Assessing and managing signs of neurological disease in birds

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Captive birds frequently present as emergency cases when suffering from clinical signs of neurological disease.

Table 1. Differential diagnoses for neurological signs in avian patients

	Commonly affected birds	Additional notes
Chlamydia psittaci (psittacosis)	Parrots, columbiformes, galliformes	Zoonotic
Listeria monocytogenes		
Botulism	Waterfowl	Typically seen in autumn
Mycobacteria species	Waterfowl, Amazon parrots	Low zoonotic species typically
Sarcocystis species	Pigeons	
Isospora (atoxoplasma) serini	Passerines	Diagnosed from buffy coator impression smears (**Figure 5**)
Toxoplasma gondii		
Baylisascaris procyonis		Not present in the UK
Proventricular dilation syndrome (avian bornavirus)	Parrots	
Paramyxovirus	Pigeons, passerines, poultry	Newcastle disease is notifiable in chickens
Hypocalcaemia	Parrots, juvenile birds	
Hypoglycaemia		
Hyperglycaemia	Goshawks	
Hyperlipaemia	Parrots	
Athehrosclerosis	Parrots, raptors	
Hypovitaminosis B	Raptors, poultry	
Vitamin E/selenium deficiency	Juveniles	
Herpesvirus	Poultry, raptors (especially owls)	
West Nile disease	Passerines, raptors	Not in the UK
Avian influenza	Waterfowl, poultry, pelagic birds	High path strains are notifiable
Cryptococcus neoformans		
Aspergillosis (including spinal form)	Penguins, parrots, raptors	
Organophosphate/carbamate toxicity	Free-flying birds	
Chloroquine toxicity	Penguins	
Heavy metal intoxication (lead, zinc, copper)	Parrots, raptors, waterfowl	
Egg binding	Parrots, chickens	
Testicular neoplasia	Budgerigars, pigeons	
Traumatic injury to head/ limbs/spine		

Table 1. Differential diagnoses for neurological signs in avian patients.

Some of these conditions are easily managed and carry a good prognosis, but others may be

terminal and of significant infectious potential, so it is important to be able to triage and investigate cases appropriately at an early stage.

Birds tend to present late in the course of clinical disease and deteriorate rapidly, so awareness of likely causes for the species and clinical signs enables rapid targeted diagnostics and offers the best potential for recovery. Differentials for neurological clinical signs in birds are listed in **Table 1** and the most common conditions in UK birds are discussed in this article.

Parrots

Hypocalcaemia

Clinical hypocalcaemia is a common condition for pet parrots, particularly African grey parrots (*Psittacus erithacus*). Husbandry is frequently implicated with parrots fed on a seed diet developing a deficiency in calcium due to low dietary levels.

A seed diet and lack of exposure to natural or artificial UV light also leads to a vitamin D deficiency, compounding the problem by reducing dietary absorption of the available calcium. Rarely renal disease results in a similar clinical process.

Initial compensatory mechanisms result from low circulating calcium levels with the release of parathyroid hormone, resulting in liberation of mineral from the bone, decreased renal calcium losses and increased intestinal uptake. When this becomes a chronic situation, bone stores are gradually depleted and serum calcium levels cannot be adequately restored, leading to clinical signs.

Hypocalcaemia results in neurological signs, including ataxia, tremors and seizures. Other changes may include bone deformities, delayed clotting and dystocia. Clinical signs develop rapidly in young, growing animals and bone deformities are often much more marked (**Figure 1**).

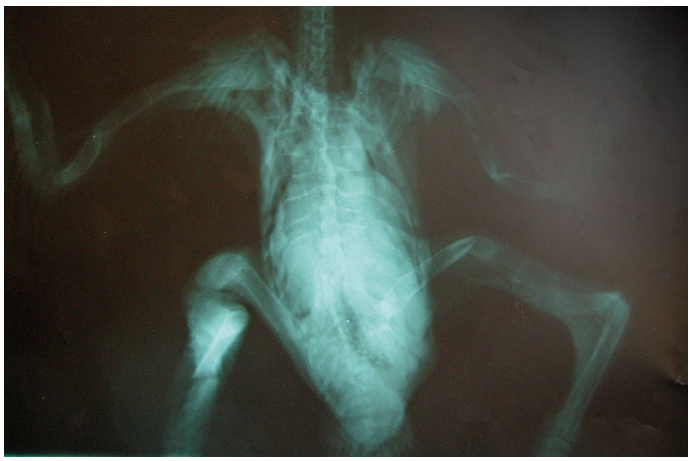

Figure 1. Osteodystrophy in a juvenile Harris' hawk fed an exclusively whole meat diet and deprived of UV light.

