



Abstract

Otitis media results from a multitude of factors that cause inflammation of the lining of the tympanic cavity and/or structures of the middle ear, including infections, middle ear polyps, and neoplasms. Cats with otitis media can be asymptomatic or exhibit clinical signs of otitis externa, neurologic deficits, or upper respiratory tract disease. A therapeutic challenge is presented by cats' unique, almost complete, septum bulla in the ventral cavity of the middle ear. Diagnosis of otitis media relies on visualization of material in the middle ear by otoscopy or imaging. Treatment requires management of the underlying cause as well as removal of the material in the middle ear by minimally invasive (e.g., myringotomy and bulla lavage) or surgical (e.g., ventral bulla osteotomy) techniques.



DERMATOLOGY

Diagnostic and Therapeutic Approach to Otitis Media in Cats

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Otitis media (OM) is inflammation of the structures (auditory ossicles, septum bulla) and/or modified mucosal membrane of the middle ear cavity, known as the tympanic cavity, frequently accompanied by fluid accumulation.^{1,2} The causes are multifactorial and include ascending infections associated with nasopharyngeal disease; neoplasia; inflammatory aural polyps; descending infections from the external ear canal; or, rarely, hematogenous spread.²⁻⁵ Clinical signs of OM are often missed because cats may exhibit nonspecific signs.⁶

Diagnosis requires advanced imaging in most cases or video otoscopy (VO). The middle ear is lined by a modified respiratory mucosal epithelium that comprises 3 areas⁷: the epitympanic recess, which houses the 3 auditory

ossicles; the midsection (tympanic cavity proper), which is directly adjacent to the tympanic membrane and contains the manubrium of the malleus (**FIGURE 1**); and the ventral cavity, which is the largest area and occupies the ventral portion of the tympanic bulla and contains a bony septum (septum bulla). The septum bulla is almost complete in felids, creating dorsolateral and ventromedial compartments (DLC and VMC, respectively) that communicate via 2 small openings.⁷ The unique septal anatomy of cats warrants special consideration when diagnosing and managing OM.

ETIOLOGY

In general, the etiology of OM can be classified as primary or secondary and as ascending,

Take-Home Points

- Otitis media in cats is relatively common and underreported.
- Clinical signs are not always present, but clinically affected cats may demonstrate neurologic deficits, otalgia or otorrhea, or sinonasal disease.
- Diagnosis necessitates video otoscopy or imaging to reveal material in the bulla.
- Treatment should address the root cause and management of the condition through medical or surgical intervention.



FIGURE 1. A normal-appearing tympanic membrane. The “comma” (arrowhead) represents the manubrium of the malleus, which attaches to the umbo of the pars tensa of the tympanic membrane. The tympanic membrane appears intact and transparent, affording visualization of the manubrium.

descending, or hematogenous (according to the route of infection or material migration into the middle ear) (TABLE 1). Primary OM in cats is thought to occur by auditory tube dysfunction resulting from ascending infectious or inflammatory processes, and secondary OM is thought to occur by descending (e.g., external ear disease) or hematogenous routes.⁸

In humans, auditory tube dysfunction plays a major role in the pathogenesis of OM with effusion.^{9,10} The auditory tube equalizes pressure, ventilation, and mucociliary clearance of middle ear secretions.¹¹ Disruption of these functions catalyzes the

development of OM. For humans with allergic rhinitis, chronic rhinosinusitis, or upper respiratory infection, risk for development of auditory tube dysfunction is higher.¹² A similar role is suspected to occur in cats; experimental inoculation of bacterial endotoxin and obstruction of the auditory tube has led to high rates of OM.¹³ Other studies have shown that a relatively high proportion of cats with OM have sinonasal disease (28%)³ and exhibit nasopharyngeal signs (18% to 45%).^{14,15} Although the retrospective nature of these studies does not prove a causal relationship between upper respiratory disease and OM, it is suspected that ascending routes of infection likely contribute to the pathogenesis of OM in cats.^{3,14-16} Another, rarer, cause of auditory tube dysfunction in cats is soft palate abnormalities.¹⁷

