The recognition of avian chlamydiosis, psittacine beak and feather disease (avian circovirus infection) and heavy metal poisoning (lead, zinc, copper, mercury) in caged and pet birds

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Introduction

Avian Chlamydiosis and Psittacine Beak and Feather Disease are two of the most common and important infectious diseases of caged birds at this point in time. Heavy metal poisoning is one of the most common non-infectious diseases I encounter in caged and pet birds.

Yet the diagnosis of these diseases is often overlooked. Clinical signs associated with these diseases are often not recognised or are mistakenly attributed to other processes.

Let us therefore review some of the clinically significant aspects of these diseases and find clues which may alert us to the possible presence of these diseases in birds.

Avian Chlamydiosis

The term "avian chlamydiosis" has been used to specifically distinguish this form of chlamydiosis (which is associated with *Chlamydia psittaci* infection) from that form of chlamydiosis known to our medical colleagues as a venereal disease (which is associated with *Chlamydia trachomatis* infection) and *Chlamydia pneumoniae* infection. This distinction can be important when one is trying to diagnose, or trying to help our medical colleagues diagnose, *Chlamydia psittaci* infection in the "owners" of infected birds. Unfortunately, unless the distinction is made clear, clients requesting that they be tested for chlamydiosis are more likely to have a genital sample examined than one more appropriate for avian chlamydiosis.

Avian chlamydiosis is sometimes known as psittacosis, ornithosis and "bird fever". Psittacosis is a name applicable to chlamydiosis affecting psittacine birds. Ornithosis is a name applicable to chlamydiosis in non-Psittacine birds, other animals and man. "Bird fever" is a non-specific term which usually refers to chlamydiosis. For the sake of this article I will from herein refer to avian chlamydiosis as "chlamydiosis"!

Chlamydia psittaci is an endemic acid-fast obligate intracellular potential pathogen of most bird populations. Many birds are thought to carry live *Chlamydia psittaci* organisms without developing recognisable disease. Clinical disease is precipitated mainly by human-induced chlamydiosis is thought to develop when the immune system of the bird is unable to limit the number of *Chlamydia psittaci* organisms in the body to a level compatible with health. The organisms exert their adverse effects via the

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release of endotoxins (endotoxicosis) and lysis of the host cells. There are many possible reasons for this, some of which will be discussed below, but one of which is the ability of the surface antigens of the organisms to change during replication. *Chlamydia psittaci* organism show a predilection or tropism towards columnar epithelial cells and mononuclear macrophages. The replication cycle of *Chlamydia psittaci* is complex. *Chlamydia psittaci* strains can show marked variations in virulence and antigenicity and additionally five avian serovars are now recognised (as well as non-avian ones): psittacine, pigeon I, duck, turkey and pigeon II. These serovars do not confine themselves to the named species of bird. Antibodies generated against most of the surface antigens do not confer protective immunity.

Interspecies transfer (e.g. in quarantine stations, breeding farms, multispecies aviaries and pet shops) of *Chlamydia* can change the antigenic composition, the toxic components and the host spectrum of the *Chlamydia*.

Low doses of a nonvirulent (for the individual bird) strain do not stimulate lysis of the infected macrophages which are subsequently converted to long-lived epithelioid cells that remain chronically infected. These chronic infections favour shedding of large numbers of *Chlamydia* that might be highly virulent for other avian species.

Because *Chlamydia psittaci* varies so much in its pathogenic characteristics and avian chlamydiosis is so variable in its appearance, chlamydiosis should always be considered as a potential component of the diagnosis, if not necessarily the sole diagnosis, for diseases of individual birds which show any one or combination of the following clinical signs:

Signs of disease possibly associated with chlamydiosis in individual birds.

