

Pseudallescheria boydii infection of the nasal cavity of a horse

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- ▶ Nasal infection with *Pseudallescheria boydii* is uncommon in horses but should be considered in the differential diagnosis when horses with malodorous nasal discharge are examined.
- ▶ The tissue phase of *P boydii* is similar to that of *Aspergillus* spp, making culture the only way to definitively and accurately diagnose nasal mycosis.
- ▶ Nasal infection with *P boydii* can be successfully treated with topical application of 2% miconazole cream and systemic administration of sodium and potassium iodide.

An 18-year-old 454-kg (1,000-lb) American Quarter Horse gelding was referred to the Auburn University Large Animal Clinic for evaluation of chronic intermittent malodorous right-sided nasal discharge. Five months earlier, the horse had been evaluated at the clinic because of bilateral, mucopurulent nasal discharge. On radiographs of the skull obtained at that time, density of the roots of the right upper fourth premolar and first molar and of the left upper first and second molars appeared to be less than normal, and fluid lines were evident in the left and right rostral and caudal maxillary sinuses. The sinuses on the right side contained a greater amount of fluid than the left. The reduced density of the tooth roots was considered to be a result of normal aging processes, and no evidence of a tooth abscess was found during a dental examination. No other abnormalities were detected. Because of budget limitations, nasal endoscopy was not performed at that time. A diagnosis of bilateral maxillary sinusitis was made, and the left and right rostral maxillary sinuses were each flushed with 1 L of physiologic saline solution containing 5×10^6 U of penicillin G potassium once daily for 6 days through catheters placed through holes drilled through the maxilla and into each rostral maxillary sinus. The horse was also treated with trimethoprim-sulfamethoxazole (22 mg/kg [10 mg/lb] of body weight, PO, q 12 h for 21 days). Antibiotics were selected on the basis of empirical judgment. The horse was discharged after 6 days of hospital treatment, and the owner reported that clinical signs had resolved until 1 week prior to reexamination. At this time, the trainer noticed that the horse had malodorous breath.

At the time of reexamination, the horse had purulent discharge from the right nostril, and its breath was malodorous. Airflow from both nostrils was normal. Rectal temperature was 38 C (100.3 F), pulse rate was

32 beats/min, and respiratory rate was 20 breaths/min. Oral examination revealed an erosion on the rostral medial aspect of the right upper first molar. On radiographs of the skull, density of the tooth roots was unchanged from previous radiographs. A radiopaque density was evident in the right caudal maxillary sinus, but no fluid lines were seen.

Endoscopy of the nasal passages was performed, and the nasomaxillary openings of the caudal maxillary sinus appeared larger than normal bilaterally (Fig 1). Large white plaques were seen in the nasal cavity adjacent to the nasomaxillary opening of the right caudal maxillary sinus (Fig 2). No other abnormalities were detected.

The abnormally large nasomaxillary openings allowed exploration of the caudal maxillary sinuses, as well as the frontal sinuses, with the endoscope. These areas were normal in appearance. Samples of the white plaques in the nasal cavity were retrieved with a biopsy probe and submitted for cytologic examination and fungal culture and susceptibility testing. Cytologic evaluation of the samples revealed numerous fungal hyphae and conidia (Fig 3), and a presumptive diagnosis of fungal sinusitis was made. The isolate was identified as *Pseudallescheria boydii* (Fig 4) and was susceptible in vitro to miconazole, ketoconazole, natamycin, and clotrimazole.

The following day, under endoscopic guidance, a balanced electrolyte solution was flushed through a mare uterine catheter to loosen the more persistent



Figure 1—Endoscopic view of the right nasal passage of a horse with *Pseudallescheria boydii* infection of the nasal cavity. Notice the large nasomaxillary opening of the caudal maxillary sinus (arrow).

