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Case Report

Inflammatory Polyp of the Tympanic Cavity After Total Ear Canal Ablation with Lateral Bulla Osteotomy in a Dog

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Abstract

A 12-year-old dog presented with neurologic signs three years after bilateral total ear canal ablation with lateral bulla osteotomy (TECA-LBO). Based on clinical history, diagnostic imaging, and cytologic findings, cholesteatoma was suspected. Medical management was ineffective, and aggressive surgical excision was performed despite neurologic risk. However, histopathology confirmed an inflammatory polyp. This case highlights that inflammatory polyps may occur within the tympanic cavity following TECA-LBO in dogs and should be included among the differential diagnoses for tympanic cavity mass-like lesions, with consideration of this possibility during surgical planning.

Keywords: Dog, Total ear canal ablation, Lateral bulla osteotomy, Tympanic cavity, Inflammatory polyp

Introduction

Inflammatory polyps are non-neoplastic proliferative lesions arising from the epithelium of the middle ear, auditory tube, or nasopharynx, and are well recognized in cats but rare in dogs [1]. These polyps may extend into the external ear canal or nasopharynx and can cause clinical signs associated with local inflammation or mass effect [2]. Their etiology is unknown but may be associated with chronic inflammation, congenital malformations, and infection [2,3].

Total ear canal ablation with lateral bulla osteotomy (TECA-LBO) is widely performed as a salvage procedure for end-stage otitis externa or external ear canal masses [4]. Reported postoperative complications include facial nerve paralysis, Horner's syndrome, persistent discharge, para-aural abscess, fistula, and cholesteatoma [4,5]. This report describes a dog that developed an inflammatory polyp within the tympanic cavity three years after bilateral TECA-LBO. In dogs presenting with a mass-like lesion within the tympanic cavity after TECA-LBO, inflammatory polyps should also be considered among the differential diagnoses, in addition to cholesteatoma. Consideration of this possibility during diagnostic imaging, cytologic evaluation, and surgical planning may help guide clinical decision-making in similar cases.

Case History

Written informed consent was obtained from the owner prior to inclusion of the animal in this case report.

A 12-year-old female spayed Pomeranian with underlying diabetes mellitus initially presented to the internal medicine department of our hospital for episodes of generalized convulsions and nystagmus. The neurologic signs temporarily improved after dextrose supplementation at a local veterinary clinic but recurred, prompting referral to our hospital. On initial presentation, the patient showed a right-sided head tilt, bilateral vertical nystagmus, and vestibular ataxia. Vital signs were unremarkable. Serum chemistry revealed severely decreased glucose (9 mg/dL; reference interval [RI]: 65-118) and a mildly increased alkaline phosphatase (167 U/L; RI: 29-97). Blood gas analysis demonstrated a mild decrease in potassium (3.75 mmol/L; RI: 3.9-5.1). All other blood test results

were within normal limits. The marked hypoglycemia was considered the primary cause of the neurological signs and was suspected to be iatrogenic, likely related to inadequate insulin dose adjustment during diabetes management. The patient was hospitalized for blood glucose stabilization and further management, during which horizontal nystagmus was also observed.

Despite blood glucose stabilization, the vestibular signs persisted, making hypoglycemia an unlikely sole cause. Therefore, magnetic resonance imaging (MRI) was performed to further investigate the underlying cause of the neurologic signs *(Fig. 1)*. A markedly expanded right tympanic cavity containing a rim-enhancing lesion with diffusion restriction was identified on MRI, a finding that had not been detected on physical examination. The left tympanic cavity showed only mild expansion with non-enhancing material. Brain parenchyma appeared normal. The patient had undergone bilateral TECA-LBO at a local veterinary clinic three years previously for chronic otitis externa, and therefore the external ear canals were absent on imaging. Computed tomography (CT) further confirmed a contrast-enhancing soft-tissue mass occupying the right tympanic cavity with bony destruction, while the left tympanic cavity contained non-enhancing soft-tissue material *(Fig. 2)*. Based on the imaging findings, the neurologic signs were considered to be caused by the right tympanic cavity mass. The relevance of the left-sided lesion was uncertain. Fine-needle aspiration identified keratinized epithelial cells and cocci, with keratin debris accounting for approximately 20% of the sample, and bacterial culture yielding positive. Antibiotic susceptibility testing demonstrated susceptibility to minocycline, and antimicrobial therapy was adjusted accordingly. However, clinical signs did not improve despite antimicrobial therapy. Therefore, surgical intervention was considered necessary to remove the underlying cause, and the patient was referred to the surgical service. Resection of the right tympanic cavity mass was subsequently performed.

