Common medical conditions in pet psittacines

Here, Tom Dutton and Neil Forbes provide an exploration of four commonly encountered medical conditions in pet psittacines presented to their practice.

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Declaration of interests: Tom Dutton and Neil Forbes both work for the Vets Now group in Swindon.

CPD

Aspergillosis

Aspergillosis is a fungal disease affecting pet aviary birds. Aspergillus fumigatus is by far the most commonly isolated species. Aspergillus niger is the next most common isolate. The fungus is ubiquitous in the environment. A healthy bird in a normal environment will cope with the expected day to day level of Aspergillus spp. spores that they encounter. One or two scenarios, however, result in clinical disease:

1. If the bird is immune-compromised due to stress or concurrent disease (for example, PBFD) their immune defences can often no longer cope with normal Aspergillus spp. levels. Traditionally, this was most commonly seen in imported wild caught parrots, many of whom would die en route or soon after arrival due to the stress of confinement and transportation.
2. Clinical disease can occur when a parrot is exposed to abnormally high levels of Aspergillus spp. spores. The fungus grows on damp or decaying vegetable material. The worst possible source of Aspergillus spp. spores is hay. However, with regards to parrots, in the author's opinion, the commonest source is sunflower or other seeds that have been harvested or stored damp (with a moisture content of over 16 per cent).

This is most often a problem when the shipment of sunflower seeds has suffered weevil attack, so that some seeds have a small hole bored into them. Moisture gains access into the seed and fungal growth can proliferate. Any owner feeding a seed based diet is advised to select a good quality seed. On purchasing any new batch, it is advisable to take half a mug full of seed, break open the shells and check the seeds inside. The inner seed should be clean, white and dry. If there is any sign of brown, green or black dust or any other deposits, then the batch of food must be discarded.

Clinical Signs

Clinical disease affects three different anatomic loci. Aspergillosis can be classified as:

Type 1. Syringeal/ tracheal form

Localised to the distal trachea and syrinx. This is characterised by a change in voice, inspiratory, expiratory or biphasic respiratory stridor. These lesions are generally only visible on tracheal endoscopy (tracheoscopy). Unlike other forms of Aspergillosis, haematology is typically unremarkable. Suspect cases should be submitted for tracheoscopy as an emergency (**Figure 1**). Where significant dyspnoea is present, an air sac breathing cannula should be placed to stabilise the patient while diagnosis is confirmed and specific treatment is instigated (**Figure 2**).

Type 2. Pneumonic form

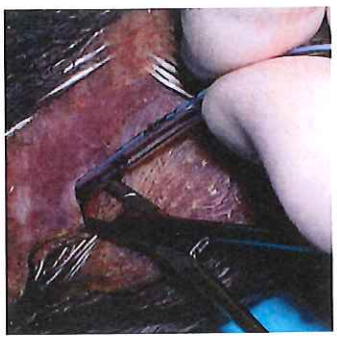
Most commonly seen with excessive environmental spore contamination. Severe respiratory distress, cyanosis and a grave prognosis.

Type 3. Air sac form

Aspergillosis is contained within the air sacs. The caudal air sacs are most likely to be affected. In cases where the air sacs are involved, typically no respiratory signs are observed. Instead, patients tend to suffer from non-specific clinical signs; weight loss, inappetence, green urates/faeces. Haematology reveals a severe leukocytosis. Biochemistry often shows a derangement in the liver enzymes. Focal air sac consolidation will be seen on the radiographs. A diagnosis is confirmed with coelomic

Figure 1. *Syringeal aspergilloma as viewed via a 2.7mm 0° rigid endoscope.*

Figure 2. *Placement of an air sac breathing cannula between ribs seven and eight on the left hand side.*



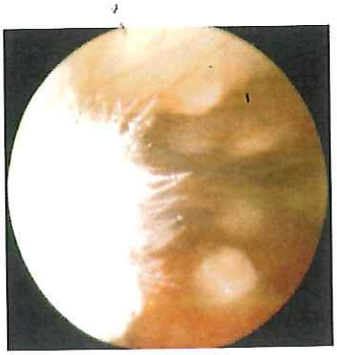


Figure 3. *Air sac viewed during coelomic endoscopy with a typical aspergillus granuloma.*

