Bornavirus, Avian (ABV, virus)

For diagnosis and treatment, ABV now clinically generalizes to **Ganglioneuritis, Avian (AG)**. Originally observed due to Wasting Disease and **PDD**. These are immuno-neurological diseases in which anti-bodies attack protein inflammation and damage neurons. The proteins and damage cause tissue and organ malfunctions and failure.

ABV is an infection primarily passed vertically from mother through egg to chick.[1] Cockatiels exposed intranasally and by gavage feeding did not contract ABV. It does not spread by normal horizontal means unless the potential hosts are immunocompromised.[2] It occurs in a third of all birds, not just parrots.[1] It remains dormant in the nuclei of neurons showing no clinical signs in the lifetime of at least 90% of those infected. It does not harm the host cell.[1]

When activated by stress or disease, ABV produces inflammatory proteins that the immune system attacks causing nerve damage.[1] Clinical disease is observed using a blood test called an Anti-Gliadin Antibody Assay (**AGAA**). It measures the antibodies fighting the proteins (gliadin).[3] A high value is typically associated with ABV infection,[4] particularly values at or above 200.[1]

When activated, ABV also multiplies to spread to other parts of the body. It sheds in urine and feces where it has a fragile and short life outside the body from 5–6 hours.[1] Fecal samples are best given to the lab at the beginning of the week so they don't sit over the weekend and breaking down.[5]

> The ones with the highest number of positive (ABV) results are cockatoos, Amazons, eclectus, African greys and macaws. Quakers and lovebirds have been minimally represented. Those with the lowest number are cockatiels, budgerigars, pionus, and conures.[6]

PCR or ELISA tests for the ABV RNA may report its presence, but that does not mean it is of clinical concern. ABV is related to **Paramyxo** and **Newcastle** but is not so contagious.[1] Newcastle & Orf vaccines were not found effective against ABV.[7]

Vaccination can teach the body to identify and go directly after the RNA of a disease like ABV. This can prevent infection or force ABV to remain ineffective within cell nuclei. You do not want to the body to go after the proteins as that causes nerve damage.

- **Modified Vaccinia Virus Ankara** (MVA/PaBV-4) "vaccinated cockatiels were completely protected against subsequent PaBV-2 challenge infection and PDD-associated lesions."[7]
- **ABV** viral RNA synthesis may be **inhibited** by **Ribivirin** OR
- a combination of **Celebrex** with
  - **BVT** (Bee Venom Therapy) injections[8] OR
  - **BCG** Vaccine (Bacillus Calmette Guerin Tuberculin).[9]

BCG is difficult to obtain due to an ongoing global shortage.[10] It is more difficult in the US due to it creating false positives on TB tests.[11]

Symptoms & Treatment: see **Ganglioneuritis, Avian (AG)**.

Ganglioneuritis (AG or AGN, endemic)

Avian ganglioneuritis (AG) is a disease of neural lesions that develops when a mature immune system attacks neurons in response to pus free inflammatory disease. The lesions connect inflammatory diseases to stressors that can be hormonal, environmental, dietary, psychological, or other diseases. The most common causes of lesions are **PDD** and **Bornavirus, Avian (ABV)**. Paramyxo, Chlamydia and other diseases can also cause lesions.[1]

Avian ganglio•neur•itis is named for the inflammation (~itis) of neural gangliosides. Gangliosides are part of cell membrane composition that modulates signaling by interacting proteins.[12] When these proteins (gliadin) are defined incorrectly by a disease like ABV, the immune system develops anti-gliadin antibodies (AGA) observable in an AGA Assay (AGAA).[3]

Dr. Dahlhausen reports that, "AGAA most accurately detects clinically diseased birds" whether they are ABV positive or not. The ABV Antibody test is not reliable. The PCR test is inconsistent among laboratories, where negative just means it wasn't observed, and positive is not necessarily cause

for concern. The concerns are for the proteins and nerve damage.[1]

High numbers suggest ABV infection.[13] **Numbers above 200 correspond with clinical disease**. To stop the body from damaging itself and possibly recover to original condition, **aggressive treatment** and early treatment are advised.[1]

Dr. Dahlhausen's March 15, 2025 lecture[1] divides AG effects into four categories:

- **Central Nervous System (CNS)** damage includes the brain and spinal cord. CNS symptoms include focal and general seizures, imbalance, lost coordination, motor function, inability to identify where body parts are (proprioception).
  Old world birds including African greys are prone to CNS damage.[1]
  For **seizures** see **Medications** chapter + **Gabapentin**, **Ketamine**, **Phenobarbital**.
  Cortical blindness can be caused by ABV affecting the part of the brain interpreting vision. Caught early enough and treated with anti-inflammatories, vision can be restored.[1]
- **Autonomic Nervous System (ANS)** regulates the heart, respiration, kidneys, sexual arousal, etc. ANS damage to the heart can appear as arrhythmia, picking feathers or mutilating the region around or over the affected organs (Figure 1-1 Mutilation), and organ failure including sudden death by heart attack. New world birds (macaws and Amazons) are prone to ANS damage.[1]

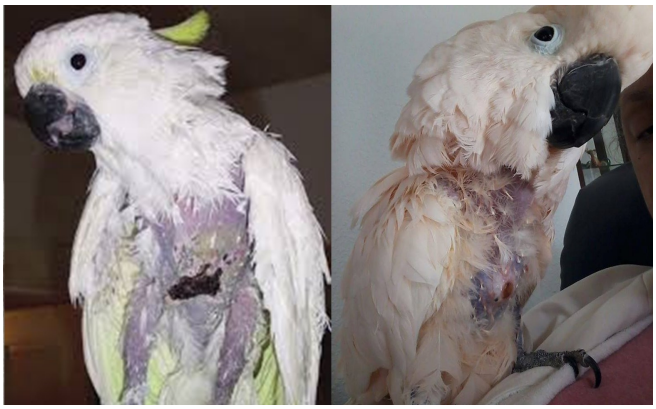

Figure 1-1 Mutilation

- **Gastrointestinal (GI)** neurons regulate digestion and are technically part of the autonomic nervous system (ANS). The GI tract is also the primary vector of PDD. Nerve damage in the proventriculus causes muscles to go unused and atrophy. This is where birds "chew their food." Consequently the food can't digest right or be absorbed. Food passes undigested and birds wastes slowly to death.[1]

Figure 1-2 Peripheral: Feather Destructive Looks

- **Peripheral Nervous System (PNS)** connects the CNS to sensory inputs, motor and organ functions. Common PNS symptoms include excessive itching, feather destruction (Figure 1-2), barbering (chewing/cutting feathers), and mutilation, dermal hypersensitivity (a hives feeling). Stressors can also cause general feather loss.[1]

Figure 1-3 Hypersensitivity (courtesy R. Dahlhausen)

Dr. Dahlhausen further observes that new world birds (especially macaws) are prone to GI and other ANS damage. Old world birds (cockatoos and African greys) are particularly prone to CNS damage. African greys are also more prone to PNS damage. Many examples he gave remind us: susceptibility is not a limitation.

Treatment is divided into categories: inflammation, prevention, **dietary support**, AG specific support, and other diseases that can aggravate AG. The outline below primarily follows the advisement of Drs. Dahlhausen & Orosz[9] updated with Merck and a November 2024 lecture by Dr. Dahlhausen[14]

- **Inflammation** COX-2 enzyme inhibitors depending severity and tolerance:
  - **Meloxicam** may be adequate to keep a minor flare in check, as with **Heavy Metal Toxicity** and **Arthritis**. One suspect study showed it as ineffective for treating ABV or PDD.[15]
  - **Celecoxib** (**Celebrex**), "initially 30-40 mg/kg divided BID PO, Long term 15-30 mg/kg BID PO. 60-80 mg/kg divided BID PO for central nervous system involvement." (**BID**=twice daily; **PO**=orally). This treatment is not as effective as Onsior therapy.
  - **Robenacoxib (Onsior)**: Onsior therapy is ideal and fast acting. It is stored cold and very thick cold. It needs to be drawn with a large needle then the syringe warmed using body heat or warm water to be administered by a more reasonable needle with less discomfort.
    Injectable formulated for cats and dogs is diluted to **20 mg/mL**. A script of **10 mg/kg** needs to be adjusted to fit dilution → **10/20 mL/kg = ½ cc / kg body mass**.
    2 – 10 mg/kg IM every 5-7 days for 4-8 weeks as needed.
    Small birds may metabolize faster and need more frequent treatments even to every 3 days.[10]
    The worst cases may be as much as 20 mg/kg.

