“Amos’s Aural Dilemma”

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Introduction

The following case report will highlight the most common disease of the canine external ear canal, otitis externa.[2] Dogs of any age or breed may develop this disease, although some groups are at higher risk.[3] These patients can present with a wide variety of clinical signs including head shaking, pruritus of the ear, otic discharge, malodor, head tilt, swelling, and pain.[2,3,6] Otitis externa is defined as inflammation of the ear canal, and often leads to secondary infection with bacterial and/or yeast pathogens.[2,3] Overtime, this may lead to irreversible, pathological changes within the ear resulting in chronic, recurrent otitis externa.[2,7] This case emphasizes the importance of addressing the primary and secondary causes as well as the predisposing and perpetuating factors when managing cases of chronic otitis externa for successful resolution. This case report will review the pathophysiology, clinical signs, diagnostics, and treatment of chronic otitis externa as well as the outcome of the patient, Amos, who was seen by the Dermatology Department at Mississippi State University, College of Veterinary Medicine.

History and Presentation

Amos is an approximately 11-year-old, male neutered, mixed breed dog. He presented to Mississippi State University College of Veterinary Medicine's Dermatology Department on May 10, 2021, for chronic, recurrent otitis externa of the left ear. His owners reported a long-term history of intermittent ear infections since November 2018. They described intermittent, acute episodes which were characterized by dark brown to red, waxy aural discharge from the left ear canal, head shaking, and paw licking. Diagnostics performed prior to referral included ear cytology as well as culture and sensitivity testing. Ear cytology consistently yielded yeast and cocci bacteria. Culture and sensitivity testing had also been performed. The owner stated this

yielded susceptibility to enrofloxacin, however, the official results were not provided. Multiple medical treatments, such as Claro Otic Solution® (florfenicol, terbinafine, and mometasone furoate), TrizUltra + Keto® otic flush (USP, USP tris-EDTA, ketoconazole), Mometamax® (gentamicin sulfate, mometasone furoate monohydrate, and clotrimazole), dexamethasone injections, Apoquel® (oclacitinib), oral and injectable enrofloxacin, and medical shampoos, had been used. At the time of presentation, Amos was only receiving TrizUltra+Keto® otic flush once a day along with medicated baths using aloe and oatmeal shampoo. Response to medical treatment was reported as poor with only marginal improvement in clinical signs and persistent recurrence of the infection. A complete dermatologic history included the following. Amos lives in a single dog household in the Mississippi Delta. He spends the majority of his time indoors, however, his outdoor environment consists of flat, grassy farmland. He receives K9 Advantix® II monthly for flea and tick prevention, as well as ProHeart® 12 for heartworm prevention. His diet consists of Rachel Ray Nutrish® dog food and occasional human food. He is up to date on all core vaccinations and is apparently healthy otherwise.

On presentation, Amos was bright, alert, and responsive. He weighed 42.3 kilograms with a body condition score of 8/9. His mucous membranes were pink and moist with a capillary refill time of less than two seconds, indicating an adequate hydration status. Vital parameters included a temperature of 101.8° F, a heart rate of 120 beats per minute, and a panting respiratory rate. Cardiopulmonary auscultation was within normal limits with no murmurs, arrythmias, crackles, or wheezes noted. His abdomen was soft and non-painful on palpation. Dermatologic examination revealed scaling along the dorsum as well as multiple subcutaneous masses. There was a 2mm hard, nodular mass present on his chest, a 1mm pedunculated, pigmented mass located in the inguinal area, and a 1 mm mass on the dorsal aspect of the head.

A serosanguinous, ceruminous, malodorous discharge was present in the left external ear canal. Otoscopic examination revealed severe erythema and inflammation of the left external ear canal, as well as an aural mass which was located at the junction of the horizontal and vertical ear canal. The tympanic membrane was unable to be visualized due to obstruction of the canal. The remainder of the physical exam was within normal limits.

