Psittacine Beak And Feather Disease
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PBFD is a viral infection believed to affect all parrot species, causing progressive damage to growing feathers and the germinal epithelium of the beak and claws. Concurrent immunosuppression is a common debilitating factor in infected birds and results in potentially fatal secondary infections. Clinical disease was first reported in 1888 in wild red-rumped parrots showing feather and beak changes, but the virus responsible was not identified until the late 1980s. PBFD is known to be well established in wild parrot flocks, with 20% of cockatoo flocks in Australia showing clinical signs and a seroprevalence within flocks of 60-80%. Capture of wild birds for the pet trade has lead to spread of the virus within captive collections worldwide and disease has been reported in over sixty psittacine species. Several virus strains are seen that may be species specific.

PBFD is caused by a circovirus which is very stable in the environment, resistant to many disinfectants and able to remain infectious for years. Natural infection can occur by inhalation of contaminated feather dust, and oral intake of fresh or dried faeces, or crop secretions. Viral replication within the mouth, oesophagus, crop, intestines, bursa of fabricius, and liver can result in viral contamination of faeces. Vertical transmission and fomite spread are also possible so artificial incubation of eggs does not prevent infection of chicks from infected parents. A similar syndrome has been reported in columbiformes, attributed to a similar but antigenically distinct circovirus.

The virus targets dividing cells with initial replication occurring in the bursa and gastrointestinal lymphoid tissue, and secondary replication in the liver, thymus and other organs. The epidermis is selectively affected due to high cell activity, with damage predominantly seen in the dividing cells in developing feathers. Clinical disease following infection with this virus is only seen in psittacines; most commonly budgerigars, lorikeets, lories, Eclectus and African Grey Parrots. It is suspected that Cockatoos may be common carriers of the virus, only showing signs when immunocompromised with concurrent infection, particularly Chlamydophila or reovirus.

[Figure 1]

Disease course is very variable, depending on age at infection, maternal antibody protection, viral challenge and immune status. Four categories of disease are recognised, differentiated by clinical signs and the age of bird affected:

1. Acute
   
   This is seen in newly hatched birds, most commonly grey parrots. Sudden death or severe, rapidly fatal pneumonia, enteritis or acute hepatitis are seen. There are no pathognomonic gross post-mortem signs.

   Older nestlings up to a months old present as systemically ill, with non-specific signs. Anorexia, regurgitation, lethargy, diarrhoea and secondary infections are commonly seen. The feathers may be affected, with loss of powder down, malformation, fracture and colour changes. Unlike the chronic form, all feathers can be affected in a short period of time. Leucopaenia and anaemia may also occur, but leucocyte numbers can show dramatic variation over a 24 hour period so a normal count does not exclude disease.
At post-mortem there are often signs of anaemia, hepatopathy and secondary infections, especially aspergillosis and enteric bacterial infections. High levels of virus can be isolated from the liver. Birds may recover from this acute clinical phase and become chronically infected.

3. Chronic

This usually affects birds of 8 months to 3yrs, and is the most common stage to be presented in general practice. There is a typical progressive feather loss and deformity. Manifestation of the feather disease only occurs with a moult so may not be evident for many months. Dystrophic feathers gradually begin to replace normal ones as they are moulted. Symmetrical feather loss or changes are seen, usually starting with the tail feathers, and growing feathers become curved, clubbed and prone to breakage. Damaged blood feathers lead to haemorrhage.

[Figure 2]

Dystrophic feathers may be short with fault lines across the vanes, thickened or retained feather sheaths, haemorrhage and annular constriction of the calamus or curling.

[Figure 3]

Eclectus parrots may only show delayed moulting and poor quality feathering. Pulviplumes (down feathers) are consistently moulted so are often the first feathers to show signs. They become fragile or develop an abnormally thickened outer sheath that fails to disintegrate to produce normal powder down. The failure of production of this powder down leads to a dull plumage and a glossy beak.