- Susceptibility to other self-evident disease
- Sick bird look
- listlessness
- Respiratory signs including sneezing, nasal discharge, soiled, stained or caked feathers around one or both nostrils, sinusitis, facial or peri-ocular swelling, choanal discharge, dyspnoea, exaggerated movements of the tail, abdomen, pectoral regions or mouth breathing or extended neck breathing, abnormal respiratory sounds, rales, clicks, reduced exercise tolerance, pneumonia, air sacculitis, thickened or cloudy air sacs (on endoscopy). Radiographic signs of lower respiratory tract disease.
- Gastrointestinal signs including inappetence, vomiting, diarrhoea in many guises, reduced faecal output, tacky droppings, soiled vent, soiled underside of tail, dried debris on face and/or head, weight loss, flat or concave pectoral muscles, prominent keel bone. Inflamed intestines.
- Liver signs including liver enlargement, liver extending caudally beyond sternum, green, cream or yellow discoloured uric acid and/or urine component of droppings, green or yellow expanded water rings around the faeces, overgrowth of beaks and claws, especially if accompanied by interlaminal haemorrhages, signs of bruising or haemorrhage within the layers of the beak or nails; caudally displaced gizzard; radiographic signs of enlarged swollen liver. Liver with rounded edge and lack of uniform colour, often with apparently thickened capsule or diphtheritic membrane. Liver may be shrunken and fibrotic in some chronic cases.
- Signs of enlarged spleen seen radiographically or at necropsy.
- Neurological signs including unresponsiveness, convulsions and feather picking.
- Self mutilation.(About 30% of self mutilating sulphur crested cockatoos in a Melbourne specialty practice are doxycycline responsive).
- Haematological signs especially marked leucocytosis, anaemia.
- Biochemical signs referable to liver and gastrointestinal tract disease.

Obviously there are many other diseases with which one or more of the above signs may be associated. It is important to consider these in your differential diagnosis but unless specific tests are done to "rule

out" avian chlamydiosis it should remain as part of your differential diagnosis.

The big questions then arise.

- What should you do if you suspect chlamydiosis?
- How do you confirm a diagnosis of avian chlamydiosis?
- How accurate and reliable are the tests for chlamydiosis?
- Can they be done in a veterinary practice or do they need to be done at a specialist laboratory?
- Should you treat it? With what and how should you treat it?
- For how long should you treat it?
- How effective and reliable are currently available treatments?
- How soon after treatment is the bird likely to be re-exposed to *Chlamydia psittaci*?

These are questions which I hope to have the opportunity to comment upon or answer at some future date.

Psittacine beak and feather disease (avian circovirus disease)

We have come a long way since I first described my first case of Beak Rot in a Sulphur Crested Cockatoo back in 1972. When I first coined the name "A Psittacine Beak and Feather Disease Syndrome" I was just putting together or linking a pattern of diverse disease signs which are now well known to most avian veterinarians around the world. "A Psittacine Beak and Feather Disease Syndrome" soon became abbreviated to "Psittacine Beak and Feather Disease" but it took another 8 years or so before Dr. David Pass and I proved the disease is virus-associated. That virus has been subsequently been found to be minute, about 17 nanometers in diameter and to contain a single stranded loop of DNA. It has been classified as a Circovirus.

Two other circoviruses are known to man: that referred to as Chicken Anaemia Agent and the Porcine Circovirus.

Until recent times the Circovirus of Psittacine Beak and Feather Disease (PBFD) was thought to be restricted to Psittacine Birds. However, there is increasing documentation that this is not so, with the infection being recognised in doves and pigeons in Australia and a limited number of positive antigen tests being found in other species. Hence, it seems likely that we face another name change.

"Avian Circovirus Disease" is up for grabs, but how do we distinguish our meaning when referring to the circovirus which commonly infects psittacines, and that which infects poultry?

Be all this as it may, PBFD circovirus is now regarded as perhaps the most important and globally widespread viral infection of psittacine birds.

Whereas emphasis and most publicity has been given to the feather and beak changes associated with PBFD Circovirus infection, relatively little attention has been given to other signs of disease relating to circoviral hepatitis, enteritis and immunosuppression.

Psittacine Beak and Feather Disease may appear in many guises, some of which are very easy to recognise and others of which are very difficult to recognise clinically.

