Acknowledgments

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Fungal pneumonia in a captive black rhinoceros

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A captive black rhinoceros (*Diceros bicornis*) with a hoof abscess was treated with long-term antibiotic therapy. After 9 months of treatment, there was rapid deterioration, marked weight loss and reluctance to stand. Profuse, bilateral epistaxis developed accompanied by collapse and the animal was euthanased. Necropsy revealed pulmonary aspergillosis with concurrent *Pseudomonas aeruginosa* infection. Though a well-recognised disease of black rhinoceros, fungal pneumonia has not been reported in this species in Australia. The cost and efficacy of treatment have been questioned, however, prophylactic antifungal drug administration will be considered in any further cases of chronic, debilitating illness in black rhinoceros at Western Plains Zoo.

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Fungal pneumonia is a well recognised disease of black rhinoceros (*Diceros bicornis*),[1,2] however there are no reports of this condition occurring in rhinoceros in Australia. This article documents pulmonary aspergillosis with concurrent *Pseudomonas aeruginosa* infection in a captive black rhinoceros at Western Plains Zoo at Dubbo in Australia.

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Case report

In November 1996 a 39-years-old, female black rhinoceros of estimated 1000 kg body mass at Western Plains Zoo was reported to be favouring its right foreleg. Limited examination revealed a 5 cm wide by 2 cm deep piece of sole missing from the medial aspect of the central digit of the right forefoot. No foreign body or pus were noted, but the animal resented palpation of the foot. The area was flushed with dilute povidone iodine solution and the animal was placed on a course of oral flunixin meglumine (1.1 mg/kg once daily for 5 d) and sulfadimidine-trimethoprim powder (25 mg/kg and 5 mg/kg once daily for 7 d). The lameness appeared to improve whilst on flunixin meglumine, but it recurred once the flunixin meglumine stopped. The course of sulfadimidine-trimethoprim was extended for 12 weeks, at the end of which there was little improvement.

The animal was anaesthetised with approximately 3.375 µg/kg etorphine HCl and 11.070 µg/kg acepromazine HCl (1.5 mL Large Animal Immobilon: etorphine HCl 2.25 mg/mL base and acepromazine maleate 7.38 mg/mL base,

Vericore, Dundee, UK) administered intramuscularly by remote injection using a 2 mL type C Pneu Dart with 50 mm needle (Pneu Dart, Williamsport, PA) with the barb removed fired from a Pneu-dart model 193 – cartridge fired rifle (Animal Capture and Services, Warwick, Queensland). The feet were examined and it was found that there had been little change in the lesion on the right forefoot, but a similar lesion had developed on the lateral aspect of the medial toe of the left forefoot. Both areas were debrided, flushed with povidone iodine solution, explored and infused with cloxacillin eye ointment. The results of blood collected for routine haematology and biochemistry were unremarkable. One hundred mL long-acting penicillin and 1 mg/kg nandrolone laurate was administered intramuscularly. Anaesthesia was antagonised with 0.1 mg/kg naltrexone HCl (Wildnil, 50 mg/mL, Wildlife Pharmaceuticals, Fort Collins, CO) administered intravenously and 6 µg/kg diprenorphine HCl (2 mL Large Animal Revivon, 3 mg/mL diprenorphine base, Vericore, Dundee, UK) subcutaneously and sulfadimidine-trimethoprim was continued.

There was little change in the animal's condition until July 1997, when it started to lose weight, was reluctant to walk, adopted a stance of rocking back on its hindlegs (presumably to relieve pressure on its forefeet) and lying down. There was no evidence of respiratory difficulty. Over the next 2 months its condition deteriorated markedly: it spent progressively more time lying down and lost much weight. Profuse, bilateral epistaxis was noticed in early September. The animal was anaesthetised, blood collected, and euthanasia performed with 50 mL pentobarbitone sodium intravenously. Haematology revealed a leukocytosis (21.3 x 10^9/L), marked neutophilia (15.5 x 10^9/L) and moderate left shift (2.56 x 10^9/L bands).[3] Necropsy was performed and samples collected for microbiology and histopathology.

