

Neurological disease and blindness - two case studies

Sue Jaensch¹ and Shane Raidal¹

Case report 1 - a wild peregrine falcon with nervous signs.

A mature peregrine falcon was found 7 km off the coast of Mandurah (approximately 100 km south of Perth) by a fisherman. The bird was reported to be disoriented and taken to a local raptor rehabilitator who received the bird and provided initial care. It was presented for examination 48 hours after capture when the bird was starting convulsing. X-rays showed marked bilateral hepatomegaly. The bird responded well to fluids and antibiotics and a follow-up radiograph 4 days later demonstrated a normal cardiohepatic silhouette. Haematology and serum biochemistry were within normal limits. The bird was returned to the rehabilitation centre.

One day later the bird was readmitted with intermittent convulsions and seemed normal between episodes. Treatment consisted of fluids, antibiotics and EDTA (suspected toxicosis). The bird deteriorated and died approximately 3 weeks after presentation.

At necropsy there were no gross abnormalities. Fresh samples of liver and brain submitted for bacteriology failed to yield significant bacterial growth. Samples of brain, eyes and visceral organs were fixed in buffered formalin for histopathology.

Histopathological examination revealed an extensive proliferative arteriopathy with relatively large 20-30 μ m granular basophilic intracytoplasmic cysts within endothelial cells. The arteries of the brain, optic nerve, and pecten were the most severely affected. However, similar cysts were present in some arterioles of the lung and kidney. A significant inflammatory reaction was not detected. Immunoperoxidase staining of endothelial cysts (kindly performed by Dr Dieter Palmer, Department of Agriculture, South Perth, Western Australia) demonstrated a strongly positive reaction to *Toxoplasma* antigen.

A diagnosis of possible toxoplasmosis was based on the clinical signs, histopathological findings and immunohistochemical staining reactions.

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School of Veterinary Studies, Murdoch University, Murdoch, WA, 6150

Toxoplasmosis is recognised as a sporadic cause of blindness and central nervous disease in canaries, other passerine birds and gallinaceous birds but clinical disease due to natural infection is rare in raptors (Parenti *et al* 1986; Hartley & Dubey, 1991; Dubey *et al* 1992; Vickers *et al* 1992; Bennett, 1994; Lindsay *et al* 1995). In flock situations a presumptive diagnosis of toxoplasmosis can be investigated by histopathological examination. However, a range of formalin-fixed visceral and nervous tissue should be examined to allow for variations in expression of tissue cysts. This case report emphasises that a presumptive diagnosis of toxoplasmosis can be difficult to confirm pre-mortem in individually affected birds. Paired serology, although not attempted in this case may be useful for making a diagnosis of toxoplasmosis. However, it may not have provided a rapid diagnosis in this case since the disease was probably the result of overwhelming acute infection and there may have been insufficient time for the animal to have mounted a detectable antibody response.

Case report 2 - sudden blindness in a rainbow lorikeet

A 4 year old rainbow lorikeet (*Trichoglossus haematodus*) with a two day history of sudden blindness and crashing into aviary walls was referred for necropsy examination. The bird was in good physical condition and, although blind and reluctant to fly, was otherwise neurologically normal. The eyelids, conjunctiva, third eyelid, cornea and anterior chamber of both eyes appeared normal. The pupils displayed normal hippus although tended to remain slightly dilated.

The bird was euthanased and necropsy failed to demonstrate gross pathology. Samples of liver, lung and aqueous humour were aseptically collected for routine bacteriology and cytology. The eyes, brain and major visceral organs were fixed in buffered formalin. Microscopic examination of Diff-Qik and Gram-stained smears of aqueous humour obtained from both eyes and smears of liver and lung failed to demonstrate inflammatory cells or microorganisms. Aqueous humour, and swabs of lung and liver routinely inoculated onto blood agar plates and incubated at 37°C failed to yield bacterial growth after 24 or 48 hours. Samples of the major visceral organs, brain and eyes collected at necropsy and fixed in buffered formalin were processed routinely for histopathological examination.

Histopathological examination demonstrated a severe necrotising pectenitis and focal areas of retinitis in both eyes. There was acute necrosis and a severe infiltration mainly at the base of the pecten with heterophils and macrophages. Amongst the inflammatory reaction was a myriad of monomorphic, filamentous, branching bacteria, approximately 0.5-1.0 µm thick which stained Gram-positive. The organisms were morphologically consistent with *Nocardia* spp. Abundant inflammatory cells were present free within the vitreous humour and attached to the internal limiting membrane of the retina. In some focal areas there was necrosis of the inner layers of retina associated with an infiltration of heterophils. The choroid

and uvea appeared relatively normal.

