

A review of aspergillosis in penguins (2008)

By

Melissa Orzechowski Xavier (DVM, MSc, PhD student from Brazil

Universidade Federal do Rio Grande do Sul)

E-mail: melissaxavier@bol.com.br

Aspergillosis is a rare disease in free-living penguins with little impact on the reproductive colonies of these animals, corresponding to a mortality rate about 3% [13]. However, this disease shows a very different role when related to captive seabirds. In captivity, aspergillosis has been described in a great variety of penguin species and associated with stress, change in habitat, handling, injury or other concomitant diseases which promote a high susceptibility to primary infection with *Aspergillus* spp. [11, 19, 20, 21, 25, 29].

The respiratory tract is the most important anatomic site of disease caused by the genus *Aspergillus fumigatus* is the species that corresponds to the majority of aspergillosis cases, followed by *A. flavus* and *A. niger* [2, 11, 16, 19].

Penguins' predisposition to aspergillosis is aggravated by the physical effort of migration and by anatomic, physiological and immune system peculiarities.

Anatomical factors which increase penguin susceptibility to aspergillosis are the lack of an epiglottis, which permits conidial penetration to the lower respiratory tract, the lack of a diaphragm making the cough reflex difficult, restricted distribution of ciliary epithelium in the respiratory tract and the presence of air sacs with high oxygen concentration and few blood vessels [4, 27]. In addition, the absence of surface macrophages in the lower respiratory tract which are responsible for the destruction of conidia, and the substitution of neutrophils by heterophils which have different and inferior mode of action (using cationic proteins, hydrolase and lysozyme as opposed to myeloperoxidase and oxidative mechanisms to destroy hyphae) are characteristics of penguins that contribute to a susceptibility to aspergillosis [27].

Other situations commonly found in rehabilitation centers, which increase penguin immunosuppression and increase the predisposition to infection with *Aspergillus* species are many and include - oil contamination, administration of corticosteroids and antibiotics, overcrowding and poor ventilation [4, 22, 27].

Thus in captive penguins in zoos, aquariums or rehabilitation centres, aspergillosis is commonly associated with high mortality rates, resulting in ecological and economic losses for establishments [1, 10, 12, 15, 19, 21, 23, 25]. This situation has been documented over the past 40 years in zoos [2], and the last 70 years in rehabilitation centres since the first record of an ecologic disaster due to an oil spillage ("Oregon Standard", San Francisco, California, EUA). On this occasion nearly 7000 seabirds were contaminated with oil and 6700 of these died, the majority due to aspergillosis [14, 22].

Published reports from captive penguins with aspergillosis show mortality rates associated with the disease in different regions of the world (Figure 1). Reports from zoos in the United Kingdom (London and Scotland), India (New Delhi) and the United States of America (Michigan) were published in 1949, 1990, 1977 and 2003, respectively. The studies describe the mortality due to aspergillosis in Magellanic penguin (*Spheniscus magellanicus*), Gentoo penguin (*Pygoscelis papua*), Chinstrap penguin (*Pygoscelis antarctica*) and King penguin (*Aptenodytes patagonica*) [2, 9, 16, 17]. The reports from rehabilitation centers are more recent and rare. The death of seabirds due to aspergillosis at the International Bird Rescue Research Center (IBRRC -USA) (shown in figure 1) occurred in 1991 after an oil spillage in Washington [22], and statistics from the Marine Animal Rehabilitation Center (CRAM -Brazil) refer to fatal aspergillosis in penguins in 2004 [29].