[Figure 4]

In later stages the beak and claws may become very brittle with a necrotic layer under the superficial horn. Transverse or longitudinal cracks or fractures, or delaminations can occur. In severe cases, necrosis of the beak, oral epithelium and progressive osteomyelitis can cause the beak to slough. Beak changes are a prominent feature of disease in cockatoos. Severe beak involvement is a very painful condition and euthanasia should be considered.

On the limbs, hyperkeratosis can cause skin to become prominently scaled and thickened. Chronic ulceration occasionally occurs in the skin overlying the elbows and wing tips.

[Figure 5]

Lovebirds may present with minimal feather changes and large crusting skin lesions, particularly in the peri orbital region, that must be differentiated from mite and pox infections.

Secondary infections are common, including parasitic, bacterial and mycotic proliferation due to the related immunosuppression. At post-mortem the bursa may be difficult to locate, the liver appears enlarged and pale with necrotic foci and splenomegaly is often noted.

4. Subclinical form

This is seen in older birds, particularly budgies, cockatiels and cockatoos. Virus is shed asymptptomatically, contaminating the environment and infecting other birds. These birds typically show no clinical signs but may be predisposed to repeated mild secondary infections and can progress to the chronic disease status. They act as a continuing source of infection to other birds.

[Figure 6]
A PCR test on blood can be used to detect the viral DNA. However, as the virus circulates within leucocytes in the blood, clinical cases with profound leucopaenia may test negative. For this reason, the preferred sample for viral DNA detection is feather pulp\textsuperscript{9}. However, tissue samples must be taken aseptically to avoid potential environmental circovirus contamination. False negatives can occur as not all feathers are involved, and atypical strains of circovirus may not be picked up by standard tests. Tissue samples from the thymus, bursa, bone marrow and liver should be collected at post-mortem of any suspected cases and submitted for PCR analysis.

A positive PCR result does not confirm active infection as non-replicating viral DNA may take up to 3 months to clear from the bloodstream. Retesting of live birds after 90 days is recommended if results are positive but there are no clinical signs. If the bird is still positive then this indicates that the bird is either subclinically infected or is being repeatedly exposed to the virus. Concurrent histopathological examination of feather material can help differentiate active and latent infection\textsuperscript{10}.

Histological examination of feather biopsies demonstrates large, granular basophilic intracytoplasmic inclusion bodies, particularly in macrophages and keratinocytes, and intranuclear inclusion bodies in epithelial cells\textsuperscript{11,12}. Examination of the bursa shows inclusions, necrosis of lymphoid follicles, medullary cysts and haemorrhage. Immunoperoxidase staining can be used to confirm diagnosis\textsuperscript{13}. There may be no histopathological changes in birds incubating PBFD, or in subclinical carriers.

[Figure 7]

A variant of psittacine circovirus (a virus termed PsCV 2) has been documented as the cause of feather dystrophy in a group of lories. Sequence analysis confirms that PsCV 2 has sufficient nucleic acid differences that it is not detected using standard laboratory PBFDV DNA detection tests. If a feather biopsy taken from a psittacine contains characteristic inclusion bodies but the blood PCR test is negative for PBFDV nucleic acid, then the blood sample could be retested using a less specific circovirus DNA assay to test for PsCV2. Microscopic lesions in affected lories were less severe than those seen with PBFDV. There are reports that 40% of wild loriikeets recover within two moults and captive bred loriikeets have been reported to spontaneously recover from this virus.\textsuperscript{5} Although PsCV2 can be cleared by lories and loriikeets it causes chronic progressive PBFD like disease in other psittacines with no resolution.

Therapy for PBFD is unsuccessful and chronically infected birds usually die within four years, from secondary infections. Small numbers of infected birds have survived up to fifteen years in a featherless state.

