

CANINE ALLERGIC OTITIS EXTERNA – FROM A TO Z

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Atopy (environmental allergy) and to a lesser degree, food sensitivity are the most common primary factors associated with the development of otitis externa in the dog. While it would appear that flea bite hypersensitivity is capable of inducing otitis, its prevalence is somewhat controversial. If it is seen, it is usually seen in more severely affected individuals. Contact hypersensitivity may also be noted, but is relatively rare.

ATOPIC OTITIS EXTERNA (ENVIRONMENTAL ALLERGY)

Otitis externa is noted to affect 80 – 85% of atopic individuals. In many cases (25%), otitis is the first sign of their allergic disease. Patients may have several “flares” of allergic otitis externa prior to developing more generalized signs of allergy. The areas targeted in affected individuals tend to be the proximal portion of the medial pinna and entrance to the vertical canal. With progression, the canals become involved. The development of secondary infections are also associated with the development of canal involvement. Some individuals will have only the medial pinnae involved. Acute “flares” of allergy may be associated with significant pinnal edema. In some breeds (e.g. German Shepherd Dog and Labrador Retriever), otic pruritus may be associated with an ear margin dermatitis (alopecia, variable degrees of scaling) that can look very “scabies-like”. Individuals who are severely pruritic may develop alopecia and dermatitis over the outer aspect of the pinna , these changes induced by self-trauma. Pinnal changes associated with chronicity include thickening, hyperpigmentation and lichenification. Chronic canal changes include canal wall thickening due to edema, ceruminous gland dilatation and fibroplasia. Canals may become stenotic as the thickened canal walls become “folds”. Some individuals (e.g. spaniel breeds) may develop fibroproliferative nodules. Although most individuals will have bilateral involvement, it is possible to see atopic otitis affect predominantly one ear. Occasionally, only one ear will be involved. We tend to see a higher incidence of dilatation of the pars flaccida in affected ears. Whether this is in response to head shaking or low grade posterior pharyngeal disease that results in some degree of Eustachian tube dysfunction is not known.

FOOD SENSITIVITY (ADVERSE FOOD REACTION; FOOD ALLERGY)

Otitis externa is noted in about 60% of food sensitive individuals. It may be the first sign of allergy in as many as 35% of cases. About 20% of food sensitivity patients will have otitis as the only manifestation of their allergy problem (i.e will never have more generalized signs of allergic disease). This is unlike atopic individuals who may start manifesting their allergic disease as an otitis, but eventually develop more generalized allergic signs.

Anecdotal observations that have not been proven:

It has been suggested that cocker spaniels and Labrador retrievers are more likely to present with otitis externa as the only symptom of food sensitivity. It has been suggested that dogs younger than 6 months of age who experience an acute bout of allergic bilateral otitis are more likely to have food hypersensitivity. It has also been suggested that involvement of only the ear canals (sparing the medial pinna and entrance to the vertical canal) may be more commonly associated with food sensitivity.

CONTACT HYPERSENSITIVITIES

The most common ingredients associated with the development of irritant or contact hypersensitivity reactions in the ear appear to be propylene glycol (present in many topical formulations – flushes and medicinal drops) and neomycin (fradiomycin). However, it is possible to develop sensitivities to other ingredients. A “flare” of otitis is often noted several days after the initiation of therapy. On physical examination, there is often macular to confluent patches of intense erythema over the medial pinna, entrance to the vertical canal and canals (in areas on which the medication has been applied). On occasion, the reaction can be even more severe, resulting in erosions or ulceration within the ears. Yet another presentation is the persistence of otitis (with or without complicating secondary infections) in the face of what appears to be appropriate therapy. These changes may be severe (erosion or ulceration). Irritant/contact sensitivities should always be considered on the list of differential diagnoses for refractory erosive/ulcerative otitis. Yet another presentation that appears to be more common with Otomax or Mometamax is a popular eruption (erythematous papules), usually admixed with erythematous macules.

A “tip off” to the presence of a reaction to topical medications is the finding of neutrophils on cytologic examination of exudates, when neutrophils had not been present in the original cytology. Bacterial and/or *Malassezia* numbers have often significantly decreased since the original visit, in spite of the topical reaction.