Diagnostic Approach

Following initial physical examination, a sample was obtained from both ears for Diff Quick staining and cytological evaluation. Microscopic evaluation revealed too numerous to count cocci bacteria, neutrophils, and inflammatory cells, indicating an active infection within the left ear. No abnormal cells were found within the right ear. Amos was then sedated, and a computed tomography (CT) study with contrast was performed. The resulting study revealed a smoothly marginated, ovoid, soft tissue and fluid attenuating, heterogeneously contrast-enhancing structure within the left horizontal ear canal. This structure measured to be approximately 8 x 16 x 7 mm in size. This structure also had a small tendril originating from the medial aspect. There was fluid attenuating material filling and expanding the remainder of the horizontal ear canal. The differential diagnoses for the described structure included inflammatory polyp, neoplasia, or inflammatory/infectious causes. The left tympanic bulla was filled with heterogenous fluid and soft tissue attenuating, mildly contrast-enhancing material. Additionally, the tympanic bulla was thickened and showed evidence of permeative lysis. These changes were likely due to otitis media or extension of external ear canal disease, however, cholesteatoma could not be entirely excluded. Evidence of mineralization of the auricular cartilages was present bilaterally indicating chronic otitis externa.

Following his CT, a biopsy sample of the otic mass was obtained through blunt dissection using alligator forceps. The sample was submitted for histopathological evaluation which revealed small, composed islands of hyperplastic squamous epithelium, degenerate neutrophils, and colonies of large round basophilic bacteria. There was no overt evidence of neoplasia seen. Therefore, the sample submitted was consistent with the diagnosis of epithelial hyperplasia likely secondary to the significant suppurative otitis with intralesional bacteria.

The combined results of the diagnostics performed on Amos lead to a tentative diagnosis of chronic unilateral otitis externa with secondary otitis media due to an aural mass, likely an inflammatory polyp, within the left horizontal ear canal. Since Amos' condition was deemed non-responsive to medical therapy due to the obstruction of the ear canal and changes present within the tympanic bulla, he was referred to MSU-CVM's Surgery Department for a surgical consultation. A blood sample was obtained, and a complete blood count (CBC) and serum chemistry were performed for pre-anesthetic evaluation. All values were within normal limits with the exception of a mild lymphopenia on his CBC. However, this parameter was likely decreased due to a stress leukogram and not of clinical concern. It was determined that Amos would be healthy enough to undergo anesthesia for surgical treatment.

Treatment and Management

Successful treatment of chronic otitis externa relies greatly on the identification and treatment of the underlying, perpetuating, and infectious causes. [1,3,7] Initial medical management is directed towards controlling the active inflammatory process.[3] Medical management involves cleaning and drying the ear as well as applying topical medications.[1] Topical combination therapies containing glucocorticoids, antibiotics, and/or antifungal agents are most commonly used.[1] However, it is important to recognize that topical therapy for the initial inflammation and

infection will not be curative unless the underlying factors and disease process is also addressed.[1] Failure to address the underlying process causing the inflammation often leads to persistent clinical signs and recurrent disease which further progresses to proliferative changes.[2,7] These proliferative changes promote the development off concurrent otitis media as well as chronic, hyperplastic, soft tissue changes.[2,7]Once the disease has reached this stage, medical therapy is often unsuccessful leading to a chronic, recurrent otitis, similar to what was seen with Amos.[2,7]

Surgical management should be recommended in cases of recurrent otitis that have failed to respond to medical therapy or in cases of proliferative growths and stenotic ear canals.[1,7] Several different surgical procedures can be performed, however, a total ear canal ablation with a lateral bulla osteotomy (TECA-BO) is the most common surgical procedure performed for treatment of chronic otitis externa.[3,5,6] Most animals with severe, chronic otitis externa also have concurrent otitis media.[5,6] Therefore, a lateral bulla osteotomy is performed in conjunction with this procedure to treat the middle ear disease component.[1,5,6] With a TECA-BO, the entire ear canal is excised and the tympanic cavity is exposed so that the exudate and secretory epithelium can be removed.[1,5,6] Due to Amos' history of failed medical therapy along with the evidence of a proliferative growth present within his left ear canal, a TECA-BO was recommended. He was started on Surolan® Otic Suspension, which contains polymyxin B sulfate, prednisolone acetate, and miconazole nitrate, to treat the active infection in his left ear in preparation for surgery.