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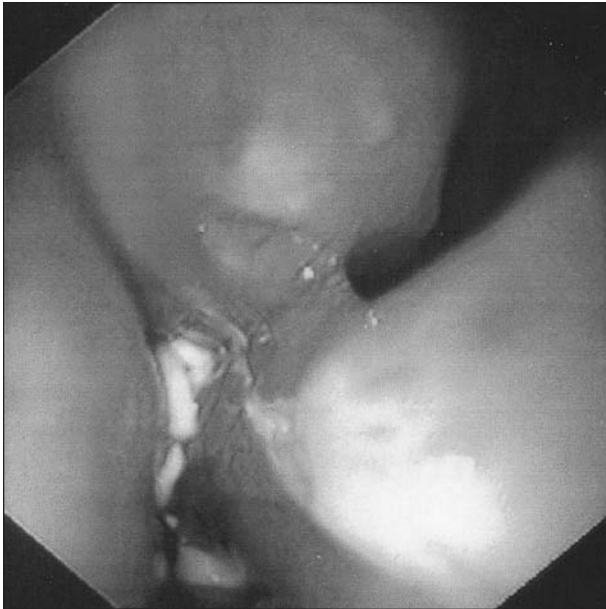


Figure 2—Endoscopic view of the right nasal passage of the horse in Figure 1. Notice the white mycotic plaque. The nasomaxillary opening is just caudal to the plaque.



Figure 3—Photomicrograph of a section of the white plaque from the horse in Figure 2. Notice the hyphae and coremia (arrows) bearing terminal conidia. Potassium hydroxide stain; bar = 500 μ m.

fungal masses from the nasal mucosa. The underlying nasal mucosa was reddened and ulcerated. On the third day, the horse was again sedated, and any remaining fungal components were debrided and flushed in the same manner. Lavage tubing was passed through a hole drilled through the frontal bone and threaded through the right frontal sinus, right caudal maxillary sinus, right nasomaxillary opening, and into the nasal passage so that the end of the tubing was adjacent to the site of fungal infection. Passage of the lavage tube through the sinuses was achieved, using the endoscope in combination with endoscopic biopsy forceps used to grasp the tube. Five grams of 2% miconazole cream was infused through the lavage tubing every 12 hours for four weeks. Sodium iodide was administered IV at a dosage of 30 mg/kg (14 mg/lb) once daily for 4 days. The horse was then treated with potassium iodide at a dosage of 0.06 mg/kg (0.03 mg/lb), PO, every 12 hours for 14 days. The horse was discharged on day 5, and the owners were instructed to observe the horse for

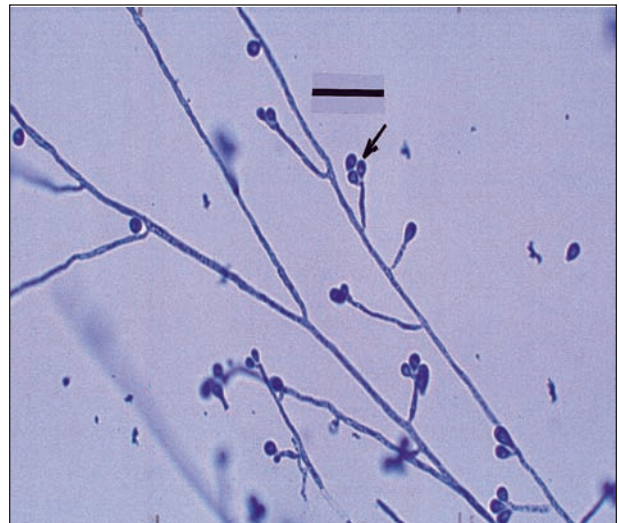
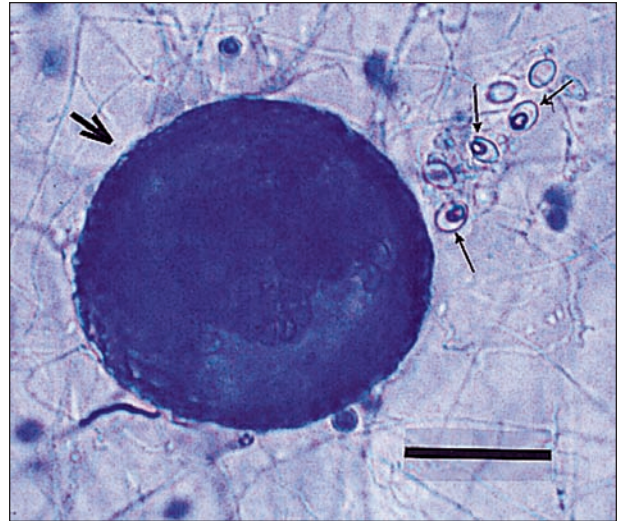


