

Blindness as a sign of proventricular dilatation disease in a grey parrot (*Psittacus erithacus erithacus*)

An approximately eight-year-old female grey parrot (*Psittacus erithacus erithacus*) was presented with a two months history of blindness. The radiographic examination showed a dilatation of the proventriculus, ventriculus and gut. Ophthalmoscopy and electroretinography revealed degeneration of the retina. A proventricular dilatation disease was suspected. The bird was euthanased because of deteriorating condition and poor prognosis. The pathological examination showed an atrophy of the ventricular muscles and lymphoplasmacytic infiltrates of the myenteric plexus of the proventriculus, ventriculus and gut as well as moderate lymphoplasmacytic infiltrates of the cerebrum with moderate neuronophagia. Lymphoplasmacytic infiltrates in the retina, indicating proventricular dilatation disease, and subsequent retinal degeneration were found. A potential common aetiology for proventricular dilatation disease and blindness is discussed.

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CASE REPORT

History and initial clinical findings

An approximately eight-year-old female grey parrot (*Psittacus erithacus erithacus*) was presented with a history of blindness for two months. The movements of the bird became hesitant, and it would climb along the bars rather than fly. When presented, the bird showed a bad body condition and weighed 383 g. The grey parrot appeared blind, would not fly and demonstrated a fear response when touched.

Ophthalmological examination

A full ophthalmologic examination was performed. Vision was bird specific tested by dropping cotton and approaching the head with a cotton bud (Steinmetz and others 2002) The bird showed no reaction to the falling cotton and responded to the cotton bud only when it touched the head or the feathers. The pupils were permanently dilated even when the bird seemed calm.

Slit lamp biomicroscopy (SL 14; Kowa) revealed no abnormalities of the anterior chamber, the iris or the lens (Fig 1). With indirect ophthalmoscopy (with 70 D lens), a normal vitreous and pecten could be seen in OU. In both eyes, the dorsomedial area of the fundus appeared brighter than expected because of severe streaky loss of pigmentation of the retinal pigmented epithelium with the choroid shining through. The intraocular pressure was 9 mmHg in OS and 6 mmHg in OD, respectively (TonoPen XI, Mentor O&O, Norwell, MA, USA, topical anaesthetic oxybuprocaine hydrochloride). An electroretinography (ERG) was performed as described for birds (Korbel and Stütz 1999) under general anaesthesia with isoflurane (Fig 2), no signal was detected. These findings led to the diagnosis of bilateral retinal degeneration.

Additional laboratory and imaging techniques and results

In swabs taken from choana, crop and cloaca *Pasteurella multocida* was isolated. Budding yeast was detectable in the cytological specimen (Diff-Quick, Hamburg, Germany) of the crop. No infections were found on parasitological faecal examination. The dropping collection and the serological screening with Widal reaction for salmonella detection were negative.

Radiography was performed in a ventrodorsal and lateral view and demonstrated increased soft tissue density, indicating proventricular enlargement (Fig 3). A proventricular dilatation disease (PDD) was suspected.

Treatment and outcome

Therapy was initiated as recommended for the treatment of PDD (Gelis 2006). Fluid substitution with amino acid solution (Aminin; Merial), Ringer's solution and glucose five per cent solution (Braun Melsungen AG) in a dosage of 20 ml/kg once a day, 300·000 IE/kg anti-infective drugs orally once a day (nystatin, Moronal Suspension; Dermapharm), 150 mg/kg amoxicillin and clavulanic acid intramuscularly

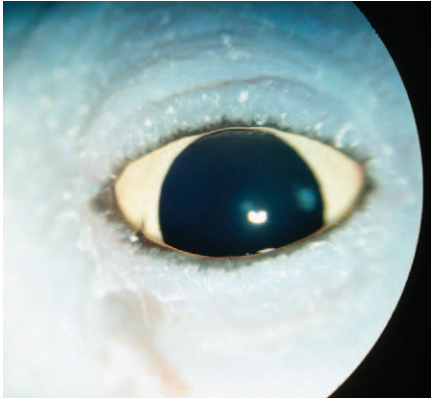


FIG 1. OS: dilated pupil, no other abnormalities



FIG 2. Electroretinography in the grey parrot

twice a day (Augmentan, GlaxoSmith-Kline) and 10 mg/kg non-steroidal anti-inflammatory drug once a day (celecoxib, Celebrex; Pharmacia) were given. The bird was tube-fed with 20 ml/kg once a day Harrison's recovery formula (HBD).