Secondary OM resulting from extension of otitis externa (OE) is believed to be less common in cats than dogs.¹⁸ The pathogenesis involves a ruptured tympanic membrane, which permits microorganisms and material to transmigrate from the external ear into the middle ear. In multiple studies, the prevalence of clinical signs or examination findings consistent with external ear disease varies greatly in cats with OM, ranging from 15% to 79%.^{2,3,6,14,15,19,20} Causes of descending OM include infectious OE, ear polyps, *Otodectes cynotis* infestation, aural masses, cholesteatomas, allergic otitis, and foreign bodies.⁸ OM acquired via the hematogenous route of infection is exceedingly rare in cats but has been reported.²¹

TABLE 1 Etiologic Factors for Otitis Media in Cats

ETIOLOGY	PATHOGENESIS
Upper respiratory disease (viral infection +/- secondary bacterial infection, chronic rhinitis)	■ Auditory tube dysfunction ■ Ascending route
Inflammatory polyp (aural, middle ear, auditory tube, nasopharynx)	■ Secondary OM (if there is a breach in the TM) or primary OM (if directly arising from the middle ear epithelium, auditory tube, or nasopharynx)
Soft palate abnormality	■ Auditory tube dysfunction
Cholesteatoma	■ Chronic inflammation and infection ■ Secondary OM (if there is a breach in the TM)
Neoplasia (auricular or extra-auricular)	■ Secondary OM (if there is a breach in the TM or destruction of the TB wall) or primary OM (if arising from the middle ear)
Hematogenous infection	■ Secondary OM via hematogenous route
External ear disease (<i>Otodectes cynotis</i> mites, allergic otitis, external ear infection, foreign body)	■ Secondary OM (descending route via a breached TM)

OM=otitis media; TB=tympanic bulla; TM=tympanic membrane



PREVALENCE

The prevalence of OM in cats is difficult to ascertain; however, in a retrospective postmortem study, 1.7% (59/3442) of cats had OM and otitis interna (OI) without ear polyps, and only 10% of those cats had neurologic signs referable to OM/OI while 29% had external ear disease.¹⁹ Later studies have found prevalence rates of OM to be higher; rates vary from 32% in cats receiving computed tomography (CT) in a veterinary hospital, 28% in cats with sinonasal disease, 10% in hoarded cats, and 28% in cats exhibiting vestibular syndrome.^{3,6,15,20} OM is likely underreported due to the absence of clinical signs in some cats and is discovered incidentally during routine imaging studies.⁶

CLINICAL SIGNS

The clinical signs of OM vary and can be neurologic, otic, and/or sinonasal (**TABLE 2**). Some cats with OM may not exhibit clinical signs and are considered asymptomatic or subclinical.^{6,19}

Neurologic Signs

Neurologic manifestations of OM and OI are attributed to damage to any of the neuroanatomic structures associated with the ear.

The ocular sympathetic trunk in cats, which is more superficial than that in dogs, predisposes cats to a higher incidence of Horner syndrome,²⁵ with clinical signs of miosis, ptosis, enophthalmos, and protrusion of the third eyelid. However, Horner syndrome is relatively uncommon in cats with OM, reported to occur in 1.7% to 33% of cats with OM.^{14,15,18-20} Absence of Horner syndrome does not preclude OM, nor does its presence indicate OM.^{14,19}

The facial nerve traverses beyond the osseous wall of the facial canal in the middle ear, rendering it particularly prone to damage from OM.²² Clinical signs are associated with facial nerve paralysis (**TABLE 2**).²² In a study of 16 cats with clinical nonsuppurative OM, 70% exhibited neurologic signs.¹⁴ In other studies, the percentage of cats with facial nerve paralysis is not reported or is reported to be absent; hence, facial nerve paralysis seems to be a rare phenomenon in cats with OM.^{18,19}

Otogenic peripheral vestibular syndrome arises from OI due to damage to the structures of the inner ear (vestibular receptor, cochlea, vestibulocochlear nerve).²² However, OI is a common sequela to extension of OM. Hence, concurrent OM and OI is typically inferred.²² The prevalence of vestibular signs in cats with OM is reported to be around 8%.^{6,19} OM and OI collectively

