

# Meningoencephalomyelitis in domestic cats: 3 cases of *Pasteurella multocida* infection and literature review

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**Abstract.** Neurologic diseases are common in domestic cats, and infectious agents are suspected to be the primary cause in 30–45% of cases. Among infectious etiologies, those of bacterial origin are only sporadically characterized in the literature, with few of these reports correlating gross and histologic findings with confirmatory bacteriologic identification. Here, we describe bacterial meningitis and meningoencephalomyelitis associated with *Pasteurella multocida* in 3 domestic cats. Purulent exudate expanding the cerebral meninges was grossly evident in 2 of the cases. In all 3 cases, histologic changes included multifocal suppurative-to-necrosuppurative meningitis and/or meningoencephalomyelitis of variable severity. Intralésional colonies of gram-negative, short rod-shaped to coccobacillary bacteria were evident histologically in only 1 case. *P. multocida* was confirmed by routine bacteriologic culture in all cases. Based on our cases, we hypothesize that the upper respiratory system serves as the main portal of entry for *P. multocida*, leading to invasion of the central nervous system and possible systemic hematogenous dissemination. A case series of meningoencephalomyelitis associated with *P. multocida* infection in cats has not been reported previously, to our knowledge. We also review briefly other causes of meningoencephalomyelitis in cats.

**Keywords:** bacterial meningoencephalomyelitis; encephalitis; feline; myelitis; neurologic signs; *Pasteurella multocida*.

Neurologic diseases are common in cats, constituting ~10% of the referral caseload in feline patients.<sup>9</sup> Infectious agents are suspected to be the primary cause in 30–45% of these cases.<sup>1,9</sup> Among infectious etiologies, bacterial infections in the central nervous system (CNS) only rarely manifest as the primary neurologic lesion; more often they develop as locoregional extension of bacterial infections affecting the middle ear and/or nasal cavity.<sup>9</sup> Bacterial causes of CNS infection in cats have an estimated prevalence of 1% among all neurologic diseases.<sup>1</sup> We describe here the gross and histopathologic features of meningitis and meningoencephalomyelitis in 3 domestic cats associated with *Pasteurella multocida* infection. Also, we reviewed reports of the most common differential diagnoses for meningoencephalomyelitis in cats.

A review of all feline pathology case submissions involving neurologic signs was conducted in the archives of the Louisiana Animal Disease Diagnostic Laboratory (LADDL), School of Veterinary Medicine, Louisiana State University (Baton Rouge, LA, USA), from January 2012 to July 2020 using the following search query in USALIMS: meningoencephalomyelitis, meningoencephalitis, encephalitis, myelitis, and meningitis. We retrieved 64 cases from the archives, and 55 of 64 (88%) had a defined infectious agent, including: feline infectious peritonitis virus (FIPV; 28 of 55, 51%), septicemia associated with *Escherichia coli* or *Staphylococcus* sp. (6 of 55, 11%), suspected bacterial (without bacteria

identification; 3 of 55, 5%), *Pasteurella multocida* (3 of 55, 5%), *Streptococcus canis* (2 of 55, 4%), and other miscellaneous agents (13 of 55, 24%). Relevant information (signalment, clinical presentation, and postmortem findings) for the 3 cases with *P. multocida* were obtained from the reports (Table 1). A postmortem examination was performed in these cases within 24–72 h after death. Tissues were collected, fixed in 10% neutral-buffered formalin, sectioned at 4 μm, and stained using hematoxylin and eosin. Selected sections were subjected to a Gram stain following standard procedures.

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**Table 1.** Signalment, clinical features, and gross lesions in 3 cats with meningitis and meningoencephalomyelitis associated with *Pasteurella multocida* infection.

