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Otitis in small animals – an update

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Abstract

Otitis externa, a prevalent condition in small animals (7.5–18 % dogs, 2–7% cats), arises from primary triggers such as allergies, endocrine disorders, parasites, and foreign bodies, compounded by secondary microbial overgrowth (e.g., *Staphylococcus*, *Malassezia*, *Pseudomonas*) and predisposing/perpetuating factors like conformational defects and canal stenosis. Progression leads to epithelial hyperplasia, glandular changes, tympanic rupture, and otitis media, manifesting as head shaking, malodour, and purulent discharge. Diagnosis relies on history, otoscopy, cytology, culture, and imaging (CT for bullae). Treatment emphasises thorough cleaning, topical antimicrobials/glucocorticoids, systemic therapy for severe cases (e.g., cephalexin 22 mg/kg q12h, prednisolone 1–2 mg/kg), and surgery (TECA/BO) for end-stage disease. Addressing underlying causes prevents recurrence and structural damage.

Keywords: Otitis, tympanic membrane, allergy

Introduction

Otitis in small animals means inflammation of the ear, and it is usually divided into otitis externa (outer ear canal), otitis media (middle ear), and less commonly otitis interna (inner ear).

Ear structure and why it matters

The outer ear in dogs and cats has a long L-shaped canal, with a vertical part and then a horizontal part leading to the eardrum (tympanic membrane). This shape traps moisture, wax, and debris, especially in breeds with hairy, narrow, or pendulous ears, which is why they are at higher risk of otitis.

The middle ear lies behind the eardrum inside the tympanic bulla and contains air, the small hearing bones (ossicles), and the opening of the Eustachian tube. When infection crosses the eardrum or travels through the Eustachian tube, the middle ear can fill with fluid, pus, or keratin, creating a “hidden” reservoir that keeps feeding infection back into the external canal.

The inner ear has the cochlea (hearing) and semicircular canals (balance). Disease reaching this level can cause deafness, head tilt, nystagmus, and severe balance problems, so preventing extension beyond the middle ear is critical.

Otitis externa: how the disease develops

Primary causes: what starts the fire

Primary causes are the true “triggers” that make a normal ear become inflamed.

- **Allergic skin disease:** In atopy or food allergy, the whole skin barrier is defective and inflamed, and the ear canal skin is simply a continuation of that skin.

This leads to:

1. Increased ear canal humidity and wax production
2. Microfissures in the skin that allow microbes and allergens to penetrate
3. Strong itching that makes the animal scratch and shake its head, adding trauma

- **Endocrine and keratinization disorders:** Conditions like hypothyroidism, Cushing’s disease, seborrhoea, or vitamin/zinc-responsive dermatoses alter keratin turnover and sebum production. This creates:

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1. Greasy, thickened skin with excess cerumen
 2. A rich nutrient medium for yeast and bacteria
 3. Narrowing of the canal as the lining thickens
- **Foreign bodies and parasites:** Grass seeds, hair, or dirt mechanically irritate the canal; ear mites (*Otodectes*) and other ectoparasites directly damage the skin and provoke intense itching. The local trauma plus allergic-type inflammation around mites or foreign material quickly sets up otitis.
 - **Immune-mediated, autoimmune, and drug reactions:** Conditions such as pemphigus, lupus, or erythema multiforme can preferentially affect thin skin areas like pinnae and ear canals. Topical drug reactions can similarly cause erythema, erosion, and exudation, which then is secondarily infected.

Secondary causes: what keeps feeding the fire

Secondary agents grow in an ear that is already abnormal.

- Bacteria (cocci, rods), yeasts (*Malassezia*, *Candida*), and filamentous fungi flourish in the warm, moist, inflamed canal.
- Over-cleaning or harsh products further damage the epithelium, making it easier for microbes to invade deeper.

With time, microbial flora shift:

- Early or mild cases often show mostly cocci and *Malassezia*.
- Chronic, previously treated ears frequently evolve to rod-dominated infections such as *Pseudomonas*, which are more destructive and harder to treat.

Predisposing and perpetuating factors: why some ears never fully recover

Predisposing factors make it easy for disease to start but do not cause inflammation by themselves.

- Ear conformation (long floppy pinnae, hairy canals, stenosis) reduces airflow and traps moisture and wax.
- External factors (humidity, swimming, grooming) add moisture and macerate the skin.
- Obstructions (polyps, tumors, excess tissue) create pockets where secretions stagnate and cannot be cleaned or drained.

Perpetuating factors are structural and functional changes that develop after inflammation and make the disease self-perpetuating.

