common neurological findings observed in this group were ataxia (29/41), depression (23/41), cranial nerve deficits (19/41), tetraparesis (17/41), central vestibular signs (15/41), abnormal postural reactions (15/41) and paraparesis (12/41). The neurological localisation was the brainstem in 22, multifocal in 12, the spinal cord in six cases and forebrain in one case. The most frequent general clinical finding in group B was apathy (23/41 cats). The laboratory abnormalities included pleocytosis on CSF examination (13/41), hyperglobulinaemia (10/41), anaemia (6/41) and leukocytosis (3/41). Treatment was attempted in nine cases and included glucocorticoids, non-steroidal anti-inflammatory and antibiotic drug administration, but no clinical improvement was noticed. Except for one cat which died, all other cats were euthanased at the owners' request because of deterioration of the clinical signs.

Pathological findings

A full postmortem pathological examination was performed in 22 cats. Significant changes consistent with a diagnosis of FIP were found in 13 cats and included granulomas in kidneys and liver, chorioretinitis and interstitial purulent pneumonia.

A gross pathology examination of the CNS was performed in all 55 cats of this study. The neuropathological findings were consistent with those reported in the reference literature (Summers et al 1995) and included severe pyogranulomatous meningitis, ependymitis and choroiditis in the area of the posterior brainstem and fourth ventricle in all cats. The inflammation invaded the periventricular and submeningeal tissues to various extents. Significant degeneration of subpial parenchymal structures, notably white matter, was observed in several animals. In a few cats, a considerable amount of exudate and inflammatory cells accumulated in the ventricles. In addition to massive perivascular infiltration with lymphocytes, plasma cells, macrophages and neutrophils, necrotising arteritis was noted in several animals. Thrombosis and haemorrhage were seen in a few cats. The inflammatory process extended rostrally along the ventricular and meningeal spaces, involving third and lateral ventricles and cerebral meningeal spaces to variable extents. Mild to marked ventricular enlargement as well as variable degrees of parenchymal involvement of the cerebrum were apparent in many animals. Semi-quantitative assessment of ventricular enlargement, extension of the inflammatory process in the forebrain and the degree of parenchymal involvement of the cerebral cortex in epileptic and non-epileptic cats are summarised in Table 1.

Statistical findings

Differences in breed and sex between groups A and B were statistically analysed using the FET. Due to the small number of animals of different breeds, the population was divided into domestic shorthairs and other breeds. There was no significant difference in sex ($P = 0.21$) or breed ($P = 1.00$) between the two groups. Neither was there a significant difference in age, the median age in both groups being 12 months (Kruskal–Wallis ANOVA on ranks, $P = 0.64$).

The semi-quantitative histopathological findings, comparing cats with seizures (14) to a randomly selected group of cats without seizures (14) by means of cross-tabulation and FET, revealed a significant ($P = 0.035$) relationship between forebrain damage and seizures (Table 1). Ventricular enlargement and extension of the inflammation in the forebrain also showed positive association with the occurrence of seizures, however, these findings were not statistically significant ($P > 0.05$).

Discussion

The purpose of the present study was to evaluate prevalence and characteristics of seizures in cats with the neurological form of FIP. In addition, we

Table 1. Cross-tabulation report and FET of the neuropathological examination of cats with seizures ($n = 14$) in relation to a comparable group of cats without seizures ($n = 14$)

Parameter	Total (n)	Seizures (%)	FET, P-value
Ventricle enlargement			0.153
None	5	20.0	
Mild and severe	23	60.9	
Forebrain involvement			0.255
None	13	38.5	
Mild and severe	15	66.7	
Forebrain damage			0.038
None	20	40	
Mild and severe	8	87	

Cats with seizures show a significantly ($P = 0.038$) higher degree and extent of forebrain damage than cats without seizures.

attempted to relate occurrence of seizures with breed, age, sex and neuropathological findings. Cases were selected based on typical histological findings in the CNS. Immunohistochemical demonstration of FIP antigen could have been used to confirm the diagnosis. Apart from the fact that this technique has not been validated in any great number of feline inflammatory CNS lesions, the neuropathology of FIP is highly characteristic (Summers et al 1995). Because of their immunopathological nature FIP lesions differ totally from other viral infections. Suppurative inflammation could also occur in bacterial and mycotic infections, which would rarely completely mimic the FIP lesions in respect to distribution and morphology and organisms could be readily detected. Therefore, we believe that the inclusion of cases in the present study strictly based on neuropathological criteria was a reasonable approach.

