Nasal Granuloma Caused by Scedosporium apiospermum in a Dog

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A 10-month-old male American Staffordshire terrier was presented to the Autonomous University of Barce-
loña Veterinary Teaching Hospital because of a 6-month history of a mucopurulent bilateral nasal discharge.
The dog had not responded to antibiotics. A follow-up X ray revealed a mixed pattern of osteolysis and in-
creased radiodensity confined to the nasal cavity. Histologic sections of the biopsy specimens revealed the pre-

cence of granules containing numerous septate hyphae that were hyaline to pale brown and smooth, one-celled,
subpherical-to-elongate conidia that were hyaline to brownish green, and bacteria. Cultures yielded numerous
colonies belonging to Scedosporium apiospermum. Susceptibility tests were performed on the isolated strain. The
isolate was sensitive to ketoconazole, intermediate to clotrimazole, and resistant to amphotericin B, 5-fluoro-
cytosine, flucnazole, and itraconazole. The dog was treated with oral ketoconazole. During the treatment a
general improvement in the lesions was observed. To our knowledge, S. apiospermum has not been implicated
previously as an etiologic agent of nasal disease in dogs. This report provides its first description as such.

Fungal infections are a common cause of nasal disease in dogs and cats. Aspergillus fumigatus is the species most com-
monly isolated from infections in the nasal cavities of these animals. However, Aspergillus niger, Aspergillus nidulans, and
Aspergillus flavus have also been recovered from this location. Penicillium spp. are rarely reported as causing invasive nasal
disease (12, 16).

Pseudallescheria boydii is an ubiquitous saprophytic ascomycetace that has two anamorphs. It has been isolated from soil,
water, vegetation, and sewage. The Scedosporium apiospermum synanamorph is most commonly isolated from clinical cultures
(1) and from all strains of P. boydii (5).

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provides its first description as such.

Case report. A 10-month-old male American Staffordshire terrier was presented to the Autonomous University of Barce-
loña Veterinary Teaching Hospital because of a 6-month history of a mucopurulent bilateral nasal discharge and some
sneezing. The disorder started 10 days after the dog suffered a crash and the owners did not want to do the necropsy.

The animal was mildly depressed and had a temperature of 39.6°C. There was bilateral thick yellow discharge and deformed
external nares. The dog had a mature neutrophilic leu-

kocytosis (24,000 leukocytes/μl). The serum chemistry profile and urinalysis were normal. A follow-up X ray revealed a
mixed pattern of osteolysis and an increased radiodensity confi-

ded to the nasal cavity that was greater on the right side. The
turbinate pattern was less apparent than in normal dogs on
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The baseline laboratory analysis (complete blood count, chemistry profile, and urinalysis) were normal. After a month,
the nasal discharge decreased and the sneezing disappeared. Unfortunately, 1 month after the last checkup, the dog died in a car

crash and the owners did not want to do the necropsy.

Discussion. Reported cases of S. apiospermum infections in dogs are few. This species has been recognized as a causative
agent of mycosis mainly in the abdominal cavity (1, 6, 8, 15). A disseminated infection has been reported (2). It has been cited
in other locations such as the epidermis (chronic eczematous lesion) (10), eyes (keratomycosis) (14), and testicles (1).

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FIG. 1. Intraoral radiograph showing lysis of the vomer bone (arrow) and increased radiodensity confined to the nasal cavity, which is more pronounced on the right side (asterisks). The turbinate pattern is less apparent than in normal dogs on both sides (black areas in the nasal cavity).

FIG. 2. Detail of a Grocott-methenamine-silver-stained section of a tissue biopsy sample showing a granule containing hyphal and conidial elements. Bar ~ 10 μm.
ditionally, in horses, two reports of *P. boydii* causing infection of the nasal chamber have been published (3, 7).

In humans, the wide spectrum of diseases caused by *P. boydii* includes sinusitis, brain abscess, meningitis, pulmonary colonization, fungus ball, invasive pneumonitis, endocarditis, arthritis, osteomyelitis, thyroid abscess, endophthalmitis, disseminated systemic disease, cutaneous and subcutaneous granulomata, keratomycosis, and mycetoma (9). Infection in humans most frequently occurs in soil-contaminated wounds on the hands or feet. However, especially in a compromised host, infection by inhalation causing sinusitis is not uncommon. Pulmonary lesions may also occur, and the angioinvasive nature of the fungus may result in spread to other areas of the body (3).

In our report, except for the parvoviral enteritis process and antibiotic treatment, no other predisposing factors such as neoplasia, immunodeficiency, depression, trauma, and exposure to high levels of fungal spores were identified. No sign of disseminated systemic disease was detected. The infection was limited to the nasal chambers. Nevertheless, lysis of the vomer bone was observed.

Some authors (13) have stated that in the treatment of canine nasal aspergillosis, topically administered enilconazole (via indwelling tubes, implanted bilaterally through trephine holes in each frontal sinus) is more effective than the oral administration of thiabendazole, ketoconazole, fluconazole, or itraconazole. In our case the isolate belonging to *S. apiospermum* was sensitive only to ketoconazole, and for this reason the dog was treated with oral ketoconazole.

Although antifungal susceptibility tests for filamentous fungi remain unstandardized and the in vivo outcome cannot always be extrapolated from the in vitro results, the resistance of *P. boydii* to different antifungal agents such as amphotericin B, clotrimazole, 5-fluorocytosine, griseofulvin, and nystatin and its mixed responses to ketoconazole, miconazole, and natamycin have been mentioned (14). On the other hand, different strains of *S. apiospermum* and *P. boydii* appear to have various degrees of sensitivity to antifungal agents (11) (for these strains the MICs of miconazole, ketoconazole, and itraconazole are lowest) and they appear to be resistant to fluconazole.
REFERENCES