Affected ears should be flushed out thoroughly with saline. Efforts should be made to minimize the use of topical medications in the ears. Anti-inflammatory and germicidal effects can be achieved with parenteral therapy (i.e. glucocorticoids, antibiotics, anti-fungal). Flushing is often encouraged to remove debris from the ears. The flush of choice for the author has been dilute vinegar and water (1:2 to 1:3). The greater dilution (1:3) is used for more sensitive ears. Flushing is often only necessary once every other day. If it is deemed necessary to use topical therapies, consideration would be given to alternatives that are devoid of those ingredients known to have created problems (e.g. enrofloxacin and dexamethasone sodium phosphate mix, 1:3). In spite of the fact that neomycin is an aminoglycoside (as is Gentamicin), the author has used Otomax (containing Gentamicin) in ears that

have proved sensitive to Tresaderm or Panalog (both containing neomycin). The product appeared to be tolerated well.

Contact hypersensitivities have been most commonly associated with reactions to topical medications (e.g. neomycin, propylene glycol). Affected individuals usually have a “flare” of otitis in the face of therapy. The finding of neutrophils in the cytology of affected areas, where neutrophils were not present originally can be a helpful “tip off” to the presence of contact hypersensitivity.

SECONDARY INFECTIONS IN ALLERGIC OTITIS

Malassezia pachydermatis is the most common organism associated with secondary colonization and infection in these hypersensitivity disorders. It is important to note that dogs may also develop hypersensitivities to *Malassezia* and its by-products and, as such, relatively few organisms may contribute significantly to inflammation and pruritus within the ear. Secondary bacterial infections are less commonly encountered. *Staphylococcus pseudintermedius* is most commonly seen in both acute and chronic cases. Cytologically, these are seen as cocci and diplococci on Diff Quick staining. Gram negative infections (*Pseudomonas*, *E. coli*, *Klebsiella* etc.) are more likely to develop in chronic cases and/or those that have been intermittently treated with topical antibiotics. These infections are generally seen as rods on cytologic examination. These secondary infections (*Malassezia* and/or bacteria) contribute significantly to the overproduction of wax within the ears, inflammatory and proliferative changes noted previously.

DIFFERENTIATING OTITIS EXTERNA DUE TO FOOD SENSITIVITY FROM ATOPY

This is best done by assessing response to a restrictive diet trial. As much as possible, secondary ear infections and proliferative changes should be resolved prior to or during the earlier stages of the restrictive diet trial. A restrictive diet (e.g. novel protein, hydrolysate or home prepared diet) is also begun. The author generally does not use a home prepared diet because these are often not nutritionally balanced and the diet trials to determine the relative importance of food sensitivity to otitis externa are often long (i.e. 3-4 months). Clinical/otoscopic/cytologic examinations should be repeated every 2 - 3 weeks during the diet trial. As the ears come under control, one gradually drops out of the various anti-inflammatory medications (oral steroids first, then topical steroid containing product) until the ear is controlled with flush alone. This will allow for interpretation of the effectiveness of the diet alone. It does appear to take longer to see the onset of benefit of a restrictive diet when evaluating otitis externa as compared to evaluating more generalized pruritus/dermatitis. As noted above, diet trials to assess the importance of food sensitivity to otitis externa frequently last 3-4 months. The patient is challenged with his/her regular diet to prove the presence of a food sensitivity as the cause of the otitis problem.

ROUTINE TREATMENT OF ACUTE AND INFREQUENTLY RECURRENT ALLERGIC OTITIS EXTERNA

Most cases of acute and infrequently recurrent allergic otitis externa are treated with topical medications : anti-fungal / antibiotic / anti-inflammatory proprietary preparation (choice dictated by cytology findings) and an ear cleanser (when moderate or larger amounts of wax are in the ear) (See Otolaryngology Pearls Parts I and II in these proceedings). The average duration of time required to put the problem in to remission is generally 2-4 weeks. Systemic steroids, starting at anti-inflammatory dosages (e.g. prednisone or prednisolone, 0.5 – 1.0 mg/kg/day; decreasing dosage over 2-3 weeks) may quickly reduce the discomfort and swelling within more severely affected ears.