TABLE 2 Clinical Signs of Otitis Media in Cats

CLINICAL SIGNS	ANATOMIC STRUCTURE AFFECTED	PROPORTION REPORTED (ESTIMATES)
Subclinical disease (asymptomatic)		Unknown frequency (common to rare) ⁶
NEUROLOGIC		
Horner syndrome ²² Miosis, ptosis, enophthalmos, and protrusion of the third eyelid	Sympathetic ocular tract	Rare (1.7%–33%) ^{6,15,18,23}
Facial neuritis ²² Ipsilateral lip drooping, lowered ear carriage, widened palpebral fissure, inability to blink (provoked or spontaneous), absent abduction of the nostril during inspiration, and deviation of the nose toward the contralateral side (without OM)	Facial nerve	Rare (absent, 4%) ^{18,24}
Otitis interna, peripheral vestibular syndrome Head tilting, ataxia, and nystagmus	Inner ear (vestibulocochlear nerve, cochlea, vestibular receptors)	Rare (8%) ^{6,19}
OTIC		
Otorrhea, otalgia Ear canal erythema Otic pruritus	Middle ear or external ear canal	Variable, common (15%–79%) ^{2,14,15}
SINONASAL		
Nasopharyngeal signs	Nasopharynx, auditory tube	Common (18%–45%) ^{3,14,15}

OM = otitis media



contribute to approximately 43% of cases associated with peripheral vestibular disorders in cats,^{20,26} which positions OE as among the most common causes. In severe cases, ascending infection from the inner ear can extend intracranially, causing intracranial abscesses and meningoencephalomyelitis.¹⁸ Among the 19 cats studied, 63% did not have a history of ear infections.¹⁸

Otic Signs

Less specific signs of OM include otic pruritus, otorrhea, otalgia, erythema, and ear canal swelling. External ear disease can be the cause of OM through a descending route of infection or it can be secondary to OM with OM serving as a perpetuating factor. The prevalence of cats with OM that also exhibit clinical signs of OE is highly variable.^{6,15}

Sinonasal Signs

Bilateral OM is more commonly reported for cats with than without sinonasal disease.^{3,15} Signs of upper respiratory disease are unlikely to be directly related to OM but may suggest the influence of ascending infections from the nasopharynx in the development of bulla effusion.³

Subclinical Disease

The precise determination of the rate of asymptomatic or subclinical OM in cats is complicated by the lack of diagnostic confirmation of OM in routine veterinary practices. Reported prevalences of non-neurologic OM are 34% (34/101 cats)⁶ and 90% (53/59 cats),¹⁹ but the latter study demonstrated a higher prevalence of external ear disease (29%) and upper respiratory disease (18%). The high percentage of cats with OM that exhibit no apparent clinical signs underscores the limitations of relying solely on clinical signs for the diagnosis of OM in cats.

Other indicators of OM include proliferation and osteolysis of the tympanic and septum bullae,¹ which are best confirmed by cross-sectional or, to a lesser extent, radiographic imaging.

DIAGNOSIS

A diagnosis of OM relies on demonstrating material or fluid in the normally air-filled middle ear by use of advanced imaging or otoscopic (or VO) examination. Although otoscopy can aid with OM diagnosis,

imaging is necessary to visualize the entire tympanic cavity and structures including the VMC, which cannot be accessed with otoscopy. Suspicion for OM should be raised by peripheral vestibular syndrome, Horner syndrome, facial nerve paralysis, OE, or sinonasal disease but cannot be the sole basis for diagnosis. Bilateral OM is common even in cats in which clinical signs are unilateral; hence, both ears should always be examined carefully.^{16,19,24}

Additional diagnostic challenges are attributed to the limited access of cross-sectional imaging and experience needed for performing and interpreting otoscopic examination. If OM is suspected in a cat, then referral to a dermatologist is recommended for further evaluation and management. Indications for referral include detection of an external ear mass or polyp, suspicion of otogenic neurologic deficits (e.g., Horner syndrome, ataxia, head tilt, facial paralysis), chronic OE unresponsive to medical management, and other abnormal otoscopic findings (e.g., bulging pars flaccida).