Twenty-five percent of the cats in this study demonstrated seizures associated with neurological FIP which is similar to the findings in another retrospective study on cats with intracranial neoplasia, where incidence of seizures was reported to be 22.5% (Troxel et al 2003). The range of age in our population was similar to previous reports, showing that FIP can occur at any age (Rand et al 1994, Stacy 2000) and did not differ between the two groups. The claimed predisposition to FIP for some pure breeds of cat (Pesteanu-Somogyi et al 2006) was not apparent in our study. The breed distribution of the cats in our study roughly reflected the breed distribution of our overall feline hospital population. The distribution between the sexes was almost equal in our and previous studies and is consistent with the sex distribution in the overall feline population in our hospital. Therefore, it seems that age, sex and breed play little role in seizure genesis in the neurological form of FIP.

Focal and focal complex seizures are usually reported as a common type of seizures in cats with secondary epilepsy (Parent and Quesnel 1996, Kline 1998) usually resulting from structural lesions in the brain (Quesnel et al 1997). In contrast, in some studies on secondary epilepsy in cats, generalised seizures dominated (Quesnel et al 1997, Barnes et al 2004). Generalised seizures were registered in most of our cases, but the majority of ictal events were observed and described by owners in our study. Therefore, a focal seizure initiating the ictal event could have gone unnoticed. It is usually assumed that clusters of seizures or status epilepticus reflect progression of the underlying disease. Compensatory mechanisms in the brain may become exhausted due to increasing lesion volume and development of significant structural damage. Despite severe and progressive neuropathological lesions, status epilepticus was rare in our case material (1/14) and clusters of seizures were not observed. While we did not find any literature on the incidence of cluster seizures or status epilepticus associated with particular types of structural brain damage in cats our findings suggest that species-specific differences may exist in this respect. For example, intracranial neoplasia induces status epilepticus in up to 15% of human or canine patients (Towne et al 1994, DeLorenzo et al 1995, Bateman and Parent 1999, Knake et al 2001, Platt and Haag 2002, Gandini et al 2003).

The pathophysiology of viral meningoencephalitis related seizures is only partly understood, and, depending on the aetiological agent, different mechanisms are most probably involved. The pathophysiological basis of seizures associated with meningoencephalitis is related to a range of immunological, biochemical, anatomical and physiological changes that shift the balance between intra-cortical inhibitory and excitatory mechanism towards excitation (Engelborghs et al 2000, Bette et al 2004, Chen et al 2004). Neuronal and/or receptor loss or damage, changes in neuropeptides, increase of pro-inflammatory cytokines and other mechanisms have been described to be involved in seizure genesis in encephalitic brains in experimental animals (Novoa et al 1997, He et al 1998, Jalanko and Vesa 2005).

The mechanism of seizure induction in FIP has not been studied so far. Most of the examined cats in our study with neurological FIP, with or without seizures, also have lesions in other organ systems. It is impossible to specify the extent to which extra-neural lesions, notably liver disease, could have contributed to seizure genesis in our cats. Neither liver failure nor a clinical presentation suggestive of hepatic encephalopathy was seen in any of our epileptic cats. Rather, we believe that seizures in our animals were due to the observed severe intracranial lesions. In one large study on 220 dogs with inflammatory disease of the CNS, only 13% of the dogs had been suffering from seizures (Tipold 1995). It appears from our findings that, in cats too, the presence of an inflammatory process in the CNS per se is not sufficient to elicit seizures. All cats with or without seizures had severe inflammation in the meninges and ventricles, presumably with secretion of vast amount of inflammatory mediators such as

cytokines and chemokines diffusing throughout the CSF compartment. Rather, our findings suggest that secondary structural changes in the cerebrum were responsible for the seizures. The cerebral cortex is the primary element in the generation of an epileptic attack (Fischer et al 2005). Also in patients with intracranial tumours, supra-tentorial lesions are associated with higher prevalence of seizures in dogs, cats and humans (Zaki and Hurvitz 1976, Bagley et al 1999, Troxel et al 2003, Hildebrand et al 2005). A clinical forebrain localisation was much more frequent among the cats who suffered from seizures (7/14) as compared with cats without seizures (1/41) in our material. Severe obstructive hydrocephalus and marked extension of the inflammatory process in the forebrain appeared to enhance seizure incidence in our material, and the presence of parenchymal involvement of the cerebrum was significantly related with occurrence of seizures.

According to our study, the occurrence of seizures in FIP indicates significant extension of the inflammatory process and structural damage to the cerebrum and could be interpreted as an unfavourable prognostic sign.

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