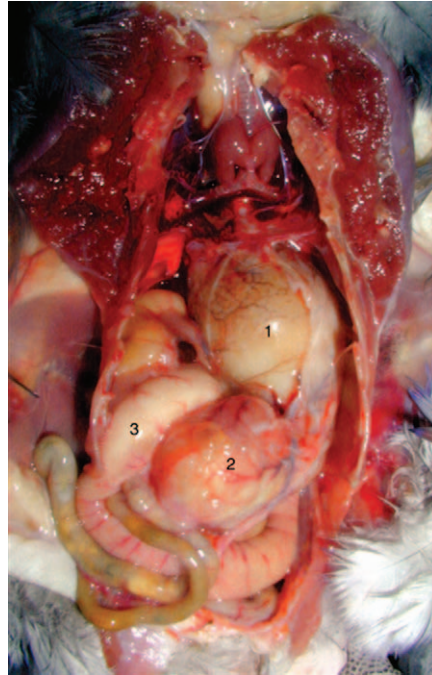


FIG 4. Section: a moderately dilated and ingesta-filled proventriculus (1), ventriculus (2) and gut (3) as well as an atrophy of the muscles of the ventriculus

After 17 days of treatment, the bird was euthanased because of its deteriorating condition and progressive weight loss.

Necropsy and pathological results

The main gross lesions included poor body condition, a dilated (about 4×4×4 cm³) and paste-filled proventriculus (Fig 4).

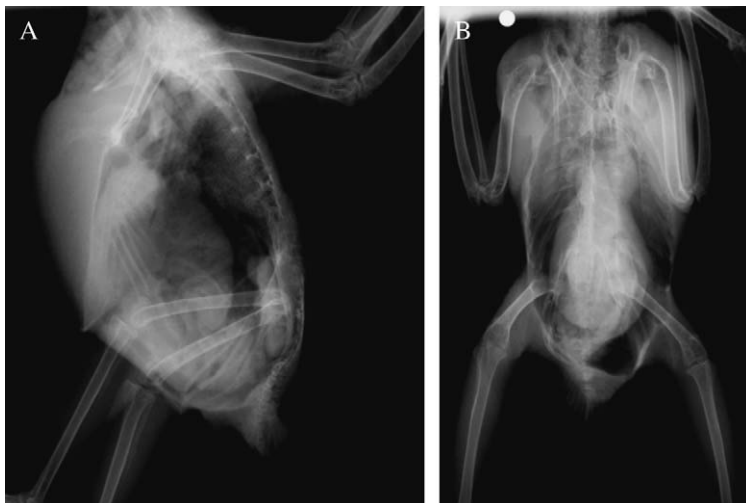


FIG 3. X-ray, laterolateral (left) and dorsoventral recumbency (right): proventricular enlargement

Histological lesions were characterised by severe lymphoplasmacytic infiltration of the myenteric plexus of the proventriculus, ventriculus (Fig 5) and gut as well as moderate lymphoplasmacytic infiltration of the cerebrum with moderate neuronophagia.

Considerable histological lesions of the bulbus oculi were characterised by the following findings: there were multi-focal signs of retinal degeneration characterised by loss of photoreceptors (mainly of the rods), partial retinal ablation, activation of microglia and mild lymphoplasmacytic infiltration with partial retinoschisis. The multi-focal atrophy involved the papilla and two thirds of the retina including the fovea. A multi-focal mild inflammatory response consisting of predominantly lymphoplasmacytic infiltration and discrete heterophilic infiltration could be seen (Fig 6). Chorioretinal scars were detected. The histological lesions in the ocular system were comparable to the lesions found in other organs of the grey parrot.