Many of the signs of PBFD. appear to be the same as those for avian chlamydiosis.

Many of the signs of PBFD also mimic signs of Heavy Metal Poisoning.

All three of these disease can be associated with gastrointestinal, liver, and neurological dysfunction, as well as with immunosuppression with associated predisposition to concurrent diseases.

Whereas the appearance of advanced chronic PBFD affected birds is almost pathognomonic, the peracute, acute, subacute and early chronic forms of the disease often give rise to confusion and/or oversight.

The recognition of subtle forms of feather dystrophy is dependent in part on knowledge of and familiarity with the appearance of the normal feather anatomy of the species concerned. This knowledge and familiarity is honed by careful examination of the feathers of each bird presented by clients, irrespective of whether or not it is ill.

PBFD or Avian Circovirus Infection can be at least suspected when feather dystrophy affects more than one quill bilaterally, where there is beak dystrophy or deformity and where there is nail dystrophy or deformity, especially when these are seen in a known susceptible species.

In those susceptible species which have powder downs, these feathers are likely to be affected early in the course of the subacute and chronic forms of disease.

Powder down feathers in psittacine birds are most easily located growing in the skin overlying the hips or femoral head area of the birds . In *Cacatua* sp. and galahs these feathers extend in a band of tracts from forward of the hip to behind the hip, and from there they tend to continue towards the cloaca ventrally and towards the preen gland dorsally.

The powder down feathers usually exist as blood quills, that is , the great majority of them are normally actively growing at any one time. However only a very short part, perhaps 2-3 mm, of the exposed feathers closest to the skin, is ensheathed. That part of the feathers visible within the skin is quite wide or thick and of uniform diameter distally to that part where the barbules of the feather are breaking into fine powder. The distal end of normal powder downs in these birds is normally fluffy and powdery. The exposed part of the feathers is normally about 1 cm long. When one rubs one's finger through these feathers it should be liberally coated with feather powder. Failure of this to happen suggests feather dystrophy, be it gross or very subtle.

Other signs of dystrophy of the powder downs suggestive of PBFD include narrowing of any part or parts of the feather, fault lines in the feathers, extension of the sheath of the feather, elongation of the powder downs, sparseness of the powder downs, the appearance of "empty" feather follicles between "occupied" powder down feather follicles.

Absence of or reduction of the powder formed by powder downs through the plumage and on the beaks, skin, legs and feet , gives rise to a number of other signs of PBFD in those birds which normally produce a lot of powder. These signs include an almost polished texture to the beak, at least in the early stages of the disease before it becomes obviously dystrophic, such that the beak has a "gloss" to it. On analysis, this is due to lack of the normal healthy powder film. In those species with blackish beaks this sign is made more obvious, because the powder is normally white and normally gives the beak a gray colouration, whereas when it is absent, the beak appears much darker, almost black. The same applies to the featherless skin of the legs and feet.

With a little practice, the clinician can become very alert for and adept at recognising this sign during the distance examination.

As soon as one notices that the beak and legs are darker gray or more intense in their colouration, one should become suspicious until proven otherwise that the bird may have PBFD or avian circovirus infection. (Loss of normal powder downs due to feather plucking or barbering for whatever reason is an

important differential diagnosis. Less common differential diagnoses include contamination of skin and/or plumage with an ointment or cream and the misuse of shampoos designed for dry skin.)

Another sign resulting from lack of normal powder down powder production is the altered colour of the bird's plumage, particularly the body plumage. Birds which are normally white often appear off white or "dirty', and the feathers often have a pale brown tinge. This is not readily removed by bathing or washing the bird. Galahs which are normally pale pink and pale gray often become dark pink and dark gray when their plumage lacks the film of almost white powder that is normally present.

In species which normally have little or no powder down powder through their plumage, one must be alert for other signs of PBFD such as:

- untidy plumage
- more blood quills through the plumage than expected
- abnormally coloured feathers
- beak and claw dystrophies
- reversal to more juvenile behaviour than expected for age
- susceptibility to other infections (often despite apparently good management).

