PEER REVIEWED

Managing Chronic Otitis Externa

PSPP…have you heard about this approach?

Brittany Lancellotti, DVM, DACVD
Dermatologist at Veterinary Skin and Ear in Los Angeles, CA.

Chronic otitis externa (OE) can be a frustrating relapsing disease leading to pet discomfort, permanent disfigurement, increased caregiver burden, damage to the human-animal bond, and financial strain. A systematic approach using the Primary, Secondary, Perpetuating, and Predisposing (PSPP) classification systemic allows the practitioner to consider each of these important aspects for more successful management.[1-4]

Primary Diseases

The source of the disease

Food, flea, and environmental allergies can all trigger otitis externa. Hormonal diseases (hypothyroidism, Cushing's syndrome) often cause recurrent infections. The presence of masses or foreign objects (grass lawns) can be a trigger. Ectoparasitic (Sarcoptes, Demodex spp.), fungal (Dermatophyte), and epithelialization (primary seborrhea, sebaceous adenitis, zinc responsive dermatosis) disease can also occur.

Secondary Infections

Bacteria and/or yeast

Cytology is a key tool for evaluating the presence and response to topical treatment of secondary Staphylococcal, Malassezia, and Pseudomonas infections. Culture and systemic treatment are typically only warranted with otitis media or severely proliferative deep horizontal canals.

Perpetuating Factors

Chronic changes

Excess cerumen and damaged epithelial migration, biofilm production, glandular hyperplasia, mineralization, edema and stenosis of the canal, development of cysts, scar tissue and chronic inflammatory changes are all perpetuating factors that interfere with resolution and control of clinical signs.

Predisposing Risks

Anatomic risk factors

Certain breed-influenced conformation factors will increase risk of frequency and severity of infection when primary disease is present, including narrowed horizontal canals (e.g. brachycephalics), excessive hair (e.g. poodles and mixes), pendulous pinnae (e.g. bassett hounds), and primary secretory otitis media (e.g. cavalier King Charles spaniels).

Hypersensitivity dermatitis is the most common cause of chronic OE and can include environmental, food, and insect triggers.5 Up to 40% of patients with canine adverse food reactions (CAFR) may develop signs before 1 year of age.[6] A well performed 8-12 week strict hydrolyzed or novel protein diet trial is recommended to evaluate control of clinical signs with elimination of food triggers.[7] Other common clinical signs of CAFR include gastrointestinal upset (vomiting and diarrhea), conjunctivitis, anaphylaxis, and increased frequency of stools.[8] Addressing pruritus and inflammation with glucocorticoids when initiating an elimination diet trial may allow for abbreviated trials if pruritus and inflammation do not relapse within two weeks of finishing the course of glucocorticoids.[9] After 8-12 weeks of an elimination diet trial, challenge feeding by reintroducing the pet's original diet may result in a relapse of clinical signs within five days and fourteen days for 50% and 90% of dogs with CAFR respectively.[10] Relapsing OE despite control of food allergens may indicate ongoing environmental triggers and warrant referral to a dermatologist and discussion of allergy immunotherapy for long term control.[11,12]

Endocrinopathies, such as hypothyroidism and Cushing's syndrome (hyperadreno-corticism, HAC), may result in recurrent secondary infections from a poorly functioning immune system.[13] A detailed history may reveal other systemic signs,

such as lethargy, weakness, weight gain, appetite change or thinning hair coat. Thyroid testing should include Total T4, free T4 by equilibrium dialysis, and thyroid stimulating hormone (TSH) in order to differentiate between euthyroid sick syndrome and true hypothyroidism. Complete blood count and serum chemistry (CBC/Chem) can increase clinical suspicion of HAC if common abnormalities such as thrombocytosis or elevated alkaline phosphatase are present, but normal CBC/Chem should not rule out HAC as a primary trigger for OE.