Case	Age	Sex	Breed	Clinical features	Gross lesions
1	5 y	SF	American Shorthair	Recurrent ear infection, progressive neurologic signs (3-d course; lateral recumbency, stiff limbs, vocalization, anisocoria, nystagmus). Treated with clindamycin (10 mg/kg IV) and mannitol (0.5 g/kg IV). The cat had cardiorespiratory arrest.	CNS: opaque, white-to-green-tinged purulent exudate coating the meningeal surface of the caudodorsal aspect of the left cerebral hemisphere. Kidneys: unilateral focal chronic infarct.
2	2–3 mo	SF	Mixed breed	Upper respiratory signs, ocular discharge, neurologic signs (nystagmus, paraparesis, head tilt that progressed to lateral recumbency [unknown duration]). Treated with broad-spectrum antibiotics for the respiratory signs. The cat was found dead by the owner ~1 wk after having been introduced to its new home.	Skin: periocular yellow-to-brown crusts. Liver: white pinpoint foci.
3	2 mo	M	Mixed breed	Upper respiratory signs, sneezing, ocular and nasal discharge, and lethargy (unknown duration). No specific neurologic signs observed by the owner. Treated with broad-spectrum antibiotics for the respiratory signs. The cat was found dead by the owner a day after it had still been seen playing with the other kittens.	CNS: purulent exudate expanding the subdural space on the dorsal and ventrolateral aspect of the right cerebral hemisphere and interhemispheric fissure, and circumferentially around spinal cord segment C1. Nasal cavity and frontal sinuses were filled with mucopurulent exudate. Skin: periocular and nasal dark brown crusts. Thymus: inconspicuous. Intestines: mild nematodiasis ( <i>Toxocara cati</i> ).

CNS = central nervous system; IV = intravenous; M = intact male; SF = spayed female.

Refrigerated fresh brain samples from all cases were submitted for aerobic bacteriologic culture.

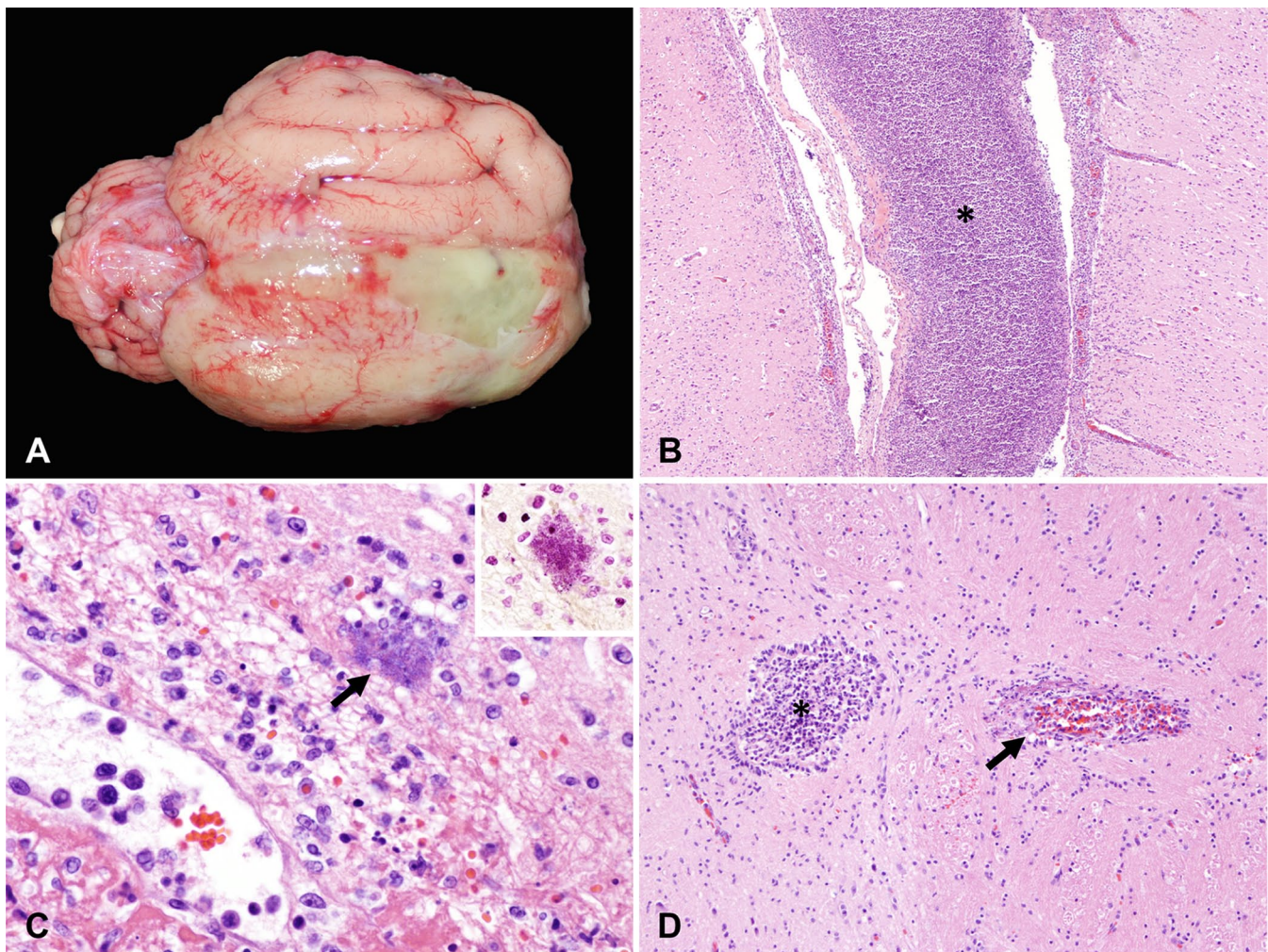
Gross findings in the CNS were similar in cases 1 and 3 but more severe in the latter, with abundant purulent exudate either partially filling the calvaria (case 1) or extensively expanding the meninges of the brain and cervical spinal cord (case 3; Fig. 1A). Additionally, the nasal cavity and the frontal sinuses of case 3 were filled with similar exudate, the eyelids and nose were coated with crusts, and the thymus was inapparent. No gross lesions were evident within the CNS in case 2 and, despite the previous history of recurrent otitis in case 1, no gross abnormalities associated with otitis were evident. No other significant gross lesions were noted in any of the animals.

