



Try_my_best/shutterstock

Topic Overview

Otitis is a common disease among dogs. Without early and effective intervention, it can progress to chronic and recurrent otitis externa (CROE), which can be challenging to treat. The clinical framework of primary causes, secondary contributors, predisposing factors, and perpetuating factors is useful for creating treatment plans. General physical examination helps practitioners prioritize primary causes. Otoscopic examination can identify early and reversible chronic changes. External palpation of the ear canals can help detect suspicion for mineralization. The most valuable diagnostic tool is cytology because it enables identification and targeted treatment of the organisms involved in the infection. For treatment, most patients with CROE will benefit from oral and/or topical glucocorticoids, the selection of appropriate antimicrobials, ear cleaning, and control of the primary causes. Regular recheck examinations evaluating otalgia and treatment response are crucial to treatment success, after which maintenance therapy is needed to prevent relapse.

**GET CONTINUING
EDUCATION CREDIT**



Scan the QR code
after reading this
article to take the
quiz at vetfolio.com
and earn free CE.



DERMATOLOGY

Diagnosing and Treating Chronic and Recurrent Otitis Externa

K. Helena Montin Mills, DVM, Animal Dermatology Clinic, Robbinsville, New Jersey

Annette Lundberg, DVM, MS, DACVD, University of Minnesota College of Veterinary Medicine, St. Paul, Minnesota

Otitis remains one of the most common diseases among dogs.¹ To help categorize clinical cases and formulate therapy, a recent review proposed definitions for recurrent and chronic otitis (see **GLOSSARY**).² Successful management and treatment of chronic and recurrent otitis externa (CROE) can be challenging because they require an understanding of the pathogenesis, diagnostics, and multimodal therapy.²⁻⁵ Preventing chronic and irreversible changes to the ears requires early recognition and medical intervention.^{2,3,5} Furthermore, patience and adherence from clients and patients are vital for positive outcomes.^{3,6} Appropriate intervention leads to better patient outcomes, client satisfaction, improved antimicrobial stewardship,^{2,6} and ultimately saves ears from end-stage CROE requiring surgical removal

through total ear canal ablation and bulla osteotomy (TECABO).^{3,7-9}

PATHOGENESIS

Understanding the primary, secondary, predisposing, and perpetuating (PSP) factors of otitis externa (OE) can help clinicians create effective treatment plans (**TABLE 1**).^{2,3,5,10,11}

Identifying the causes through a detailed history and physical examination prioritizes differentials and diagnostic workup.^{2-5,10} CROE may have more than one primary cause and multiple other contributing factors.^{3,5,10}

Primary Causes

Primary causes of OE are conditions that can

Learning Objectives

- Successful management of chronic and recurrent otitis externa requires an understanding of primary causes, secondary contributors, predisposing factors, and perpetuating factors.
- Oral and/or topical glucocorticoids can reverse early chronic changes to the ear canals such as hyperplasia, but mineralization cannot be reversed.
- Bacterial culture and susceptibility is controversial for otitis externa, but may have a place in identifying organisms and in a public health perspective.
- Chronic and recurrent otitis externa is the result of inadequately controlled primary causes.
- Recheck examinations with cytology are crucial in monitoring patient comfort and treatment response.
- Maintenance therapy with ear cleanings, low-potency corticosteroids, and control of underlying diseases can help to prevent relapses.

Glossary

Otitis Inflammation of the pinnae and/or ear canals

Ear infection Clinically significant microbial overgrowth or infection

Recurrent Clinically significant ear inflammation and/or infection within 3 months of complete resolution of previous episode

Acute Otitis without acquired proliferative changes in the ear canals

Chronic Otitis with acquired proliferative changes in the ear canals

induce clinical disease in a healthy ear with no secondary, predisposing, or perpetuating factors (i.e., dermatologic diseases that also affect the ear epithelium).^{2-4,10} Atopic dermatitis is the primary cause

in 43% to 75% of dogs with OE and in 31% to 50% of dogs with CROE.¹²⁻¹⁴ Cases of unilateral otitis should prompt prioritization of other causes such as foreign bodies (e.g., plant awns), otitis media (OM), polyps, and neoplasia (particularly in cats as allergic otitis is less common).¹⁵ For successful management of CROE, obtaining an accurate diagnosis for the underlying cause and its appropriate management is crucial.^{2-4,10}

Secondary Contributors

Secondary contributors to otitis lead to clinical disease in abnormal ears,³⁻⁵ which compound the effects of primary causes, exacerbating clinical signs.^{2,3} Bacterial or yeast infections of the ear can incite inflammation³; however, they result from a combination of primary causes, predisposing factors, and perpetuating factors.³ Ear infections become chronic or recurrent despite treatment of the infection itself because of uncontrolled underlying disease.³

TABLE 1 Primary, Secondary, Predisposing, and Perpetuating Factors of Otitis Externa^{2-5,10,11}

CAUSE	EXAMPLES
PRIMARY CAUSES	
Autoimmune disorder	Pemphigus complex, lupus erythematosus, erythema multiforme
Endocrinopathy	Hypothyroidism, hyperadrenocorticism, sex hormone imbalance
Foreign material	Plant, sand/dirt, dessicated medication, hairs
Hypersensitivity disorder	Allergies (e.g., environmental, fleas, food), contact dermatitis, adverse drug reaction
Immune-mediated	Juvenile cellulitis
Keratinizing disorder	Sebaceous adenitis, idiopathic seborrhea
Obstructive ear disease	Polyp, feline ceruminous cystomatosis, feline proliferative and necrotizing otitis externa, neoplasia (e.g., ceruminous gland adenoma/adenocarcinoma, squamous cell carcinoma, basal cell tumor, papilloma, sebaceous gland hyperplasia)
Parasites	Ear mites (<i>Otodectes cynotis</i>), <i>Demodex</i> species, <i>Sarcoptes</i> species, ticks, chiggers (<i>Trombicula</i>)
Trauma	Hair plucking, cotton-tipped applicators
SECONDARY CONTRIBUTORS	
Infectious	Bacteria (<i>Staphylococcus</i> , <i>Pseudomonas</i> , <i>Proteus</i>), yeast (<i>Malassezia</i>), or fungi (<i>Aspergillus</i>)
PREDISPOSING FACTORS	
Conformation	Pendulous pinnae, congenital stenosis, auricular hypertrichosis
Breed predisposition	Cocker spaniels, Labrador retrievers, springer spaniels, Shar-Peis, Chow Chows, brachycephalic breeds
Excessive moisture	Swimmers, hot and humid environments
Overtreatment	Excessive moisture from cleaning, maceration, impactions, iatrogenic damage (cotton-tipped applicators, hair plucking)
PERPETUATING FACTORS	
Acquired pathologic changes	Disrupted epithelial migration, hyperplasia, proliferation, stenosis, calcification