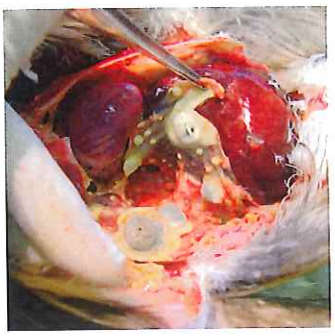


Figure 4. *Air sac aspergillomas on a post-mortem specimen.*

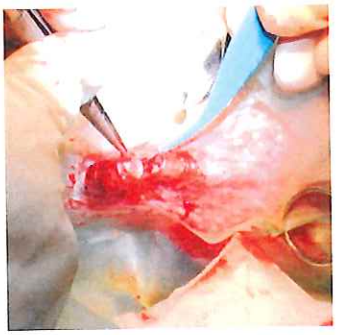


Figure 5. *Resection and anastomosis of tracheal aspergilloma.*

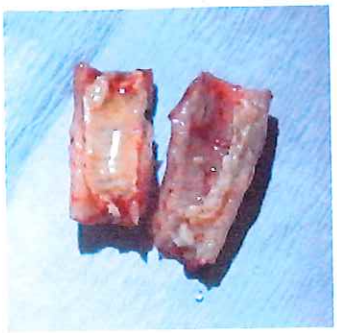


Figure 6. *The resected portion of trachea with its associated aspergilloma.*

endoscopy and cytology/culture. Visible yellow/green masses are associated with Aspergillis fumigatus. Brown masses are associated with Aspergillis niger. In-house cytology of biopsy samples reveals tubular filaments with regular septa and irregular branching at a 45 degree angle (**Figures 3 and 4**).

Treatment

Traditional medical therapy for Aspergillosis involved the use of itraconazole (toxicity reported in grey parrots) or amphotericin B (decreased efficacy has been reported with time). Voriconazole, a second generation triazole, has significantly improved reported recovery rates in birds with Aspergillosis (Di Somma et al, 2007). In a study using voriconazole in human patients with systemic mycosis, where treatment had failed with amphotericin B or itraconazole, nine out of thirteen patients had a favorable response. Despite advances in medical treatment, pulmonic or systemic Aspergillosis are associated with a very guarded prognosis.

In some cases of trachea Aspergillosis, resection and anastomosis of a portion of the trachea is required to excise a well-adhered granuloma (**Figures 5 and 6**). In most psittacines, up to six tracheal rings can be safely removed. Some soft granulomas in the trachea or syrinx can be removed by endoscopic guided suction following the placement of an air sac breathing cannula. Medical therapy with antifungal agent should be maintained for two months. Haematology should be repeated at the end of the treatment course and at one, three and six months after completing therapy so any relapse can be identified at an early stage of the disease.

Psittacosis

Psittacosis is caused by the obligate intracellular bacterium Chlamydophila. Prevalence of infection among pet psittacines has been reported to range from between 10 and 30 per cent. It is a zoonotic infection; human cases are commonly associated with exposure to a pet bird. Disease severity in humans varies from subclinical infection to sepsis and organ failure.

Typical clinical signs include persistent flu-like symptoms, headache, respiratory distress, fever, confusion, or cough. Dyspnoea progressing to pneumonia, and elevated hepatic enzymes are also seen. Most immune-competent people infected show only mild signs. Atypical presentations with greater severity include renal disease, hepatitis, pancreatitis and inflammatory arthritis. Cardiac manifestation of the disease is seen with mortality approaching 50 per cent. Carers/owners of suspected cases should be provided with written advice about the zoonotic potential of the infection.

Chlamydophila psittaci's elementary bodies are environmentally stable and can survive for three months in soil and one month in avian faeces. Infection follows inhalation of an elementary body, which attaches itself to a eukaryotic cell – most frequently a respiratory epithelial cell. The incubation period is very variable (three days to several weeks).

Vertical transmission does occur and the young are very susceptible to clinical disease, and may die soon after hatching or while still in the nest. Asymptomatic infection is common especially in pigeons, waterfowl and passerines, but also in psittacines (particularly cockatiels). Stress (reproduction, nursing young, deficient husbandry, etc) all increase the chance of clinical disease resulting from infection.