Mortality

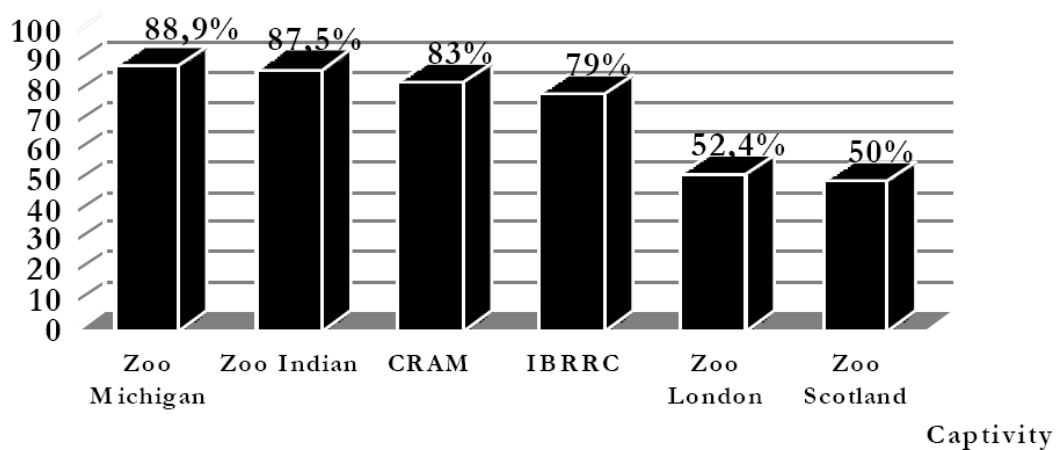


Figure 1-Mortality rates due to aspergillosis in penguins at different places of captivity.

Clinical presentation of aspergillosis in penguins is characterized by a diffuse disease of the lower respiratory tract, with air sac and lung involvement. The tracheal bifurcation is another region frequently involved due to the narrowness of the lumen and the air turbulence which allows the conidia to escape the expiratory flow and to be deposited in the epithelium [4]. This tracheal form of aspergillosis can either partially or totally obstruct the passage of air with granulomatous nodules or necrotic debris [1, 4, 15, 19].

Systemic aspergillosis results from fungal dissemination by air sacs or blood vessels. In these cases lesions can be observed in the respiratory tract, liver, gastrointestinal tract and kidney. Birds can show some clinical signs as dyspnoea, swelling, lethargy and anorexia or sudden death [1, 5, 15, 19], whilst both the tracheal and systemic forms of aspergillosis are frequently observed in captive penguins [2, 7, 15, 16, 29].

Difficulties in the diagnosis of penguin aspergillosis “in vivo” are attributed to the rapid and progressive nature of the disease without specific clinical signs, or sudden death. Thus, post-mortem exams are the most common method

of proving the diagnosis [1, 6, 19, 28]. At necropsy the lesions are characterized by white-yellowish nodules, isolated or grouped, 0,1cm to 7cm of diameter. Additionally, yellow exudates, fibrous tissue, fungal colonies, lung hyperemia and congestion, thickened air sacs and hepatomegaly can be observed [3, 4, 5, 27, 28].

Histopathological findings include granulomatous nodules with necrotic centers, macrophages and heterophils. In chronic cases giant cells and capsules of fibrous tissue can be observed [4, 5]. Hyaline, septate and branching hyphae (45°) are seen, as well as complete sporulation structures of *Aspergillus* in some tissues [1, 4, 5, 26]. The presence of asexual reproductive structures of *Aspergillus* in tissues is a common find in birds suffering from aspergillosis, due to the presence of cavernous air sacs, a high body temperature and avian sensitivity to gliotoxin. These factors, in addition to the paucity of blood vessels, low number of immune cells and the ready availability of oxygen and nutrients in the air sacs provide an ideal habitat for fungal germination and development [27].

Although a few reports have described successful therapy with cetoconazole [19] and with itraconazole [24], the factors which make the diagnosis of aspergillosis in penguins difficult are the same that contribute to inefficacy of treatment. Thus, preventative methods are fundamental to the successful maintenance of captive penguins. Adequate temperature and ventilation (15 air changes per hour) are necessary, as well as a good and hygiene with the removal of the organic material and disinfection of all facilities [1, 3, 15, 18, 19; 21, 22, 23, 25, 29].

These measures are frequently adopted in zoos in order to minimize fungal proliferation and conidial air concentration. However, in rehabilitation centers, which frequently house many sick and injured animals in smaller facilities, the environmental conditions are more difficult to control and contamination is higher due to the accumulation of organic material and overcrowding [22].

