INFLAMMATORY POLYP IN THE MIDDLE EAR WITH SECONDARY SUPPURATIVE MENINGOENCEPHALITIS IN A CAT

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A 15-month-old male Maine Coon Cat presented with persistent auricular discharge and progressive head tilt, ataxia, and loss of blink on the right side. Using computed tomography a hyperattenuating, contrast-enhancing material within a thickened right tympanic bulla and contrast enhancement of the adjacent cerebellum were identified. Marked suppurative inflammation was identified on cerebrospinal fluid analysis with no growth on bacterial culture. Ventral bulla osteotomy was performed to remove a soft tissue mass, and an inflammatory polyp with chronic severe suppurative inflammation was confirmed using histology. Staphylococcus auricularis was grown on aerobic culture and Fusobacterium necrophorum and Peptostreptococcus anaerobius were grown on anaerobic culture. The cat was treated for 10 weeks with amoxicillin/clavulanic acid and metronidazole. Dramatic improvement in body weight, appetite, energy level, balance, and resolution of right-sided facial paralysis were noted, but the cat retained a head tilt.

Key words: Inflammatory polyp, Otitis media, Otitis interna, Meningoencephalitis, Computed tomography

Introduction

A FIFTEEN-MONTH-OLD male Maine Coon Cat presented with a chief complaint of persistent auricular discharge and progressive head tilt, ataxia, right-sided facial paralysis and a depressed appetite over a 1-month period. The cat was treated appropriately for otitis externa with antibiotics, but the clinical signs continued to progress. The cat was a breeding male that lived strictly indoors and was current on vaccinations for rabies and FVRCP.

On presentation, the cat was in good body condition but febrile (103.2°F). There was a moderate amount of brown discharge from the right ear, and the right mandibular lymph node was moderately enlarged. The cat had a right head tilt and a mildly ataxic gait. A ventral strabismus of the right eye and a left rotary nystagmus bilaterally were noted. The cat had right-sided facial paralysis with lack of menace and palpebral responses as well as a mild facial droop. There was a right-sided Horner's syndrome. All other cranial nerves, postural reactions, and spinal reflexes were normal. There was no evidence of cranial or spinal hyperesthesia. A right-sided peripheral vestibular lesion with concurrent facial paralysis and Horner's syndrome was suspected. Differential diagnoses included otitis media/interna, inflammatory middle ear polyp, and neoplasia (lymphoma, squamous cell carcinoma, sebaceous adenocarcinoma).

There was a mild neutrophilia (10,270/µl; reference range 2500–8500/µl) identified on complete blood count. Serum biochemical panel, urinalysis, FeLV, FIV, and serum T4 were unremarkable. Serum toxoplasma titers were negative.

The cat was premedicated with glycopyrrolate (0.01 mg/kg) and butorphanol (0.3 mg/kg) subcutaneously prior to induction of anesthesia with propofol (4.7 mg/kg) intravenously. Anesthesia was maintained with 2% isoflurane and oxygen. The cat was placed in sternal recumbency, and the tympanic bullae and ear canals were examined using a third generation CT scanner.* Transverse and dorsal 3 mm contiguous slices of the bullae were acquired and there was hyperattenuating material within the right tympanic bulla that was contrast enhancing. There was apparent thickening of the right bulla. In addition, there was an area of contrast enhancement in the adjacent cerebellum. (Fig. 1, A and B).

The cat was placed in right lateral recumbency, and cerebrospinal fluid was collected from the cerebellomedullary cistern. There was marked neutrophilic pleocytosis. White blood cells were 303/µl (normal <8/µl) and protein was 136 mg/dl (normal <25 mg/dl). There were 85% segmented non-degenerate neutrophils, 10% small lymphocytes, and 5% large mononuclear cells. Aerobic bacterial culture of the cerebrospinal fluid yielded no growth.

The cat was placed in dorsal recumbency, and a ventral bulla osteotomy was performed on the right side. A one

*Sytec 4000 CT Scanner (General Electric Company, Milwaukee, WI).
A large amount of purulent fluid was lavaged from the ear canal. Upon recovery from anesthesia, the cat began rolling severely and was markedly disoriented. The cat was treated with acepromazine 0.02 mg/kg intramuscularly, and the signs resolved within 1 hour after recovery. The histologic diagnosis was inflammatory polyp with severe chronic suppurative inflammation, fibrosis and neovascularization. Aerobic bacterial culture from the middle ear grew *Staphylococcus auricularis*. Anaerobic bacterial culture grew *Fusobacterium necrophorum* and *Peptostreptococcus anaerobius*. Treatment was initiated with amoxicillin/clavulanic acid at 12 mg/kg by mouth daily for 12 hours and metronidazole 12 mg/kg by mouth daily for 12 hours for 10 weeks based on Kirby-Bauer susceptibility testing.

Six weeks after surgery, there was dramatic improvement in the cat’s ataxia, energy level and appetite. Physical examination was unremarkable except for persistent right head tilt. Four weeks later the cat had gained weight, had a normal appetite, energy level, and balance despite a slight persistent right head tilt.

**Discussion**

CNS complications secondary to middle ear disease are very uncommon in all species but have been well described in humans, most commonly related to chronic otitis media. These complications in humans have become less common with appropriate antibiotic therapy but remain an important consideration due to the prognostic implications of CNS involvement. Meningitis and brain abscesses are the most common complications seen. Rarely, brain herniation through the osteitic temporal tegmen into the middle ear or mastoid cavity occurs with chronic disease. CNS involvement may occur via direct extension through soft tissue planes, suture lines, or along cranial nerves most commonly to the temporal lobe of the cerebral cortex and the cerebellum adjacent to the tympanic bulla. Thrombophlebitis and hematogenous spread have also been implicated in causing meningitis or abscessation.