Predisposing Factors

Predisposing factors are present before the onset of otitis, alter the ear canal anatomy and physiology, and increase the likelihood of OE development.^{3,11} Alone, each factor is unlikely to cause disease.^{2,11} However, the presence of any predisposing factor(s) warrants active surveillance for the development of primary causes, which may then lead to otitis.^{10,11} A classic example is the pendulous pinnae of basset hounds, especially because not every dog breed with pendulous pinnae is at increased risk for OE.^{11,16} Some individuals and breeds may have multiple predisposing factors.^{3,11,17}

An often overlooked predisposing factor is iatrogenic trauma from hair plucking or vigorous cleaning that induces inflammation or injury to delicate epithelial cells.¹¹ The authors believe that hair plucking may be required for select patients (e.g., those with entrapped biofilm or matting) and should be done responsibly with use of appropriate analgesia and anti-inflammatory medications.⁸

Perpetuating Factors

Perpetuating factors include changes to the normal anatomy and physiology resulting from chronic inflammation and OM; ultimately, they are the result of uncontrolled otitis.^{2,18} These changes are initially very mild but become prominent with time. The significance of perpetuating factors is that they sustain otitis regardless of resolution or control of the primary cause.^{2,3,5}

The first change detectable during otoscopic examination is a cobblestone appearance of the lining of the canal walls, resulting from epidermal nodular and glandular hyperplasia (**FIGURE 1**).^{2,3,5} Treatment at this stage can most effectively reverse the changes and prevent end-stage otitis; however, if untreated, the changes progress to epidermal and dermal hyperplasia, stenosis, fibrosis, and finally mineralization (**FIGURE 2**).^{2,3} Although medical management may be able to reverse hyperplasia and fibrosis, it cannot reverse mineralization of the canal; therefore, ears with mineralized canals are often called end-stage ears,^{2,3} at which stage a TECABO may be recommended to improve patient comfort by removing a source of pain.^{2,3,7,9}

The above changes can all result in OM and tympanic membrane alterations, if not rupture.^{2,3,18,19} The natural, self-cleaning mechanism of the ear becomes disrupted, and cerumen along with other debris will no longer migrate out of the canal.^{2,3,5,8}

Diagnosis of otitis media/interna (OMI) can be more apparent when patients exhibit Horner syndrome, facial paralysis, or peripheral vestibular signs.^{3,19} However, in a study evaluating 121 dogs with CROE and no overt evidence of OMI in either their history or clinical presentation, magnetic resonance imaging (MRI) detected subclinical OM in 20.8% of dogs.¹⁸ OM should be considered for patients with recurrent otitis, an abnormal tympanic membrane, and a history of treatment failures.^{3,19,20}



FIGURE 1. Video otoscopy image of classic cobblestone appearance and moderate erythema in a horizontal canal of a dog.



FIGURE 2. Video otoscopy image of marked ceruminous and nodular hyperplasia with stenosis, fibrosis, and purulent material in a dog. The deep structure may represent an abnormal opaque tympanic membrane or a mass.



CLINICAL PRESENTATION

History

Patients with CROE can exhibit a variety of clinical signs (e.g., head shaking, malodorous ears, hearing loss).³ Hearing loss caused by OE can be conductive or sensorineural.^{3,5,21,22} In a study of dogs referred for chronic OE, hearing deficits were bilateral for 53% and unilateral for 25%.²¹ The more severe cases of hearing loss were statistically significantly associated with OM; therefore, patients with hearing loss should be referred for advanced imaging so they can receive appropriate diagnosis and treatment.²¹

OE always results from an underlying disease that must be addressed to minimize recurrence. A detailed history regarding recurrent skin infections, pruritus or pruritic behaviors, and seasonal changes may reveal concerns for atopic dermatitis. Alternatively, changes in appetite, thirst, urination, and energy levels may indicate an endocrinopathy.

Physical Examination

The entire patient should always be examined because general examination can provide valuable insight into the underlying cause, perpetuating factors, or adverse drug effects. Oral examinations are relevant for detecting potential diseases affecting the middle ear or temporomandibular joint.^{3,19}

The clinical signs of OE depend on the primary cause, secondary infections, and chronicity.³ Ear canal palpation is a valuable part of examination, tolerated to some degree by most patients. A healthy ear canal is made up of soft and pliable cartilage.^{2,3,23,24} Chronic inflammation makes the ears firm, thickened, and nonpliable, often corresponding to the degree of hyperplasia and calcification.^{2,3} Palpation also helps with assessment of facial asymmetry and identifying unilateral versus bilateral disease.

External visual examination of the ear canal can reveal hyperplasia and stenosis of the vertical canal (**FIGURE 3**),^{3,8} which may be reversible depending on whether the cartilage in the canal has mineralized.³ A subset of patients will have purulent exudate with erosions to ulcerations, which may indicate *Pseudomonas* species infection.^{2,25} These conditions typically are particularly painful, although pain is likely underrecognized in most patients with chronic otitis.^{3,25} Characterizing the exudate can provide insight as to

expected cytologic findings; however, ear cytology should never be replaced by smell or macroscopic visual inspection.

