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Otomycosis due to *Aspergillus* spp. in a dog: case report and literature review

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Editor's Note
Contemporaneously with the preparation for publication of this paper, another case report of canine *Aspergillus* otitis was published (Ghibaudo G, Peano A. Chronic monolateral otomycosis in a dog caused by *Aspergillus ochraceus*. Vet Dermatol, 21:5; 2010), and therefore regrettably could not be cited in the literature review.

Abstract

This report describes the clinical findings, clinicopathology and treatment of otomycosis caused by *Aspergillus* spp. in an atopic dog affected by chronic unilateral purulent otitis externa unresponsive to topical and oral antibiotics and antifungal treatments. Cytology of otic exudate revealed neutrophils and septate fungal hyphae, and otic culture grew *Aspergillus* spp. and no bacteria. Treatments included allergen-specific immunotherapy, topical and oral antifungal therapy and anti-inflammatory steroid therapy. Final resolution occurred after treatment of the underlying hypersensitivity disorder, administration of topical ketoconazole and debridement of infectious ear exudate. Otomycosis due to filamentous fungi may, as in humans, occur in dogs with ear canals compromised by pre-existing allergic or bacterial otitis, and possibly previous antibiotic therapy. Antifungal medications provided clinical improvement, but the key to successful treatment was the restoration of the normal physiology of the external auditory canal.

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Introduction

In dogs, otitis externa is a common multifactorial inflammatory disorder. Primary causes of otitis include atopy, food allergy, contact hypersensitivity to topical ear medications, otic tumours or foreign bodies, otic parasites, keratinization or glandular disorders, immune-mediated disease and fungal infections with dermatophytes or *Sporothrix schenkii*.[1] Secondary infections with bacteria or *Malassezia pachydermatis* commonly complicate treatment. Otomycosis, often due to *Aspergillus* spp., is a common complication in humans post-operatively and after chronic antibiotic therapy,[2–20] and has also been described in companion animals in a few publications; however, treatment of canine otomycosis has not been documented.[21–26] This case report describes the clinical findings, clinicopathology and treatment of otomycosis due to *Aspergillus* spp. that complicated atopic otitis in a dog.

Case report

A 3.5-year-old spayed female 36-kg golden retriever was presented with a 1-year history of chronic right otitis that had been diagnosed cytologically as yeast and bacterial infections and treated unsuccessfully with multiple topical ear medications, several courses of oral ketoconazole, two courses of oral marbofloxacin (Zeniquin®; Pfizer, New York, NY, USA), multiple injections of triamcinolone (Vetalog®; Fort Dodge, Animal Health, Overland Park, KS, USA; last dose 9 months prior to referral) and a hypoallergenic diet (Hill's Science Diet z/d Ultra, Hill's Pet Nutrition Inc., Topeka, KS, USA). Eight months (240 days) prior to referral, video-otoscopy identified a ruptured right tympanum and otic culture revealed *Xanthomonas maltophila* and *Aspergillus glaucus* (labelled as 'flush', but with no indication of whether the source was the outer or middle ear). This led to treatment with ketoconazole at 5.5 mg/kg orally twice daily for 6 weeks, marbofloxacin (Zeniquin®; Pfizer) at 2.75 mg/kg orally twice daily for 7 days and topical neomycin–thiabendazole–dexamethasone eardrops (Tresaderm®; Merial Ltd., Duluth, GA, USA). Two months prior to referral, the dog was again treated with ketoconazole 5.5 mg/kg orally twice daily for 15 days, marbofloxacin (Zeniquin®; Pfizer) 2.75 mg/kg orally once daily for 10 days, and topical neomycin–thiabendazole–dexamethasone eardrops (Tresaderm®; Merial). Otic cytology at that visit indicated an absence of organisms, but the otitis persisted and the dog was then referred.