Pressure sores were associated with the lateral elbows, hocks, carpi and points of the animal's hips. The teeth showed signs of wear and there was marked dental calculus. There were multiple well-organised adhesions between the lungs and chest wall. Approximately 15% of lung tissue appeared grossly normal, the rest appeared collapsed and contained necrotic, 10 to 15 cm diameter areas of green-grey material. Some bronchi and alveoli appeared fibrosed and were filled with creamy white-yellow pus, others contained yellow, rubbery plaques adherent to the walls of the bronchi.

A section of an abscess from the lung was examined for microbiology. Wet preparation revealed fungal hyphae and low numbers of Gram-positive cocci were present. No acid-fast bacilli were seen using Ziehl-Neelsen stain, and no fluorescent bacilli using Auromine-O stain. No mycobacteria were isolated using mycobacterial culture. Routine culture of the swab from a terminal bronchus and the section of abscess from the lung grew a heavy mixed growth of *Pseudomonas aeruginosa* and an α-haemolytic *Streptococcus* sp. *Aspergillus glaucus* was identified by fungal culture.

Histopathology confirmed the severe pneumonia and indicated significant fungal infection. There was severe chronic, active pyogranulomatous bronchopneumonia with large numbers of foamy macrophages in the airways. Some of the macrophages contained orange-yellow material. In one portion of the lung there was a fungal pneumonic lesions with many branching fungal hyphae with spherical conidiospores (Figure 1 and 2). There was no evidence of tuberculosis.

Discussion

Fungal pneumonia in black rhinoceros was reviewed recently[2] and predisposing factors, prevention, diagnosis and treatment were discussed. A survey of US institutions holding black rhinoceros identified eight animals that had fungal disease at the time of death. The animals' ages were not stated, however all were older than one year of age. A prevalence of 14 % of all deaths in black rhinoceros was ascribed to this condition from 1980 to 1994. The predominant genus involved was *Aspergillus*, however the species involved were not presented. All animals had concurrent disease. Premortem diagnosis using tracheobronchial lavage, percutaneous lung biopsy and serology were concluded to be of questionable value because fungal hyphae could be present in normal animals, biopsy may miss the

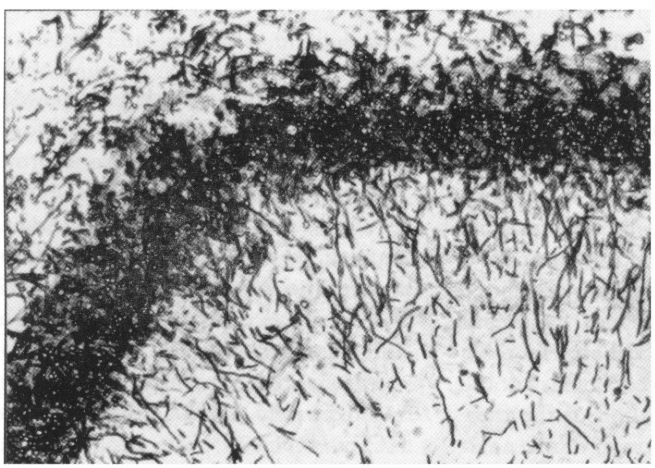

Figure 1. Section of lung showing fungal hyphae at the edge of a pneumonic lesion. Gomori's silver, x 200.

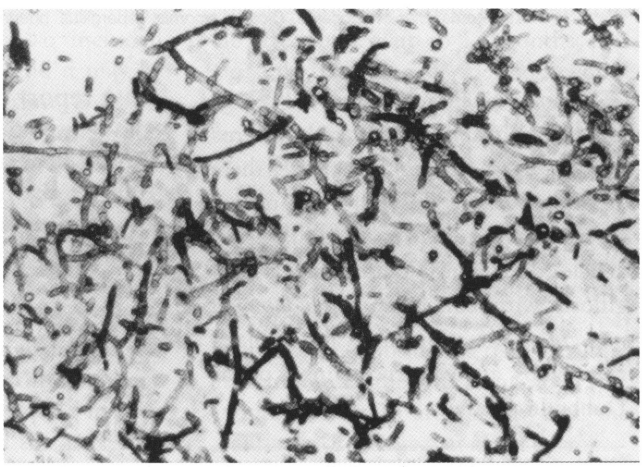

Figure 2. Section of lung showing branching fungal hyphae with spherical conidiospores. Gomori's silver, x 500.

site of infection, and serology could be positive due to environmental exposure. It was suggested that systemic antifungal drugs could be considered for treatment although their cost and efficacy were questioned and no examples were presented. They concluded that the association with other severe diseases, the difficulty of premortem diagnosis, and lack of effective and available therapy created a clinical challenge, and that it was important to be aware of fungal pneumonia as a potential factor when treating a sick black rhinoceros.