When otoscopic examination is possible, a cobblestone appearance of the canal epithelium indicates ceruminous gland hyperplasia. Concurrent stenosis or masses can be seen.^{2,3,5} For dogs of certain breeds, especially brachycephalic breeds, the normal horizontal canal is often slightly stenotic, which is a predisposing factor for OE and should not be confused with pathologic stenosis.^{3,8,25} Hair may be present within the canals of breeds such as poodles, bichon frises, and terriers, making deep structures difficult to visualize.^{3,8}

A healthy tympanic membrane is translucent, but when chronically inflamed it becomes opaque and thickened and/or ruptures.^{2,3,5,19} Depending on their size, partial ruptures can be difficult to diagnose. In stenotic ears, visualization of the tympanic membrane may not be possible.^{18,26} Note that an intact tympanic membrane does not rule out OM.^{3,18,27,28}

OMI may be identified during examination of facial symmetry and neurologic examination. Head tilt can sometimes be a nonspecific finding because it can result from pain, causing abnormal head carriage, or from temporary or permanent sequelae to vestibular syndrome.³ Middle ear disease can damage the postganglionic sympathetic neurons, leading to Horner



FIGURE 3. Canine hyperplastic otitis.

Figure3: Courtesy Dr. Sandra Koch. Opposite Figure 4: Courtesy Dr. Kim Devos Davies.

syndrome: miosis, third eyelid elevation, enophthalmos, and ptosis.^{3,22} In dogs, it is more common to also see facial nerve paralysis from middle or inner ear disease.^{3,22} These dogs can have neurogenic keratoconjunctivitis sicca, making tear production evaluation relevant.^{3,22} Patients with neurologic deficits benefit greatly from early referral for diagnosis and intervention.

DIAGNOSTICS

Ear Cytology

Ear cytology is a foundational part of chronic otitis management because it enables quick identification and quantification of infectious organisms and inflammatory cells. Ear cytology should be performed at every visit to help measure treatment effectiveness and particularly at the end of a treatment course to prove infection resolution. Cytology may reveal cocci (usually *Staphylococcus* species), rods (e.g., *Pseudomonas*, *Proteus*), or yeasts (e.g., *Malassezia* species).^{2,5,29,30} Rarely, filamentous fungi (e.g., *Aspergillus*) are also identified.^{3,30} Inflammatory and red blood cells are not seen in healthy ears and enter the lumen of the canal only through ulceration, exudate, masses, or middle ear effusion.²⁹ The presence of rods and inflammatory cells is associated with a higher risk for OM.^{18,28}

Biofilm can be produced by a variety of organisms, including *Malassezia* and *Staphylococcus*, but predominantly *Pseudomonas*.^{2,31} Most bacteria noted on cytology are in their planktonic form; however, a

biofilm is an aggregate of bacteria suspended in a liquid matrix.^{2,31} Presence of biofilm can be suggested by clinical and cytologic evaluation of sticky mucus and a net-like cluster of bacteria and neutrophils (FIGURE 4).^{2,31} However, a definitive diagnosis requires specialized testing, such as periodic acid–Schiff staining.³² Identification of biofilm is valuable because biofilm makes the bacteria less susceptible to antibacterial agents and increases adherence to the epithelium, both of which can make infections more difficult to resolve.^{2,31}

Bacterial Aerobic Culture and Susceptibility

The usefulness of ear aerobic bacterial culture and susceptibility for OE treatment is controversial. When interpreted along with cytology, culture results can provide insight to predominant pathogens.^{2,3,29} The controversy arises from the lack of clinical breakpoints for topical formulations, and the in vivo therapeutic concentrations for topical therapy are significantly higher than plasma drug concentrations.^{2,3,5,33} Culture results do not seem to be predictive of in vivo response.³⁴ The literature reports that cultures are potentially useful for cases of atypical bacterial infections or failure to respond despite adequate treatment and adherence.^{2,5} It is the authors' opinion that with adequate treatment of the underlying disease and anti-inflammatory therapy, proper application technique, and selection of medications and cleanser, along with compliant clients and patients, true treatment failure resulting from resistance is uncommon. However, from a public health perspective, identifying multidrug-resistant bacteria is useful because resistance genes can be shared between bacteria. In addition, bacterial culture and susceptibility of material collected from within the tympanic bulla during a myringotomy is recommended because OM is frequently treated with a systemic antibiotic, whereas OE is treated topically.¹⁹ Although the relevance of ear cultures will remain controversial, a smaller sample study showed cultures better correlated with cytology than with 16S rRNA gene profiling.³⁵

Advanced Imaging

Advanced imaging is crucial for detecting masses, congenital abnormalities, mineralization of the external ear canals, and OMI. The decision between computed tomography (CT) and MRI depends on case presentation, differentials, and availability.^{18,28} CT is

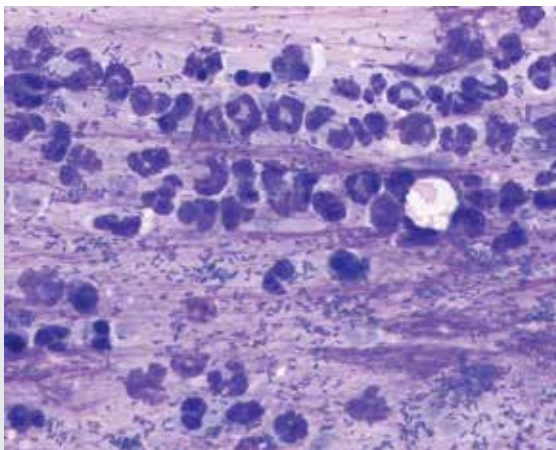


FIGURE 4. Oil immersion showing biofilm containing rod bacteria and inflammatory cells from a dog with chronic otitis externa secondary to a mass. Diff-Quik stain, 1000× magnification.



better for evaluating osseous tissue, including evaluating the thickness of the bullae and mineralization of the external canal (**FIGURE 5**).²⁸ For patients with neurologic deficits, depending on the constellation of clinical signs, MRI may be preferred because it enables better evaluation of soft tissue (**FIGURE 6**). When a mass is suspected, contrast should be used if available and not contraindicated.