On initial examination (day 0), the dog was receiving only a prescription hypoallergenic diet (Hill's Science Diet z/d Ultra) and an oral glucosamine–chondroitin supplement. Both outer ears were mildly inflamed, with mild external exudation. Interdigital and perivulvar erythema with brownish hair discolouration were observed. External otic cytology revealed scattered cocci and rods in both ears, and perivulvar cytology showed a mild yeast overgrowth with occasional rod-shaped bacteria. Otoscopic examination under sedation demonstrated scant debris and a thickened tympanum in the left ear canal; in the right ear dark debris occluded the horizontal canal and

the tympanum was ruptured. Cytology of debris from the right bulla showed neutrophils, cerumen and scattered *Malassezia* but no bacteria. Intradermal testing revealed multiple reactions, and allergen-specific immunotherapy (ASIT) was prescribed in conjunction with generic ciprofloxacin at 15 mg/kg orally once daily for 4 weeks (to combat the potential for bacteria found in the external ears to descend into the right middle ear via the ruptured tympanum), generic ketoconazole (Taro Pharmaceuticals, Hawthorne, NY, USA) at 5.5 mg/kg orally once daily for 6 weeks for *Malassezia* otitis and dermatitis, and a tapering anti-inflammatory course of generic methylprednisolone (Qualitest Pharmaceuticals, Huntsville, AL, USA; 0.22 mg/kg orally twice daily for 5 days, then 0.22 mg/kg once daily for 5 days, then 0.22 mg/kg every other day for 10 days) to reduce ear canal inflammation. The ear canal and bulla were flushed to remove debris while the dog was sedated for intradermal testing, and EpiOtic® (Virbac US, Fort Worth, TX, USA) was prescribed twice weekly to clean the ears. The Hill's z/d Ultra hypoallergenic diet was replaced with Royal Canin rabbit/potato formula and the glucosamine–chrondroitin supplement was discontinued.

One month later (day 30) the left ear was erythematous, contained scant cerumen and the tympanum was now normal; cytology showed cerumen and rare *Malassezia*. The left ear remained unchanged thereafter for the duration of treatment. The right ear was inflamed and full of dark brown exudate which obscured the tympanum. Right otic cytology showed scattered neutrophils, rare *Malassezia*, no bacteria and scattered filamentous fungal hyphae. Persistent otomycosis due to previously cultured *Aspergillus* was suspected; the owner did not permit ear culture. Owing to persistence of fungal organisms on cytology despite ketoconazole treatment, antifungal therapy was changed to compounded itraconazole at 5 mg/kg orally once daily for 6 weeks [Itraconazole powder; CBS Chemicals (China; validated by Analytical Research Labs, Oklahoma City, OK, USA); compounded into 110 mg capsules by Roadrunner compounding pharmacy, Phoenix, AZ, USA] and topical gentamicin–clotrimazole–betamethasone (Otomax®; Schering-Plough, Union, NJ, USA). The ear canal was again flushed to remove debris, and twice weekly ear cleaning and the ASIT injections were continued. The rabbit/potato diet was continued for two more weeks; then the owner was instructed to challenge the hypoallergenic diet trial by reintroducing a previously fed commercial maintenance dog food, and to monitor the dog for flare of otic or pedal inflammation.

Four weeks later (day 60) the owner reported no flare of pedal pruritus with diet challenge, but flatulence was increased and beef ingestion caused increased ear inflammation. The right external ear was 50% improved, with less inflammation and exudate; cytology showed neutrophils, inflammatory debris, only rare fungal hyphae and no bacteria. Otoscopic examination of the right ear under sedation showed a moderate amount of tan exudate, which obscured the tympanum, but ear flushing revealed that it was ruptured with visible bulla. The bulla was flushed to remove exudate, and the dog was discharged on continued itraconazole for 4 weeks, topical gentamicin–clotrimazole–betamethasone (Otomax®; Schering-Plough), weekly ear cleaning and continuation of ASIT. An over-the-counter fish- or duck-based hypoallergenic diet was recommended to reduce flatulence and eliminate exacerbation of signs due to beef exposure.