Tissues from the CNS examined microscopically included cerebrum, cerebellum, and brainstem in all cases, and spinal cord (cervical, thoracic, and lumbar) in cases 2 and 3 (Table 2). Microscopically, cases 2 and 3 had similar histologic alterations (lesions were most severe in case 3); changes in case 1 were subtle and observed primarily in the leptomeninges. In cases 2 and 3, the subdural space and leptomeninges overlying the brain and spinal cord were multifocally to diffusely expanded by purulent exudate, comprised of necrotic cell debris, degenerate neutrophils, and fibrin (Fig. 1B). Rare-to-occasional intralesional colonies of short rod-shaped to coccobacillary bacteria were evident histologically in case 3

(Fig. 1C). The inflammatory exudate extended into and blended with the underlying neuroparenchyma. Vasculitis with fibrinoid necrosis characterized intralesional blood vessels, mainly small venules in the leptomeninges. Multifocal, occasionally extensive, areas of the underlying cortical lamina within the cerebrum and periventricular areas were characterized by rarefaction; numerous infiltrating viable and degenerate neutrophils; shrunken, angular, and hyperchromatic neurons; and blood vessels delimited by gitter cells and neutrophils. Also, in cases 2 and 3, the inflammatory exudate partially filled the lumen of the lateral ventricle, with evidence of suppurative choroid pleinitis, and extended to both the molecular layer of the cerebellum and the medulla oblongata. Although in case 3 the gross changes involved mostly the right cerebral hemisphere, the histologic degree of inflammation was bilaterally similar, with unilaterally more pronounced interstitial edema within the right neuroparenchyma.

Throughout all segments of the spinal cord (cervical, thoracic, and lumbar from cases 2 and 3), the central canal was filled by abundant degenerate neutrophils (Fig. 1D) that extended into the circumjacent gray matter, which had multiple blood vessels cuffed by a few neutrophils and occasionally effaced by necrotizing vasculitis. These histologic changes were most severe in case 2. All white matter tracts had multifocal mild neutrophilic infiltrates as well as digestion chambers and scattered spheroids, with no defined pattern.





