CASE REPORT

Presumptive meningoencephalitis secondary to extension of otitis media/interna caused by *Streptococcus equi* subspecies *zooepidemicus* in a cat

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A 5-year-old castrated male domestic longhair cat was presented to the Ohio State University Veterinary Medical Center for evaluation of a 12-h history of falling over. The cat lived exclusively indoors, and was current on vaccines.

Physical and neurological examination revealed dizziness, a left-sided head tilt and vestibular ataxia, characterized by falling to the left when walking. Cranial nerve examination revealed miosis, ptosis, enophthalmos and third eyelid protrusion on the left eye (OS), consistent with complete Horner’s syndrome. Marked neutrophilic pleocytosis was identified on cerebrospinal fluid analysis. *Streptococcus equi* subspecies *zooepidemicus* (SEZ) was isolated from the cerebrospinal fluid. Intracranial extension of otitis media/interna is relatively infrequent in small animals. There are no reports of otitis media/interna caused by SEZ in dogs or cats. This is the first report of otitis media/interna and presumptive secondary meningoencephalitis caused by SEZ in a cat.

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fluid (CSF) collection. Anesthesia consisted of intramuscular dexmedetomidine (20 μg/kg), induction with intravenous propofol (2 mg/kg) and maintenance with inhalatory isofluorane and oxygen using mechanical ventilation. The CT study consisted of 1.3 mm contiguous transverse acquisitions, pre- and post-contrast administration (iohexol, Omnipaque 240 mg/ml, dose: 2 ml/kg IV). Hyperattenuating material was noted completely filling the left tympanic bulla, with mild contrast enhancement after iohexol administration (Fig 1). The left retropharyngeal lymph node was mildly enlarged (Fig 1C). No brain parenchyma abnormalities were noted. However, CT has inherent limitations when imaging soft tissues, so the lack of brain parenchyma abnormalities in this case may have been related to the limitations of this imaging modality. The differentials considered were left OMI or a polyp, with neoplasia considered less likely based on the presence of only mild contrast enhancement and no lytic lesions. CSF was collected from the cerebellomedullary cistern. The fluid was colorless and slightly hazy, with a total protein of 19.9 mg/dl (RI < 25 mg/dl), a white blood cell (WBC) count of 1368 cells/μl (RI < 5 cells/μl) and a red blood cell (RBC) count of 99/μl (RI < 5 cells/μl). Cytology revealed 61% non-degenerate neutrophils, 27% large mononuclear cells and 12% lymphocytes (Fig 2). The large mononuclear cells were vacuolated and interpreted as reactive. The lymphocytes were small and well differentiated. No evidence of hemosiderin, erythrophagia, etiologic agents or neoplastic cells was seen. The findings were consistent with a neutrophilic pleocytosis with mild blood contamination. Based on the combination of the CT images and CSF results, a bacterial meningoencephalitis secondary to extension of OMI was considered the most likely presumptive diagnosis. Other differentials included cryptococcosis, viral infection (feline infectious peritonitis) and toxoplasmosis.

Cerebrospinal fluid was cultured on trypticase soy agar with 5% sheep’s blood (TSAII, Becton-Dickenson, NJ, USA) and incubated at 35°C in 5% CO2; reduced thioglycolate broth (Becton-Dickenson, NJ, USA) was inoculated to recover fastidious organisms and anaerobes. A polymerase chain reaction (PCR) was performed for Toxoplasma gondii, feline coronavirus and feline leukemia virus.

Streptococcus equi subspecies zooepidemicus (SEZ) was isolated from the CSF in high numbers in pure culture. The organism was speciated using Lancefield grouping (Streptocard, Becton-Dickenson, NJ, USA) and conventional biochemicals (API-20 STREP System, Biomerieux, MO, USA). PCR results were negative. Cryptococcus species antigen enzyme immunoassay was negative.

The cat recovered uneventfully from anesthesia. Treatment was initiated with ampicillin—sulbactam (30 mg/kg IV q 8 h, Unasyn; Pfizer), enrofloxacin (5 mg/kg IV q 24 h, Baytril; Bayer), dexamethasone—sodium phosphate (0.15 mg/kg IV q 24 h for 2 days) and famotidine (0.5 mg/kg PO q 12 h), pending

![Fig 1](image_url)  
(A) Transverse pre-contrast image at the level of the tympanic bulla. Note the hyperattenuating material completely filling the left tympanic bulla. No lytic lesions were noted. No abnormalities were noted involving the brain parenchyma. (B) Transverse post-contrast image, obtained at the same level as image (A), which shows mild contrast enhancement of the material present within the left tympanic bulla. (C) Transverse pre-contrast image at the most caudal level of the tympanic bulla. Note the enlargement of the left retropharyngeal lymph node (white arrow). (D) Dorsal pre-contrast reconstruction at the level of the tympanic bulla, showing hyperattenuating material completely filling the left tympanic bulla.
Improvement (7 versus 1368 WBC/cytes). The results of the second CSF showed marked trophils, 3% large mononuclear cells and 95% lymphocytes.