Weekly disinfection of all the surfaces of all penguin housing is recommended, using different chemical products in order to prevent the development of fungal resistance. To improve hygiene, neither the cage floor nor the walls should be made of porous material [23]. In addition, professionals must be careful to minimize penguins stress. Thus, handling must be done only when necessary, the cage must be calm and a visual barrier can be placed in the cages [1,3,10,15,19]. Adequate nutrition is another important factor in the prevention of respiratory alterations, as hypovitaminosis A which modify the stratified squamous epithelium of the syrinx region developing hypertrophy that can predispose the *Aspergillus* spp. infection [4, 10].

Antifungal prophylaxis with itraconazole (15-25mg/Kg/day for one week) is indicated in the rehabilitation protocols for juvenile penguins and/or debilitated penguins (as with low weight and/or oiled birds) [18, 21, 22, 25]. The same procedure is described in zoo protocols for the maintenance of captive penguins, however in these cases the antifungal has to be administered when the seabirds are in the adaptation period [8]. In addition, zoo

protocols recommend the serological monitoring of anti-*Aspergillus* antibodies in penguins by ELISA every three months, to facilitate early diagnosis and increase the chance of successful therapy [8, 19].

In conclusion, aspergillosis is one of the major causes of death in captive penguins of great economic importance and also in ecology, since it has been cited as a major problem in the rehabilitation of seabirds that suffer following ecological disasters such as oil spills [7, 22, 29]. This mycosis in captive penguins was first described many years ago [2] but continues to have many facets which need to be studied and mechanisms to be elucidated, such as the pathogenesis of the disease and early diagnostic methods to improve treatment and save birds.

REFERENCES

1. Abundis-Santamaria E. Aspergillosis in birds of prey. 2003. Available at: <http://www.aspergillus.org.uk> Accessed: October 2007.
2. Ainsworth GC, Rewell RE. The incidence of aspergillosis in captive wild birds. *Journal of Comparative Pathology and Therapeutics* 1949; 59: 213-224.
3. Andreatti Filho RL. Enfermidades micóticas, In: Berchieri Júnior A & Macari M. Doenças das aves. Campinas: FACTA, 2000: 369-375.
4. Bauck L. Mycoses. In: Ritchie BW, Harrison GJ, Harrison LR. *Avian Medicine: Principles and Application*, Florida: Wingers Publishing, 1994: 997-1006.
5. Beytut E, Özcan K, Erginsoy S. Immunohistochemical detection of fungal elements in the tissues of goslings with pulmonary and systemic aspergillosis. *Acta Veterinaria Hungarica* 2004; 52(1): 71-84.
6. Blanco JL, Guedeja-Marrón J, Caballero J, García ME. Aspergilosis: mecanismos de patogenicidad implicados y aproximación al diagnóstico de laboratorio. *Revista Iberoamericana de Micología* 1998; 15: 10-15.
7. Carrasco L, Lima Jr JS, Halfen DC, Salguero FJ, Sanchez-Cordón P, Becker G. Systemic Aspergillosis in an Oiled Magallanic Penguin (*Spheniscus magellanicus*). *Journal of Veterinary Medicine* 2001; 48: 551-554.
8. Diebold EN, Branch S, Henry L. Management of penguin population in North American zoos and aquariums. *Marine Ornithology* 1999; 27: 171-176.
9. Flach EJ, Stevenson MF, Henderson GM. Aspergillosis in gentoo penguins (*Pygoscelis papua*) at Edinburgh Zoo, 1964-1988. *Veterinary Record* 1990; 126(4): 8185.
10. Fowler GS, Fowler ME. Order Sphenisciformes (Penguins), In: Fowler ME, Cubas ZS. *Biology, Medicine and Surgery of South American Wild Animals*, 1 ed. USA: 2001: 53-64.
11. Graczyk TK, Cockrem JF. *Aspergillus* spp. seropositivity in New Zealand penguins. *Mycopathologia* 1995; 131: 179-184.
12. Graczyk TK, Cranfield MR. Maternal transfer of anti-*Aspergillus* spp. Immunoglobulins in African Black-footed Penguins (*Spheniscus demersus*). *Journal of Wildlife Diseases* 1995; 31(4): 545-549.
13. Hocken AG. Cause of death in blue penguins (*Eudyptula m. minor*) in North Otago, New Zealand. *New Zealand Journal of Zoology* 2000; 27: 305-309.
14. IBRRC. Oil spill history. Available at: <http://www.ibrrc.org/spill_history.html>

Accessed: December 2006.