One month later (day 90) video-otoscopy established that the right ear canal was still full of grey debris, which when flushed revealed friable and eroded canal walls and an intact but thickened and abnormal tympanum. Cytology of the right ear revealed no bacteria but neutrophils and fungal hyphae, established as *Aspergillus versicolor* on fungal culture. Antifungal therapy was changed to generic fluconazole (Glenmark Generics, Mahwah, NJ, USA) at 4.2 mg/kg orally twice daily for 6 weeks, and the ASIT and twice weekly ear cleaning were continued.

Four weeks later (day 120), the right ear was less inflamed and contained considerably less, drier otic debris. Otic cytology showed epithelial cells and rare cocci, but no neutrophils or fungal organisms. Fluconazole was continued for 3 months, and an ear cleaner with ketoconazole (T8 Keto flush®; DVM Pharmaceuticals, Miami, FL, USA) was prescribed. The ASIT was continued, as was the fish-based hypoallergenic diet, which the owner felt was providing acceptable control of flatulence.

Eight weeks later (day 180) the right outer ear was erythematous and the horizontal canal was full of dark brown debris that obscured the tympanum. Cytology showed epithelial cells and cerumen but no organisms, and repeat bacterial and fungal cultures were negative. As much debris as possible was flushed out without sedation, and more frequent at-home cleaning to reduce debris was recommended.

Four weeks later (day 210), less exudate was present in the right horizontal canal; however, a ceruminolith was now noted at the level of the tympanum. This was removed under sedation using alligator forceps because vigorous flushing failed to displace it. The ceruminolith was palpably adhered to the tympanum and when removed revealed an intact but thickened underlying tympanum. Cytology of the ceruminolith revealed ceruminous debris and fungal hyphae similar to those observed previously; no bacteria were observed. In addition to daily use of T8 Keto flush® (DVM Pharmaceuticals), topical fluocinolone acetonide 0.01% and dimethyl sulfoxide 60% (Synotic®; Fort Dodge) were prescribed to reduce canal inflammation post ear flush. The ASIT and hypoallergenic diet were continued. Ten days later, on otoscopic examination, there was no exudate or recurrence of ceruminolith in the right ear and only a small amount of cerumen at the tympanum, which was no longer thickened.

For a year after resolution of the *Aspergillus* otitis, both ears were otoscopically examined and cleaned every 2–4 weeks because the dog was too fractious for effective at-home ear cleaning. The left ear canal and tympanum were always normal, and the right horizontal canal walls were invariably lined with dry circular flakes of tan epithelial debris that were mildly adherent but easily removed with light ear flushing, suggestive of a defect in epithelial clearance. The right tympanum was consistently normal and translucent, and cytological examinations revealed only epithelial cells and cerumen, with no

inflammatory cells or infectious organisms. The dog was maintained on ASIT injections, with intermittent use of fluocinolone acetonide 0.01% and dimethyl sulfoxide 60% (Synotic®; Fort Dodge) eardrops, a hypoallergenic diet, and ear cleaning once or twice a month with T8 Keto flush® (DVM Pharmaceuticals). Repeat fungal culture for a study on otic fungal flora in dogs was performed recently on both ears (day 670), after 2 weeks withdrawal of topical therapy. Otic cytology revealed only rare cocci bacteria and rare *Malassezia* in the right ear; fungal culture of a left ear swab grew *Sporidiobolus johnsonnii*, while the right ear sample yielded no growth.

Discussion

Otomycosis accounts for 9–50% of cases of otitis externa in humans; associated fungal organisms may be superimposed upon prior bacterial otitis, or act as primary pathogens.[2,3,5,12,17] Development of otomycosis is prevented by an intact ear canal surface and glandular secretions that maintain the protective keratin layer by normalizing hydration and providing an acidic, fungistatic environment. Also, the constant external migration of the lining of the external ear canal aids the removal of microbes and allows replacement of damaged epithelium with healthy new tissue.[4] Alteration of these barriers to infection or the metabolic equilibrium of the microbial flora in the external ear canal enables colonizing fungal organisms to proliferate.[4] Factors that promote such proliferation include changes in the epithelial lining (dermatological diseases, microtrauma due to bacterial infection or topical use of irritating substances), increased pH of the external ear canal, altered cerumen composition, systemic factors (alterations in immunity, debilitating diseases, corticosteroids, antibiotics and neoplasia) and environmental factors (heat and excessive humidity).[2,4,5,14,15] Although there has been controversy regarding whether the fungi are true infective agents or merely colonization species as a result of compromised local host immunity secondary to bacterial infection, most clinical and laboratory evidence to date supports the view that otomycosis is a true pathological condition, with *Candida* and *Aspergillus* spp. as the most common fungal species isolated.[2]