**Figure 1.** Meningoencephalomyelitis caused by *Pasteurella multocida* in cats (case 3). **A.** Thick, yellow exudate expands the subdural space on the dorsal and ventrolateral aspect of the right cerebral hemisphere and interhemispheric fissure. **B.** Microscopically, the meninges are severely expanded by abundant necrotic cell debris, degenerate neutrophils, and fibrin (asterisk), with extension of the inflammatory process into the underlying neuroparenchyma. H&E. Original objective 10 $\times$ . **C.** Rare-to-occasional intralesional colonies of gram-negative (inset; Gram stain) short rod-shaped to coccobacillary bacteria (arrow) are visualized. H&E. Original objective 40 $\times$ . **D.** The central canal of the spinal cord was dilated and filled with abundant degenerate neutrophils and necrotic debris (asterisk); necrotizing vasculitis was also present (arrow). H&E. Original objective 20 $\times$ .

**Table 2.** Brain and spinal cord lesions in 3 cats with meningitis and meningoencephalomyelitis associated with *Pasteurella multocida* infection.

Case	Olfactory bulbitis	Leptomeningitis	Encephalitis	Periventricular encephalitis	Choroid plexitis	Ventriculitis	Myelitis
1	NA	Neu, H, MC, Mil	L, MC, Mil	U	U	U	NA
2	NA	Neu, H, Fib, VL, MC, Mod	Neu, GC, VL, MC, Mil	Neu, Fib, M, Mod	Neu, MC, Mod	Neu, H, M, Mod	Neu, Fib, VL, MC, Mar
3	Neu, Fib, B, Ext, Mar	Neu, Fib, B, VL, D, Mar	Neu, GC, Fib, VL, MC, Mod	Neu, Fib, B, MC, Mar	Neu, D, Mar	Neu, H, Fib, D, Mar	Neu, Fib, VL, MC, Mod

B = bacteria; D = diffuse; Ext = extensive; Fib = fibrin; GC = gitter cells; H = histiocytic; L = lymphocytic; M = multifocal; Mar = marked; MC = multifocal-to-coalescing; Mil = mild; Mod = moderate; NA = not available; Neu = neutrophilic; U = unremarkable; VL = vasculitis.

Histologic changes in case 1 were limited mostly to the cerebral leptomeninges, which were expanded only mildly by interstitial edema and minimal-to-mild neutrophilic and

histiocytic infiltrates. A few blood vessels at the level of the hippocampus were delimited by delicate lymphocytic cuffs. No vasculitis was observed in this case. Bacteriologic culture

yielded moderate-to-heavy growth of *P. multocida* (exclusively) in the brain of all cases (Suppl. Table 1).

Given that the upper respiratory tract can serve as a portal of entry into the CNS, we evaluated the nasal cavity in cases 2 and 3. Based on the available specimens, we performed either partial (case 2) or thorough evaluation of the nasal cavity and associated structures (case 3), which included the nasal cavity, frontal sinuses, cribriform plate, olfactory nerve fascicles and olfactory bulb, as well as the middle and inner ears. The nasal epithelium was disrupted by fibrinosuppurative inflammation that also expanded the submucosa and, along with scattered colonies of coccobacillary bacteria, filled the nasal and paranasal sinus lumina. Additionally, in case 3, the olfactory neuroepithelium was extensively disrupted by similar inflammation, with frequent fibrinoid necrosis of the submucosal (nasal) blood vessels and marked osteolysis of the subjacent turbinate bone. In this cat, the nerve fascicles forming the olfactory nerve as well as the extracranial segment of the optic nerve were encircled by the inflammation, accompanied by fibrinoid necrosis of meningeal vessels, bilateral liquefactive necrosis of the olfactory bulb, and flooding of the olfactory recesses by abundant fibrinosuppurative exudate. In case 3, there was also marked bilateral suppurative otitis media; however, the inner ears were unremarkable. The eyes exhibited multifocal mild conjunctivitis and uveitis.