Diagnosis is by identification of related clinical signs, radiography demonstrating decreased bone mineralisation and/or dystrophic changes, and assessment of markers of calcium homeostasis on biochemistry. Ionised calcium levels are preferred for assessment as the amount of total calcium is an unreliable measure of physiologically available calcium.

In mild chronic cases, ionised calcium may appear normal due to compensatory mechanisms, but in clinical cases exhibiting neurological symptoms a depressed ionised calcium level is expected. Normal ranges are not established for many species, but in African grey parrots a suggested reference range is 0.96mmol/L to 1.22mmol/L, with values less than 0.75mmol/L supporting a diagnosis of clinical hypocalcaemia. Reduced serum vitamin D_3 and elevated parathyroid hormone levels are preferred for confirmation of diagnosis, but assays are not commercially available.

Treatment of established hypocalcaemia involves controlling the neurological symptoms and stabilising the calcium levels. In mild cases, correction of serum calcium alone can lead to rapid cessation of neurological symptoms. Oral or injectable calcium salt preparations can be used. In advanced cases, where seizures are evident, it may be necessary to use sedatives alongside administering calcium.

Long-term husbandry improvements are needed to address the underlying causative factors and prevent recurrence. Simple measures include conversion to a nutritionally complete diet, provision of UV light at wavelength 315nm to 280nm for a minimum of four hours per day and calcium supplementation until bone stores are restored.

Psittacosis

Chlamydophila psittaci is an obligate intracellular bacterium widespread in pet parrots and capable of infecting many bird families and their human carers. Inhaled or ingested elementary bodies lead to bacteraemia and migration to organs – primarily the liver and respiratory system, causing clinical signs.

Not all birds develop clinical disease and asymptomatic carriers can act as a reservoir of infection, or develop disease at a later stage if immune function is suppressed. Where clinical disease is seen it can include lethargy, anorexia, upper and lower respiratory signs, diarrhoea or neurological symptoms, such as opisthotonus, ataxia, seizures and tremors.

In atypical forms of chlamydiosis, neurological signs can occur in isolation, complicating diagnosis. Diagnostic methods include PCR for *C psittaci*, performed on faecal matter (which can result in false negatives due to intermittent shedding) or detection of antibodies in serum.

Treatment of neurological psittacosis involves supportive care, sedatives or anticonvulsants, where necessary, and appropriate antibiotic therapy to attempt to eliminate the pathogen. Tetracycline antibiotics, most frequently doxycycline, are used for continuous six-week treatment and effective in the majority of cases.

Azithromycin has recently shown promise as an alternative therapy in cockatiels and fluoroquinolones have partial efficacy in treatment, but may not eliminate the disease. It is recommended to repeat testing following treatment to confirm the absence of bacteria on PCR or a decline in serum antibody levels to support successful treatment.

Proventricular dilatation syndrome

Figure 2. Undigested seed in the faeces of a cockatiel with suspected proventricular dilatation syndrome.

Psittacine proventricular dilatation syndrome (PDS) can cause central and peripheral neuropathies as well as common intestinal signs, including weight loss, undigested food in faeces and delayed crop emptying (**Figure 2**). Clinical signs result from progressive neuronal damage and disruption of neurotransmitter activity, most frequently affecting the nerves to the intestinal tract.

It is thought viral infection not only damages nerves directly, but that viral coat proteins resemble neurotransmitters, so antibodies against these may cross-react with the host's own proteins. The resulting autoimmune destruction of neurotransmitters complicates the neurological dysfunction.

The most marked changes tend to be seen in the proventriculus that, with flaccid paralysis, can increase dramatically, leading to the common name for the condition (**Figure 3**).

Comparison of proventricular diameter to keel height allows an objective assessment of proventricular size. It has been demonstrated the proventricular diameter in a bird without

proventricular disease (including PDS and other infectious, obstructive and neurological conditions) should be no more than 48% keel height.

Histology of a crop biopsy has been used for diagnosis and if the biopsy shows lymphoplasmacytic ganglioneuritis alongside proventricular dilation, then a diagnosis of PDS can be made. A suspected aetiological agent of avian bornavirus was identified in recent years, allowing more targeted testing by PCR and serology becoming the preferred testing regimen.