Aspergillus spp. are ubiquitous environmental saprophytic filamentous fungi that live and grow on soil and organic debris. They produce numerous small airborne conidia, which land on most inanimate and animate objects,[27] and thus isolation of *Aspergillus* spp. from the skin and hair of normal mammals represents transient contamination in most cases. However, *Aspergillus* spp. can produce opportunistic infections by invading mucosal or cutaneous surfaces in compromised individuals.[1,27] Positive fungal culture without either histological or cytological evidence of associated inflammation can be misleading.[27]

The clinical diagnosis of otomycosis in humans is made from clinical signs, the character of otic exudate, identification of fungal organisms (otoscopically or by cytology), exclusion of bacterial infection, lack of clinical response to antibiotic therapy and a positive response to antifungal treatment.[2,12] Some authors believe that fungal culture is rarely needed and does not alter clinical management, the most important therapeutic strategy being selection of a specific treatment based on the efficacy and characteristics of the drug regardless of the causal agent.[2,12,13] Others consider it important to culture and identify the causative fungal agent and select an appropriate antimycotic treatment on the basis of susceptibility results.[3,19] *Aspergillus* is a well-recognized causative factor in human fungal otitis (9–98% of cases); most commonly *Aspergillus niger*, although *Aspergillus flavus, Aspergillus fumigatus* and *Aspergillus terreus* have also been isolated.[2–20] An increase in the incidence of fungal otitis in the period after widespread use of ofloxacin has been reported, and suggested pathogenetic explanations include elimination of the competing normal bacterial flora and increased ear canal pH; *Aspergillus* grows optimally at pH 6, and the pH of topical ofloxacin is 7, compared with a pH of 3–4 in the normal human ear canal.[4,15]

The most common symptoms of human otomycosis are pruritus, sensation of fullness in the ear, pain, otic discharge (which can be scant exudate, a light to dark dry ceruminous plug or moist, thick fibrinous exudate), ear canal swelling and erythema, and in cases of fungal otitis media, tinnitus and diminished hearing.[2,3,5,7,11] *Aspergillus fumigatus* infections cause a fluffy white discharge, whereas *A. niger* produces black colonies.[4] In fungal otitis media, the tympanum is usually opaque, sometimes with central or multiple defects and middle ear lesions, such as polyps, granulation tissue or cholesteatoma; osteomyelitis of the mastoid process or temporal bone may also be seen.[5,6]

Treatment of human otomycosis involves a combination of daily cleansing/debridement of the infectious exudate with topically administered antifungal medications. According to one author, the use of antimycotic agents alone is generally insufficient to achieve complete recovery, and treatment must also be directed towards restoring the physiology of the external ear canal.[14] Topical medications described for treatment of otomycosis include clotrimazole, ketoconazole, fluconazole, itraconazole, miconazole, econazole, natamycin, terbinafine, acetic acid, boric acid, tolnaftate, amphotericin B, ciclopiroxolamine, thimerosol, 5-fluorocytosine, 1% mercurochrome, Gentian violet, cresylate and pigmentum Castellani.[2–7,14,16,19] A review of 18 studies found that topical clotrimazole was the most widely used agent, with a cure rate of 95–100%.[3] Other azole antifungals, such as 2% ketoconazole cream, 1–4% fluconazole suspension and 2% miconazole cream, were also effective in 90% of cases.[3,5] Topical antifungal products that are not considered to be potentially ototoxic and have been recommended for human use include clotrimazole, fluconazole, ketoconazole, econazole and miconazole.[3,4,19,29] Conversely, topical antifungal products that have been found to be ototoxic include acetic acid, boric acid, cresylate and Gentian violet.[2–4,29] Oral azole antifungal medications are sometimes added for immunosuppressed patients and for cases of fungal otitis externa and otitis media refractory to topical medications alone, and in one case report, oral and topical terbinafine were used successfully to treat a case of external otomycosis due to *Aspergillus versicolor*.[5,10,18,20,28] A review of *in vitro* susceptibilities of 120 cases of human otomycosis associated with *Aspergil-*