- Epithelium becomes hyperplastic and hyperkeratotic, so the canal lining thickens and produces more keratin and wax.
- Glands become enlarged and inflamed (ceruminous and sebaceous hyperplasia, hidradenitis), increasing oily secretions.
- Perichondral fibrosis and calcification make the canal cartilage rigid, permanently narrowing the ear canal and making medical therapy less effective.
- The tympanic membrane thickens, loses transparency, may rupture, develop permanent holes, or form pockets that trap debris and drops (false middle ear).
- The middle ear fills with inflammatory material or keratin (cholesteatoma), becoming a chronic source of infection.

Once these changes are advanced, the ear behaves like

“end-stage” disease: even if infection is cleared temporarily, anatomy is so distorted that relapse is expected without aggressive control or surgery.

Clinical signs and discharge: interpreting what you see

Animals usually present with one or more of: head shaking, ear scratching, crying when ears are touched, foul odour, ear redness, and sometimes hearing loss.

Chronic cases may also show thickened, “cauliflower-like” ear canals and hyperpigmented pinnae.

Discharge character is a useful clinical clue:

- Dry black “coffee-ground” material strongly suggests *Otodectes* mites.
- Moist, dark brown discharge is typical of mixed *Staphylococcus*/yeast infection in allergic or seborrheic ears.
- Creamy yellow, purulent discharge suggests Gram-negative rods such as *Pseudomonas*.
- Very waxy, greasy yellow-tan exudate is commonly associated with *Malassezia* overgrowth.
- Excess, relatively clean cerumen without infection often points to chronic hypersensitivity or keratinization disorders.

A logical diagnostic work-up

History and general examination

A detailed history reveals whether otitis is acute, chronic, or recurrent, whether it is unilateral or bilateral, and whether there is pruritus or skin disease elsewhere.

Important questions include diet, seasonality, previous ear treatments, prior systemic drugs, and presence of other allergic signs (face rubbing, paw chewing, ventral dermatitis). Full physical and dermatological examination is essential because otitis is often only one manifestation of a generalized skin problem such as atopy, CAFR, or endocrine disease.

Otoscopy

Otoscopy allows visual assessment of the canal and tympanic membrane and identification of foreign bodies, polyps, masses, and the type and location of discharge.

A painful animal may require sedation or anaesthesia to examine thoroughly and safely, especially if deep cleaning or flushing is planned.

Always examine the less affected or normal ear first to avoid contamination and to understand the patient’s normal anatomy. The presence of a ruptured, thickened, or invisible tympanic membrane changes drug choice and cleaning strategy due to risk of ototoxicity.

Cytology

Cytology is the cornerstone of otitis diagnosis and monitoring.

- Smears made from the horizontal canal or deepest reachable part are stained and examined for:
 - Cocci and rods
 - Yeasts (often *Malassezia*)
 - Neutrophils and other inflammatory cells
 - Phagocytosed bacteria (indicating active infection)

Cytology should be repeated during follow-up to confirm that microbial load and type are improving in response to therapy, not just judged clinically.

Culture, histopathology, and imaging

Culture and susceptibility are especially indicated in:

- Rod-rich infections (suspected *Pseudomonas*)
- Chronic, recurrent otitis not responding to empirical therapy
- Suspected otitis media or when systemic antibiotics are planned long term

Histopathology is useful when ear canal biopsies are required to rule out tumors, immune-mediated disease, or to characterise severe proliferative changes.

Radiography or CT of the tympanic bullae is recommended when otitis media is suspected (neurologic signs, pain on jaw opening, chronic non-resolving otitis, or abnormal tympanum).

CT gives much better detail of bony changes and soft tissue densities compared with plain radiographs.

Treatment: practical step-wise approach

General principles

Successful treatment means:

1. Removing or controlling the primary cause,
2. Treating secondary infections,
3. Reducing inflammation, pain, and swelling,
4. Correcting predisposing and perpetuating factors as far as possible.

Ignoring the primary cause (for example, underlying allergy) often leads to repeated flares that gradually drive the ear towards permanent damage.

Pain management and sedation

Pain relief allows proper examination and cleaning and improves welfare.

- Topical local anaesthetics like lidocaine or proparacaine can be used for short procedures when the tympanic membrane is known to be intact.
- Systemic sedatives/anaesthetics such as xylazine, butorphanol, ketamine, and diazepam are used during deeper cleaning, flushing, or when the patient is aggressive or extremely painful.

These drugs are also used peri-operatively for surgical procedures like canal ablation or bulla osteotomy.

Ear cleaning: how and what to use

Cleaning is not a one-time event but a repeated process until the epithelium is smooth and non-inflamed and discharge is gone.

Ceruminolytic cleansers contain strong surfactants such as Ca/Na docusate (DOSS) or triethanolamine polypeptide oleate condensate that dissolve cerumen and oils.

- They are very effective but must be thoroughly flushed out and must not remain in the middle ear because they can damage inner ear structures (ototoxicity).