Abnormally coloured feathers show the following trends: normally Blue feathers tend to develop black flecking; normally green feathers often become partly yellow but also may develop black flecking in the green part; normally red or crimson feathers may develop black flecks and/or change to yellow or orange; normally gray feathers of galahs often develop pink bands or tinting; white feathers often develop pale brown or creamy tinting; black feathers may become dull and grayer. Fault lines and dystrophic "windows" may appear in the vanes of the feathers. The dystrophic "windows" are most apparent when the feathers are transilluminated and appear to be associated with defective barbules so there is less interlocking of the barbs than normal.

Beak dystrophies can show may forms: look for irregularities, particularly asymmetrical one, yet both sides of a beak will be affected, in the surface texture, the colour, the blood supply, the contour and the wearing of the beaks. In most affected birds, the beaks tend to become too long in the earlier stages of chronic disease but later may, but do not always become very short and develop "rot". Always remember to also examine the oral or lingual surface of the beaks for necrosis and dystrophy.

Replacement of normally developed mature feathers by short dystrophic blood quills which cease growth while still a dystrophic blood quill and which are shed with pinched off proximal ends, tends to occur in an approximately bilaterally symmetrical pattern. However the areas of the body affected in the early stages of disease seem to correlate best with the age of the bird and the stage of its moulting cycle that it was in when it first succumbed to PBFD.

Birds can become affected by PBFD when mature but the majority of those affected are young immature birds. As noted above they tend to become more juvenile in their behaviour than their chronological age would indicate. Hand raised and hand-tame birds frequently revert to "begging for food' and almost constantly demand attention when otherwise well. Birds which are not hand tame will often become more timid and in a flock situation will lose rank in the flock, that is, they will drop down in the "pecking-order".

The above signs can all be relatively easily related to PBFD. However there are many other signs which often suggest concurrent disease which may be PBFD associated irrespective of whether or not additional data supporting a diagnosis of another disease can be found. In other words, unless you now a parrot or cockatoo to be immune to PBFD via vaccination or laboratory testing, always keep in mind that you may be dealing with PBFD, particularly if the bird is immature, if the bird shows signs referable to gastrointestinal, liver, kidney or neurological disease. Even if it only shows respiratory signs, consider the possibility of immunosuppression induced by or associated with PBFD infection.

Immunological and antigen tests for avian circovirus have become the cornerstones for diagnosing PBFD in birds showing non-specific signs of disease and subtle feather and/or beak dystrophies. Both types of test have a role to play. Birds can show significant levels of antibodies to PBFD and yet still be infected with PBFD virus and still be shedding virus into the environment to place other susceptible birds at risk. However, present methodology necessitates that the tests be performed in highly specialised laboratories. We hope that simplified tests for in-practice use will be developed in the near future.

Recognition of Heavy Metal Poisoning in Caged and Aviary Birds

Heavy metal poisoning can involve poisoning, usually via ingestion, with any one or combination of the metals lead, zinc, copper and mercury and/or their soluble salts. The poisoning may be acute or chronic, severe and fatal or mild and subtle.

There are many potential sources of heavy metal poisoning to which pet, caged and aviary birds have access. These include:

- galvanised wire
- lead flashing
- lead sinkers
- lead curtain weights
- solder
- mercury from the rear of mirrors
- lead based paints
- copper fly wire
- copper tie wire
- brass ornaments
- copper water pipes
- Lead and tin foil from wine bottles.