Clinical signs

Clinical signs in pet psittacines are non-specific. None of the clinical signs are unique to Chlamydiosis or pathognomonic. Common signs reported include:

- Ocular and nasal irritation and discharge
- Reduced appetite, anorexia
- Dyspnoea – clouded air sacs on radiographs and endoscopy
- Lethargy
- Weakness and depression
- Diarrhoea
- Bright green urates – due to hepatic disease
- No signs at all

Diagnosis

A number of tests are available for diagnosing Chlamydophila psittaci in pet psittacines. Antibody detections have a high sensitivity but low specificity. Antigen detection has a low sensitivity and high specificity with a high incidence of false negatives. The most common post-mortem findings are splenomegaly, hepatomegaly and clouding of the air sacs (**Figures 7 and 8**). None of these findings are pathognomonic. Cytological examination of impression smears may reveal organisms. If culture is to be performed, the liver and spleen are the most commonly sampled organs. Antibody detection only confirms exposure to the bacteria, not infection, and can therefore lead to false positives.

In recently infected birds (up to two weeks), seroconversion may not have occurred. However, due to intermittent shedding of bacteria (particularly in asymptomatic carriers), antigen detection can lead to false negatives. The National Association of State Public Health Veterinarians in the USA has recently published guidelines that advise against prophylactic treatment.

To date no antibiotic resistance has been reported

with Chlamydophila psittici, but resistance is reported in other Chlamydophila species.

Treatment

The recommended treatment for Chlamydophila psittaci has traditionally been doxycycline for 45 days. Chlamydophila is not susceptible to antimicrobials while intracellular and therapeutic duration greater than the macrophage lifespan is required. A long acting form of injectable doxycycline (Vibravenous) can be imported from Europe under licence, and is the treatment of choice for pet birds. Large collections and aviary birds can be treated with medicated food or water.

Pharmacokinetic studies looking at a shorter course of treatment have shown initial success in experimentally infected birds but have yet to be evaluated in naturally infected psittacines. As the pathogen is spread in feather dander and faeces, all the birds sharing the same air space should be treated.

Prevention and biosecurity

Infected or potentially infected birds should be isolated. Good husbandry and a stress free environment are important. Veterinary facilities treating birds should have adequate isolation to avoid spread from infected or untested patients. Personal protective equipment (PPE) should be worn by all staff members in contact with potentially infected birds. Nutritional and supportive care should be provided in clinically sick birds.

Prior to its removal, faecal material and contaminated substrate/toys should be sprayed down to prevent aerolisation of the bacteria. Full disinfection and cleaning, including fogging of the environment with a suitable disinfectant (such as F10SC), should be performed regularly throughout treatment.

Heavy metal toxicity

In veterinary medicine, the term "heavy metal" is generally defined as any metal that is potentially toxic. In the true chemical sense, a heavy metal is a metallic substance that does not naturally occur in the body. Trace elements such as iron, copper and zinc are therefore not technically heavy metals, although are usually covered under the heading of heavy metal.

Zinc is by far the most common metal toxicity in psittacine birds. Lead was traditionally the most commonly diagnosed toxicity, but as lead has been removed from the home (paint, pipes, etc) due to human toxicity concerns, the number of pet avian cases has also decreased.

Common sources of avian zinc toxicity include (please note, this list is not exhaustive):

- Galvanized wire cages
- Galvanized toys - the process of galvanization may include coating with a metal alloy that is more than 98 per cent zinc and can contain 1 per cent lead
- Food and water dishes (especially when new)
- Children's toys
- Metallic fixtures and fittings (C-clamps, padlocks, etc)
- Zinc batteries
- Pennies - ingestion of pennies is often quoted in the literature but this occurs in the USA where pennies contain a high percentage of zinc

> "Zinc is by far the most common metal toxicity in psittacine birds"

Clinical signs

Clinical signs of zinc toxicity may include:

- Lethargy/weakness
- PUPD
- Diarrhoea, sometimes haemorrhagic (**Figure 9**)
- Regurgitation - crop stasis
- Neurological signs
- Haemoglobinurea
- Breeders may see decreased fertility
- Routine blood work will often reveal an anaemia and/or heterophilia
- Feather colour changes

Diagnosis

Survey radiographs should be performed to evaluate for metallic densities within the gastrointestinal tract (**Figure 10**). However, in many cases no metallic foreign objects are found. Plasma zinc concentrations up to 32mmol/l are suggestive of toxicosis. All blood samples collected for zinc assays must be collected in syringes without a rubber top. Haemolysis of the blood sample can lead to falsely elevated zinc levels on many in-house biochemistry analysers. The liver, kidney and pancreas are the most useful tissues to be analysed at post-mortem examination.