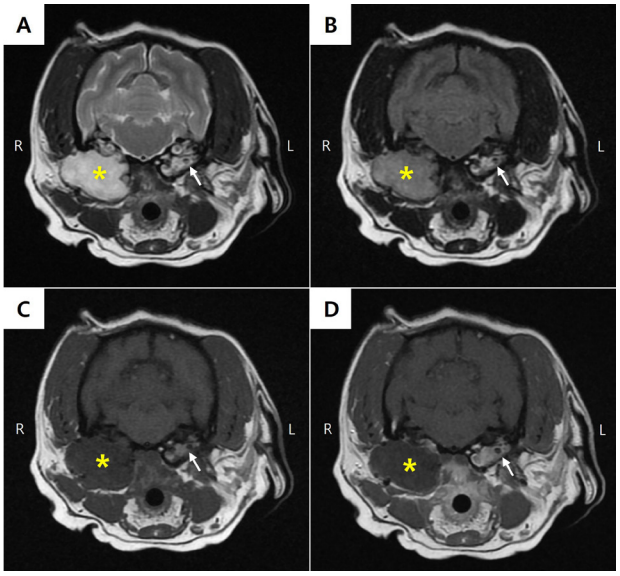

Fig 1. Magnetic resonance images of transverse T2-weighted (T2W) **(A)**, FLAIR **(B)**, and pre-contrast and post-contrast T1-weighted (T1W) sequences **(C, D)**. The right tympanic cavity (*yellow asterisks*) is markedly expanded and filled with material that appears hyperintense on T2W, iso- to hyperintense on FLAIR, and hypointense on T1W, demonstrating peripheral rim enhancement on postcontrast T1W. The left tympanic cavity (*white arrows*) is mildly expanded and contains material that appears hyperintense on T2W, hyperintense on FLAIR, and hyperintense on T1W images, without appreciable contrast enhancement

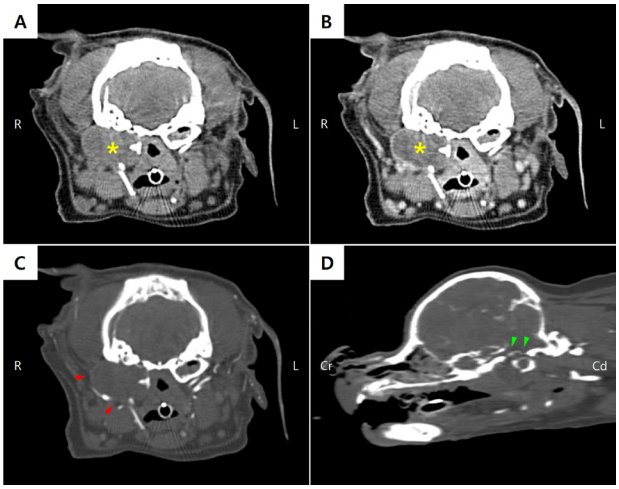

Fig 2. Transverse precontrast **(A)** and postcontrast **(B)** CT images in a soft tissue window, and precontrast CT images in a bone window in transverse **(C)** and sagittal **(D)** planes. The right tympanic cavity contains a space-occupying, expansile soft-tissue mass (*yellow asterisks*) that shows weak internal contrast enhancement with peripheral rim enhancement **(A, B)**. The ventrolateral bulla wall is absent (**C**, *red arrowheads*). Osteolysis extending to the petrous temporal bone and the ventral trigeminal canal is observed (**D**, *green arrowheads*). The left tympanic cavity contains soft-tissue attenuation material without appreciable contrast enhancement

Premedication was performed with midazolam (0.1 mg/kg IV), and anesthesia was induced with propofol (3 mg/kg IV) and maintained with isoflurane in oxygen. Intraoperative analgesia was provided by remifentanil administered as a constant rate infusion (CRI; 0-8 µg/kg/h, titrated to effect) throughout surgery.