There are sporadic reports of CNS extension secondary to otitis media/interna in many species. Brain stem extension of bacterial otitis media/interna has been reported in dogs and cats. Acute meningoencephalomyelitis resulting from *Pasteurella multocida* otitis interna was described in a 4-month-old rabbit. Meningoencephalitis secondary to otitis interna in a gorilla and a llama have also been reported.

Inflammatory polyps are nonneoplastic masses of unknown etiology that typically develop in young cats although polyps have been reported in cats as old as 15 years. Any breed can be affected, but domestic short hair cats, Abyssinian, Persian, Himalayan, Siamese, Maine Coon, and Rex breeds are most commonly affected. Proposed etiologies include congenital development from branchial arches, calicivirus, and chronic inflammation secondary to otitis media. Most polyps arise from their attachment within the tympanic bulla or middle ear and extend through the tympanic membrane into the external ear canal or through the auditory tube into the nasopharynx. Clinical signs are referable to obstruction or compression of the areas affected. Otitis media/interna may also contribute to progression of clinical signs including development of Horner’s syndrome and related vestibular signs, and as described in this cat, may result in central nervous system extension. The most commonly recognized neurologic com-
plications of middle-inner ear disease are peripheral nerve dysfunction including Homer’s syndrome, and if the structures within the petrous temporal bone are involved, facial paresis/paralysis and peripheral vestibular signs. The cat in our report presented with ataxia, head tilt, facial paresis, and Horner’s syndrome. All of these deficits could be attributed to middle and inner ear involvement alone, and the cat did not have other clinical neurologic deficits to prompt suspicion of CNS involvement. The cerebellar involvement in this cat was an incidental finding on computed tomography; however, on cerebrospinal fluid analysis there was a neutrophilic pleocytosis consistent with extension of otitis media. Unfortunately, bacterial cultures of cerebrospinal fluid were negative, so bacterial infection could not be confirmed. Feline infectious peritonitis was considered, but was unlikely based on the cat’s response to antibiotic therapy. Toxoplasma gondii infection was considered unlikely based on negative serum titers. Neoplasia was considered unlikely based on the age of the cat and response to therapy.

The middle ear cultures from the cat in this report were unique in that Staphylococcus auricularis as well as two anaerobic organisms, Fusobacterium necrophorum and Peptostreptococcus anaerobius, were isolated. The most common organisms isolated in cases of otitis media in dogs and cats include Staphylococcus intermedius, Pseudomonas aeruginosa, Proteus and Streptococcus spp. Anaerobic organisms are much less commonly isolated. Pseudomonas aeruginosa (70%) and Staphylococcus aureus (23%) are the most common aerobic organisms isolated from human patients. Anaerobic organisms are more commonly isolated from human patients with otitis media (50%) with Bacteroides and Peptostreptococcus spp. comprising the majority of these.

Inflammatory polyps may be diagnosed by otoscopic examination if they are present in the external ear canal or by oral examination or digital palpation and retraction of the soft palate if they affect the nasopharynx. Imaging may be used for patients with suspected middle ear disease when polyps are not grossly evident or in patients with pronounced neurologic deficits. Skull radiographs may allow identification of soft tissue masses in the nasopharynx or tympanic bulla, osteolysis, or enlargement of the affected bulla. Unfortunately, radiographs are not a sensitive imaging modality. Hence, lack of radiographic changes does not rule out disease. In addition, radiographs do not provide information regarding CNS involvement. Computed tomography (CT) and magnetic resonance (MR) imaging are more sensitive, particularly for evaluating middle ear, tympanic bullae, and brain involvement. The use of CT to evaluate the middle ear, tympanic bullae, and nasopharyngeal areas of cats with inflammatory polyps has been described. The location and extent of the inflammatory polyp and osseous changes could be accurately assessed. CT is the imaging modality of choice for most middle ear disease in humans because it provides excellent detail of the thin and complex bony structures of the middle ear, while MR imaging provides poor osseous imaging. In a recent report the CT imaging characteristics of normal structures of the middle ear, including the tympanic membrane, auditory ossicles, tympanic bulla, cochlea, internal acoustic meatus, and semicircular canals in dogs are described. When compared with CT, MR imaging provides superior resolution of inner ear structures and the brain stem. For this reason, human patients with middle ear diseases who have facial paralysis, vertigo, or other neurologic deficits indicating inner ear and brain stem involvement are imaged with MR.

In this cat, CT was adequate for imaging the middle ear as well as identifying the cerebellar involvement that would not have been identified otherwise. Antimicrobial therapy was continued for a longer period postoperatively (10 weeks) than if only middle ear disease had been present because CNS extension was identified on CT scan. Had CNS involvement not been identified, inadequate duration of antimicrobial therapy may have led to progression of disease. Therefore, imaging of this cat strongly influenced management and presumably outcome.

In conclusion, this report illustrates that CNS extension may occur and should be considered in cats presenting with inflammatory polyps of the middle ear and concurrent otitis media/interna with or without overt signs of intracranial involvement. CT imaging was beneficial in identifying CNS extension in our patient. Therefore, CT imaging should be considered in cats presenting with middle ear diseases in part to help rule out subclinical or overt concurrent CNS disease.

REFERENCES

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