Treatment

Treatment is based around removing ingested heavy metal and chelation therapy. If large metallic objects are present, techniques such as endoscopic removal, gastric lavage or surgical removal can be utilised.

If small non-retrievable particles are present, cathartics can be used to expedite their removal. The chelation agent of choice is CaEDTA. Fluid therapy should accompany chelation therapy to prevent possible nephrotoxicity. Treatment should continue for five days after the zinc source has been removed, although longer treatment regimes are occasionally required.

Penicillamine is another chelation agent that can be used in milder toxicities. It can be given via an oral route - regurgitation is the most common side affect. Supportive therapy, nutritional and fluid support should be provided.

Proventricular dilatation disease (PDD)

PDD is predominantly a disease of pet psittacines and is a common cause of morbidity and mortality in parrots in the UK. Species

Figure 7. *Ventro-dorsal radiographic projection from a grey parrot with Chlamydophila psittaci showing bilateral diffuse cloudy opacity of the air sacs.*

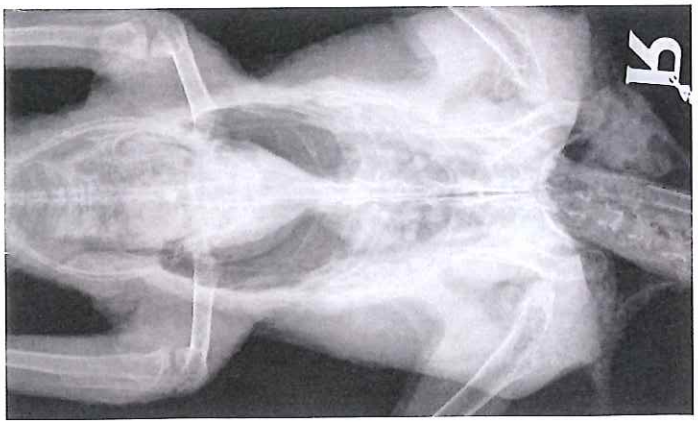


Figure 8. *Post-mortem specimen with marked splenomegaly as commonly seen with psittacosis.*

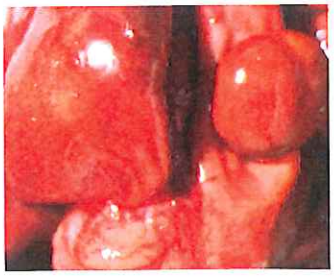


Figure 9. *Haemorrhagic diarrhoea in an Amazon parrot with leaf poisoning.*

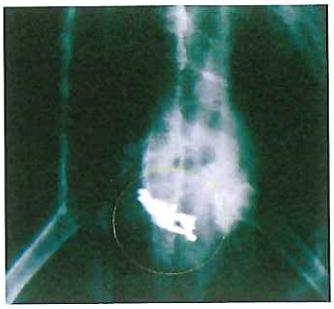


Figure 10. *Metallic density foreign body within the intestinal tract of a parrot with zinc toxicity.*

other then psittacines have also been affected and the role of wildlife acting at a reservoir is still being investigated. PDD is a fatal neurologic disease that uniquely affects the enteric nervous system.

Avian bornavirus (ABV) has been identified as the cause of PDD in psittacines. The clinical signs relate to an immune mediated reaction to the virus. Gangliocides are produced that cause changes to the nerves and disease. Many healthy birds are infected with ABV, and the development of PDD in such cases is not understood and is unpredictable.

Detecting ABV in a sick bird is not confirmation that it is suffering from PDD and detecting ABV in a healthy bird does not indicate it will become sick. ABV is not restricted to psittacines and a PDD-like disease has been diagnosed in canaries. Bornavirus has been shown to have a high prevalence in North American waterfowl, however, no evidence has been produced showing that these waterfowl genotypes can cause disease in psittacines.

The faecal/urate oral route of disease transmission is considered the most significant. The respiratory tract has also been suggested as a route of transmission. ABV has been isolated from the lung of infected birds and high-volume air sampling has detected ABV in the air of infected aviary environments. ABV has also been detected in feather calami, and feather dander is a suspected source of the air contamination.