  These are all **NSAIDs** that affect **kidney and liver function** in mammals. It has not been confirmed to affect birds. Monitoring is still recommended.[16]
- **Prevention**
  - **Stress Triggers**: account for reproductive, medical, psycho-social, environmental, and dietary. For reproductive stress: Adjust diet to reduce hormones (See **Symptoms: Hormones**) **Leuprolide acetate** (Lupron) shots or **Deslorelin implants** to counter or inhibit hormones.
  - Appropriate **antibacterial and antifungal therapy** for anerobes, yeasts, and Macrorhadu.
- **Dietary Support** see **Refeeding Syndrome** and **First Aid:Crop Feeding**.
  - Semi-elemental diet:
    Omega fatty acids: fatty fish (salmon, mackerel, tuna), chia seeds, walnuts, vegetable oils (flaxseed, soybeen, canola)
    Emeraid Omnivore and Carnivore are easily digested and absorbed with essential nutrients and Omega fatty acids.
  - **GI microbiome** stablize with pre and probiotics (sprinkled on food).
  - **GI prokinetic agents:** to improve digestion.
    Cisipride (**Propulsid**) and **Metoclopramide** (**Reglan**) in early therapy.
  - **Herbal liver and GI** support: **milk thistle**, organic **ginger** root.
- **AG Specific Support**
  - **N, N-Dimethylglycine (Vetri DMG)** supplement possibly helps with immune response, neural function (seizures & cognition), heart and skin health. Found in "many foods such as beans, cereal grains, brown rice, pumpkin seeds, and liver."[17]
  - **Seizures**: see **Medications** chapter + **Gabapentin**, **Ketamine**, **Phenobarbital**.

- **Neural pain**: see **Medications: Gabapentin**.
- **Mutilation** requires consistent wound care and hardware to protect area.
- Immunomodulation therapy.

PDD (endemic)

Diagnosis and treatment, of PDD clinically generalizes to **Ganglioneuritis, Avian (AG)**. These are immuno-neurological diseases in which anti-bodies attack protein inflammation and damage neurons. The proteins and damage cause tissue and organ malfunctions and failure.[1] PDD was originally observed as inflammation of the proventriculus in x-rays (Figure 8-1)[18] of birds suffering from wasting disease.

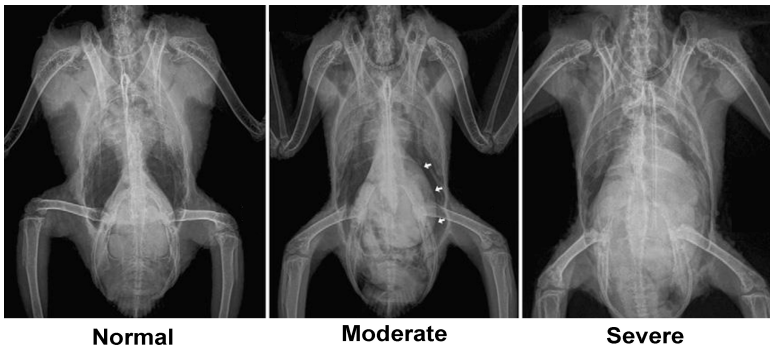

Figure 1-4 PDD X-rays

Proventricular Dilation Disease (PDD) is described as "inflammation of the autonomic nervous system of the upper GI tract, nervous tissue, and cardiac tissue." Other clinical signs of neural or gastro-intestinal diseases such as ABV may not occur.[19]

The most common vector of PDD is the GI tract, especially the proventricular area where birds internally "chew" their food. Failure to break up the food here causes it to not digest properly or absorb. Initially the subject begins to lose body mass, then starts passing undigested food in their stool.[1]

PDD is an RNA strand native to its neuron. It is a function of evolution, of adaptation to changes in diet and behavior. It is not packaged for distribution like a virus. It cannot be spread in the bloodstream or shed like ABV. It also cannot be observed directly by the immune system, or register on tests like PCR or ELISA. PDD **does not communicate to other birds**.

PDD can only be communicated to neighboring neurons in the same organ or system. For this reason it can spread across other organs in the GI tract (primary vector). On the other hand, when PDD afflicts the heart or another disconnected organ (secondary vectors), it will tends to remain isolated to that organ unless it somehow communicates to the Central Nervous System. Once in the CNS is can potentially migrate anywhere.

As RNA, PDD will conditionally produce proteins. Conditions can include a variety of AG stressors (hormones, diet, environment, psychology, other disease). The proteins cause inflammation and disrupt communications within the neuron and between the neuron and tissues it regulates. Muscles and other tissues then misbehave or go unused and atrophy.[1]

The proteins also evoke an immune response that damages host neurons. The immune response is clinical disease best observed by the AGAA antibody test. As with inflammation from ABV, PDD must be immediately and aggressively treated with antiinflammatories to prevent further nerve damage.[1]

Treatment: see **Ganglioneuritis, Avian (AG)**.

PDD is essentially a conflict with evolution. The prevalence among greater species, where genetic bottlenecking and vast care differences occur between generations and individuals, presents a case for further study. Recognizing the impact of captivity and differences in care as a cause for organ disease is important to us adapting to their needs for the sake of prevention.

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