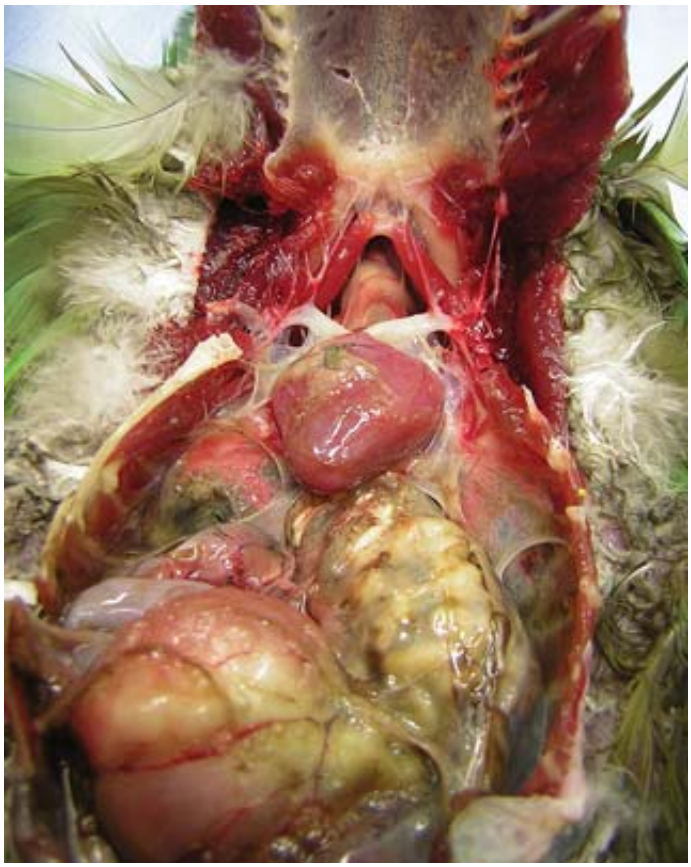

Figure 3. Markedly distended, thin-walled proventriculus in an Amazon parrot with PDS.

Treatment has been limited to controlling the immune reaction to the virus and secondary damage to nerves and meloxicam has been widely used with variable effects. Celecoxib, a COX-2 specific NSAID, has shown more consistent benefits, but does not come in a convenient liquid formulation.

Corticosteroids are not used due to the immunosuppression they induce in avian patients. No treatment resolves the condition and the aim is to manage the inflammation and immune response, slow the degenerative process and prolong lifespan, but clinical PDS remains a fatal condition.

Sciatic nerve compression

Sciatic nerve compression can result from the passage of a large egg or presence of a space-occupying mass, and presents with a bird unable to stand on one or both legs. Egg binding is often associated with uterine inertia secondary to calcium homeostasis disruption and management of both aspects of the condition is necessary.

Supportive care in a quiet environment with glucose administration, fluid therapy, calcium administration, analgesia and nesting site provision may be sufficient to encourage laying. If this fails, or the bird is sufficiently compromised to be unlikely to lay the egg, then aspiration and decompression of the egg per cloaca or coeliotomy to remove the egg is advisable.

Testicular tumours are most frequently seen in budgerigars and may be virally induced. Hindlimb weakness, lameness and paralysis are seen where sciatic nerve root compression occurs with large neoplasm development. Some sertoli cell neoplasms are functional, with hormone secretion resulting in cere colour changes in budgerigars.

Complete surgical removal of testicular neoplasia has been attempted, but is technically highly demanding due to the extensive nature of these tumours and small size of patients. Deslorelin implants have, anecdotally, had good responses in a number of budgerigars, but are not a consistently reliable treatment due to the varied response of different tumour types to endocrine suppression.

Chickens

Gallid herpesvirus 1 (Marek’s disease) is a highly contagious retrovirus that induces lymphoproliferative disease in poultry. The virus is widespread and persists for long periods in the environment. Clinical disease is widely recognised in chickens, but has also been reported in turkeys and Japanese quail.

Neoplastic leukocytes infiltrate various tissues, including peripheral nerves. The disease frequently manifests in young birds at the point of laying, presenting with leg weakness, lameness, dropped wings, opisthotonus and paralysis, despite an apparently alert and responsive demeanour.

Supportive care can prolong survival and cases of spontaneous recovery have been reported, but this is unlikely. Prevention is by vaccination of eggs or chicks at one day of age, prior to potential exposure.

Raptors

Figure 4. A goshawk with knuckled over feet secondary to lead intoxication.

Heavy metal intoxication is a common cause of neurological symptoms in pet and wild birds. Lead is most commonly seen in captive and free-living raptors where shot quarry is consumed.