Secondary infections should be investigated with cytology to determine the presence and type of microorganisms. Cytology guides selection of appropriate topical antimicrobials and/or ear cleaners, as well as evaluates response to treatment. Culture of infections is typically not recommended for OE and is more appropriate for otitis media.[5] Resistance profiles can vary significantly between samples depending on the region of the ear canal sampled. In addition, topical antimicrobial therapy allows for significantly higher concentrations of antimicrobials than systemic antibiotics. Because susceptibility testing often evaluates for expected response to concentrations of systemic antibiotics, topicals may often overcome reported resistance due to the significant increase in antimicrobial concentration when applied directly to the external ear canal, rather than passing through systemic absorption.

Perpetuating factors may result in ongoing secondary infection despite appropriate antimicrobials. Inflammation leads to significant changes such as glandular hyperplasia, edema of the canal, damage to normal epithelial migration, and stenosis.[14] Antiinflammatory medications such as glucocorticoids and modified cyclosporine are helpful in reducing acute and chronic inflammation respectively.[5] Prednisolone at 1mg/kg can help treat acute inflammation and then be tapered to minimize adverse effects. Owners should be informed of possible adverse effects with glucocorticoids, both short and long term. Modified cyclosporine (Atopica; Elanco) is a calcineurin inhibitor indicated for the control of atopic dermatitis in dogs weighing at least four pounds. A significant reduction in inflammation is achieved about 4 weeks after starting 5mg/kg daily and steroid-sparing benefits of this medication can be used in the long term management of chronic, proliferative OE.[11,12] Modified cyclosporine may be an effective alternative to total ear canal ablation surgery in the management of end-stage ear disease.[15]

Biofilm is most often produced by Pseudomonas bacteria, however, other microorganisms such as Staphylococcus and Malassezia have been shown to produce biofilm.[16] This extracellular matrix of exopolysaccharides makes microorganisms 10 to 1000 times more resistant to topical antibiotics as the result of impaired drug penetration, reduced growth of microorganisms having entered a quiescent state, and the expression of multi drug resistance pumps.[17] Although certain topical agents have demonstrated anti-biofilm efficacy, including Triz-EDTA, N-acetyl cysteine, chlorhexidine, polyhexanide, and hypochlorous acid, biofilm is most effectively treated by physical removal during deep ear cleaning with video otoscopy.[5]

Predisposing factors can increase the risk of frequency or severity of OE in animals with a primary disease and make management more challenging. These risk factors include (but are not limited to) copious ear hair, narrowed or congenitally stenotic canals, pendulous pinnae and excess moisture (swimming, bathing, excessive cleaning). Ear hair may prevent penetration of topical therapy and therefore resolution of secondary infection. When considering hair plucking, sedation, analgesia, and antiinflammatory treatment is recommended. Maintenance hair plucking should not be necessary if the primary disease and perpetuating factors are managed.

Ear flushing is a helpful tool in removing excess cerumen, adjusting pH, and providing additional antimicrobial action.[18,19] Practitioners should familiarize themselves with available ear flushes and select an appropriate cleaner(s) based on each individual pet's needs. Cooperative care techniques and use of technical staff to educate clients can help improve the client's confidence and capability in performing this helpful treatment for long term management.

Communicating to clients that OE is often caused by chronic, lifelong primary diseases can help manage expectations, minimize frustrations, and improve compliance with workup of the underlying cause and implementation of the long term plan.

References

1 Koch S. The challenge of chronic otitis in dogs: from diagnosis to treatment. Today's Veterinary Practice. April 14, 2017.