lus spp. revealed all isolates to be susceptible to both voriconazole and amphotericin B, and nine to be resistant to itraconazole.[8]

In dogs, *Aspergillus*, most commonly *A. fumigatus*, causes infection of the nasal cavity and sinuses, and can disseminate to cause systemic disease (especially in immunocompromised animals); *A. terreus* is the most common species isolated from disseminated fungal infections.[1,27] Treatment of *Aspergillus* nasal infections with oral ketoconazole, itraconazole or fluconazole has a response rate of only 43–70%, and topical intranasal administration of enilconazole or clotrimazole is recommended as being more effective.[27,30] Additionally, surgical removal of nasal or sinus fungal granulomas or mycetomas may be necessary.[30] Although systemic antifungal treatment with itraconazole and amphotericin B has been effective in some dogs with disseminated aspergillosis, the prognosis is typically poor.[27] Newer triazole antifungal drugs, such as voriconazole, posaconazole and ravuconazole, have better activity against *Aspergillus* spp. but are rarely utilized in veterinary medicine owing to cost.[8,27,30–32] Other antifungal medications, such as terbinafine and caspofungin, have not been well studied in dogs for treatment of aspergillosis, although the latter has shown efficacy in treatment of invasive aspergillosis in humans.[27,33]

In dogs, cutaneous infections with *Aspergillus* are most often associated with disseminated infection and can manifest as nodules, abscesses, draining tracts, blepharitis and oral ulcers; these lesions have been rarely described in otherwise healthy dogs.[1] *Aspergillus* as a factor in the development of canine otitis has been poorly characterized. *Aspergillus* spp. were isolated from 0.8–1.1% of canine ears affected with otitis externa but not from normal ears, and have been cultured from 1.4% of 279 normal 'clean' ears, 1.2% of 84 'waxy' ears, and 0.7% of 115 ears clinically affected by otitis; associated cytology was not described.[1,21] *Aspergillus* spp. were cultured from a high percentage of 200 normal dog ears (9.5% *A. niger*, 7.5% *A. flavus*, 15% *A. fumigatus* and 4.5% *A. nidulans*), but none was visible cytologically.[22] *Aspergillus* spp. was isolated from 28% of 50 ears affected by otitis (26% *A. niger* and 2% *A. flavus*), accompanied in most cases by pathogenic bacteria and/or *Malassezia* yeast, but in three cases *A. niger* was the only organism cultured; in two of these cases, mycelial organisms were also visible on cytology.[22] It was concluded that *A. niger* cannot infect normal ear canals but can colonize those infected with bacteria or other fungi or yeasts.[22] In a large survey of canine otitis externa in Israel, 4.6% of 8750 dogs sampled over a 3-year period exhibited clinical otitis, and *Aspergillus* spp. were cultured in 0.7% (three cases). In one case, fungal organisms consistent with *Aspergillus* were also found on cytology of the affected ear.[23] Kumar *et al.*[24] cultured *Aspergillus* spp. in 7% of 99 dogs with healthy ears and 9% of 101 dogs with otitis externa; associated cytology was not described. In a report from Venezuela, *Aspergillus* was cultured from 7.5% of 53 dogs with acute or chronic otitis (3.75% *A. flavus* and 3.75% *A. niger*); cytology was not performed.[25] Lyskova *et al.*[26] cultured *A. fumigatus* from 2.1% of dogs with clinical otitis externa, but not from 178 healthy dogs; again cytological information was not provided.[26] No treatment information was provided in the above studies where *Aspergillus* was cultured.