TMS solved over the following weeks. The total duration of the neurological signs, gradually improved and recovered fully from surgery. A severe left Horner’s syndrome was noted postoperatively, which along with the rest of the pleocytosis. The cat recovered uneventfully.

Culture results. The day after presentation the cat developed severe hypersensitivity to light, touch and sound, and self-inflicted multiple bite wounds to his limbs. The cat was started on a dexmedetomidine constant rate infusion (3 µg/kg/h IV) for sedation, which was slowly weaned off over the next 8 h. The intravenous catheter was removed on the third day of hospitalization because of poor patient tolerance. After obtaining the culture results, the cat was started on trimethoprim–sulfamethoxazole (TMS–SMZ, 15 mg/kg PO q 12 h), as treatment for the meningoencephalitis, and amoxicillin–clavulanic acid (62.5 mg PO q 12 h, Clavamox; Pfizer) to prevent infection from the self-inflicted bite wounds. The neurologic status of the patient improved gradually. A left ventral bulla osteotomy was performed 5 days after presentation. A large amount of purulent material was removed from the bulla. Histopathology revealed marked supplicative and lymphoplasmacytic otitis media with no signs of a polyp. No etiologic agents were noted but the inflammation was suggestive of a chronic bacterial infection. Aerobic, anaerobic and Mycoplasma species cultures of the material removed from the bulla were negative. A second cerebellomedullary cistern CSF sample was obtained at the time of surgery. The fluid was colorless and clear, with a total protein of 8.0 mg/dl (RI < 25 mg/dl), a WBC count of 7 cells/µl (RI < 5 cells/µl) and an RBC count of 3/µl (RI < 5 cells/µl). Cytology showed 2% non-degenerate neutrophils, 3% large mononuclear cells and 95% lymphocytes. The results of the second CSF showed marked improvement (7 versus 1368 WBC/µl) in the magnitude of the pleocytosis. The cat recovered uneventfully from surgery. A severe left Horner’s syndrome was noted postoperatively, which along with the rest of the neurological signs, gradually improved and resolved over the following weeks. The total duration of TMS–SMZ therapy was 8 weeks. Upon last contact with the owners 8 months after diagnosis, the cat remained neurologically normal.

Central nervous system (CNS) complications of OMI have been recognized in animals, although they are considered uncommon. In people, the incidence of these complications has decreased with the wider availability of antibiotics; however, they are still associated with mortality rates ranging from 5 to 31%. The case reported here made a full recovery. A variety of organisms have been isolated from the few feline cases of intracranial extension of OMI reported to date, including Pasteurella multocida, Escherichia coli, Enterococcus species, Staphylococcus aureus, Mycoplasma species, and Streptococcus canis.

Streptococcus equi subsp zooepidemicus is considered a commensal organism of the mucous membranes and skin of various animals, notably horses. It frequently acts opportunistically in horses, causing respiratory infections, wound infections, endometritis, and abortion. This bacterium is not regarded as a component of the commensal flora of neither dogs nor cats. Over the last few years, SEZ has been reported as an emerging pathogen in dogs, associated with severe hemorrhagic pneumonia in shelter dogs. Only recently, two reports have documented infections caused by SEZ in cats. One report described an outbreak of respiratory disease in a cattery. Four of the cats necropsied showed signs of pyogranulomatous meningoencephalitis. The other report described two cases of rhinitis and meningitis caused by SEZ in two cats housed in separate shelters. Neither of the two cats or their attendants had any known exposure to horses. In our case, no exposure to horses or farm animals was identified upon questioning the owner. As there were no clinical signs or history of otitis externa, it is likely that the route of infection into the middle/inner ear was via the oral mucosa and/or the nasopharynx. Negative bacterial culture from the tympanic bulla is likely due to the 5 days of antimicrobial therapy given to the cat between the original CSF collection and the bacterial culture from the tympanic bulla is likely.

Infection with SEZ is a rare cause of meningitis in humans with only 22 cases reported so far. The majority of these cases were caused by contact with animals (mostly horses) or ingestion of unpasteurized dairy products. The reported mortality rate was 24%. In this case, antimicrobial therapy using TMS–SMZ was elected. This is a bactericidal drug that penetrates both normal and inflamed meninges and achieves therapeutic levels in the CSF. Two doses of intravenous dexamethasone were also administered, starting with the first dose of antimicrobials. In spite of the controversy regarding the use of steroids in bacterial meningitis, we elected to use it in our patient following the most current recommendations for treatment of acute bacterial meningitis in people. A recent meta-analysis, which reviewed 24 randomized controlled trials of corticosteroids use for acute meningitis, found no difference in mortality between treated and control groups.
bacterial meningitis in people, revealed a lower rate of short-term neurologic sequelae and a trend toward lower mortality in the corticosteroid-treated group in adults in high-income countries.\textsuperscript{17}

To the authors’ knowledge, this is the first report of OMI and secondary meningoencephalitis caused by SEZ in a cat. Clinicians should be aware of the rare zoonotic disease potential of this agent, which seems to be an emerging pathogen in small animal companion species.

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