Birds may present with central or peripheral nerve defects. These may include leg weakness, plantigrade stance, ankle grasping, abnormal posture or toes knuckled under (**Figure 4**). Central signs can vary, from apparent normality, but reduced flight performance to cluster seizures.

Radiography may show radiodense metallic particles within the proventriculus, but birds of prey can cast (regurgitate) with other indigestible pieces of food as part of their normal feeding strategy, leaving no trace on radiographs. Blood analysis may reveal mild leukocytosis, elevation of liver parameters and hypochromic anaemia. Definitive diagnosis involves blood lead assays, but if suspicion is high, chelation therapy may be commenced after sample collection, but prior to result availability.

Where possible, metallic particles should be removed by careful proventricular flushing under general anaesthesia, or endoscopic retrieval. Sodium calcium edetate injections are the heavy metal chelation therapy of choice and should be administered in clinical cases, even if particle removal is successful to chelate remaining ionic lead that has already been absorbed or remains in intestinal content.

Fluid therapy is essential to minimise renal compromise from both absorbed metallic salts and the potentially nephrotoxic effects of chelation. Post-therapeutic relapses can occur with lead intoxication due to rebound increase in blood levels following the release from bone deposits. In such cases, repeat chelation is indicated until the reservoirs are exhausted.

Hypoglycaemia

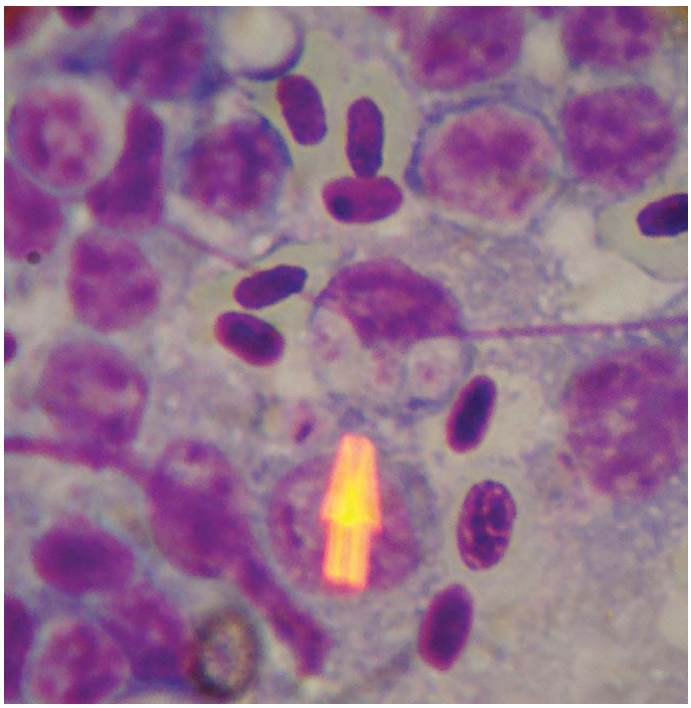

Figure 5. *Isospora serini* inclusions in an impression smear from a finch.

Hypoglycaemia is associated with septicaemia, chronic stress, exertion of an underweight bird, or acute food deprivation.

Wild birds with concurrent pathology or captive raptors kept at a very low flying weight are more susceptible. Birds may present as weak, collapsed or showing neurological signs, such as incoordination or convulsions.

A blood glucose below 5mmol/L in a bird with clinical signs supports clinical hypoglycaemia requiring intervention.

Uncomplicated hypoglycaemia can be rapidly corrected with intravenous or oral glucose, or dextrose-containing fluids. Endocrine and hepatic disease may cause secondary hypoglycaemia, so a failure of recovery, despite glucose therapy, merits further investigation.

Hyperglycaemia

A clinical syndrome of stress-induced hyperglycaemia leading to seizures has been reported in goshawks, but is not recognised in other species. Supportive care, oral hypoglycaemics and insulin therapy have all been used for management.

Trauma

Limb fractures and spinal subluxation are not uncommon collision injuries in wildlife casualties and captive raptors. A known history of trauma, or acute onset paresis in a free-flying bird, indicates radiographic assessment of the skeleton is required.

Separation of the spinal regions of the notarium and synsacrum can present atypically as a slow-developing paresis, secondary to progressive compression of the spinal cord due to spinal instability and expanding callus formation, and is best diagnosed with advanced imaging, such as CT or MRI.

- Some drugs mentioned are used under the cascade.

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