Atopy predisposes dogs to the development of otitis due to a combination of allergic otic inflammation and self-inflicted trauma, which alter cerumen production, increase bacterial adherence and often lead to secondary infections.[1,34] Up to 55% of dogs with atopic dermatitis have concurrent otitis externa, and 3–5% of cases exhibit otitis externa as the only clinical sign of atopy.[35,36] The present case had been treated previously with numerous topical and oral antibiotics which may have predisposed it to development of secondary *Aspergillus* fungal infection as described in humans. *Aspergillus* was probably an opportunist and not the initial or primary cause of the otitis; opportunistic infection was evidenced by persistent inflammatory otitis in the absence of bacterial infection, cytological visualization of filamentous fungal organisms on four separate occasions and culture of *Aspergillus* spp. in the absence of other organisms. The species of *Aspergillus* cultured differed from that cultured 8 months prior to referral. The previous *Aspergillus* isolate may have been a contaminant, or the dog may have had a unique susceptibility to recurrent secondary *Aspergillus* otitis by different species. In retrospect, it is unfortunate that fungal cultures were not performed at each visit to document causative fungal species; although fungal hyphae appeared identical at each visit, the causative organisms were not specifically identified and were assumed still to be *Aspergillus* spp. Otomycosis persisted despite topical and systemic steroids and antibiotics, topical clotrimazole, topical thiabendazole, oral ketoconazole and compounded itraconazole. Otitis and otomycosis finally resolved after ASIT, prolonged therapy with oral fluconazole, topical therapy with ketoconazole, flushing and removal of a large ceruminolith adherent to the tympanum. Although the canal and tympanum of the affected ear were clinically improved, and cytology and culture of otic swabs revealed no fungal organisms after fluconazole and topical ketoconazole therapy, the fungal hyphae present in the ceruminolith illustrate the importance of complete removal of ear canal debris that can protect organisms from antifungal medication. It is difficult to assess the effect of systemic antifungal medication on the final resolution of the otomycosis, but most of the improvement appeared to result from topical ketoconazole treatment and more aggressive aural toilette, as described in human otomycosis treatment. Resolution may have occurred more quickly if the ear canal had been lavaged and suctioned daily as is recommended in humans, but owing to the fractious nature of the dog, this would have necessitated daily sedation. Additionally, addressing the underlying original allergic cause of the otitis was crucial in re-establishment of a more normal ear canal physiology and prevention of otomycosis recurrence.

The results illustrate the need to identify and treat opportunistic fungal organisms, such as *Aspergillus* spp., which may complicate cases of chronic otitis in dogs, with an awareness of the potential consequences of increased use of topical fluoroquinolones that may, as in humans, increase the occurrence of *Aspergillus* otitis. Further comparison of fungal organisms cultured from

the ears of normal dogs with those from cases with atopic and infectious causes of otitis is currently in progress.

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Résumé Cet article décrit les signes cliniques, clinicopathologiques et le traitement de l'otomycose due à *Aspergillus* spp. chez un chien atopique atteint d'otite externe purulente unilatérale chronique ne répondant pas aux antibiotiques oraux et topiques et aux traitements antifongiques. La cytologie de l'exsudat auriculaire a révélé la présence de neutrophiles et d'hyphes fongiques septés. La culture auriculaire a permis la croissance *Aspergillus s*pp. mais pas de bactérie. Les traitements consistaient en une immunothérapie spécifique d'allergène, une thérapie antifongique topique et orale ainsi que des anti-inflammatoires stéroïdiens. La guérison a été obtenue après traitement de l'hypersensibilité sous-jacente, l'administration de kétoconazole topique et le débridement de l'exsudat infectieux auriculaire. L'otomycose due à des champignons filamenteux peut, comme chez l'homme, toucher les chiens dont les conduits auriculaires sont modifiés par une otite allergique ou bactérienne pré-existante et peut-être une antibiothérapie. Les antifongiques ont permis une amélioration clinique mais la clé de réussite du traitement a été la restauration de la physiologie normale du canal auriculaire externe.