Additional histologic findings of significance included either mild, multifocal, portal-to-random, lymphoplasmacytic-to-suppurative hepatitis (cases 1, 2), or scattered hypertrophied (reactive) Kupffer cells, with clear cytoplasm and vesiculate nucleus, and pulmonary edema and leukocytosis (case 3). Heavy growth of *P. multocida* was obtained from the liver and lung of case 3, with frequent circulating clusters of gram-negative coccobacillary bacteria within hepatic sinusoids. Light growth of *Staphylococcus haemolyticus* was obtained from the liver and lung of case 2; no microorganisms were evident in H&E- and Gram-stained sections, however. No microorganisms were evident either in tissue sections of case 1, of which no samples other than the brain were available for bacteriologic culture. Additional ancillary testing included virus isolation (to rule out felid herpesvirus 1 [*Felid alphaherpesvirus 1*] and feline calicivirus; cases 2 and 3), fluorescent antibody tests for rabies virus (*Rabies lyssavirus*) and feline coronavirus (*Alphacoronavirus 1*; case 2), and reverse-transcription real-time PCR assays for eastern equine encephalitis and West Nile viruses (case 2), all of which yielded negative results. Hence, we based the diagnosis of meningitis or meningoencephalomyelitis associated with *P. multocida* on gross, microscopic, and bacteriologic findings.

We have described here the gross and histologic findings associated with *P. multocida* infecting the CNS of cats. Bacterial meningoencephalomyelitis is often a lethal disease process of challenging management given difficulties in accurate and prompt diagnosis.<sup>9,16</sup> In another study, 3 cases of bacterial

meningoencephalitis in cats were reported with identification of intralésional bacterial colonies microscopically, and further characterized by electron microscopy; however, no specific microorganism was confirmed as the causative agent.<sup>1</sup> Additionally, a series of 10 cases of bacterial myelitis failed to identify the microorganism(s) involved.<sup>17</sup> As evidenced by these studies, the role of bacterial organisms in the pathogenesis of meningoencephalitis and myelitis in cats is poorly characterized. In our cases, moderate-to-heavy growth of *P. multocida* was obtained from the brain of all 3 cases, unequivocally implicating *P. multocida* as the causative agent. In one previously reported case, the diagnosis of meningitis by *P. multocida* was achieved through bacteriologic culture of cerebrospinal fluid<sup>18</sup>; however, no pathology analysis was performed to confirm the suspected meningoencephalomyelitis or CNS abscess and, therefore, no absolute causal relationship was demonstrated.

*P. multocida* is a highly versatile gram-negative rod or coccobacillus frequently associated with the normal oropharyngeal microbiota of animals, with the highest carriage rate among cats (70–90%) and dogs (20–55%).<sup>24</sup> It is an important cause of disease in swine (atrophic rhinitis), ruminants (pneumonia), rabbits (snuffles and septicemia), and poultry (fowl cholera).<sup>6</sup> In animals, descriptions of *P. multocida* leading to CNS lesions are uncommon and are frequently related to respiratory signs and septicemia.<sup>2,8</sup> The majority of human cases caused by *P. multocida* are associated with soft tissue infections, following animal bites or scratches, and leading to the development of cellulitis and/or abscesses.<sup>16,24</sup> In children and immunocompromised patients, septicemia, meningitis (frequently suppurative leptomeningitis), and peritonitis have been described.<sup>16,24</sup> Immunosuppression related to stressors, such as recent changes in environment and/or diet (case 2), and inferred by thymic atrophy (case 3) could have predisposed to the development of the CNS lesions associated with *P. multocida* in our cases. Feline leukemia virus and feline immunodeficiency virus also play an immunocompromising role. However, the status of infection with these viruses in our cats is unknown. Although felid herpesvirus and feline calicivirus were not detected in the cases that were tested (cases 2, 3), preceding upper respiratory tract infections associated with these viruses is possible and could have acted as predisposing factors to opportunistic *P. multocida* infection.<sup>2,13</sup>