Several microscopic nodules of granulomatous inflammation were randomly distributed in the lungs and kidneys. These were demarcated by giant cells and heterophils and contained a central core of necrotic heterophils and acellular eosinophilic material. Filamentous bacteria were not seen in these sites. The liver, spleen, alimentary tract and brain appeared histologically normal.

In most psittacine birds the pecten is a highly vascularised, folded vane-like structure attached to the optic disc that projects into the vitreous humour. Rhythmic oscillations of the pecten facilitate perfusion of the retina (Pettigrew *et al.*, 1990). The vascular network of the pecten might be expected to be a site favourable for lodgement of bacteria during bacteraemia or septicaemia. The present case confirms this theory.

The cause of blindness in the lorikeet described in the present case was a severe, acute, septic pectenoretinitis. Failure to culture and identify the bacteria present in the lesions was probably due to the acute nature and localisation of the lesions rather than inappropriate sampling. Despite this the morphology and staining characteristics of the bacteria present within the histological lesions and the nature and distribution of the latter were consistent with acute nocardiosis.

Nocardiosis is an acute, subacute or chronic disease of animals and humans caused most commonly by opportunistic primary infection of the lower respiratory tract following inhalation of *N. asteroides* (Long *et al.*, 1983; Parnell *et al.*, 1983; Koneman *et al.*, 1988). This organism belongs to the nocardioform actinomycetes which are branching, filamentous bacteria widely distributed in soil and organic material. In birds the sinuses of the upper respiratory tract have been reported as a primary site of infection (Baumgartner *et al.*, 1994). Haematogenous dissemination of the organism, as probably occurred in the present case, from the lungs to almost any organ occurs in about 50% of human patients (Koneman *et al.*, 1988). Despite this, blood cultures are not reliable for demonstrating infection. This, along with probably few bacteria in the relatively mild inflammatory nodules present in the lungs and kidneys and localisation of bacteria in the posterior rather than the anterior chambers of the eyes did not favour isolation of bacteria from the samples collected in the present case.

REFERENCES

Case 1

Bennett R.A. (1994) Neurology. In *Avian medicine, principles and application*. Chapter 28 ed.

Ritchie, Harrison, Harrison. pp 723-747

Dubey J.P. *et al* (1992) Toxoplasmosis in owls. *J. Zoo Wildlife Medicine*. 22, 98-102.

Hartley W.J. & Dubey J.P. (1991). Fatal toxoplasmosis in some native Australian birds. *J Vet Diagn In vest*. 3, 167-169.

Lindsay D.S., Gasser R.B., Hanigan K.E., Madill D.N. & Blagburn B.L. (1995). Central nervous system toxoplasmosis in roller canaries. *Avian Diseases*, 39, 204-207

Parenti E. *et al* (1986). Spontaneous toxoplasmosis in canaries and other small passerine cage birds. *Avian Pathology*, 15, 183-197.

Vickers M.C., Hartley W.J., Mason R.W., Dubey J.P. & Schollam L. (1992). Blindness associated with toxoplasmosis in canaries. *J. Amer. Vet. Med. Assoc.* 200, 1723-1725

Case 2

Baumgarmer R., Hoop, R.K. & Widmer, R. (1994). Atypical nocardiosis in a red-lored amazon parrot (*Amazona autumnalis autumnalis*). *journal of the Association of Avian Veterinarians*, 8, 125-127.

Koneman E.W., Allen, S.D., Dowell, V.R., Janda, W.M., Sommers, H.M. & Winn, W.C. (1988). Nocardioforms and aerobic actinomycetes. In *Color Atlas and Textbook of Diagnostic Microbiology*. 3rd Edition, J.B. Lippincott Philadelphia pp 381-385.

Long, P., Choi, G. & Silberman M. (1983). Nocardiosis in two Pesquet's parrots (*Psittichas fulgidus*). *Avian Disease*, 27, 855-859.

Parnell, M.J., Hubbard, G.B., Fletcher, K.C. & Schmidt, R.E. (1983). Nocardia asteroides infection in a purple-throated sunbird (*Nectarinia sperapa*). *Veterinary Pathology*, 20, 497-500.

Pettigrew J.D, Wallman, J. & Wildsoet, C.F. (1990). Saccadic oscillations facilitate ocular perfusion 'from the avian pecten. *Nature*, 343, 362-363.