Otoscopic Examination

The intertragic incisure, a groove sitting behind the tragus, is a useful landmark in which to insert the otoscopic cone or scope.⁷ The ear canal in cats has a sharp angle between the vertical and horizontal ear canal and is marked by a prominent cartilaginous ridge located at the upper part of the junction. The L-shaped



FIGURE 2. Suppurative effusion behind the tympanic membrane. Note the manubrium cannot be visualized.



canal makes visualization of the tympanic membrane challenging because the cartilaginous ridge often obstructs the view.⁷ The authors find it helpful to grab and lift the ear pinna to help straighten the canal and elevate the cartilage ridge, which allows better visualization of the horizontal canal and tympanic membrane. The pars tensa (tense membrane of the tympanic membrane) should normally be transparent, denoted by the comma-shaped manubrium, the arm of the malleus (**FIGURE 1**). In some patients, effusion in the dorsal aspects of the middle ear can be confirmed by otoscopic examination revealing an abnormal tympanic membrane (**FIGURE 2**). A bulged pars flaccida (flaccid membrane of the tympanic membrane, located above the pars tensa) suggests the presence of fluid or increased positive pressure in the middle ear (**FIGURE 3**).²⁷ A ruptured pars tensa of the tympanic membrane suggests the presence of OM.¹⁴

Other changes to the tympanic membrane include an opaque or gray pars tensa; however, these changes may not indicate OM in all cases and may instead be indicators of chronic inflammatory external ear disease with or without OM. Conversely, cats with OM do not always have an abnormal tympanic membrane or external ear disease. In 1 study, the tympanic membrane appeared normal in 47% (7/15) of cats with OM.¹⁸ Therefore, a normal-appearing tympanic membrane should not rule out OM. Aural polyps are commonly associated with OM and can often be easily visualized with routine otoscopic examination (**FIGURE 4**).^{2,28}



FIGURE 3. The pars flaccida of the tympanic membrane bulging out convexly. This cat had effusion in the dorsal aspect of the middle ear.

Myringotomy and Ancillary Testing

An abnormal tympanic membrane is amenable to VO-guided myringotomy under general anesthesia, which gives access to the DLC. Cytology and microbial culture and sensitivity testing of the effusion are recommended. Negative culture may represent noninfectious causes of OM as sterile effusion.²⁵ Cytologic samples should be placed on a glass slide, air-dried, preserved in fixative, and stained (e.g., modified Wright-Giemsa, Diff-Quik). The slide is then reviewed using light microscopy at various magnifications, including an oil immersion objective to identify any microorganisms (e.g., cocci, coccobacilli, bacillus-shaped bacteria, yeast). Cytologic findings should be correlated with the culture and sensitivity findings.



FIGURE 4. Aural polyp. **(A)** An aural polyp in the horizontal ear canal, preventing visualization of the tympanic membrane. **(B)** After removal of the aural polyp, a perforation is visualized in the tympanic membrane (**arrowhead**) through which the polyp had breached from the middle ear into the external ear canal. Secondary otitis media developed in this cat.



Cross-Sectional Imaging

CT and magnetic resonance imaging (MRI) are considered the gold standard for diagnosing OM, offering the highest sensitivity and specificity for detecting fluid or soft tissue material within the tympanic bulla.²⁹ An additional benefit of CT (FIGURE 5) and MRI is the ability to detect extra-auricular changes that may contribute to the pathogenesis of OM (e.g., tumors, polyps). The disadvantages are the high operating cost, lack of availability in first-opinion practices, and need for heavy sedation or general anesthesia.