Key virulence factors of *P. multocida* identified to date include the capsule and lipopolysaccharides. Five *P. multocida* capsular serotypes are routinely identified based on capsule antigens (A–F).<sup>11</sup> The capsule is involved in bacterial avoidance of phagocytosis and resistance to complement; lipopolysaccharides are critical for bacterial survival in the host. Other virulence factors include *P. multocida* toxin, putative surface adhesins, and iron acquisition proteins.<sup>11</sup> Unfortunately, in our cases, we were unable to determine the virulence factors involved in the lesions because of limitations of microbial typing with matrix-assisted laser



**Table 3.** Epidemiology, predisposing factors, and lesions for the most common differential diagnosis of meningoencephalomyelitis in cats.

Agent	Age	Predisposing factor	Remarks on brain and/or spinal cord lesions	Reference
<b>Viral</b>				
Feline peritonitis virus	Young cats (<2 y old)	Recent stress	Fibrinopyogranulomatous periventriculitis, meningitis, perivasculitis, segmental myelitis, hydrocephalus	2,22
Feline immunodeficiency virus	All ages can be affected	Aggressive behavior, fighting, access to outdoors, concomitant infection	Gliosis within the hilus of the dentate gyrus and granule cell axonal sprouting, lymphocytic meningoencephalomyelitis	19
Feline leukemia virus	All ages can be affected	Access to outdoors	Diffuse white-matter degeneration, myelin sheaths devoid of central axons and containing infiltrating gitter cells	3,15
Rabies virus	All ages can be affected	Access to outdoors, contact with the vector	Nonsuppurative meningoencephalomyelitis, neuronophagia, intracellular inclusions	7
Pseudorabies virus	All ages can be affected	Consumption of contaminated raw pork	Brainstem lesions consistent with mononuclear cuffing, pronounced proliferation of astrocytes and microglial cells, weak eosinophilic viral inclusion bodies in the nuclei of astrocytes and neurons	12
Feline herpesvirus 1	Young cats	Access to outdoors, intensively housed	Severe nonsuppurative meningoencephalitis, chromatolysis of neurons, satellitosis, and gliosis with perivascular lymphocytic cuffing	13
<b>Bacteria</b>				
<i>Streptococcus canis</i>	All ages can be affected (kittens appear to be more susceptible)	Intensively housed, closed-colony animals	Fibrinosuppurative meningitis, thrombosis, intralesional bacterial aggregates	8,21
<i>Pasteurella multocida</i>	All ages can be affected (kittens appear to be more susceptible)	Upper respiratory infection Skin wounds	Necrosuppurative meningoencephalomyelitis	18
<b>Fungi</b>				
<i>Cryptococcus</i> sp.	All ages can be affected	Immunocompromise	Granulomatous meningoencephalitis with intralesional fungi (“soap-bubble” appearance)	23
<b>Parasites</b>				
<i>Sarcocystis neurona</i>	Young (1–7 y old)	Use of corticosteroids, concomitant viral (FeLV) infection, chemotherapy	Pyogranulomatous meningoencephalitis with intralesional parasitic structures	10
<i>Cytauxzoon felis</i>	All ages can be affected	Access to outdoors, wooded environment, or tick exposure	Intravascular schizont-laden macrophages, occlusion of arterioles, venules, and capillaries by protozoal organisms, random areas of necrosis	4
<i>Toxoplasma gondii</i>	All ages can be affected (kittens appear to be more susceptible)	Immunosuppression, concurrent infection	Nonsuppurative encephalomyelitis, polyradiculoneuritis with intralesional protozoa	5
<i>Cuterebra</i> spp.	Young to middle age	Access to outdoors, summer months, upper respiratory infection	Eosinophilic meningoencephalitis with parasite tracts	14

desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry.

