Cockatiel Upper Respiratory Disease - An Overview

Adrian Gallagher[1]

Introduction.

To the clinician upper respiratory disease is one of the most common presentations in companion birds. It is also seen in aviary birds although not to the same extent. Here I will try to document an overview of clinical signs, causative agents, diagnostic workup, therapy both medical and surgical, and preventative strategies. A case study of an aviary outbreak is also documented.

Clinical Signs.

Cockatiels are probably the most common companion bird presented at my practice. Apart from enteric disease, upper respiratory disease would rank second. Sinusitis/rhinitis/ conjunctivitis is the primary presentation. These signs can be acute or chronic.

Clinical Signs.

- Sneezing
- Head shaking
- Yawning(Neck stretching)
- Stained or moist nares
- Unilateral or bilateral periorbital swelling
- Unilateral or bilateral periorbital redness
- Nare hyperaemia
- Irregular nare shape or size
- furrows in beak associated with chronic nasal discharge.
- Conjunctival swelling
- Scleral exposure.
- ocular pruritis
- Nare picking with claw

Most of these signs can be uni- or bilateral, acute or chronic. Some birds can show

[1] Brighton Veterinary Surgery, 353 Beaconsfield Tce., Brighton, QLD. 4017

minimal signs of distress leading to the owner not recognising signs of disease. Other birds are “sick birds”, showing depression, lassitude, anorexia and decreased vocalisation.

Causative agents.

In Australia the most common cause of upper respiratory disease is probably *Chlamydia psittaci*. These infections can be solitary or complicated by secondary bacterial infections. These bacterial infections can also be primary. Eg. *Pseudomonas, Staphylococcus, Streptococcus, Pasteurella*.

Mycoplasma is also considered to be a significant respiratory pathogen but as yet culture has failed to produce any positive evidence of infection. This belief is based on response to therapy.

Viral disease occurs in some psittacines but is not thought to be a common cause. Numerous fungal diseases occur, often producing chronic disease, eg. *Aspergillus, Cryptococcus, Candida*.

Protozoal infections of the sinus as an extension of buccal infections have also been recorded. The role of nutrition in the development of respiratory disease can not be overlooked. Hypovitaminosis A is a common predisposing factor. Low Vitamin A levels produce squamous metaplasia of the mucous membranes of the mouth, nasal cavity and sinuses. Vitamin A deficiency can be suspected if there is loss of structure to the choanal slit and/or thickening of the palps which extend from the hard palate at the point of attachment of the upper mandible.

Diagnostic Workup.

The preliminary workup of any condition should be the same.

- thorough history taking
- distant examination
- close (Physical) examination

During the history taking it is necessary to ask

- How long has the condition been present?
- What diet the bird has been on?
- Condition of air quality eg. Fumes, Smoke, Poor ventilation
- How long has the owner had the bird?
- Has there been any exposure to other birds?

Distant Examination.

On distant examination we should be assessing just how sick to bird is. Is it active and

vocalising etc.? It is when we look for any obvious signs of disease eg. Periorbital swelling or sneezing. Any diagnostic aids should be made available if required to minimise handling.

Physical Examination.

Handling should be kept to a minimum. Examination should include the nares, the buccal cavity and choanal slit, the eyes and periorbital region. Examination of the rest of the body including auscultation of the air sacs should continue.

Samples should be taken at this point. A swab of any discharge is required for cytology, culture or Chlamydia testing. Specimens can also be taken from the conjunctival sac, nares or choanal slit. If sinus swelling is evident a sinus aspirate may be indicated.

Further Investigations.

Any material collected should first of all have a *Chlamydia* test performed to exclude this disease. This is for therapeutic and public health reasons. The specimens should be examined on a wet mount and a gram stain performed.

Culture and sensitivity may also be required from these specimens.

Therapy.

Respiratory disease, especially sinusitis, can be very difficult to treat medically in chronic cases. I classify sinusitis into three primary types;

- Serous
- mucoid
- caseous

Serous sinusitis is generally a relatively acute condition. This generally involves a rhinitis and some periorbital swelling with some conjunctivitis and/or scleral exposure. Often this is and irritant condition and no causative agent can be found. Improving air quality often produces a resolution of signs. Chlamydia is also often involved.

Mucoid sinusitis is often a transition stage between serous and caseous disease. This stage is difficult to control. Very regularly there is no rhinitis as the nares are blocked. Periorbital swellings of the sinuses are virtually always a feature. A secondary conjunctivitis is often the presenting complaint. Because of the poor vascular supply to the sinuses perfusion of drugs into the sinus is poor if given systemically. The drainage from the sinuses is often reduced or non existent. A primary aim of therapy is to establish drainage. Flushing of the nares with a saline or antibiotic solution can help establish drainage to the choanal slit. Flushing of saline or antibiotics directly into the sinuses can also be performed. Often the sinuses will rupture through the conjunctival sac. If drainage

can be established, the bird should be placed on systemic therapy with frequent nebulization performed. This allows the drug therapy direct contact with the infection. Sadly, in my experience therapy at this mucoid stage of disease progression is often unrewarding. If initial therapy fails, and the patient is well, then I often let the condition mature to the caseous stage (This may take many months). If the bird is depressed and clinically unwell with the sinusitis then surgery can be performed, but once again this is often unrewarding. The anatomy of the sinuses is very complex and unless all the mucoid material can be removed, then recurrence is inevitable. Surgical technique will be described shortly.

Caseous Sinusitis is the eventual progression of most sinus infections. This is where the liquid infectious material in the sinus becomes cheese like or caseous. This stage of disease is refractory to medical therapy. Surgical removal of the caseous material is required. It should be remembered that the primary causes of the condition will also require therapy eg. hypovitaminosis A, poor ventilation.