2 Bajwa J. Canine otitis externa - treatment and complications. Can Vet J. 2019;60(1):97-99.

3 Saridomichelakis MN, Farmaki R, Leontides LS, Koutinas AF. Aetiology of canine otitis externa: a retrospective study of 100 cases. Vet Dermatol. 2007;18(5):341-347. doi:10.1111/j.1365-3164.2007.00619.x

4 O'Neil DG, Volk AV, Soares T, Church DB, Brodbelt DC, Pegram C. Frequency and predisposing factors for canine otitis externa in the UK - a primary veterinary care epidemiological view. Canine Med Genet. 2021;8(1):7. doi:10.1186/s40575-021-00106-1

5 Nuttall T. Managing recurrent otitis externa in dogs: what have we learned and what can we do better? Journ Am Vet Med Assoc. 2023;261(S1):S10-S22. doi:10.2460/javma.23.01.0002

6 Olivry T, Mueller RS. Critically appraised topic on adverse food reactions of companion animals (7): signalment and cutaneous manifestations of dogs and cats with adverse food reactions. BMC Vet Res. 2019;15(1):140. doi:10.1186/s12917-019-1880-2

7 Mueller RS, Olivry T. Critically appraised topic on adverse food reactions of companion animals (4): can we diagnose adverse food reactions in dogs and cats with in vivo or in vitro tests? BMC Vet Res. 2017;13(1):275. doi:10.1186/s12917-017-1142-0.

8 Mueller RS, Olivry T. Critically appraised topic on adverse food reactions of companion animals (6): prevalence of noncutaneous manifestations of adverse food reactions in dogs and cats. BMC Vet Res. 2018;14:341.

9 Favrot, C. et al. "The Usefulness of Short-Course Prednisolone during the Initial Phase of an Elimination Diet Trial in Dogs with Food-Induced Atopic Dermatitis." Vet Dermatol, vol. 30, no. 6, Dec. 2019, p. 498–+.

10 Olivry T, Mueller RS. Critically appraised topic on adverse food reactions of companion animals (9): time to flare of cutaneous signs after a dietary challenge in dogs and cats with food allergies. BMC Vet Res. 2020;16(1):1-4.

11 Olivry T, DeBoer DJ, Favrot C, et al; International Committee on Allergic Diseases of Animals. Treatment of canine atopic dermatitis: 2015 updated guidelines from the International Committee on Allergic Diseases of Animals (ICADA). BMC Vet Res. 2015;11:210. doi:10.1186/s12917-015-0514-6.

12 Outerbridge CA, Jordan TJM. Current knowledge on canine atopic dermatitis: pathogenesis and treatment. Adv Small Anim Care. 2021;2:101-115. doi:10.1016/j.yasa.2021.07.004.

13 Hensel P, Santoro D, Favrot C, Hill P, Griffin C. Canine atopic dermatitis: detailed guidelines for diagnosis and allergen identification. BMC Vet Res. 2015;11(1):196. doi:10.1186/s12917-015-0515-5.

14 Tabacca NE, Cole LK, Hillier A, Rajala-Schultz PJ. Epithelial migration on the canine tympanic membrane. 2011;22(6):502-510. doi:10.1111/j.1365-3164.2011.00982.

15 Hall JA. Oral cyclosporin in the treatment of end-stage ear disease: a pilot study. Vet Dermatol. 2003;14:212.

16 Robinson VH, Paterson S, Bennett C, Steen SI. Biofilm production of Pseudomonas spp. isolates from canine otitis in three different enrichment broths. Vet Dermatol. 2019;30(3):218-e67. doi:10.1111/vde.12738.

17 Chan WY, Hickey EE, Page SW, Trott DJ, Hill PB. Biofilm production by pathogens associated with canine otitis externa, and the antibiofilm activity of ionophores and antimicrobial adjuvants. J Vet Pharmacol Ther. 2019;42(6):682-692. doi:10.1111/jvp.12811.

18 Paterson S. Topical ear treatment - options, indications and limitations of current therapy. J Small Anim Pract. 2016:57(12):668-678. doi:10.1111/jsap.12583.

19 Buckley LM, McEwan NA, Nuttall T. Tris-EDTA significantly enhances antibiotic efficacy against multidrug-resistant Pseudomonas aeruginosa in vitro. Vet Dermatol. 2013;24(5):519-e122. doi:10.1111/vde.12071.