Sick birds within collections should be euthanased due to pain associated with beak and claw changes, psychological distress and trauma potential in birds rendered flightless, and transmission risk. In multi-bird households or breeding situations euthanasia of birds consistently testing positive, regardless of clinical status, should be a serious consideration. Virus-infected birds with feather abnormalities shed large concentrations of virus in their feather dust, which can be easily carried to other birds by the wind or by personnel. All areas, supplies and equipment that could potentially be contaminated with feather dust should be repeatedly cleaned and disinfected, using glutaraldehyde products\textsuperscript{10}. Clinically affected single pet birds can be treated
supportively with vitamin A and probiotics, with antibiotic/antifungal treatment as necessary for secondary infections as long as the bird is able to maintain a good quality of life. Autogenous vaccines, immunoglobulin injections and immunotherapy may help for short periods but most are not commercially available in the UK\textsuperscript{3}.

Prevention involves good hygiene, with testing and quarantine of any new birds in disease free collections. New birds should be tested and positive cases retested after 90d to see if virus has been eliminated or if the first sample may have been contaminated. Carrier birds may appear clinically normal but produce diseased young, hence it is sensible in commercial situations to breed birds in quarantine. Persistent positives must be kept in isolation or culled. PBFD is impossible to control with quarantine alone due to the variation in incubation length and the existence of subclinical infection.

Psittacine Beak and Feather Disease is an important disease of parrots, which is presented relatively frequently in general practice but often remains unrecognised. Prompt diagnosis is important as it is a fatal disease process but also highly infectious, risking contamination of both the keepers facility and the veterinary practice. Viral particles remain viable, and pose an infectious risk, for at least two years. Many laboratories now offer screening tests, and screening for this and other infectious diseases should be discussed with new owners, particularly where a new bird is to be introduced into a household or collection with other psittacines.

Acknowledgements

References available on request from the author.
Figure 1
Loss of feather cover and progressive abnormal colouration of feathers in an African Grey

Figure 2
Clubbing of feathers in an Umbrella cockatoo
Figure 3
Fret bars across tail feathers

Figure 4
Loss of powder down, distortion and elongation of beak
Hyperkeratosis and hyperpigmentation in cockatoo, caused by PBFD (left), normal cockatoo foot for comparison (right)

<table>
<thead>
<tr>
<th>Feather changes</th>
<th>Skin changes</th>
<th>Sudden death in juveniles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polyoma virus</td>
<td>Bacterial dermatitis</td>
<td>Bacterial septicaemia</td>
</tr>
<tr>
<td>Self-mutiliation</td>
<td>Fungal dermatitis</td>
<td>Hypo/hyperthermia</td>
</tr>
<tr>
<td>Hepatic dysfunction</td>
<td>Traumatic injury</td>
<td>Clostridial enteritis</td>
</tr>
<tr>
<td>Systemic disease</td>
<td>Knemidokoptes infestation</td>
<td>Pacheco's Disease</td>
</tr>
<tr>
<td>Polyfolliculitis (lovebirds)</td>
<td>Pox virus</td>
<td>Polyoma virus</td>
</tr>
<tr>
<td>Physical feather damage</td>
<td>Self-mutilation</td>
<td>Salmonellosis</td>
</tr>
<tr>
<td>Heavy metal toxicity</td>
<td>Polyfolliculitis</td>
<td>Congential abnormality</td>
</tr>
<tr>
<td>Amino acid deficiency</td>
<td></td>
<td>Trauma</td>
</tr>
</tbody>
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| Immunosuppression                        |                                   | Toxicity                  |
|------------------------------------------|                                   | HPAI                      |
| Proventricular Dilation Syndrome         |                                   |                           |
| Systemic Disease                         |                                   |                           |
| MalNutrition                             |                                   |                           |
| Chronic stress                           |                                   |                           |
| Psittacosis (Chlamyphilia)               |                                   |                           |
| Pacheco's Disease                        |                                   |                           |
| All chronic infections                   |                                   |                           |

Figure 6
Differential Diagnoses for PBFD symptoms
Figure 7
Section through feather follicle demonstrating basophilic intracytoplasmic inclusion bodies.
References


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