*P. multocida* can enter the host through the mucous membranes of the upper respiratory tract, conjunctiva, and cutaneous scratch or bite wounds. *P. multocida* is an opportunistic

bacterium that can cause mucopurulent rhinitis and sinusitis.<sup>24</sup> Infection of the CNS can occur as result of extension of bacterial infection from neighboring tissues, such as the middle ear or the nasal cavity.<sup>9</sup> We hypothesize that, in our cases, *P. multocida* reached the CNS by direct extension from either

the nasal and sinus passages (cases 2, 3) or possibly the ear (cases 1, 2). In the cat, otitis media occurs primarily as a sequela of upper respiratory tract disease, associated with bacteria translocated via the Eustachian tube into the tympanic bulla.<sup>20</sup> Evaluation of the ears in cases 1 and 2 was limited to the postmortem examination; however, these cats reportedly had recurrent ear infection and vestibular signs, leading to the possibility that the CNS lesions developed as extensions from ear infections. In cats with peripheral vestibular disease, inner ear disease is most often the result of extension of middle ear disease,<sup>20</sup> which may have gone unnoticed grossly in these cases. Although a skin wound is a plausible portal of entry for *P. multocida* as well, none was evident at the time of postmortem examination in our cases.

Other means of dissemination to be considered in our cats include hematogenous and retrograde parasellar venous infections. In cases of parasellar venous infection, the ventral system of dural venous sinuses (ventral cerebral veins and ventral petrosal and cavernous sinuses) drains the ventral forebrain and the face, including the nasal cavity; therefore, infective material from those areas can drain directly into this system of sinuses and spread to the neuraxis.<sup>2</sup> Extension of the inflammation from the olfactory recesses (case 3) and the choroid plexus (cases 2, 3) explains the presence of the exudate in the cerebral ventricular spaces in 2 of our cats. The spinal cord lesions in these animals likely occurred via cerebrospinal fluid and hematogenous dissemination. Systemic hematogenous dissemination is common in cases with bacteremia, such as observed in case 3, which had lesions in multiple tissues consistent with septicemia and from which *P. multocida* was also cultured from the lung and liver.

We performed a literature search for the epidemiologic and predisposing factors as well as lesions for the most common differential diagnoses of meningoencephalomyelitis in cats (Table 3). Most infectious causes of neurologic disease result in neurologic signs that are acute, progressive, and frequently related to a systemic disease process. A detailed clinical examination in addition to complementary ancillary tests is therefore essential to reach an accurate diagnosis.<sup>9</sup> Approximately 15–20% of neurologic disease cases in cats are caused by feline infectious peritonitis virus (FIPV; *Alphacoronavirus 1*) and are related to the non-effusive (or “dry”) form of the disease, seen in infected cats with marked humoral immunity but only partial cell-mediated immunity.<sup>9</sup> The main differential cause of bacterial meningoencephalomyelitis in cats is *Streptococcus canis*. This bacterium has been described as a cause of meningitis in intensively housed, closed-colony, young cats, affecting more than 150 cats during 3 time-limited outbreaks.<sup>21</sup> *S. canis* is a  $\beta$ -hemolytic gram-positive zoonotic bacterium associated with chronic upper respiratory infections, urogenital infection, and neonatal septicemia in cats.<sup>8</sup> Young cats can be predisposed to *S. canis* infection by primary viral infections, such as felid herpesvirus and feline calicivirus.<sup>21</sup> Clinical signs and gross and histologic lesions are similar in *S. canis* and *P. multocida*

infections. Gram staining of tissue sections and bacteriologic culture are therefore needed to reach a presumptive and confirmatory diagnosis, respectively, in these cases.

Our retrospective study unequivocally links *P. multocida* infection with the occurrence of bacterial meningitis and/or meningoencephalomyelitis in cats and highlights the importance of considering this microorganism among the potential etiologies in neurologic cases. Even though of rare occurrence, *P. multocida* infection should be considered as a differential cause of CNS infection in cats, especially in cats with associated rhinitis of sinusitis and otitis media.

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### Supplemental material

Supplemental material for this article is available online.

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