With the patient in left lateral recumbency, a skin incision was made over the right tympanic cavity, and the subcutaneous tissues were bluntly dissected. A capsulated mass was identified and dissected using sterile cotton swabs and monopolar electrocautery. The medial aspect of the mass was adherent to the bone and lacked a distinct capsule. The mass was incised to obtain samples for bacterial culture and antimicrobial susceptibility testing, and the adherent portion was removed *(Fig. 3)*. Intraoperative bleeding was controlled with compression and a hemostatic powder. The cavity was irrigated, inflammatory debris was aspirated, and a drainage tube

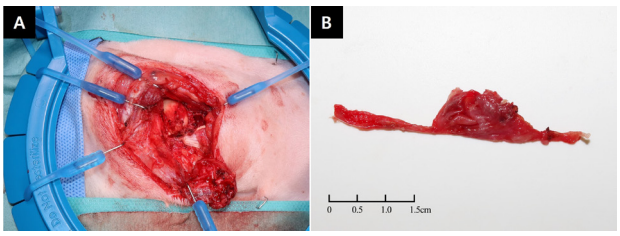

Fig 3. Intraoperative findings and gross appearance of the inflammatory polyp. **(A)** Lonestar retractors were applied to optimize surgical exposure, allowing identification of an inflammatory polyp occupying the right tympanic cavity. **(B)** The excised inflammatory polyp, which had been incised intraoperatively for culture sampling, exhibited an irregular surface owing to its detachment from the adjacent bony structures

constructed from a 19-gauge butterfly catheter and a plain tube was placed to provide active suction. The resected mass was fixed in 10% neutral buffered formalin and submitted for histopathologic evaluation (IDEXX Laboratories, Inc., USA). Examination revealed an inflammatory polyp with pleocellular otitis media, lined by stratified squamous epithelium *(Fig. 4)*.

Postoperative recovery from anesthesia was uneventful. Postoperative analgesia consisted of a continuous remifentanil infusion during the initial postoperative period, followed by transition to a fentanyl transdermal patch (1.66 µg/kg/h), with the patient discharged on postoperative day 3. The drain produced approximately 1 mL of fluid within the first 3 hours after surgery and was subsequently removed as no further drainage was observed. The surgical site healed without complications, and the suture removal was performed 14 days after surgery. Based on the results of bacterial culture and antimicrobial susceptibility testing, the postoperative antibiotic was changed to ciprofloxacin in consultation with the internal medicine team, initiated 1 week after surgery and continued for one month. Facial nerve paralysis was noted immediately after surgery, characterized by right-sided lagophthalmos, loss of the palpebral reflex and drooling. Right-sided head tilt persisted postoperatively, and bilateral horizontal nystagmus was intermittently observed with excitement. The patient received ocular supportive care with hyaluronic acid eye drops and lubricant ointment, but corneal edema developed 20 days postoperatively, for which a bandage contact lens was applied. With continued ocular management, the cornea remained healthy, although facial nerve dysfunction persisted throughout the 5-month follow-up period. The owner was informed of the need for continued ocular care and has been compliant with the recommended management.

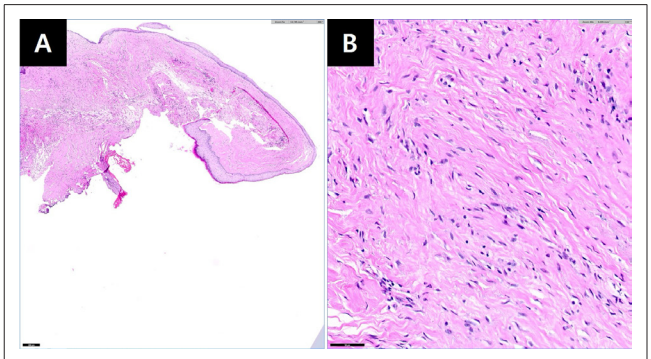

Fig 4. Histopathologic features of the inflammatory polyp. **(A)** The polyp is lined by well differentiated keratinizing stratified squamous epithelium, and mixed inflammatory infiltrates consistent with pleocellular otitis media are present (Hematoxylin and eosin stain, Magnification ×50, Scale bar 200µm). **(B)** The collagenous stroma of the polyp is observed (Hematoxylin and eosin stain, Magnification ×400, Scale bar 50 µm)

Discussion

Incomplete removal of epithelial remnants during TECA-LBO may allow these cells to persist within the tympanic cavity and, under chronic inflammation, proliferate and lead to complications including persistent infection, para-aural fistula formation or cholesteatoma [5-7]. In the present case, the patient underwent bilateral TECA-LBO three years previously for chronic otitis externa, and a soft tissue lesion was identified within the right tympanic cavity. These findings, together with the absence of brain parenchymal abnormalities on MRI, suggest that the observed vestibular signs were most likely associated with peripheral vestibular dysfunction secondary to the tympanic cavity lesion [8,9]. Based on previous reports of cholesteatoma as a postoperative complication of TECA-LBO [6], the lesion was presumed to be a cholesteatoma and surgical intervention was performed. However, histopathologic examination confirmed an inflammatory polyp lined by stratified squamous epithelium. This finding indicates that inflammatory polyps can occur within the tympanic cavity after TECA-LBO and may mimic cholesteatoma clinically.