Radiography

Radiography is not routinely performed for diagnosing OM due to the disadvantages of requiring heavy sedation or anesthesia, multiple radiographic projections, and superimposition of multiple complex osseous and soft tissue structures. These factors contribute to the high rates of false-negative and false-positive findings.²⁹

Ultrasonography

Bulla ultrasonography is a novel technique for detecting fluid within the VMC of the tympanic cavity (FIGURE 6). The advantages of ultrasonography are its wide availability as a quick point-of-care instrument, less invasive technique without the need for general anesthesia or heavy sedation in most patients, and relatively lower cost.²⁹ However, the operator dependence of ultrasonography makes interpretation challenging. In addition, ultrasonography cannot image

the DLC of the tympanic cavity, potentially leading to underdiagnosing some OM cases.²⁹ The accuracy of bulla ultrasonography for detecting bullous fluid in feline cadavers is 96%, which is higher than that of radiography but lower than that of CT.²⁹ The benefits of ultrasonography as a point-of-care imaging modality in cats with naturally occurring OM remain to be investigated.

TREATMENT

Treatment of OM requires identifying and managing the underlying cause, removing the material in the tympanic bulla, and addressing any infection and inflammation. Cases of suppurative bullous effusion should be treated like an abscess that requires drainage, lavage, and antimicrobial therapy. However, whether all cats with OM should be managed is in question, especially when OM is discovered incidentally in subclinical cases during routine CT, MRI, or otoscopy. Due to lack of long follow-up periods and longitudinal studies, it is unclear whether clinical signs will develop later in life in cats with subclinical OM or if the effusion will spontaneously resolve. In a study evaluating incidence of OM in cats undergoing imaging of the head, only 2 of 22 cats with subclinical OM required treatment for ear disease and none of the other cats reportedly required treatment for ear disease after a median 12-month follow-up period.⁶ The authors generally recommend pursuing management of OM for cats with clinical signs suggestive of OM or OI and/or when otoscopic or imaging findings are abnormal. Treatment of OM, whether by minimally invasive myringotomy and lavage or by surgery, should be referred to a board-certified dermatologist or surgeon, respectively, with the available resources.

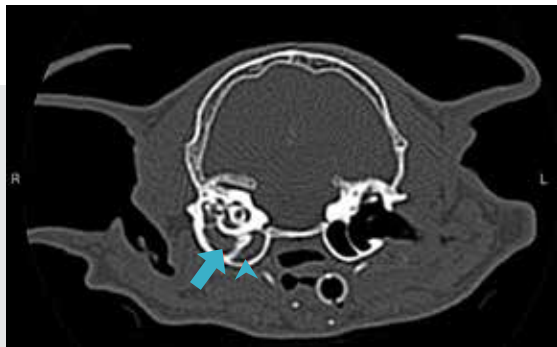


FIGURE 5. Transverse computed tomographic image (bone window) of a cat with unilateral otitis media in the right ear. The right tympanic cavity is filled with soft tissue-attenuating material in both dorsolateral (**arrow**) and ventromedial (**arrowhead**) compartments. The left middle ear is gas-filled (normal).

Myringotomy and Deep Ear Irrigation

Cats require special consideration for this procedure because the nearly complete septum bulla prevents access to the VMC via myringotomy. Only material in the DLC is amenable to lavage from the external ear canal, which presents a problem due to material accumulating in both compartments in 94% to 97% of patients compared with either compartment (DLC or VMC) alone.^{6,24} One study demonstrated that 71% (17/24) of cats with OM, of which 97% had material in both compartments, exhibited clinical recovery after a single myringotomy, bulla flushing, administration of oral steroids, and systemic antibiotics.²⁴



A novel technique involving septum fenestration under VO guidance, which has the potential to allow access and lavage of both compartments in cats with bicompartamental OM, has been reported.³⁰ Describing this procedure in detail is outside the scope of this article; further reading elsewhere is advised.