On May 20, 2021, Amos was taken to surgery at MSU-CVM for a total ear canal ablation and bulla osteotomy (TECA-BO). Under general anesthesia, he was positioned in right lateral recumbency. The left ear was clipped and aseptically prepped with betadine scrub and draped in a sterile manner. A #10 scalpel blade was used to make an incision ventral to the tragus as well as circumferentially around the external opening of the vertical canal. The subcutaneous tissue

was dissected around the ear canal, and when the proximal ear canal was freely moveable, it was retracted and manipulated using Allis tissue forceps. Senn retractors were placed to improve visualization, and further dissection was performed until the level of the external acoustic meatus was reached. The previously described aural mass was found within the horizontal ear canal, and blunt dissection was used to remove the entirety of it. Love-Kerrison rongeurs were used to remove the ventrolateral aspect of the bulla to expose the middle ear. A culture swab was then used to obtain a sample of the middle ear exudate. Curettage was used to thoroughly remove the epithelial tissue lining from the tympanic bulla. The cavity was copiously lavaged with warm, sterile saline. Nocita® (bupivacaine liposome) was then injected into the deep and superficial tissue layers for post-operative pain management. Lastly, the deep tissues were closed using 3-0 PDS in a cruciate pattern. The subcutaneous tissue layer was closed using 3-0 Monocryl in a simple continuous pattern. The skin/cartilage of the ear was apposed using 3-0 Nylon in a simple interrupted pattern, and the skin ventral to the tragus was closed using 3-0 Monocryl in an intradermal pattern.

Post-operatively, he was maintained on hydromorphone and trazodone for pain and anxiety control. He was also receiving Optixcare® eye lube in the left eye every 4 hours to prevent the formation of a corneal ulcer in the instance of post-op facial nerve paralysis. He was pain scored and monitored closely for any signs of pain or discomfort every 4 hours. His surgical site was also evaluated for signs of dehiscence, infection, and excessive swelling. He was hand walked and offered food every 6 hours. A full physical exam was performed every 6 hours throughout the remainder of his stay.

The exudative sample obtained from the middle ear during surgery was submitted for aerobic and anaerobic culture and sensitivity. The culture showed light growth of two types of

Staphylococcus intermedius. Both *Staph spp.* showed susceptibility to amikacin, chloramphenicol, and rifampin.

The aural mass and associated ear canal was submitted for histopathology. It was noted that the exophytic mass was composed of dense, mature, fibrous connective tissue covered by hyperplastic, stratified squamous epithelium. Moderate fibrosis and hemorrhage was present. Numerous cocci bacteria were also present. No neoplastic cells were seen. The changes seen were determined to be consistent with diagnosis of chronic otitis externa and subsequent otitis media due to an inflammatory polyp within the left ear canal.

Case Outcome

Overall, Amos' surgery was uneventful, and his recovery was without complications. One day post-op, Amos was bright, alert, and responsive and was showing no overt signs of pain. It was deemed his pain had been adequately managed, and he was switched to oral pain meds, acetaminophen with codeine (Tylenol 4) and carprofen, prior to his discharge on May 19, 2021. He was to continue to receive the Tylenol 4 for ten days, the carprofen for five days, and the Trazodone for fourteen days. He was also sent home with an Elizabethan-collar which was to be kept on until his sutures were removed. Additionally, his owners were sent home with instructions to keep him on strict activity restriction for the next two weeks to allow ample time for his incision to heal. They were also advised to monitor the surgical site for swelling, discharge, pain, and redness at least twice a day.

TECA-BO procedures generally have a great prognosis as they provide increased comfort to the animals and medicating the ears is no longer needed, however, there is a potential for several post-operative complications.[1,5,6] Due to the anatomy of the ear, the most common

complication seen with this procedure is facial nerve paralysis as this nerve courses just ventral to the horizontal ear canal and tympanic bulla.[5,6] This results in an absence of the palpebral reflex, drooping of the ipsilateral ear or lip, and/or loss of parasympathetic nerve innervation to the lacrimal glands.[1,6] Fortunately, this complication can be managed by administering artificial tears or eye lubrication to prevent the animal from acquiring dry eye or developing a corneal ulcer.[5] Amos did develop these clinical signs and was sent home with Optixcare eye lube. However, this is often times a temporary complication that will resolve on its own a few week post-op.[1,5] Although, if the nerve is transected or severely stretched it may be permanent.[1,5] Superficial wound infection may also occur as a result of surgical manipulation of the infected tissue, inadequate closure of dead space, inadequate drainage, or antibiotic resistance.[1,5] Hearing loss may occur with this procedure, however, it is not always recognized as a significant complication as most of these patients likely already have some degree of hearing loss prior to surgery.[5,6] Additionally, vestibular dysfunction, chronic fistulation or abscessation, and avascular necrosis of the pinna are also potential complications that may be seen following this procedure.[1,5]

Amos presented to the MSU-CVM Surgery Department on June 2, 2021, for his 14 day post-op re-check appointment. The surgical site was determined to be healing appropriately, and his sutures were removed. His owners reported that since the procedure his energy levels were increased, however, he was unable to fully blink and was getting large amounts of food stuck in his mouth and shaking his mouth when eating. It was determined he was still suffering from facial nerve paralysis. It was also reported that approximately three days prior to his appointment, Amos began shaking his head. He was examined by his referring veterinarian and prescribed chloramphenicol based off the culture and sensitivity results. Following this

appointment, his owners were instructed to continue administering the antibiotics as prescribed by his referring veterinarian, as well as applying the Optixcare® eye lubricant as previously directed.