TREATMENT

At-Home Ear Cleaning

When done correctly, ear cleaning is a crucial component of managing OE because it removes exudate, debris, and foreign material, which affect the effectiveness of medications and serve as nidi for infection.⁸ Clients must be shown how to correctly clean the ears, including introducing the product into the ear, gently massaging the ears if it is comfortable for the patient, and then allowing the patient to shake its head.⁸ Clients can gently clean debris from the visible parts of the ears but should never insert objects (e.g., a cotton-tip swab) into the canal as it can cause an impaction and trauma.⁸

The market for ear cleaners is continually changing; therefore, being familiar with desired active ingredients is recommended (**TABLE 2**).^{8,36} Many of the active ingredients can potentially irritate the middle ear or be ototoxic; therefore, when the status of the tympanic membrane is unknown, a good option for minimizing

risk for ototoxicity is use of tromethamine (tris) and EDTA-tromethamine (tris-EDTA).³⁷

Patients with CROE may be very sensitive to topical medications.⁸ If a contact reaction is suspected, discontinue topical therapy and allow the ears to recover; saline should be considered as a safe alternative if cleaning is necessary.⁸ Some ear cleaners may also contain topical glucocorticoid(s). If one of these cleaners is to be used, choose a product with low-potency glucocorticoid(s) and use it infrequently to avoid the adverse effects of steroids.^{8,20}

In-Clinic Ear Cleaning

At-home cleaning may be sufficient for patients with acute OE; however, in-clinic cleaning or deep ear flushing may need to be performed in clinic for patients with more chronic OE. When material is tightly adhered deep in the canal, as is common for patients with CROE, deep ear flushing by a veterinarian is usually recommended. Recent studies on collaborative care showed deep ear flushing leads to faster patient improvement and resolution of otitis.^{6,38,39} The distinction between in-clinic ear cleaning and deep ear flushing is that in-clinic ear cleaning using commercial cleaners can be helpful when the client is unable to perform ear cleaning at home⁴⁰; however, deep ear flushing involves visualization of the canal and tympanic membrane to remove as much material as



FIGURE 5. Computed tomography scan showing end-stage otitis externa with hyperplasia and mineralization of the ear canals (**circles**) and left-sided otitis media (**arrow**) in a dog.

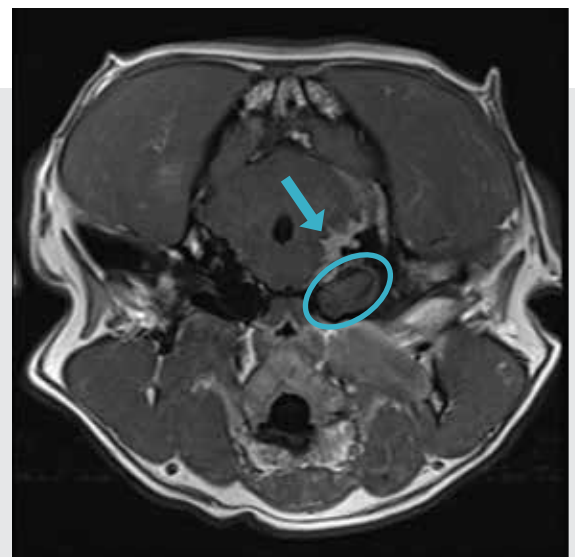


FIGURE 6. Magnetic resonance image of T1-weighted fluid-attenuated inversion recovery after contrast administration, showing otitis media (**circle**) and interna (**arrow**) with meningeal enhancement secondary to otitis media in a dog.

possible while often using specialized equipment to mitigate damage.^{8,20}

Patients undergoing deep ear flushing need to be anesthetized with a protected airway in case of tympanic membrane rupture or compromise because the fluid will drain through the auditory canal and into the oropharynx.^{8,20} Ideally, irrigation is performed through a video otoscope.^{3,8,20} If a video otoscope is not accessible, a hand-held otoscope with a red rubber catheter and a syringe may enable adequate flushing,

although visualization throughout the procedure may be more difficult.^{3,8,20} Saline is typically considered the best option for flushing because it is safe for the middle ear and provides excellent visibility.^{3,8,20} Soaking with a ceruminolytic product could be considered before flushing.⁸

Complications may result from deep ear flushing and anecdotally seem to be more common among cats; therefore, the risks should be discussed with clients before flushing.⁸ Commonly reported complications

TABLE 2 Active Ingredients in Ear Cleaning Products^{3,8,36}

CATEGORY	PURPOSE	APPLICATION	EXAMPLES
Ceruminolytic	Soften and disrupt cerumen	Mildly waxy ears	- Propylene glycol - Lanolin - Glycerine - Squalene - Butylated hydroxytoluene - Cocamidopropyl betaine - Mineral oils
Ceruminolytic surfactant	Emulsify debris and maintain in a solution	Ceruminous and purulent debris	- Diethyl sodium sulfosuccinate (or docusate) - Calcium sulfosuccinate - Other detergents
Foaming agent	Disburse debris and aerate canal	Break down large clumps of debris	- Urea-carbamide peroxide - Sodium lauryl ether sulfate
Astringent	Dry ears	Dry ears and antimicrobial	- Isopropyl alcohol - Acetic acid - Boric acid - Benzoic acid - Salicylic acid - Sulfur - Aluminum acetate - Silicon dioxide
Antimicrobial agent	Slow microbial growth	Prevent recurrent infection	- Chlorhexidine - Parachlorometaxyleneol
Anti-inflammatory agent	Reduce inflammation	Control inflammation	- Hydrocortisone - Prednisolone

TABLE 3 Select Veterinary Products With Reported Ototoxicity^{42,44}

TYPE	PRODUCTS
SYSTEMIC MEDICATIONS	
Antibacterials	Aminoglycosides (amikacin, gentamicin), macrolides (erythromycin, minocycline), chloramphenicol
Chemotherapy agents	Carboplatin, cisplatin, nitrogen mustard compounds
Other	Aspirin products, loop diuretics (furosemide), potassium bromide
TOPICAL MEDICATIONS	
Antibacterials	Aminoglycosides, chloramphenicol, polypeptides (bacitracin, polymyxin), tetracycline (minocycline)
Antiseptics	Quaternary ammonium compounds, ethanol, chlorhexidine, iodine compounds, acetic acid, propylene glycol
Antifungals	Amphotericin B, griseofulvin
Other	Dimethyl sulfoxide



include ruptured tympanic membrane, peripheral vestibular syndrome, facial nerve paralysis, Horner syndrome, and hearing loss.^{3,8,20}

Antimicrobial Therapy

Topical

Historically, topical antimicrobial therapy has been empirically based on cytology and tympanic membrane status.^{2,3,41,42} If tympanic membrane integrity is compromised, ototoxic medications (**TABLE 3**) should not be used.^{3,41-44} It has recently been suggested to also base therapy on antibiotic tiers, similar to those outlined in the International Society for Companion Animal Infectious Diseases guidelines for antimicrobial use for pyodermas.^{2,45,46}

