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Catherine J. Pelelo
Iowa State University

Mike Denson
Iowa State University

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Proventricular Dilatation Syndrome in Psittacines

Catherine J. Pelelo, D.V.M.*
Mike Doolen, D.V.M.**

Introduction
Proventricular dilatation syndrome is a disease syndrome of psittacine birds that is most often characterized by a wasting bird suffering from malnutrition and/or one suffering from a nervous disorder. It affects most notably the central nervous system and the nerves of the gastrointestinal system. Specifically it causes an obliteration of the nervous tissue by inflammatory cells. Its clinical signs are caused by damage to the nerve supply of a specific organ, most often the proventriculus.

Most information on proventricular dilatation syndrome (PDS) is anecdotal, as it is a more recently discovered disease and little is known about it. As of yet, no one has discovered the cause of this syndrome. Epidemiology and histological changes are suggestive of a viral etiology, but attempts to isolate a virus and serological testing for known viruses have proven unsuccessful. Various researchers have advanced evidence in support of a number of possible viral agents, but perhaps the most convincing argument may be made for paramyxovirus. To date, the exact mode of transmission and susceptibility patterns remain unclear. Currently, there is no cure for PDS, only supportive treatment.

History
PDS was first reported in a group of macaws that were imported from Bolivia in 1977 and quarantined in California. Since then, it has been reported in a variety of psittacine birds and found worldwide. Synonyms include, macaw wasting (or fading) syndrome, myenteric ganglioneuritis, psittacine encephalomyelitis, and neuropathic gastric dilatation in psittaciformes.

Clinical Signs
There are two main categories of clinical signs: those related to the gastrointestinal tract, and those related to the central nervous system. Birds may present with one or both types of signs. The most commonly reported gastrointestinal signs are weight loss and a gradual decrease in condition with persistent or intermittent regurgitation, depression, anorexia, dehydration, and passing of watery stools which may contain seeds or other evidence of maldigestion. These signs are related directly or indirectly to the affect of the disease on the nervous system. Birds may show none, some, or all of these signs. The CNS signs include ataxia, lameness, leg weakness, paralysis, a proprioceptive placing deficit, seizures or other general signs. Additional signs have been reported. For example, evidence of aspiration pneumonia is fairly common. Some clinicians have noted an apparent sex predilection for females. The age of onset ranges from two weeks to eighteen years. One clinician has stated that a classic presentation is that of a bird reverting to baby-like behavior, such as wanting to be hand-fed again. A variable course of disease has also been reported. Often, the progression of the disease is rapid once the initial signs are noted. In some cases, the progression is more chronic.

The clinical outcome typically reported is an inevitable death, however, a few have reported keeping birds alive for a limited time.

Radiographs
A survey radiograph often shows an overall increase in coelomic cavity density, with loss of the normal hourglass formed by the heart, liver, and proventriculus. The ability to differentiate organs is also often diminished.

*Dr. Pelelo is a 1993 graduate of the College of Veterinary Medicine at Iowa State University.

**Dr. Doolen is a 1991 graduate of the College of Veterinary Medicine at Iowa State University and is currently completing a Masters Degree in Physiology.
Gas and fluid may be seen in both the proventriculus and the small intestine. Contrast radiography shows an increased transit time beyond the normal four hours through the entire GI system, a thin-walled, impacted proventriculus, and possibly an impacted ventriculus. Various organs may be shown to be dilated with contrast, most notably the proventriculus and the ventriculus, and the duodenum may also be dilated.

Ultrasoundography may show a homogeneous opacity, which proves useful for ruling out foreign bodies.

**Clinical Pathology**

No consistent changes in blood parameters are found. In some cases, a leukocytosis is seen with counts two to three times normal. In addition, protein/albumin loss, increased phosphorus, increased uric acid levels, and increased Vitamin D levels may be seen. Occasionally, anemia is seen. An elevated CK has been reported, but is not a consistent finding.

**Necropsy**

On necropsy, the most common finding is a dilated proventriculus filled with food, as is the crop, the intestine, and the esophagus. Some report the proventricular impaction in one hundred percent of PDS birds necropsied. The ventriculus may be pale white with or without hemorrhage into the mucosa or serosa. Pressure from dilated organs may cause atrophic visceral organs. Evidence of secondary infections is common. Atrophy of the pectoral muscles accompanies the common clinical signs. The rarest lesions are similar cellular accumulations in other organs. A histiocytosis and plasmacytosis has been reported in the spleen, with follicular lymphoid hyperplasia, likely due to increased hematopoiesis. In addition, some researchers report mononuclear cell infiltrates of the adrenal gland in up to sixty percent of the cases. The myocardium may be affected with cellular accumulations, as may the kidney, which present as a lymphocytic plasmacytic interstitial nephritis.

Many secondary changes may be present. In the proventriculus, ulcers and erosions may be present, with submucosal or serosal bleeding. The gut muscle may be thickened focally. One histopathologist has reported a case with a nephrocalcinosis and liver thrombosis.

**Diagnosis**

There are many antemortem differentials for diarrhea and or wasting in birds, such as Giardia, Salmonella, bacterial or mycotic thickening of the proventricular muscosa, parasites (Tetrameres americana), chlamydia or Mycobacterium avium. In addition, foreign bodies, tumors, or a rapid swelling of seeds with resultant impaction can mimic PDS. Differentials for the CNS signs include a Vitamin E deficiency and various toxins. The most notable of these toxins is lead. Others are protoporfin, zinc, salt, nitrates, etc. Diagnosis is by identification of characteristic histopathologic lesions in the proventriculus, ventriculus, or CNS.
present 6,9,13,18. Contrast radiographs showing increased transit time and a large proventriculus is suggestive, but can be mimicked by foreign bodies 10.

Since the histological lesions may be present in the crop, a sample may be taken from this organ. This procedure results in significantly fewer postoperative complications than with a proventricular biopsy but may not be diagnostic if lesions are not present 9.

Treatment

Treatment is primarily supportive, with clinicians reporting either some success or none at all. Most recommend electrolytes, warmth, liquid feed (such as rice baby food, preferably predigested), vitamins, a bacterial inoculum, antibiotics, and an antifungal agent such as Nystatin for secondary infections. It is helpful to tube feed these patients 2,5,8,12,14,15,17. Some try metoclopramide to increase motility 5,12 but others dismiss this as ineffective 17. Some have tried corticosteroids to decrease the inflammatory response 12,17. Interferon has been used with apparent short term success 14.

Surgical Intervention

A surgical ingluviotomy may be performed to gain access to the proventriculus. A stainless steel gavage tube is passed into the proventriculus and the organ is flushed with saline to remove impacted food. The patient may then be placed on a soft food diet to prevent proventricular impaction 9.

The duodenum may be cannulated via a duodenostomy tube. The patient may be fed a liquid diet which has been predigested with pancreatic enzymes 9.

Conclusion

Certainly, there is much to learn about PDS. It must be determined if the disease has a long incubation, an interim host, fomite transmission, or a latent period. Once the disease has been better characterized, studies to investigate the control and cure can begin.

References


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