Galvanised wire poisoning is the most common form of heavy metal poisoning recognised in the author's practice, and is probably the most common non-infectious disease recognised in cage and aviary birds presented for examination. Very often birds are kept in cages made from wire which is far too fine or thin in diameter for the species of bird such that it can easily bend and sometimes break the wire with its beaks. Concurrently many cages are made of unpainted galvanised wire which is often poorly finished such that the bird can easily dislodge fragments of galvanising from the wire, especially where it has been welded. Furthermore many people are under the delusion that weathering galvanised wire for 6-12 months renders it safe; weathering for 6 months, or even for 2 years, does not render the wire safe. While galvanised wire continues to be used for aviary and cage construction it will continue to pose a threat via poisoning to the birds. It is thought that the risk of poisoning from galvanised wire can be reduced by washing the wire in acetic acid or vinegar for 20-30 minutes concurrently with scrubbing the wire with a wire brush to dislodge loose fragments, then hosing off the vinegar and drying the wire quickly.

Recently the author was contacted by a representative of a bird cage manufacturer who was dealing with a complaint about one of their painted cages. It was ascertained that the paint used contained toxic amounts of heavy metals. It appeared that the manufacturer had been unaware that this is important until this complaint had been received.

Although signs of heavy metal poisoning are usually associated with signs referable to Gastro-intestinal tract, renal and neurological disorders, it is suspected that chronic heavy metal poisoning can be immunosuppressive and may interfere with the normal growth of feathers.

Signs of heavy metal poisoning referable to the gastro-intestinal tract can include:
- vomiting
- anorexia
- crop stasis and slow emptying of the crop
- sour crop
- diarrhoea.

Signs of heavy metal poisoning referable to the renal system include:
- polyuria polydipsia
- excess urates
- haematuria in some species particularly with acute poisoning, sometimes evident as "tomato soup made on milk" droppings.

Signs of heavy metal poisoning referable to the nervous system include:
- crop and intestinal stasis
- depression
- ataxia
- convulsions
- paresis and/or paralysis of one or more legs, feet, wings or neck
- respiratory difficulty
- clenched feet
- rigidly extended toes (not often)
- difficulty swallowing.

Signs of heavy metal poisoning referable to the liver may also be present and usually include:
- green or yellowing of the normally white uric acid component of the faeces.

Conclusions and Practice.

It is apparent from the above that many of the more subtle signs of chlamydiosis, PBFD and Heavy Metal Poisoning are either the same or very similar. One is therefore faced with the challenge of distinguishing one disease from the others. (Remember that any two or three can occur concurrently in the same bird!) So what does one do?

The protocol that I adopt is to first of all obtain a history and in that determine whether or not the bird is ever allowed out of its cage, and whether or not the cage in which the bird has been brought for examination is the cage in which it normally "lives" (sic). If the cage is its normal cage then a quick inspection can usually determine whether or not heavy metal poisoning is likely to be involved. Heavy metal poisoning is much more likely in the older birds which are either frequently allowed out of their cages or which are confined in galvanised wire aviaries. I often ask whether or not there have been any recent repairs to the aviary (i.e. new wire used).

I ask whether or not the bird has contact with wild or recently introduced birds, and if so what kind they are.

It is important to determine how long the bird has been sick and in what order the various signs appeared.

I also ask what was the first treatment you tried at home or some other question likely to reveal details of treatments already given.

Having obtained a basic history I begin my clinical examination paying particular attention initially to the

bird's droppings. I look at the colour texture quantity bulk and form of the faeces (dark green normally cylindrical), the normally white uric acid, and the normally narrow clear urine ring surrounding the solid phases.

I routinely perform low and high magnification examinations of coverslip wet unstained smears of the freshest warmest dropping I can find. I take great care when preparing the smear to minimise the uric acid in it. I often add a little warm saline or Hartmans solution (I now avoid using tap water which is often chlorinated or otherwise contaminated). I search for recognisable signs of pathogens such as ova, oocysts, fungal hyphae, yeasts, *Giardia* sp. , Megabacteria and highly motile bacterial rods. I also look for undigested food, starch granules, avian cells, mites, and oil droplets. It is generally easier to see most of these (if present) in the liquid phase of the smear with the condenser of the microscope lowered.

I perform a physical examination of the bird as described elsewhere.

I then perform a crop wash and aspirate and prepare a wet smear which I examine under coverslip as for faeces, looking for *Trichomonas* sp., ova, mites, yeasts, motile bacteria, fungal hyphae, foreign materials etc.