A study evaluating chronic, unresponsive *Malassezia* infections found that within 4 weeks of initiating a course of glucocorticoids, performing a deep ear flush, and following up with otic antifungal ointment resolved the infections.³⁹ Common antifungals include the azoles (clotrimazole, miconazole, and posaconazole), allylamines (terbinafine), and polyene (nystatin).^{2,41} Empiric treatment failure is often a sign that a PSPP factor is unidentified or inadequately addressed or that cleaning and medication application have been improperly performed.^{3,5,8}

Systemic

Use of systemic antimicrobials for OE has been controversial and is now discouraged with the emergence of antimicrobial resistance and emphasis on antimicrobial stewardship as the likelihood of obtaining therapeutic drug concentrations within the ear lumen is low.^{2,41,47} Use of systemic antimicrobials should be reserved for patients with OM with or without otitis interna and ideally based on culture and susceptibility of material from within the tympanic bulla.^{2,20,41,46,47}

Glucocorticoids

Glucocorticoids provide the best chance for reversing chronic changes from inflammation and should be considered for OE patients with stenosis, erythema, ulcerations, or pain.^{48,49} Early use of systemic glucocorticoids is associated with better outcomes and fewer relapses of CROE.⁶ Prescribing considerations include case selection, treatment phase (induction or maintenance), formulation, potency, and route of

administration. Patients that are sensitive to glucocorticoids may experience fewer adverse effects from different formulations of triamcinolone, methylprednisolone, or dexamethasone.⁴⁹ For some patients, glucocorticoids are contraindicated and alternative therapy should be investigated, which may make treatment challenging.

TREATMENT PHASES

Induction

The goals for the induction phase are to improve patient comfort, identify and address PSPP factors, treat infection, and determine whether permanent physiologic changes are present.⁴⁸ The success of the treatment plan strongly depends on patient and client adherence.^{2,3} Patients often have otalgia and form negative associations with the ears.^{2,3,50} Reassessing pain and behavior at every visit enables the practitioner to intervene with pain medication and anxiolytics to improve patient comfort and therefore adherence to the treatment plan.

Typically, an induction dose of glucocorticoids starts at the high end of the anti-inflammatory dosing range and continues for 1 to 3 weeks.^{2,20,41,48,49} Depending on the degree of pain, it can take a few days of systemic glucocorticoids before the patient will tolerate topical ear medications, after which it is recommended to start a topical glucocorticoid for a local effect.⁴⁹ Similar to systemic glucocorticoids, the potencies of topical formulations vary (**TABLE 4**). Fluocinolone, which is in Synotic (fluocinolone acetonide 0.01% and dimethyl sulfoxide 60%; Zoetis, zoetis.com), is among the most potent glucocorticoids. Synotic also has the benefits of dimethyl sulfoxide, and together these ingredients can effectively address hyperplasia.^{20,41,48,49} Betamethasone, dexamethasone, hydrocortisone aceponate, and mometasone furoate are all potent glucocorticoids and are appropriate for use during the induction phase. Hydrocortisone aceponate and mometasone furoate are considered “soft steroids” and are less likely to cause local and systemic adverse effects.⁵¹ However, even these steroids can be systemically absorbed.^{48,49,52,53} The steroid vehicle also contributes to potency.^{3,41,49} Ointments are more potent because they form an occlusive barrier.⁴⁹ For adequate effect, recommended frequency for administration of most otic glucocorticoids is twice daily.⁴⁹

During the induction stage, ear cleaning and topical antimicrobials are started. The topical antimicrobial selection should be based on cytology results. Many commercially available otic medications contain a combination of a topical steroid, an antibacterial agent, and an antifungal agent. However, more targeted treatment is based on cytology (e.g., using a sole topical azole for *Malassezia* infections). Furthermore, an in vitro study showed that miconazole has some antistaphylococcal activity.⁵⁴ Because mechanical removal of material from the ear canal via cleaning is a valuable component of treatment, it is the authors' opinion that long-lasting topical gels be used only for patients for which at-home treatment is not possible and that lanolin-based products be avoided entirely. When used, long-lasting topical gels should be applied after thorough in-clinic cleaning and verification of an intact tympanic membrane. If otalgia precludes medicating at home, long-lasting topical gels can be considered to improve patient comfort while clients can practice veterinarian-guided behavior modification to help reset negative associations with ear interactions to enable future at-home ear treatment.

To monitor treatment response, patients with CROE should ideally be evaluated every 2 to 4 weeks until resolution of infection and abnormal otoscopic findings.⁴¹ The induction phase ends when only irreversible chronic changes remain and infection is resolved.

Maintenance

The goal for the maintenance phase is to prevent recurrence of OE. The focus should be to reduce inflammation in the ear as a noninflamed ear canal is less likely to support bacterial or yeast overgrowth.^{5,55} After the infection is treated and stenosis is improved,

systemic glucocorticoids should be tapered down to the lowest effective dose and frequency.^{48,49} PSPP factors should be reassessed to ensure that infection and stenosis remain controlled. Depending on the primary etiology, this can be a time to transition to other allergy medications. For patients with severe cases, modified cyclosporine is frequently a good option for long-term control and may prevent the need for surgery.⁵⁶

For patients with CROE, at-home maintenance ear cleaning should be performed 1 to 2 times weekly.⁸ Regular intermittent administration of the least-potent steroid possible can help decrease systemic medications needed for long-term control and prevent flares.^{48,55} In addition, topical otic solutions containing aqueous solution of aluminium triacetate (Burow's solution) or enzymatic solutions can be used a few times per week to help prevent infection return.^{48,49}

EFFECTS OF CROE

Treating CROE is associated with a significant emotional and financial burden for clients.^{2,6,38} Patients and clients experience reduced quality of life as a result of CROE, and it can also fracture the human–animal bond.^{6,38} In the authors' experience, initial encounters at the referral level for patients with CROE can be very emotionally charged, particularly when concern for end-stage otitis is raised. Recent studies showed that outcomes are worse for patients that have had CROE for more than 6 months from diagnosis to referral.^{6,38} Another benefit of early referral is availability of the special equipment for video otoscopy and deep ear flushing, which has been associated with improved patient outcomes.^{6,38,39}