"Cockatoos, grey parrots and macaws appear most susceptible to PDD"

Clinical signs

Initial clinical signs are generally non-specific. Birds present as lethargic and off-colour. Insidious weight loss is often noticed and undigested food can occasionally be found in the faeces. Crop impaction, delayed crop emptying, vomiting, regurgitation and coelomic distension are the most common GI signs. Birds often have a good appetite but occasionally anorexia is observed. In a small percentage of cases acute peripheral and central nervous system signs are seen including; blindness, fits, seizures and falling from the perch.

Within a collection of birds, isolated cases may be diagnosed but equally epidemics of infection affecting a large percentage of the collection can occur with a fast progression of disease from birds being acutely ill to death within 11 days. Other birds will show a more insidious disease progression with gradual weight loss and gastrointestinal signs. Some birds in the collection will be asymptomatic but, if endoscopy or radiography is performed, will have mild/moderate signs of proventricular dilation. Other birds within the collection will be unaffected.

Cockatoos, grey parrots and macaws appear most susceptible to PDD. However, the disease has been seen in over 50 species of bird, so all psittacines must be considered susceptible. All age groups can be affected. Incubation period is from as little as 11 days to more than seven years. The disease is not considered to be highly infectious and the pathogen is labile – not surviving outside the host longer than 48 hours.

Diagnosis

Proventricular dilatation disease is difficult to diagnose. Clinical signs are non-specific. If a clinician suspects PDD then radiographs (ideally with contrast instilled in the proventriculus in the anaesthetised bird) or fluoroscopy can help detect proventricular dilation (**Figures 11 and 12**). Dilation of the proventriculus is present if the depth of the proventriculus is greater then 48 per cent of the greatest depth of the carina of the sternum, when viewed on a lateral projection. Traditionally, a crop biopsy was performed (examined for signs of myenteric ganglioneuritis) as part of the diagnostic protocol; however, this test is only 55 to 76 per cent sensitive.

Differential diagnosis for PDD include:

- Foreign body ingestion
- Heavy metal toxicity
- Gastrointestinal parasitism
- Fungal/bacterial gastrointestinal infection
- Any other cause of GIT obstruction or atony

To make an informed diagnosis of PDD a clinician should rule out potential differential diagnosis and obtain evidence of ABV infection. It is, however, important to remember that many clinically healthy birds shed ABV, adding to the challenge of making an accurate ante-mortem diagnosis.

Studies at Texas A&M University have suggested reverse transcriptase–PCR as an efficient method of determining the presence of ABV RNA. Four serotypes of ABV are currently recognised and many laboratories do not currently test for all serotypes. The selection of appropriate samples for PCR testing is vital to obtaining a meaningful result - urates/faeces as well as conjunctival, choanal

Figure 11. *A dilated proventriculus is identified on lateral projection.*

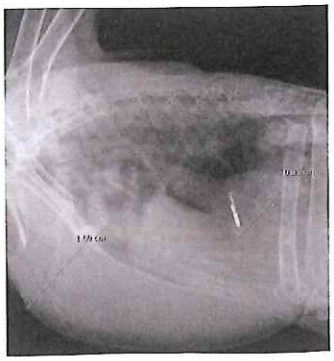
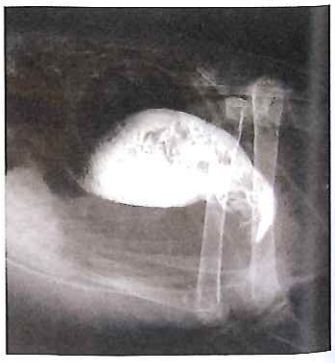


Figure 12. *Barium contrast instilled under anaesthetic allows more accurate identification of the proventricular wall.*



and cloacal swabs. ABV is shed in greatest volumes in urates and faeces, however shedding is intermittent and false negatives are therefore a problem. Serology has the potential of being a useful diagnostic test as there appears to be correlation between antibody titres and disease development. Also, sudden seroconversion has been seen to occur just prior to the onset of clinical disease.

Treatment

Treatment has historically been based around the use of anti-inflammatory medication. This was based on the observed histopathologic lesions being inflammatory in nature. The author's treatment of choice is celocoxib (Dalhausen et al, 2002). Birds were treated for six to twelve weeks with celocoxib at 10mg/kg bid, and showed marked clinical improvement. Fluoroscopy can be used to monitor treatment success and the dose of celocoxib titrated down to the minimum affective dose. Many birds that first present with PDD have secondary bacterial/fungal crop infections and/or enteritis, which when treated lead to significant clinical improvement.