Pathophysiology

Otitis is defined as inflammation of the ear, however, it is classified based off the anatomical structures in which it is affecting.[1] The ear is composed of three parts: the inner, middle, and external ear.[1,4,5,6] The external ear consists of the horizontal and vertical canal as well as the external auditory meatus.[4,5,6] The horizontal and vertical canal form what is often termed as the "external ear canal."[4,5] This canal is composed of cartilaginous and bony tissue, and it extends from the opening of the external acoustic meatus to the tympanic membrane.[4,5] Inflammation of this canal as well as the surrounding structures is known as otitis externa.[1,2,3] The middle ear is formed by an air-filled chamber, known as the tympanic cavity.[1,4,5,6] The tympanic cavity is separated from the external ear by a structure known as the tympanic membrane.[1,4,5,6] Three auditory ossicles (the stapes, malleus, and incus) traverse the cavity and connect the tympanic membrane to the inner ear.[1,4,5] When otitis externa extends through the tympanic membrane, it is termed as otitis media.[1] Over half of dogs with chronic otitis externa eventually develop subsequent otitis media.[1] The inner ear is composed of a membranous and osseous labyrinth, consisting of the cochlea, vestibule, and semicircular canals which function for hearing and balance.[1,4,5] Rarely, extension of infection from otitis media into the petrosal bone may result in inflammation of the inner ear, termed otitis interna.[1]

Otitis externa is the most common disease of the canine external ear.[2] The etiology of this disease is multifactorial, and therefore, the pathophysiology may vary from patient to patient.[3,7] Otitis may occur due to primary causes, secondary causes, predisposing factors, perpetuating

factors, or a combination thereof .[2,7] Primary causes of otitis externa are processes or factors that create disease in a normal, healthy external ear canal.[3] Examples of commonly seen primary causes include, but are not limited to underlying dermatologic disease (allergies), ectoparasites, and foreign bodies.[2,3] Secondary causes induce and contribute to otitis in an abnormal ear.[3] The ears are often infected with bacteria and/or yeast pathogens.[3] *Corynebacterium* spp, *Escherichia coli, Proteus mirabilis, Psuedomonas aeruginosa, and Staphylococcus psuedintermedius* are the most common isolated pathogens.[1,2,3] However, it is important to note that in these cases these pathogens are considered opportunistic as the infection is secondary to an underlying cause of otitis.[2] Likewise, overcleaning and topical medications may also serve as secondary causes by causing inflammation that leads to changes in the physiology or anatomy of the ear.[3] Predisposing factors are present prior to the development of ear disease and increase the risk of developing otitis externa.[2,3] These are often anatomic or conformational factors such as stenotic ear canals, long pendulous ears, and excessive hair in the canal.[2,3] These factors promote retention of moisture and exudate within the ear which leads to maceration of the epithelial lining and contributes to overgrowth of yeast and bacteria.[1,3] Lastly, perpetuating factors do not initially cause the disease, but exacerbate and maintain the inflammation, even if the primary factor is eliminated.[3] They prevent resolution and promote permanent changes in the ear such as hyperplastic changes in the epithelium, otitis media, cartilage calcification, and tympanum rupture.[2,3]

To outline the pathophysiology seen with this case, it is important to recognize that any chronic obstruction to the ear canal, such as inflammatory polyps, will interfere with the normal physiologic function and result in irreversible pathological changes.[7] This further leads to chronic inflammation which results in progressive thickening of the epidermis as well as a

significant decrease in the patency of the lumen.[7] This also leads to increased secretion of cerumen and exudate within the ear canal.[7] The obstruction within the ear canal promotes accumulation of the exudate, predisposing the ear to secondary infection.[2,7] Furthermore, if the obstruction prevents release of secretions through the external acoustic meatus, the suppuration may extend and infect surrounding soft tissues leading to otitis media and/or interna.[1] Therefore, if these pathological changes are not addressed, this becomes a viscous cycle of chronic, recurrent otitis externa.[7]

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