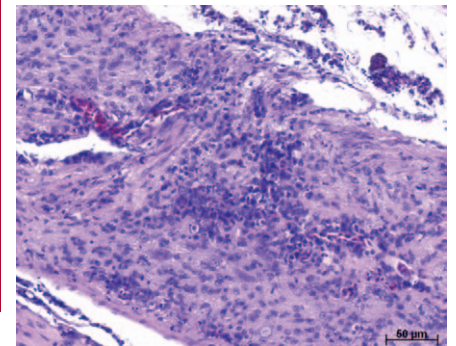


FIG 5. Histopathology of the myenteric plexus (H&E staining ×20): severe lymphoplasmacytic infiltration

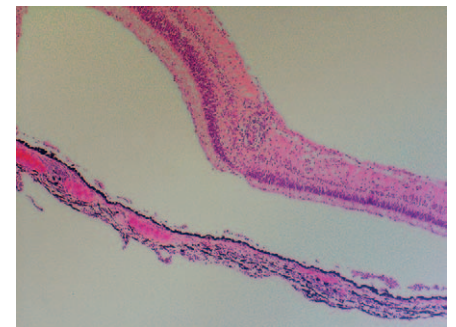


FIG 6. Histopathology of the retina (H&E staining, ×20): multi-focal mild inflammatory response consisting of predominantly lymphoplasmacytic infiltration

DISCUSSION

Blindness after traumatic insults is a result of haemorrhages arising from the pecten oculi and retinal detachment following shrinking of intravitreal fibrin clots (amotio retinae) (Korbel 1999). The funduscopy in the grey parrot revealed no signs of an older or an acute traumatic insult.

Macrophages containing the tachyzoite form of *Toxoplasma gondii* were not seen in this case, so that toxoplasmosis could be ruled out.

PDD, also named neuropathic gastric dilatation, has been found worldwide in more than 50 psittacine species including grey parrots (Gregory 1995). The origin of this disease is still unknown, although current research suggests a yet unidentified neurotropic virus as primary trigger for the mononuclear cell inflammatory response associated with PDD (Gregory 1995, Phalen 2006). Clinical signs are correlated with innervation deficiencies of various organs. The most common finding is the gradual wasting as a result of disturbed motility of the gastrointestinal tract, often combined with undigested seeds in the faeces. This was observed in the present case during hospitalisation. As reported in literature, proventricular dilatation could be seen on radiographs and at necropsy in this case (Phalen 2006). Prognosis for PDD-affected birds is poor, death because of circulatory collapse or food aspiration is common (Gregory 1995).

Today, the only definite diagnosis for PDD is the finding of lymphoplasmacytic infiltration in the ganglia and nerve plexus, especially the myenteric plexus of the gastrointestinal tract (Schmidt and others 2003). In the present case, lymphoplasmacytic infiltration was observed in the myenteric plexus of the ventriculus and gut. These infiltrations are reported in nerve tissue in different organs and also in the optic nerve (Schmidt and others 2003).

Ophthalmoscopy revealed large streaky brightened areas in both eyes. Furthermore, the absent signal in ERG confirmed the diagnosis of retinal degeneration and showed a functional loss of the complete retina and the visible histological changes confirmed the diagnosis. Histopathological examination revealed lymphoplasmacytic infiltration and subsequent degeneration of the retina, whereas the optic nerve showed no abnormalities.

The dilatation of proventriculus, ventriculus and gut, as well as the atrophy of the muscles of the ventricle in connection with the lymphoplasmacytic infiltration of the myenteric plexus of the proventriculus, ventriculus and gut, as well as a moderate lymphoplasmacytic infiltration of the cerebrum with moderate neuronophagia confirm the diagnosis of PDD in the present case. Similar lymphoplasmacytic infiltration of the retina and subsequent retinal degeneration and blindness seen here have as of yet not been described. So it is assumed that the initially recognised blindness in the present case was part of the

PDD, as confirmed by histopathology and absence of other disease processes that could be responsible for the blindness.

Conclusion

Blindness as a result of retinal degeneration may be one of the first signs of PDD in birds. In a bird that potentially suffers from PDD, vision should be tested. Ophthalmoscopy and ERG (if possible) should be performed if vision is decreased. Signs of retinal degeneration could stand in context to the general disease PDD of this bird.

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