Many have progressed to using meloxicam, however, a recent study using this drug demonstrates it may be contraindicated. Early trials with alternative drugs including ribavirin (antiviral) and cyclosporine have not been encouraging.

Prevention and biosecurity

Preventing disease in collections is based around good hygiene and biosecurity. Any new birds, sick birds or ABV-positive birds should be isolated or quarantined. Healthy ABV-negative birds should be visited first and traffic from infected/ill birds to the rest of the facility should be avoided. In an effort to keep a disease free flock, all newly presenting birds should be tested using multiple PCR tests and ideally also serology. If it is the owners aim to eradicate ABV from their collection, all birds should be tested using repeated PCR tests and serology. Birds should be grouped and, if required, isolated based on the results of this testing. It has been reported that within a positive group of birds, a small number of infected individuals are persistent high-level shedders of the virus. These birds should be targeted and removed as a priority. Due to the intermittent shedding and inconclusive serology testing, testing and separating birds may require years to obtain ABV-negative aviaries. ■

CPD Questions

1. A grey parrot (Psittacus erithacus) is presented in severe respiratory distress and a brief clinical history reveals the bird has lost its voice. What is your suspected diagnosis?

   A. Syringeal obstruction
   B. Air sac disease
   C. Pharyngitis

2. True or False. A negative Bornavirus PCR rules out proventricular dilatation disease as a cause of regurgitation in a pet macaw?

3. What specific precaution should be taken when obtaining a blood sample for analysis of zinc levels?

   A. Sample must be frozen immediately
   B. Sample must be taken into syringe without a rubber bung
   C. Sample must be collected in EDTA

Answers
1. A 2. False 3. B

Humane euthanasia of fish: (Vol 2 Issue 1) Update

There is now a product launched by Vetark called Aqua-Sed. The active agent is phenoxyethanol and the product is used under the exception scheme for small pet animals.

Specialist disposal of the product is required from trade premises (such as, veterinary practice); however, in domestic situations burial of the domestic waste is permitted. The dose rate for anaesthesia is 500-700ppm and for euthanasia 2000-2800ppm.

For euthanasia the fish is placed in a small volume of water and Aqua-Sed is added to make a 2000-2800ppm solution. The fish is left in the solution for one hour, which is sufficient time for euthanasia. The data sheet advises the fish then be placed in the freezer until it can be disposed of appropriately.

References

Cong W, Huang SY, Zhang XX et al (2014). Chlamydia psittaci exposure in pet birds. J Med Microbiol 63(4): 578-81.

Santos F, Leal DC, Raso TF et al (2014). Risk factors associated with Chlamydia psittaci infection in psittacine birds. J Med Microbiol 63(3): 458-63.

Evans E (2011). Zoonotic Diseases of Common Pet Birds: Psittacine, Passerine, and Columbiform Species. Veterinary Clinics of North America: Exotic Animal Practice 14(3): 457-476.

Lane RF (2003). Diagnostic testing for fungal diseases. Veterinary Clinics of North America: Exotic Animal Practice. 6(2): 301-314.

Orosz SE (2003). Antifungal drug therapy in avian species. Veterinary Clinics of North America: Exotic Animal Practice.. 6 2): 337-350.

Gigolashvili T (1999). Update on antifungal therapy. Cancer Practice 7: 157-59.

Lightfoot T and Yeager J (2008). Pet Bird Toxicity and Related Environmental Concerns. Veterinary Clinics of North America: Exotic Animal Practice 11(2): 229-259.

Hoppes SM, Tizard I and Shivaprasad HL (2013). Avian bornavirus and proventricular dilatation disease: diagnostics, pathology, prevalence, and control. Veterinary Clinics of North America: Exotic Animal Practice 16(2): 339-55.

Dennison SE, Adams WM, Johnson PJ et al (2009). Prognostic accuracy of the proventriculus: keel ratio for short-term survival in psittacines with proventricular disease. Vet Radiol Ultrasound 50(5): 483-86.

Dahlhausen R, Aldred S and Colaizzi E (2002). Resolution of clinical proventricular dilatation disease by cyclooxygenase 2 inhibition. Proc Annu Conf Association Avian Vet. 9-12.

Di Somma A, Bailey T, Silvanose C and Garcia-Martinez C (2007). The use of voriconazole for the treatment of aspergillosis in falcons (Falco species). J Avian Med Surg 21(4): 8-12.