I then prepare a thin smear of faeces and crop aspirate on separate areas of one microscope slide and proceed to Gram stain then examine these under oil immersion.

I have found that provided the bird has recently received antibiotics and is not drinking water full of disinfectant (chlorhexidene or Chloramine) one can often quickly "clue in" to heavy metal poisoning by comparing the number and variety of bacteria in the crop smear with the faecal smear. When heavy metal poisoning is present, often (but not always, it could rarely be that easy!) the crop bacterial numbers will appear normal to increased (sour crop) whereas the faecal numbers will be depleted, presumably due to the effects of the poison directly on intestinal flora. In contrast, in chlamydiosis and PBFD both faecal bacterial populations and crop bacterial populations are usually normal or increased .

Owner willing, I also do a cloacal and often combined choanal swab for *Chlamydia* testing. I usually use a Clearview test kit (and sometimes use a Surecell test kit) but I hope we will have more specific and reliable "in-house" tests for avian chlamydiosis in the near future.

Alternatively, if chlamydiosis is suspected in the differential diagnosis I will include treatment for this in my therapeutic schedule. This will be in the form of injectable or oral doxycycline or oral enrofloxacin.

Similarly, if metal poisoning is suspected, I next advise that radiography be used to check for the presence of radiodense fragments which usually accumulate in the gizzard, as well as swollen kidneys., and anything else that may be discovered. Recently with improved laboratory technology I have become able to offer (via Veterinary Pathology Services) a combined blood lead, zinc and copper analysis from a one ml sample of blood. However the cost and/or volume of blood required has so far been prohibitive, and there remains the problem of accumulating normal values to aid the interpretation of any results obtained.

Otherwise I will also include treatment for heavy metal poisoning in my therapeutic schedule. This involves Calcium EDTA injections and oral drops being given twice a day together with tube feeding a mixture of high concentrate predigested food (Poly-Aid by Vetafarm) and Metamucil (a human dietary fibre product which expands to form a gel within the alimentary canal and thus hopefully expedite the passage of heavy metal fragments from the gizzard). I sometimes also give peanut butter. This treatment is usually continued in hospital for 3 to 5 days until the bird's appetite and body functions have shown signs of returning to normal. The bird is sent home on oral calcium EDTA for an additional 10-14 days. (Surgical removal of ingested metal is rarely necessary.)

If PBFD is suspected I advise the submission to Dept Animal Health, University of Sydney, of 3 blood

quills and some plasma or serum for PBFD antigen and antibody tests, the results of which usually took 2-3 weeks to receive.

As from mid-August 1995, regretfully and unfortunately, due to lack of funding to the Department of Animal Health, University of Sydney, and grossly unsatisfactory turn around times for the test being done elsewhere, this test option will no longer be available until further announcement. (G. Cross, pers. com.).

I explain the current state of knowledge and ignorance of a reliable cure. I express the view that while the bird has quality of life, we have the opportunity to try to help, and perchance someone somewhere may find a cure.

I explain that we need to try to minimise stress and disease in every form and take a holistic approach to managing the bird. I try to balance the diet and in Eastern Medicine terms try to make it more Ying than Yang. For example, severely limit access to sunflower seed. I offer and encourage a wide range of alternative or complementary therapies aimed at stimulating the immune system and supporting body functions, but do not know if any or which ones actually benefit the patient. Provided undesirable effects are not recognised, I am happy for people to try. “Therapies” may include Australian Bush Flower Remedies, Bach Flower Remedies, Homeopathic Remedies, Herbal treatment(eg.Garlic, Echinacea), Vitamin C therapy, Brewers Yeast, Paw Paw, Guava, Propopolis, Kombucha Tea, Reiki, Tellington Touch, prayer and more.

I also use traditional Western medicines for any responsive (treatable) concurrent diseases or infections.

I advise to persist with these while indicated and while the bird still has quality of life in a protected environment provided it can be prevented from putting other birds at risk.