Before beginning the myringotomy and deep ear irrigation procedure, samples of the external ear canal should first be taken for cytologic assessment. Middle ear contents are aspirated for culture and cytologic evaluation. The middle ear is then gently flushed with warm saline and aspirated in multiple cycles until the returning aspirate is sufficiently clear. If cytology of the external ear sample or middle ear aspirate shows microorganisms, the authors instill a topical antibiotic, with or without a steroid, directly into the middle ear during the myringotomy procedure. Careful selection of a topical agent is required to prevent ototoxicity. Aqueous-based solutions are preferred over oil- or suspension-based topicals.^{25,31}

On the basis of cytology findings, topical ear therapy for the external ear canal for at-home use may also be recommended and have the benefit of providing residual effects in the middle ear before the tympanic

membrane fully heals. Ototoxic topicals should be avoided in patients with a ruptured or incised tympanic membrane.

Medical Therapy

Selection of a systemic antimicrobial should be based on susceptibility testing of the middle ear contents collected via the myringotomy or at the surgical site.^{14,18} The optimal duration for antibiotic administration for OM remains a subject of debate; however, 4 to 6 weeks is typical, accompanied by regular follow-up evaluations and cytologic and clinical assessments.^{18,23} The use of postoperative topical and/or systemic corticosteroids is recommended for patients with otalgia, external ear canal inflammation, or those that have undergone polyp removal. Oral prednisolone (starting at 1 to 2 mg/kg and tapered over 3 to 5 weeks) may reduce the recurrence of aural polyps,²⁸ although its success is not widely reported.³² However, caution should be exercised for protracted or repeated use of corticosteroids when signs recur in the event of otogenic empyema and meningitis; judicious use of corticosteroids in cases of central nervous system inflammation should be determined by the clinician.^{18,23}