Surgical technique.

I will describe two techniques for debridement of the sinuses.

The first technique is the percutaneous technique. The involves anaesthetising the bird and making an incision over the caseous mass, after careful skin preparation, trying to avoid any obvious blood vessels, and curetting out the mass. Generally the caseous material can be removed as a lump. Often the material requires peeling from the inner lining of the sinus. Care should be taken to try to remove the material as a single piece. Any material remaining can be a nidus for reinfection. Flushing of the sinus with a saline solution and administration of appropriate post operative therapy is recommended. The incision should be left open and flushed once or twice daily until you are confident of removal of all caseous material and that drainage has been established. The disadvantages with this technique are that there is often significant blood loss from cutaneous blood vessels. This is often aggravated whenever the dried blood is bathed away to open the incision to allow drainage. The second disadvantage is that there is often a lot of swelling around the wound post operatively. This is often very upsetting for the owner.

The second technique and the one I prefer is the per-conjunctival technique. This technique involves reflecting the eye lid to expose the conjunctiva and incising through the conjunctiva over the mass. The rest of the surgery and follow up procedures are as for the per-cutaneous route. The only problem I have had with this technique has been finding the incision for follow up flushing, as the conjunctival tissue is so fine.

Prevention.

The most important step in preventing upper respiratory disease especially sinusitis is for clients to bring their new bird to an avian veterinarian for a “New bird check”. This not only detects early disease but also provides the owner with important information on the

management and care of their new companion, eg, diet, air quality. This should also establish a rapport with the new client, to provide a contact for the procurement of reliable information about their bird.

Case Report.

This case report is not on a companion bird but details the problem of sinusitis in a flock of cockatiels over a four year period.

The first contact I had with this problem was in February 1992. There was an outbreak of sinusitis in predominantly young birds. Most of these birds were showing signs of disease whilst in the nest. The initial sign of infection was of conjunctival protrusion or scleral display in a crescent around the eyelid. This was associated with sinus inflammation resulting in the globe of the eye being pushed outwards displaying the sclera or conjunctiva. In some instances this was very subtle. There was also evidence of rhinitis (Hyperaemic, swollen nares with or without discharge, sneezing), choanal swelling and hyperaemia, and neck stretching (I have interpreted this sign as irritation from discharge from the sinuses draining through the choanal slit). Some neonates showed these signs very early after opening their eyes.

Up until the introduction of one cock bird the flock had been closed for some time. The first young to develop signs were from this bird. At the time most pairs would have been on their third clutch. The weather had been quite hot. The stocking rates were high as there were many young from previous nests present. There was also much fostering of eggs and neonates that facilitated spread of the disease. The hygiene and management of the flock was very professional and I am sure the problem would have been more severe if not for the astute observations of the owner.

The initial birds were tested for *Chlamydia* by faecal sample (Clearview Oxoid Australia).This initial test was negative. The affect nest and the parents were treated with Lincospectin (Upjohn) in water for 14 days. These birds did not respond to therapy. In the meantime the owner could trace the spread around the aviary complex. Spread was direct from cage to cage and also from fostered chicks to others in the nests. Some adults also developed signs. It must be pointed out that none of these birds were clinically sick. The disease was mild.

It was decided to stop breeding and separate the birds into three flocks, affected, suspect and clear. These birds also tested negative for *Chlamydia*. The affected and the suspect birds were treated with Lincospectin (Upjohn) 0.1 ml once daily for three days, then 11 days in water. The birds deemed clear received no treatment.

New cases were still occurring in the unaffected group. Three birds were taken to Veterinary Pathology Services for Autopsy, histology and culture of the sinuses. Results were unrewarding. The mycoplasmal cultures were negative. Histology revealed minimal inflammatory changes to the sinus linings ie. Mild focal accumulations of mononuclear

cells forming lymphoid follicles. Trachea was normal. The liver showed multi focal lymphoid follicles. There was mild dysplasia of hepatocytes and mild hyperplasia of biliary epithelium. Sections of the lung showed mild congestion. A gram negative was cultured from one bird This was found to be Alkaligenes sp and was sensitive to Lincospectin .

Infected birds were now dosed with Oxytetracycline (Pfizer Terramycin LA).

Infected birds failed to respond to therapy. Another bird was euthanised for autopsy. This bird showed chronic suppurative sinusitis with fibrosis. A staphylococcal organism was found. This was considered a secondary pathogen.

Six birds were admitted to the surgery for a therapeutic trial. Three received Enrofloxathin (Bayer) 0.04 ml. I.m. and three received 0.04 ml per crop. After six days of therapy there was no change in the birds condition. Nebulisation was added to the therapeutic regime. This consisted of 2 ml of Lincospectin in 15 ml. of saline. The birds were nebulised for 15 minutes twice daily. After five days all birds were asymptomatic. All birds regressed after stopping nebulisation. All affected birds were culled at this time. This represented about 30 % of the total flock.

Since that year the flock has been closed until this last season. There have been no cases of sinusitis in any adults or young since 1992. New introductions are veterinary screened and have a six month quarantine period. One clutch was obtained from the new introduction this year and these birds showed signs of the original disease in the nest. The parents were asymptomatic.

Discussion.

I am at a loss to explain this disease to the client. The client states that he is unable to introduce new birds into his collection for fear of introducing this disease. The signs, although subtle, are obvious and very common. These signs can be observed in virtually any cockatiel collection I have visited.

There are a number of points to be established.

- Is this worth worrying about if it is subclinical?
- Does this disease predispose individual birds to clinical sinusitis later in life?
- What is the aetiological agent causing this disease?
- Will it affect the cockatiel population in any other way in the long term?