Figure 4—Sexual (top) and asexual (bottom) forms of *P boydii* in culture. Notice the cleistothecium (thick arrow) and ascospores (thin arrows) in the sexual form and the nonbranching conidiophores with terminal conidia (arrow) in the asexual form. Lactophenol cotton blue stain; bar = 500 μ m.

signs of dry flaky skin, anorexia, lethargy, or epiphora, which could indicate iodide toxicosis.

Follow-up nasal endoscopy was performed 30 days after the horse was discharged. No signs of fungal infection were observed at that time. The lavage catheter was removed, and all medications were discontinued. Nasal endoscopy was repeated 30 days after all medication was discontinued, and no abnormalities were observed.

Pseudallescheria boydii is a saprophytic ascomycete that has been isolated from a variety of substrates, including soil, polluted streams, sewage sludge, and poultry and cattle manure.^{1,2} Infection with this organism is usually manifested as a mycetoma and is characterized by swelling, granule formation, and discharging sinus tracts.^{3,4} In areas with warmer climates, mycotic granulomas can be caused by a wide variety of fungal organisms, including *Aspergillus* spp.,⁵ *Coccidioides* spp.,⁶ *Cryptococcus* spp.,⁷ *Entomophthoramyces* spp.,⁸ *Pseudallescheria* spp.,^{9,10} and *Rhinosporidium* spp.¹¹ Clinical signs of nasal mycosis in horses include uni-

lateral chronic malodorous discharge, epistaxis, and dyspnea with or without reduced airflow from the affected nostril. In humans, *Pseudallescheria boydii* infection most commonly involves the extremities and is known as Madura foot.¹² In developed countries, *P boydii* infection is being diagnosed more often in immunocompromised individuals, and can be found in the paranasal sinuses, eyes, joints, subcutaneous tissue, lungs, and brain.^{12,13} In infected tissue, the organism grows as hyphae and cannot be differentiated from *Aspergillus* spp and *Fusarium* spp unless it is cultured.¹⁴

Pseudallescheria boydii has been isolated from the pharyngeal-tonsillar area of 2 of 60 clinically normal donkeys, and from horses with chronic uterine infection.¹⁰ However, nasal mycosis caused by infection with *P boydii* is rare in horses, and to our knowledge, only 2 cases have been reported previously.^{9,11} One of these involved a horse with a progressive subcutaneous and submucosal granulomatous reaction similar to that reported for humans with eumycotic mycetomas. The other involved a horse with superficial mucosal lesions that did not involve deeper underlying tissues, similar to lesions observed in the horse described in the present report. Both of these horses were euthanized because of the extent of the lesions or a lack of response to treatment. The horse described in the present report recovered uneventfully with topical treatment. The large nasomaxillary opening allowed use of an endoscope to place lavage tubing at the site of infection. This facilitated direct placement of 2% miconazole at the site of infection. Systemic iodide treatment was also used to help combat infection.

Recently, nitric oxide production in the paranasal sinuses and its protective role in disease processes of the upper part of the respiratory tract in humans have been studied. It has been shown that in humans, nitric oxide is produced primarily in the paranasal sinuses, with a small amount produced by the nasal mucosa.^{15,16} Abnormally low concentrations of nitric oxide in the nasal cavity are thought to predispose individuals to sinusitis.¹⁶ In addition, nitric oxide has antiviral and bacteriostatic properties, plays a major role in nonspecific host defenses,^{17,18} and increases mucociliary beat frequency, thereby increasing mucociliary clearance.¹⁹

Low nasal nitric oxide concentrations in children with Kartagener's syndrome are caused by dysfunction of the nasomaxillary opening,²⁰ and we believe that the large nasomaxillary openings in the horse described in the present report may have predisposed it to develop fungal infection secondary to excess loss of nitric oxide. No other predisposing factors were identified in this horse.

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