15. Kearns KS & Loudis B. Avian Aspergillosis. In: Recent Advances in Avian Infectious Diseases, Ithaca NY: International Veterinary Information Service. Available at: <<http://www.ivis.org>> Accessed: October 2007.
16. Khan ZU, Pal M, Paliwal DK, Damodaram VN. Aspergillosis in imported penguins. *Sabouraudia* 1977; 15: 43-45.
17. Kittle D. Zoo without penguins after fatal 02 illness, 06/02/2003. Available at: <http://www.potterparkzoo.org> Accessed: March 2004.
18. Mazet JAK, Newman SH, Gilardi KVK, Tseng FS, Holcomb JB, Jessup DA, Ziccardi MH. Advances in Oiled Bird Emergency Medicine and Management. *Journal of Avian Medicine and Surgery* 2002; 16(2): 146-149.
19. Redig P. General Infectious Diseases -Avian Aspergillosis. In: Fowler ME. *Zoo & Wild Animal Medicine: current therapy 3*. Denver, Colorado: W B Saunders Inc., 1993: 178-181.
20. Rochette F, Engelen M, Bossche HV. Antifungal agents of use in animal health – practical applications. *Journal of Veterinary Pharmacology & Therapeutics* 2003; 26: 31-53.
21. Ruoppolo V, Adornes AC, Nascimento AC, Silva-Filho RP. Reabilitação de pingüins afetados por petróleo. *Clínica Veterinária* 2004; 51: 78-83.
22. Russel M, Holcomb J, Berkner A. 30-Years of Oiled Wildlife Responses Statistics. *Proceedings of the 7th International Effects of Oil and Wildlife Conference*. Hamburg, Germany, 2003: 1-18.
23. Sanchez SL, Garret TD, Sanchez C. Exhibit Modifications to Reduce the Incidence of Aspergillosis and Increase Breeding in Two Penguin Species at the Denver Zoo. Available at: <<http://www.aza.org/AZAPublications/2005ProceedingsReg>>. Accessed: September 2006.
24. Shannon D. Treatment with itraconazole of penguins suffering from aspergillosis. *Veterinary Record* 1992; 130(21): 479.
25. Silva-Filho RP, Ruoppolo V. Sphenisciformes (Pingüim). In: Cubas ZS, Silva JCR, Catão-Dias JL. *Tratado de Animais Selvagens -Medicina Veterinária*. São Paulo, SP: Roca, 2006: 309-323.
26. Stevens DA, Kan VL, Judson MA, Morrison VA, Dummer S, Denning DW, Bennett JE, Walsh TJ, Patterson TF, Pankey GA. Practice Guidelines for Diseases Caused by *Aspergillus*. *Clinical Infectious Diseases* 2000; 30: 696-709.
27. Tell LA. Aspergillosis in mammals and birds: impact on veterinary medicine. *Medical Mycology* 2005; 43(suppl 1): 71-73.
28. Tessari ENC, Cardoso ALSP, Castro AGM, Kanashiro AMI, Zanatta GF. Prevalência de aspergilose pulmonar em pintos de um dia de idade. *Arquivos do Instituto Biológico* 2004; 71(1): 75-77.
29. Xavier MO, Soares MP, Meinerz ARM, Nobre MO, Osório LG, Silva-Filho RP, Meireles MCA. Aspergillosis: a limiting factor during recovery of captive magellanic penguins. *Brazilian Journal of Microbiology* 2007; 38: 480-484.