When clinical signs concerning CROE are found during examination, appropriate expectations should be

TABLE 4 Potency of Topical Glucocorticoids in Veterinary Formulations, Compared With Hydrocortisone^{40,47}

POTENCY	GLUCOCORTICOID
Very potent (more than 100× hydrocortisone)	■ Fluocinolone
Potent (25×–100× hydrocortisone)	■ Betamethasone ■ Dexamethasone ■ Hydrocortisone aceponate ■ Mometasone furoate
Moderately potent (2×–25× hydrocortisone)	■ Prednisolone ■ Triamcinolone
Mildly potent (basis for comparison)	■ Hydrocortisone



explained to the client (i.e., if these changes are not reversed, they can lead to end-stage otitis). Medical management of end-stage otitis is unlikely to be successful over the long term without flares, and affected ears are a source of pain. A TECABO is considered a salvage procedure to remove the painful and infected canals.^{2,3,7,9,57} Although surgery is not free of potential complications, if surgery goes well, clients usually report that their dog is more playful and has a significantly improved demeanor, underscoring how much unaddressed pain accompanies CROE.⁷

SUMMARY

CROE is a complex, multifaceted condition with a significant emotional and physical burden to patients and clients. Understanding all the factors leading to its development is crucial for treatment success. For patients with CROE, glucocorticoids provide the best chance of reversing detrimental changes and improving patient comfort. Topical treatment of secondary infections should be based on cytology, with or without culture. Systemic antimicrobials are indicated for treatment of OMI. Chronic cases may require advanced diagnostics and procedures that are not always available in a primary care setting. When treatment for the initial stages of CROE is successful, long-term management is necessary to prevent further recurrence. Ultimately, avoiding cycles of inflammation that can lead to end-stage otitis depends on early recognition of PSPP factors and aggressive intervention. **TVP**

References

- O'Neill DG, James H, Brodbelt DC, Church DB, Pegram C. Prevalence of commonly diagnosed disorders in UK dogs under primary veterinary care: results and applications. *BMC Vet Res*. 2021;17(1):69. doi:10.1186/s12917-021-02775-3
- Nuttall T. Managing recurrent otitis externa in dogs: what have we learned and what can we do better? *JAVMA*. 2023;261(S1):S10-S22. doi:10.2460/javma.23.01.0002
- Scott D. Diseases of eyelids, claws, anal sacs, and ears. In: Scott DW, Miller WH, Griffin C, eds. *Muller & Kirk's Small Animal Dermatology*. 7th ed. Elsevier; 2001:1185-1235.
- Angus JC. Pathogenesis of otitis externa. Paper presented at: The North American Veterinary Conference; January 8-12, 2005; Orlando, Florida. Accessed July 16, 2025. <https://www.cabidigitallibrary.org/doi/pdf/10.5555/20053195518>
- Gotthelf LN. Perpetuating factors and treatment of otitis externa. In: Gotthelf LN, ed. *Small Animal Ear Diseases*. 2nd ed. Saunders; 2005:173-186.
- Logas D, Maxwell EA. Collaborative care improves treatment outcomes for dogs with chronic otitis externa: a collaborative care coalition study. *JAAHA*. 2021;57(5):212-216. doi:10.5326/JAAHA-MS-7167
- Lanz OI, Wood BC. Surgery of the ear and pinna. *Vet Clin North Am Small Anim Pract*. 2004;34(2):567-599, viii. doi:10.1016/j.cvsm.2003.10.011
- Nuttall T, Cole LK. Ear cleaning: the UK and US perspective. *Vet Dermatol*. 2004;15(2):127-136. doi:10.1111/j.1365-3164.2004.00375.x
- Doyle RS, Skelly C, Bellenger CR. Surgical management of 43 cases of chronic otitis externa in the dog. *Ir Vet J*. 2004;57(1):22-30. doi:10.1186/2046-0481-57-1-22
- Gotthelf LN. Primary causes of ear disease. In: Gotthelf LN, ed. *Small Animal Ear Diseases*. 2nd ed. Saunders; 2005:111-125.
- Gotthelf LN. Factors that predisposes the ear to otitis externa. In: Gotthelf LN, ed. *Small Animal Ear Diseases*. 2nd ed. Saunders; 2005:141-171.
- 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
- Paterson S. A review of 200 cases of otitis externa in the dog. Paper presented at: The 18th Annual Congress of the European Society of Veterinary Dermatology-European College of Veterinary Dermatology; September 26-28, 2002; Nice, France. Accessed July 16, 2025. <https://onlinelibrary.wiley.com/doi/pdf/10.1046/j.1365-3164.2003.00343.x>
- Favrot C, Steffan J, Seewald W, Picco F. A prospective study on the clinical features of chronic canine atopic dermatitis and its diagnosis. *Vet Dermatol*. 2010;21(1):23-31. doi:10.1111/j.1365-3164.2009.00758.x
- Ravens PA, Xu BJ, Vogelnest LJ. Feline atopic dermatitis: a retrospective study of 45 cases (2001-2012). *Vet Dermatol*. 2014;25(2):95-102, e27-28. doi:10.1111/vde.12109
- O'Neill 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
- Angus JC, Lichtensteiger C, Campbell KL, Schaeffer DJ. Breed variations in histopathologic features of chronic severe otitis externa in dogs: 80 cases (1995-2001). *JAVMA*. 2002;221(7):1000-1006. doi:10.2460/javma.2002.221.1000
- Lorek A, Dennis R, van Dijk J, Bannoehr J. Occult otitis media in dogs with chronic otitis externa - magnetic resonance imaging and association with otoscopic and cytological findings. *Vet Dermatol*. 2020;31(2):146-153. doi:10.1111/vde.12817
- Gotthelf LN. Diagnosis and treatment of otitis media in dogs and cats. *Vet Clin North Am Small Anim Pract*. 2004;34(2):469-487. doi:10.1016/j.cvsm.2003.10.007
- Angus JC, Campbell KL. Uses and indications for video-otoscopy in small animal practice. *Vet Clin North Am Small Anim Pract*. 2001;31(4):809-828. doi:10.1016/s0195-5616(01)50072-8
- Mason CL, Paterson S, Cripps PJ. Use of a hearing loss grading system and an owner-based hearing questionnaire to assess hearing loss in pet dogs with chronic otitis externa or otitis media. *Vet Dermatol*. 2013;24(5):512-e121. doi: 10.1111/vde.12057
- Cook LB. Neurologic evaluation of the ear. *Vet Clin North Am Small Anim Pract*. 2004;34(2):425-435, vi. doi:10.1016/j.cvsm.2003.12.001
- Heine PA. Anatomy of the ear. *Vet Clin North Am Small Anim Pract*. 2004;34(2):379-395. doi:10.1016/j.cvsm.2003.10.003
- Kumar A, Roman-Auerhahn MR. Anatomy of the canine and feline ear. In: Gotthelf LN, ed. *Small Animal Ear Diseases*. 2nd ed. Saunders; 2005:1-22.
- Paterson S, Matyskiewicz W. A study to evaluate the primary causes associated with *Pseudomonas* otitis in 60 dogs. *J Small Anim Pract*. 2018;59(4):238-242. doi:10.1111/jsap.12813
- Töpfer T, Köhler C, Rösch S, Oechtering G. Brachycephaly in French bulldogs and pugs is associated with narrow ear canals. *Vet Dermatol*. 2022;33(3):214-e60. doi:10.1111/vde.13067
- Cole LK, Kwochka KW, Kowalski JJ, Hillier A. Microbial flora and antimicrobial susceptibility patterns of isolated pathogens from the horizontal ear canal and middle ear in dogs with otitis media. *JAVMA*. 1998;212(4):534-538.
- Belmudes A, Pressanti C, Barthez PY, Castilla-Castaño E, Fabries L, Cadierques MC. Computed tomographic findings in 205 dogs with clinical signs compatible with middle ear disease: a retrospective study. *Vet Dermatol*. 2018;29(1):45-e20. doi:10.1111/vde.12503
- Angus JC. Otic cytology in health and disease. *Vet Clin North Am Small Anim Pract*. 2004;34(2):411-424. doi:10.1016/j.cvsm.2003.10.005
- Merchant SR. Microbiology of the ear of the dog and cat. In: Gotthelf LN, ed. *Small Animal Ear Diseases*. 2nd ed. Saunders; 2005:187-202.