Aspergillus is a ubiquitous mould that grows well on a variety of substrates, including soil, water, decaying vegetation, mouldy hay or straw, and organic debris. Although over 300 species have been characterized, less than 20 species have been identified as causing clinically significant disease in humans, the species for which most data are available.[4] There is little information in the literature regarding the prevalence and nature of disease attributable to *A glaucus* in animals, however, in humans *A glaucus* has caused mycetoma[4] and has been reported as causing clinical aspergillosis less frequently than the two most commonly implicated species, *A fumigatus* and *A flavus*. The varying degrees of pathogenicity of each species of *Aspergillus* depends on their relative geographic prevalence, conidial size and shape, thermotolerance, and production of mycotoxins. Presumably *A glaucus* was the predominant species within the black rhinoceros' environment at the time of its illness. Although exposure to *Aspergillus* spores is nearly universal, impaired host defences are required for the development of invasive disease which probably developed in the animal in the present study as a result of the combination of old age, debility and chronic disease. Furthermore, the prolonged use of broad-spectrum antibiotics is likely to have compounded the problem.

A combination of aerosolised amphotericin B delivered into a sealed stall and oral itraconazole therapy may be an option for attempting treatment of this condition in black rhinoceros. Itraconazole possesses excellent in vitro activity against *Aspergillus*, however adverse effects such as gastrointestinal disturbances (nausea, vomiting, epigastric pain, and diarrhoea) have been reported in up to 20% of human patients receiving this drug.[5] Furthermore, the reported side effects associated with administration of amphotericin B, which include normochromic, normocytic anemia[5] are cause for concern in animals in which severe and often fatal haemolytic anaemia of unknown aetiology has been reported.[6] Regular venipuncture and haematologic examination may be useful in monitoring tractable animals on this treatment regime if facilities allow it. Oral fluconazole or 5-fluorocytosine, which may be less efficacious but have fewer side effects,[5] may be a safer option, though in all cases cost and administration of drugs to severely debilitated, large, and essentially wild animals is problematic.

Despite the difficulties in diagnosing aspergillosis in the live animal,[2] monitoring of debilitated animals for *Aspergillus* infection using regular culture of nasal secretions, serological testing and haematology may be useful provided that baseline values have been determined in the individual whilst it was well. Culture of *Aspergillus*, positive or rising titres, or prolonged severe neutropaenia, commonly associated with aspergillosis in humans,[5] could be used to indicate that aerosolised and systemic antifungal treatment should begin.

An alternative approach may be to attempt to prevent the onset of the disease in any debilitated animals that are under immunosuppressive or prolonged antimicrobial treatment. Simple husbandry procedures such as feeding animals off the ground, good hygiene and the avoidance of damp feed stuffs should all decrease numbers of inhaled spores. The use of high-efficiency filters and laminar flow rooms to remove spores is unlikely to be a practical option for black rhinoceros, however housing debilitated animals in well ventilated, dry conditions may also be of benefit. Prophylactic antifungal therapy to prevent primary infection with *Aspergillus* may be another option. Prophylactic intranasal amphotericin B sprays have been used in humans with conflicting results,[5] however prophylactic administration with aerosolised amphotericin B was reported to kill inhaled *Aspergillus* spores and delay the progression of disease in a rat model of pulmonary aspergillosis.[7]

As black rhinoceros are valuable animals genetically, intrinsically and to replace in case of loss, administration of prophylactic aerosolised amphotericin B, and/ or oral itraconazole, nasal fungal culture, and routine haematological and serological monitoring for signs of *Aspergillus* infection during treatment will be considered in any further cases of chronic, debilitating illness in black rhinoceros at Western Plains Zoo.

Acknowledgments

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