LONG TERM MANAGEMENT OF ALLERGIC OTITIS EXTERNA

1. Resolve secondary bacterial/yeast infections with topical antibiotic/antifungal/steroid product
2. Routine use of a “flush” such as Mal-A-Ket plus (Dechra) or Epi-Otic Advanced (Virbac) on a once or twice weekly basis. These products help to take over for the natural flushing mechanism of the ear (lateral epithelial migration). They also have some germicidal effect and help reduce the tendency towards the development of secondary bacterial and yeast infections.
3. Long term therapy with a topical glucocorticoid :
 - a. 1:1 mix of dexamethasone sodium phosphate and 1% miconazole or, if prone to recurrent Malassezia use a 1:2 dilution (i.e. more miconazole). Usually used once or twice weekly for maintenance.
 - b. Hydrocortisone aceponate (Cortavance) – twice weekly. This has been proven to reduce the frequency of “flares” of atopic otitis externa.
 - c. Less severely; inflamed ears : CortAstrin (1% hydrocortisone; aluminum acetate astringent) – once every 48 – 72 hours long term

Many allergic ears can potentially be maintained with the topical treatments noted above.

4. Food sensitivities – patients who respond to restrictive diets can often have their allergic otitis controlled on restrictive diets alone (i.e. and not require long term, maintenance topical glucocorticoid therapy).
5. Experiences with systemic therapies for atopy (environmental allergy)
 - a. Patients on oral steroids may have their otitis well controlled with just this medication (no need for long term, maintenance topical glucocorticoid)
 - b. Patients on successful immunotherapy may have their otitis well controlled with just this. However, we do see a significant number of patients whose allergies are not well controlled enough to rely on this alone (i.e. patients still benefit from long term, maintenance topical glucocorticoid therapy).
 - c. Cyclosporine – we see a significant number of patients whose dermatologic manifestations of atopy are well controlled on cyclosporine, but their atopy related otitis is not as well controlled. These patients often benefit from long term, maintenance topical glucocorticoid therapy.

d. Oclacitinib – although we do not have a great deal of experience with this drug, it would appear that there are some individuals whose dermatologic problems are not as well controlled as their otitis. These patients also benefit from chronic topical glucocorticoid therapy.

AURAL HEMATOMA

Hematomas develop within and along the auricular cartilage (likely rupture of blood vessels extending from the convex to the concave surface of the pinna through foramina in the cartilage). The etiology of aural hematomas remains controversial. In our experience, most affected individuals have evidence of inflammatory, pruritic ear disease that is allergy related (atopy and/or food sensitivity). Head shaking, producing a sinusoidal wave motion of the ear is thought to exacerbate the problem. Cartilage degeneration and fibrovascular granulation tissue filling the cartilage defect does appear to antedate hematoma formation. This finding suggests that a pre-existing inflammatory process, possibly immune mediated in nature, may play an important role in the etiopathogenesis. Therapeutic intervention is warranted because of the patient discomfort and the cosmetic consequences associated with spontaneous resolution (fibrosis, pinnal thickening, permanent distortion). It is very important that concurrent otic disease be aggressively treated in association with any hematoma management protocol. This often includes oral glucocorticoid therapy. Failure to adequately address otic disease is a very important reason for management failure or hematoma recurrence. Small to moderately sized hematomas may be managed with oral glucocorticoids alone (in the dog, usually starting at 1 mg/kg/day for 1-2 weeks, then half this dose for 1-2 weeks then slowly taper). This can be done with or without aspiration of the hematoma content. The ear is rechecked on a weekly basis and potentially re-aspirated as necessary. Similarly, small to moderately sized hematomas may be managed by aspiration and the installation of a glucocorticoid. The volume placed in the hematoma is dictated by the size of dog / size of the hematoma (e.g. 4.0 – 20.0 mg medroxyprogesterone acetate; DepoMedrol®; 1.0 – 4.0 mg dexamethasone sodium phosphate; 0.5 – 1ml triamcinolone acetonide, Vetalog®). Recheck weekly; consider re-aspiration weekly.

These more conservative therapies for aural hematomas (oral glucocorticoids; intralesional glucocorticoids) tend to be less successful with large or chronic hematomas. These may be better managed with any one of a number of techniques: incision / mattress sutures; teat cannula installation; laser (involves incision and then multiple small holes made over the hematoma surface to stimulate adhesion). In the author's experience, the routine use of a decreasing dose of oral steroid, following one of these more aggressive treatments for the hematoma, is associated with more patient comfort and a faster resolution of the hematoma. Glucocorticoids are started at anti-inflammatory dosages (i.e. 0.5 – 1.0 mg/kg/day prednisone / prednisolone and extend over 2-3 weeks).

Proliferative Canal Disease in allergic otitis – for management, see “Otology Pearls” Parts I and II in these Proceedings