31. Paterson S. Biofilms: their importance in veterinary medicine. *Companion Anim.* 2017;22(11):659-668. <https://doi.org/10.12968/coan.2017.22.11.659>
32. Parnell-Turner H, Griffin CE, Rosenkrantz WS, Keating MK, Bidot WA. Evaluation of the use of paired modified Wright's and periodic acid Schiff stains to identify microbial aggregates on cytological smears of dogs with microbial otitis externa and suspected biofilm. *Vet Dermatol.* 2021;32(5):448-e122. doi:10.1111/vde.13009
33. Papich MG. Measuring MIC and clinical breakpoints for veterinary antibiotics: essential to antibiotic stewardship. Paper presented at: North American Veterinary Dermatology Forum 2025; April 27-30, 2025; Orlando, Florida. Accessed September 9, 2025. <https://navdf2025.com/wp-content/uploads/2025/04/NAVDF-2025-Proceedings.pdf>
34. Robson D, Burton G, Bassett R. Correlation between topical antibiotic selection, in vitro bacterial antibiotic sensitivity and clinical response in 16 cases of canine otitis externa complicated by *Pseudomonas aeruginosa*. Abstract presented at: North American Veterinary Dermatology Forum; April 14-17, 2010; Portland, Oregon. Accessed June 13, 2025. <https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1365-3164.2010.00901.x>
35. Leonard C, Thiry D, Taminiau B, Daube G, Fontaine J. External ear canal evaluation in dogs with chronic suppurative otitis externa: comparison of direct cytology, bacterial culture and 16S amplicon profiling. *Vet Sci.* 2022;9:366.
36. Logas D. Ear-flushing techniques. In: Bonagura JD, Twedt DC, eds. *Kirk's Current Veterinary Therapy XV*. Saunders; 2021:471-474.
37. Mills PC, Ahlstrom L, Wilson WJ. Ototoxicity and tolerance assessment of a TrisEDTA and polyhexamethylene biguanide ear flush formulation in dogs. *J Vet Pharmacol Ther.* 2005;28(4):391-397. doi:10.1111/j.1365-2885.2005.00672.x
38. Dembele V, McHale M, Ortalda C, Robinson V, Jackson H. A prospective study evaluating the impact of chronic otitis on the quality-of-life of dogs and their owners and assessment of the short-term impact of specialist intervention. *Vet Dermatol.* Published online May 9, 2025. doi:10.1111/vde.13347
39. Boone JM, Bond R, Loeffler A, Ferguson EA, Hendricks A. *Malassezia* otitis unresponsive to primary care: outcome in 59 dogs. *Vet Dermatol.* 2021;32(5):441-e119. doi:10.1111/vde.12995
40. Corb E, Griffin CE, Bidot W, Hall M, Kirby A, Rosenkrantz W. Effect of ear cleaning on treatment outcome for canine otitis externa. *Vet Dermatol.* 2024;35(6):716-725. doi:10.1111/vde.13292
41. Morris DO. Medical therapy of otitis externa and otitis media. *Vet Clin North Am Small Anim Pract.* 2004;34(2):541-55, vii-viii. doi:10.1016/j.cvs.2003.10.009
42. Mendelsohn CL. Topical antimicrobials for otitis. In: Bonagura JD, Twedt DC, eds. *Kirk's Current Veterinary Therapy XV*. Saunders; 2021:462-465.
43. Podell M, Friberg C. Ototoxicity. In: Bonagura JD, Twedt DC, eds. *Kirk's Current Veterinary Therapy XV*. Saunders; 2021:468-471.
44. Gotthelf LN. Ototoxicity. In: Gotthelf LN, ed. *Small Animal Ear Diseases*. 2nd ed. Saunders; 2005:329-338.
45. Koch SN. The challenge of chronic otitis in dogs—from diagnosis to treatment. *Today's Vet Pract.* 2017;7(3):60-70.
46. Jessen LR, Damborg P, Spohr A, et al. Antibiotic Use Guidelines for Companion Animal Practice (2nd ed). Companion Animal Group, Danish Veterinary Association. 2019. Accessed May 13, 2025. https://www.ddd.dk/media/2175/assembled_final.pdf
47. Cole LK. Systemic antimicrobials for otitis. In: Bonagura JD, Twedt DC, eds. *Kirk's Current Veterinary Therapy XV*. Saunders; 2021:466-467.
48. Cole LK. The rational use of glucocorticoids in canine otitis. Paper presented at: The North American Veterinary Conference; January 15-19, 2011; Orlando, Florida. Accessed May 13, 2025. <https://www.cabidigitallibrary.org/doi/pdf/10.5555/20113161350>
49. Nuttall T. Topical and systemic glucocorticoids for otitis. In: Bonagura JD, Twedt DC, eds. *Kirk's Current Veterinary Therapy XV*. Saunders; 2021:459-462.
50. Gotthelf LN. Examination of the external ear canal. In: Gotthelf LN, ed. *Small Animal Ear Diseases*. 2nd ed. Saunders; 2005:23-40.
51. Wiedersberg S, Leopold CS, Guy RH. Bioavailability and bioequivalence of topical glucocorticoids. *Eur J Pharm Biopharm.* 2008;68(3):453-466. doi:10.1016/j.ejpb.2007.08.007
52. Reeder CJ, Griffin CE, Polissar NL, Neradilek B, Armstrong RD. Comparative adrenocortical suppression in dogs with otitis externa following topical otic administration of four different glucocorticoid-containing medications. *Vet Ther.* 2008;9(2):111-121.
53. Ghubash R, Marsella R, Kunkle G. Evaluation of adrenal function in small-breed dogs receiving otic glucocorticoids. *Vet Dermatol.* 2004;15(6):363-368. doi:10.1111/j.1365-3164.2004.00416.x
54. Weese JS, Walker M, Lowe T. In vitro miconazole susceptibility of meticillin-resistant *Staphylococcus pseudintermedius* and *Staphylococcus aureus*: miconazole and meticillin-resistant staphylococci. *Vet Dermatol.* 2012;23(5):400-e74. doi:10.1111/j.1365-3164.2012.01068.x
55. Léonard C, Taminiau B, Ngo J, Fantini O, Daube G, Fontaine J. Preventive use of a topical anti-inflammatory glucocorticoid in atopic dogs without clinical sign of otitis does not affect ear canal microbiota and mycobiota. *Vet Dermatol.* 2021;32(4):355-e98. doi:10.1111/vde.12977
56. Hall JA, Waisglass SE, Mathews KA, Tait JL. Oral cyclosporin in the treatment of end-stage ear disease: a pilot study. Abstract presented at: American Academy of Veterinary Dermatology and American College of Veterinary Dermatology Annual Meeting. April 9-13, 2003; Monterey, CA. Accessed September 9, 2025. <https://doi.org/10.1046%2Fj.1365-3164.2003.t01-1-00349.x>
57. Melman SA. Simple diagnosis and treatment of pruritic otitis. In: Gotthelf LN, ed. *Small Animal Ear Diseases*. 2nd ed. Saunders; 2005:265-274.