Resumen Este articulo describe los hallazgos clínicos, clinicopatológicos y el tratamiento de la otomicosis producida por una especie del género *Aspergillus* en un perro atópico afectado por otitis purulenta externa crónica unilateral que no respondía a tratamientos antibióticos tópicos ni orales ni a tratamientos antifúngicos. La citología del exudado indicaba la presencia de neutrófilos e hifas de hongos tabicadas, y el cultivo creció una especie del genero *Aspergillus* pero no bacterias. Los tratamientos incluyeron inmunoterapia especifica de alergeno, tratamiento tópico y oral antifúngico, y tratamiento antiinflamatorio con esteroides. La resolución final se produjo tras el tratamiento de la enfermedad alérgica, la administración tópica de ketoconazol y la limpieza del exudado infeccioso. La otomicosis producida por hongos filamentosos puede, como en el caso de humanos, producirse en perros con compromiso de la integridad del canal auricular debido a un proceso alérgico o a una enfermedad bacteriana, y posiblemente tras tratamiento antibiótico previo. El tratamiento antifúngico produjo mejora clínica, pero la clave del tratamiento efectivo fue la restauración de la fisiología normal del canal auditivo externo.

Zusammenfassung Diese Studie beschreibt die klinischen Ergebnisse, die klinische Pathologie, und die Behandlung einer Otomykose, die bei einem atopischen Hund mit einer chronischen unilateralen purulenten Otitis externa, die sich auf topische und orale Antibiotika und Antipilzmittel nicht verbessert hatte, durch *Aspergillus* spp. verursacht war. Die Zytologie des Exsudats aus dem Ohr zeigte Neutrophile und gegliederte Pilzhyphen, bei einer Kultur aus dem Ohr wuchs *Aspergillus* spp. und keine Bakterien. Die Behandlung beinhaltete eine Allergen-spezifische Immuntherapie, topische und orale Antipilzmittel und eine entzündungshemmende Cortisontherapie. Eine letztendliche Heilung erfolgte nach der Behandlung der zugrunde liegenden Hypersensibilitätsstörung, der Verabreichung von topischem Ketokonazol und dem Abtragen des infektiösen Ohrexsudats. Eine Otomykose aufgrund von filamentösen Pilzen kann, wie beim Menschen, auch bei Hunden vorkommen, deren Gehörgänge durch eine vorbestehende allergische oder bakterielle Otitis, und möglicherweise durch frühere Antibiotikatherapie beeinträchtigt waren. Die Antipilztherapie brachte eine klinische Verbesserung, aber der Schlüssel zur erfolgreichen Behandlung war die Wiederherstellung der normalen Physiologie des äußeren Gehörgangs.

要約 この報告はアトピー性皮膚炎の犬に発生した、外用薬や経口抗生剤、抗真菌剤の治療に不応性の、片側性の慢性化膿性外耳炎を示す *Aspergillus s*pp による耳真菌症の臨床症状、臨床病理、治療法を記述した。耳からの滲出物の細胞診では好中球と中隔壁を有するのある菌糸が示され、滲出物の培養では *Aspergillus s*pp が分離されたが、細菌は認められなかった。アレルゲン特異的免疫療法、外用および経口での抗真菌剤による治療、抗炎症性ステロイド療法などの治療が実施された。最終的な回復は基礎的な過敏症の治療と、外用のケトコナゾール塗布、 耳の感染性滲出物のデブリードメントの後に認められた。フィラメント状真菌に起因する耳真菌症は、ヒトと同様に、既に存在するアレルギーまたは細菌性外耳炎ならびに場合によっては、以前の抗菌剤治療によって犬の耳道で生じる。抗真菌剤による治療は臨床的な改善をもたらすが、しかし治療の成功への鍵は生理的に正常な外耳道の回復である。

