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ETIOPATHOGENESIS OF FELINE OTITIS EXTERNA and/or OTITIS MEDIA

The etiopathogenesis of otitis externa in the cat can generally be divided into various factors. Primary factors are those that are noted to cause inflammation in ears. The most common primary factors are ear mites (otodectes cynotis), with the next most commonly encountered being hypersensitivities (atopy, food sensitivity). Other primary factors tend to be much less common and include foreign bodies, other ectoparasites (demodex, ticks), keratinization/lipid disorders (idiopathic ceruminous otitis), autoimmune diseases (e.g. pemphigus foliaceus) and masses (aural polyps, ceruminous cysts, neoplasia).

Predisposing factors are those that make the ear more prone to the development of otitis externa. Examples include immunocompromising infections (e.g. FIV) and masses in the ear.

Perpetuating factors are those that are capable of perpetuating the otitis even if the primary factor has been removed/resolved. They include bacteria, fungi (especially Malassezia spp.), ceruminous debris (e.g. ceruminoliths), proliferative changes, otitis media and treatment errors (e.g. over or under treatment or inappropriate treatment).

In the United States, the incidence with which feline otitis externa is diagnosed in general practice has increased by 34% since 2006 (Banfield Pet Hospital data 2011). Otitis media may be infectious (bacterial, fungal) or non infectious (polyps, neoplasia, foreign body). Otitis media is most commonly seen as an extension from an otitis externa, through a perforated tympanum. Perforation may be seen with debris accumulation, more aggressive bacterial infections (e.g. Pseudomonas sp.) or as a complication of masses growing within the ear canal (e.g. ceruminous cysts, ceruminous cystadenoma or cystadenocarcinoma, squamous cell carcinoma). Less commonly, otitis media may also arise from retrograde infections through the Eustachian tube. This is seen more commonly than in dogs likely because of the greater incidence of posterior pharyngeal inflammatory disorders seen in cats (often viral in origin). The tympanum is usually intact in such cases. Obstruction of the Eustachian tube may also be a significant predisposition to the development of otitis media. Occlusion of the Eustachian tube results in the accumulation of the secretions that are normally produced within the middle ear and is most commonly associated with chronic naso/pharyngeal disease (inflammation or neoplasia -lymphoma most
common. In one study, fluid within one (30%) or both (70%) middle ears was noticed as an incidental finding in 30% of cats with sinonasal disease. In the authors experience, the fluid from affected ears is more commonly sterile but infection is possible. Inflammatory polyps and middle ear neoplasms may also cause otitis media.

**ATOPY (Environmental Allergy) AND FOOD SENSITIVITY (FS) RELATED OTITIS EXTERNA (OE)**

The incidence of otitis seen in atopic and food sensitive cats is less than that seen in allergic dogs. However, the incidence of both are significant and they tend to be under-diagnosed. In the author’s practice, atopy related OE is significantly more common that that seen with food sensitivities. With atopy or FS, otic involvement may antedate the development of other cutaneous allergy signs, be concurrent with these signs, or may be the only clinical manifestation (i.e. ears the only area affected). Pinnal involvement is variable. In some individuals, it may predominate. Atopy or FS related otitis externa are usually bilateral, but may be predominantly or entirely unilateral.

The most common secondary infections are with Malassezia pachydermatis. Secondary bacterial infections most commonly involve Staphylococcus pseudintermedius.

In the author’s practice, atopic otitis externa is the most common cause of aural hematoma seen in the cat.

Diagnosis is by history and rule out (e.g. seasonality; rule out food sensitivity by restrictive diet trial).

Therapy for a flare of allergic (usually atopic) otitis is directed at resolving secondary infections, reducing inflammation and removing debris from the ears. Because of the propensity of the cat to develop ototoxicity related to the use of various topical medications, some clinicians only use systemic therapies in managing otitis externa in the cat (i.e. oral glucocorticoids, antibiotics and/or anti-fungals). The author generally uses topical and/or a combination of topical and systemic treatments, depending on the severity of the problem and the integrity of the tympanum. When the tympanum is intact, combination products such as Tresaderm (Merial; neomycin, thiamendazole, dexamethasone) or Otomax (Schering-Plough: gentamicin, clotrimazole, betamethasone) are used. Because the ear canals of the cat are relatively much shorter than in the dog, much debris from the ears can be shaken out, without the need for ear cleansers. However, any of the commonly used ear cleansers used in the dog are also used in the cat. The frequency of flushing is often not as aggressive as that used in the dog (e.g. in the cat, beginning once every other or every third day). Ear cleansers are often not started until 2-4 days after anti-inflammatory medications are started to facilitate greater tolerance to the flushing procedure. Topical Therapies to consider when the tympanum is perforated or integrity of tympanum is unknown (i.e. less likely to cause ototoxicity if they gain access to the middle ear) include various formulated mixes that contain injectable 22.7 mg/ml enrofloxacin, injectable 4 mg/ml dexamethasone sodium phosphate and/or 1% miconazole (Conofite; Schering-Plough). For bacteria (coci): 2:1 mix of enrofloxacin and dexamethasone; for bacteria (rods): flush the ear with a trisEDTA containing product 10 minutes before the enro.:dex. mix; for Malassezia: 1:1 mix of dex. and miconazole; for bacteria and Malassezia (1:1:2 mix of dex., enrofloxacin and miconazole).

Safer ear cleansers include dilute white vinegar and water (1 : 2 or 1 : 3 dilution); TrizEDTA or Mal-A-Ket (Dechra), Cerumene (Vetoquinol) and Duoxo Micellar Ear solution (Sogeval).