K. Helena Montin Mills

Dr. Montin Mills obtained her DVM degree from the University of Minnesota College of Veterinary Medicine in 2021, after which she completed a 1-year small animal rotating internship at Ethos' Veterinary Specialty Hospital - North County, San Diego, California. She then returned to the University of Minnesota in 2022 for a residency in dermatology. After residency completion in July 2025, Dr. Montin Mills joined the Animal Dermatology Clinic in Robbinsville, New Jersey, where she continues to provide compassionate care for pets.



Annette Lundberg

Dr. Lundberg obtained her DVM degree from the University of Minnesota College of Veterinary Medicine in 2019, after which she completed a rotating internship at the ASPCA Animal Hospital in New York City before returning to the University of Minnesota for a specialty internship in dermatology and then completing a residency at Auburn University. Dr. Lundberg then returned to the University of Minnesota as an assistant professor of comparative dermatology. She is passionate about improving human and animal quality of life as well as making veterinary health care accessible to all pet owners.



CONTINUING EDUCATION ■ PEER REVIEWED

EARN CE CREDIT

Chronic and Recurrent Otitis Externa



▲
**SCAN THE
QR CODE**
to take the
quiz for free
on VetFolio

This article has been submitted for **RACE approval for 1 hour of continuing education credit** and will be opened for enrollment upon approval. To receive credit, take the test at **vetfolio.com** by searching the name of the article or scanning the QR code.

- Free registration is required.
- Questions and answers online may differ from those below.
- Tests are valid for 2 years from the date of approval.

CE QUIZ QUESTIONS

1. Which is the most common primary cause of canine chronic and recurrent otitis externa?
 - a. Atopic dermatitis
 - b. Pemphigus foliaceus
 - c. Ceruminous gland adenocarcinoma
 - d. Sebaceous adenitis
2. The first detectable change of chronic inflammation in the ears during otoscopic examination is:
 - a. Ceruminous gland hyperplasia
 - b. Nodular epidermal hyperplasia
 - c. Mineralization of the external canal
 - d. Purulent exudate
3. An intact tympanic membrane rules out otitis media.
 - a. True
 - b. False
4. A patient exhibits painful erosions in the ear and yellow, sticky discharge. Cytology shows many rod bacteria and inflammatory cells. Which is most likely the infectious agent?
 - a. *Aspergillus*
 - b. *Malassezia pachydermatis*
 - c. *Staphylococcus* species
 - d. *Pseudomonas*
5. What active ingredient should be prioritized in an ear cleaner for a young golden retriever that frequently swims in a lake?
 - a. Squalene
 - b. Chlorhexidine
 - c. Acetic acid
 - d. Calcium sulfosuccinate

NAVC Veterinary Microbiome Certification

Unlock Diagnostic & Treatment Strategies for Microbiome Health

Learn how the microbiome impacts wellness, disease prevention, and treatment outcomes. Gain valuable insights to strengthen client communication and patient care.



**Get
certified
today!**

NAVC
YOUR VETERINARY CERTIFICATIONS