Mild leave-in cleansers include ingredients like propylene glycol, glycerin, light mineral oils, butylated hydroxytoluene, and antiseptics such as parachlorometaxlenol.

- They are suitable for long-term maintenance in milder or controlled chronic cases to keep ears dry and clean.

Antiseptic/drying agents such as chlorhexidine, iodine, alcohols, aluminium hydroxide, and Tris-EDTA reduce microbial load and limit maceration by drying the canal.

- Tris-EDTA chelates divalent cations and can weaken

bacterial cell walls, thereby increasing the effectiveness of many topical antibiotics.

- Aluminium hydroxide should not be used together with fluoroquinolones due to interference with drug action.

Techniques

- In-hospital cleaning often uses otoscope-guided curettage of debris, followed by irrigation and suction, sometimes under anaesthesia.
- At home, owners use instilled ear wash and gentle massage, or bulb syringing when instructed, being careful not to traumatise the canal.

Topical medications in detail

Antibacterial drops

- Aminoglycosides (e.g. gentamicin, neomycin) and fluoroquinolones are commonly first-line choices as they achieve very high local concentrations in the canal.
- When using aminoglycosides, avoid combining them with strongly acidic cleansers and avoid use if the tympanum is ruptured because of ototoxicity risk.
- Fluoroquinolones should not be combined with aluminium-containing products in the canal.
- For severe *Pseudomonas*, topical ticarcillin may be used where available.

Topical glucocorticoids

- Agents like fluocinolone, dexamethasone, betamethasone, triamcinolone, and hydrocortisone reduce itching, erythema, oedema, and exudation and can lead to sebaceous gland atrophy over time.
- By decreasing swelling, they increase canal diameter, improve air flow, and help other drugs penetrate better.

Topical antifungals

- Common preparations include 1% clotrimazole, 1% miconazole, 0.1% posaconazole, and 0.1% ketoconazole, often combined with Tris-EDTA, benzyl alcohol, or other agents in multi-component ear drops.
- Beta-thujaplicin is another antifungal/antimicrobial substance used in some formulations.

Topical parasiticides

Formulations containing piperonyl butoxide, milbemycin with thiabendazole, selamectin, or ivermectin drops are used for ectoparasites such as ear mites.

Other topical agents

Tacrolimus can be useful in allergic or *Malassezia*-associated otitis where standard therapies have failed, due to its immune-modulating effect.

Systemic therapy: when local treatment is not enough

Systemic therapy is indicated in: otitis media, severe proliferative otitis externa with more than 50% of lumen obstructed, inability to apply topical therapy, adverse reactions to topical drugs, or failure of topical therapy.

Systemic glucocorticoids

- Prednisolone 1–2 mg/kg orally once daily and triamcinolone 0.1–0.2 mg/kg once daily reduce chronic canal thickening and inflammation.
- These are often used for a short induction phase and then tapered.

- Systemic antibiotics (based on cytology and ideally culture):
- 1. Staphylococcal infections: cephalexin 22 mg/kg every 12 h or clindamycin 10 mg/kg every 12 h orally are typical choices.
- 2. Mixed infections: ormetoprim–sulfadimethoxine (55 mg/kg on day 1, then 27.5 mg/kg once daily) can be used.
- 3. Fluoroquinolones: enrofloxacin 20 mg/kg, marbofloxacin 5 mg/kg, ciprofloxacin 20 mg/kg, orbifloxacin 7.5 mg/kg, used carefully to avoid resistance, especially in *Pseudomonas* infections.

Systemic antifungals

Ketoconazole 5–10 mg/kg once daily is a typical starting drug; itraconazole or fluconazole are reserved for cases not improving or where better tissue penetration is required.

Systemic parasiticides

- Selamectin and moxidectin are effective for *Otodectes*-associated otitis.
- Ivermectin 0.2–0.6 mg/kg subcutaneously weekly for 4–8 weeks, or orally daily, treats ectoparasitic infestations such as mites and some ticks.

Long-term systemic therapy is usually needed for otitis media, often 4–8 weeks or more, and is ideally guided by culture and imaging.

Surgical management and “end-stage” ears

When medical therapy cannot control disease due to severe structural changes, surgery is considered.

- Lateral ear canal resection improves drainage and ventilation in cases where the vertical canal is still relatively healthy but lateral opening is narrowed.
- Vertical ear canal ablation removes a severely diseased vertical canal while preserving a functional horizontal canal in selected cases.
- Bulla osteotomy opens the tympanic bulla to curette out chronic infection, granulation tissue, or cholesteatoma in otitis media.
- Total ear canal ablation (TECA) with bulla osteotomy is performed for end-stage otitis externa/media when the canal is non-functional, calcified, and painful.
- Although TECA sacrifices normal canal anatomy and often reduces hearing, it can dramatically improve quality of life by eliminating chronic pain, infection, and discharge.

Conflict of Interest

Not available

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