Inflammatory polyps and cholesteatomas are non-neoplastic mass lesions that may arise within the canine middle ear [1,5,10]. Inflammatory polyps can be treated by traction-avulsion (TA) or TECA-LBO, and some authors advocate a conservative surgical approach, with TA as an initial treatment and TECA-LBO reserved for recurrent cases [1]. Cholesteatomas are characterized by the accumulation of keratinizing stratified squamous epithelium within the tympanic cavity, resulting in progressive bulla enlargement, osteolysis, and potential involvement of adjacent neural structures with subsequent neurologic dysfunction [11]. Consequently, aggressive surgical excision is recommended, which may require thorough curettage and removal of affected bone using rongeurs or a high-speed burr [5,6]. Inflammatory polyps have a relatively low recurrence rate of approximately 8%, whereas recurrence rates of up to 50% have been

reported for cholesteatomas [1,6]. Therefore, preoperative differentiation of middle ear masses is critical for surgical planning and prognosis, and imaging and cytologic findings should be carefully considered.

On CT imaging, both lesions may appear as soft-tissue attenuation masses with tympanic bulla osteolysis, making differentiation challenging [3,12]. However, contrast-enhancement patterns differ, with inflammatory polyps typically showing intralesional enhancement whereas cholesteatomas composed of avascular keratin debris demonstrate peripheral rim enhancement without intralesional enhancement [3,12]. Reports describing MRI features of inflammatory polyps are limited, and the MRI characteristics of cholesteatomas are not yet well established [1,3,13]. Although cytological evaluation may be non-specific, as mixed inflammatory debris, bacteria, and keratinizing squamous epithelial cells can be observed in both lesions, recent literature suggests that a cytological diagnosis of cholesteatoma is supported only when keratin debris comprises 80-100% of the sample [3,6,13]. Application of these criteria to the present case allows an interpretation different from the preoperative presumptive diagnosis.

Beyond these two non-neoplastic lesions, neoplasia should also be considered in the differential diagnosis of a mass-like lesion within the tympanic cavity [14]. Primary middle ear tumors are extremely rare in dogs, and squamous cell carcinoma is the most commonly reported tumor type [10,14]. In addition, neoplasia may extend medially from residual external ear canal epithelium [14]. A recent CT-based study reported that intralesional mineralization, intralesional fluid/debris, and regional lymphadenopathy were common CT findings in canine ear canal tumors [10]. Cytologic evaluation may also provide supportive evidence for neoplasia when neoplastic cells are identified [14,15]. However, none of these CT findings were identified in the present case, and cytology did not reveal neoplastic cells, suggesting that neoplasia may be less likely.

In the present case, the intralesional enhancement on CT and the low proportion of keratin debris on cytology indicated an inflammatory polyp should have been strongly considered as a differential diagnosis [3,13].

Nevertheless, because cholesteatoma has been described as a postoperative complication of TECA-LBO [6], the lesion was presumed to be a cholesteatoma and surgery was performed. Consequently, more aggressive dissection and excision were performed [5,6], resulting in postoperative facial nerve paralysis. Had an inflammatory polyp been included in the preoperative differential diagnosis, a more conservative initial excision followed by histopathologic evaluation, with subsequent staged surgical decision-making, might have been considered.

This case suggests that inflammatory polyps should be considered an important differential diagnosis in addition to cholesteatoma in dogs presenting with a mass-like lesion within the tympanic cavity after TECA-LBO. In particular, when intralesional contrast enhancement is observed on CT and the proportion of keratin debris on cytology is low, a surgical approach that accounts for the possibility of an inflammatory polyp may be considered to help reduce the risk of complications associated with aggressive excision. Furthermore, these findings underscore the importance of thorough removal of epithelial remnants during TECA-LBO. Overall, awareness that inflammatory polyps may occur within the tympanic cavity after TECA-LBO may provide practical guidance for clinicians when making surgical and therapeutic decisions in similar cases.

Declarations

Availability of Data and Materials: The datasets analyzed during the study are available from the corresponding author (S. Lee) on request.

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Authors' Contributions: Conceptualization was done by SJ, YK and SL, data curation was done by SJ and YK, visualization was done by SJ and YK, supervision was done by SL, and the article was written by SJ, YK and SL.

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