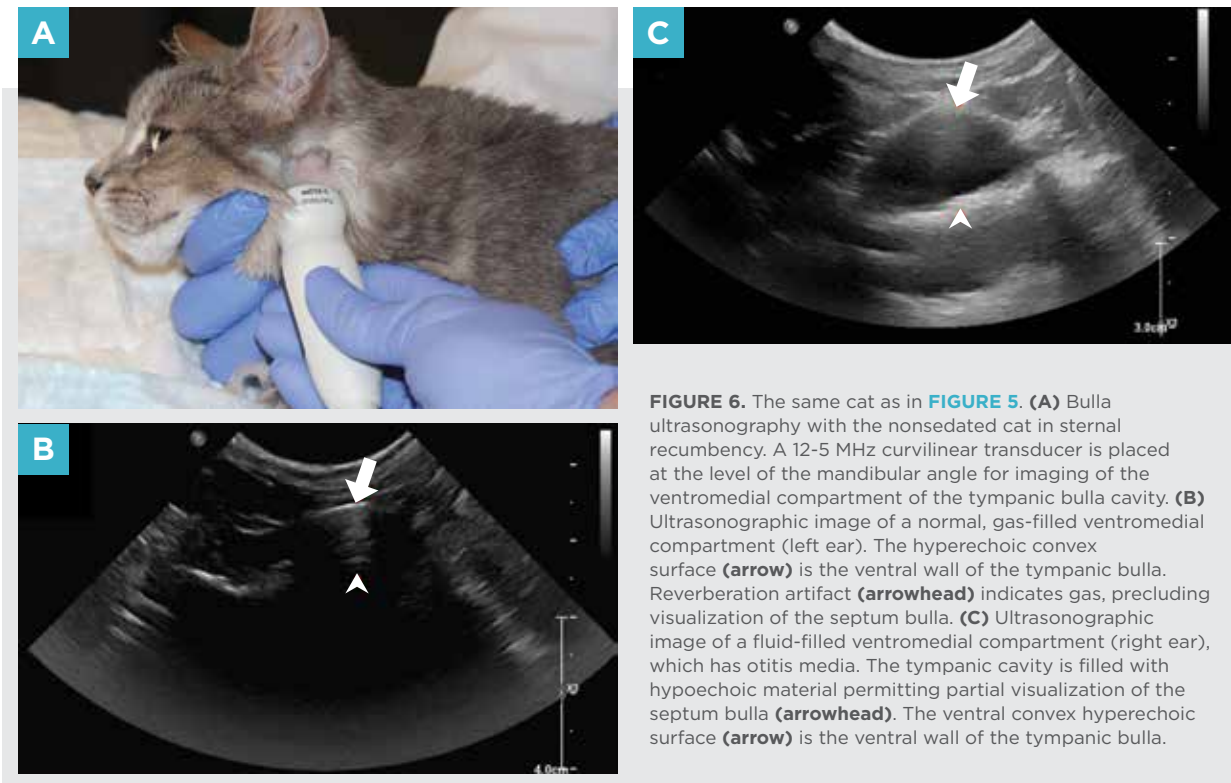


FIGURE 6. The same cat as in **FIGURE 5.** (A) Bulla ultrasonography with the nonsedated cat in sternal recumbency. A 12-5 MHz curvilinear transducer is placed at the level of the mandibular angle for imaging of the ventromedial compartment of the tympanic bulla cavity. (B) Ultrasonographic image of a normal, gas-filled ventromedial compartment (left ear). The hyperechoic convex surface (**arrow**) is the ventral wall of the tympanic bulla. Reverberation artifact (**arrowhead**) indicates gas, precluding visualization of the septum bulla. (C) Ultrasonographic image of a fluid-filled ventromedial compartment (right ear), which has otitis media. The tympanic cavity is filled with hypoechoic material permitting partial visualization of the septum bulla (**arrowhead**). The ventral convex hyperechoic surface (**arrow**) is the ventral wall of the tympanic bulla.



Surgical Treatment

Various surgical techniques exist for cases of OM not amenable to myringotomy. The benefit offered by surgical debridement over VO-guided myringotomy and lavage is that it provides access to the VMC, which cannot be accessed by the latter unless septum fenestration is performed.

This article does not cover surgical procedures in detail, and further readings elsewhere are advised. Ventral bulla osteotomy may produce a favorable outcome similar to that of medical management, although large-scale studies are lacking. In 2 studies, ventral bulla osteotomy and medical management resulted in complete resolution for approximately 80% of cats with OM in 1 study and 66% to 73% in another.^{14,18} Other surgical techniques described include per-endoscopic trans-tympanic traction removal of polyps,³³ transoral ventral tympanic bulla osteotomy,³⁴ and total ear canal ablation with bulla osteotomy.

In a study evaluating the outcome of cats with OM receiving surgical debridement via ventral bulla osteotomy, a higher risk for persistent OE was associated with surgery performed by a generalist compared to surgery done by a specialist.¹⁵ Hence, the authors recommend that any form of surgery of the middle ear be performed by an experienced, ideally board-certified, surgeon.

SUMMARY

OM is inflammation of the lining and/or structures in the middle ear, commonly associated with material accumulation and/or infection of the middle ear. The causes of OM include spreading infection from the external ear (i.e., descending) or nasopharynx or auditory tube (i.e., ascending), aural polyps, neoplasms, or cholesteatomas. Hematogenous routes of infection are exceedingly rare but reported. Clinical signs vary greatly and are absent (i.e., asymptomatic or subclinical) in a relatively high proportion of affected cats. Clinical signs may be neurologic (e.g., Horner syndrome, facial nerve paralysis, peripheral vestibular signs), otic (e.g., otalgia, otorrhea), or sinonasal. Otogenic peripheral vestibular syndrome arises from OI, which is a common sequela to OM.

Diagnosis requires demonstration of fluid or soft tissue material within the middle ear by imaging or otoscopy. Otoscopic examination is unreliable because not all cats with OM exhibit gross pathology of the tympanic

membrane and otoscopy enables visualization of only the dorsal aspects of the tympanic cavity, avoiding the ventromedial compartment. Cross-sectional imaging remains the gold standard for diagnosis. Radiographic imaging is not routinely performed due to superimposition of multiple structures, leading to an overall low predictive value. Ultrasonography of the tympanic bulla is a novel, minimally invasive technique that may be implemented, although it is less accurate than CT and further studies are needed to evaluate its benefit as a point-of-care imaging modality.

Management relies on addressing the primary cause and treatment of OM by minimally invasive management (e.g., myringotomy, bulla lavage, systemic antimicrobial and anti-inflammatory therapies) or surgery (e.g., ventral bulla osteotomy). **TVP**

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