Atopic otitis externa can often be successfully managed with adequate control of the underlying allergy. For atopy, the most successful alternatives include long term oral glucocorticoids, allergen specific immunotherapy (hyposensitization) or oral cyclosporine (5.0-7 1/2 mg/kg/day). As an alternative to systemic management of atopy or as an adjunctive therapy in those individuals who are not adequately controlled by systemic therapy, consideration can be given to chronic topical maintenance management: 1:1 mix of 1% miconazole and
dexamethasone sodium phosphate (4 mg/ml) twice weekly.

**CERUMINOLITHS**

These are concretions of wax and debris that fill the horizontal canal, in front of the tympanum. They are most commonly seen as a sequel to ear mite infestations or allergic otitis. This debris serves as a nidus for infection, is a source of irritation, prevents medication from reaching the deeper aspects of the ear canal and may perforate the tympanum. Ceruminoliths are often difficult to remove with ‘at home’ flushing. They are best removed from the ears under general anesthesia. Pre-cleaning treatment with Cerumene (Vetquinol) may facilitate removal as will the use of video otoscope guided flushing, suctioning and ‘grabbing’ forceps.

**IDIOPATHIC CERUMINOUS OTITIS EXTERNA**

This has been suggested to be an idiopathic seborrheic disorder, wherein excessive amounts of cerumen are produced within the ears. These accumulations may be associated with mild inflammation. This problem may predispose to secondary Malassezia and/or bacterial infections. At present, the author questions the existence of this syndrome and finds these to be low grade allergic ears and successfully manages them as such (see above).

**DEMODICOSIS**

A focal form of demodicosis may be restricted to the ears and manifest as an accumulation of large amounts of waxy debris. Pruritus and signs of discomfort are variable. The diagnosis is made by cytologic examination of ear swabs (debris in mineral oil). Therapy is with topical ivermectin in the ear (1:9 dilution of injectable 10mg/ml in mineral oil or propylene glycol; every other day), systemic ivermectin (0.3 mg/kg PO eod) or topical Tresaderm (Merial; q 24 hrs or BID) or amitraz (1 ml of 20% product in 30 ml mineral oil; eod).

**FELINE CERUMINOUS CYSTS / CERUMINOUS CYSTOMATOSIS**

Ceruminous cystomatosis is a non neoplastic disorder wherein ceruminous glands become cystic. These focal, blue colored lesions may be solitary or grouped and may originate anywhere from the tympanum, throughout the canals and over the medial pinnae and base of the ear. The concurrent presence of otitis externa does tend to worsen the clinical manifestations of this syndrome (more lesions, lesions larger), but otitis does not actually cause the lesions.

When ear canal cysts are small, they are usually not symptomatic. As they become larger or are present in groups they may become a predisposition to otitis by partially occluding the canal and resulting in the accumulation of debris. This produces a favorable microenvironment for yeast and bacterial proliferation. The ear becomes symptomatic with the development of these infections or if the lesion or lesions occlude the ear canal. With canal occlusion, debris accumulating behind the lesions may eventually perforate the tympanum and accumulate within the middle ear.

Unless the lesions are occluding the canals, they are often tolerated and do not require therapy. In the presence of a concurrent, allergic otitis, control of the allergic and secondary infection components (e.g. chronic topical steroid/antibiotic/anti-fungal product) may reduce the numbers and size of lesions. However, most lesions are not resolved. The most effective options for management include surgical resection, cryosurgery or laser removal. Surgical resection involves the removal of as much of the cystic tissue as possible utilizing biopsy or grasping forceps. The ears are treated with a steroid containing product for 3-4 weeks following the procedure. The incidence of regrowth is variable. Laser removal of especially the base of the cysts is very
effective for removing tissue and reducing the incidence of re-growth. Difficult to remove lesions or recurrent lesions may require ear canal ablation to resolve the problem.

AURAL POLYPS

Polyps are inflammatory lesions of unknown etiology that are most commonly noted in young cats (variably reported with a mean age of about 1 year; range of 2.5 months to 18 years) 2,3. Although most are unilateral, polyps can be bilateral. Most polyps appear to grow from the epithelial lining of the epitympanic cavity/ tympanic cavity, then extend either through the tympanum in to the horizontal canal or down through the auditory canal in to the posterior pharynx. Although uncommon, it is possible to have polyps grow in both directions (about 10% of cases4). Clinical signs associated with aural polyps include signs of otitis externa (head shaking, scratching ears, otic exudate), and/or otitis media (Horner’s syndrome, head tilt, nystagmus, ataxia) The etiology of polyps remains unknown. Recent attempts to link polyps to calicivirus, herpesvirus, Bartonella and mycoplasma have failed 4,5. The authors therapy of choice for aural polyps is to remove as much of the polyp as possible from the ear via traction/avulsion. Deeper tissue removal is facilitated by utilizing biopsy forceps directed through a video otoscope. Cytologic examination dictates the need for systemic and topical antibiotic therapy. The ear is flushed every other day with dilute vinegar and water (1:2 to 1:3). Post avulsion, the cat is treated with oral prednisolone, beginning at 2.0-3.0 mg/kg/day for 2 weeks, then 1-1.5 mg/kg/day for 2 weeks, then 0.5-0.75mg/kg/day for 2 weeks, then 0.5-0.75 mg/kg once every other day for 2 weeks (6-8 weeks of therapy). The incidence of ‘post traction’ recurrence has been reduced from 40% to 3% utilizing this steroid regimen. This has been seen by others6. Refractory cases are treated with ventral bulla osteotomy.

References