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A REVIEW OF PROVENTRICULAR DILATATION SYNDROME

Christopher R Gregory, DVM*
Kenneth S Latimer, DVM, PhD*
Frank D Niagro, PhD**
Branson W Ritchie, DVM, PhD***
Raymond P Campagnoli, MS*
Terry M Norton, DVM****
Rita McManamon, DVM*****
Cheryl B Greenacre, DVM***

*Department of Veterinary Pathology
**Department of Medical Microbiology
***Department of Small Animal Medicine
College of Veterinary Medicine, University of Georgia, Athens, GA 30602
****Riverbanks Zoological Park
500 Wildlife Parkway
Columbia, SC 29210
*****Zoo Atlanta
800 Cherokee Avenue, SE
Atlanta, GA 30315

SUMMARY

Proventricular dilatation syndrome is characterized by involvement of central and peripheral nervous tissues with lymphoplasmacytic inflammatory infiltrates. A causative agent has not been found for proventricular dilatation syndrome, although virus-like inclusions have been observed in some affected tissues. Suspicion of proventricular dilatation syndrome is based on history, clinical signs, and radiographic evidence of proventricular enlargement or dysfunction. Definitive diagnosis of proventricular dilatation syndrome requires demonstration of characteristic lymphoplasmacytic infiltrates within nerves, ganglia, and neuropil.

Proventricular dilatation syndrome was first reported in the late 1970s.[1-5] Initially, the disease seemed to be limited to macaws. This fact, in conjunction with an unknown cause, gave rise to the terms macaw wasting or fading syndrome, wasting macaw syndrome, and gastric distension of macaws.[5-7] When it became apparent that the disease occurred in psittacines other than macaws, more general terms were used to describe the syndrome including proventricular dilatation, proventricular dilatation syndrome, psittacine proventricular dilatation syndrome, psittacine wasting syndrome, proventricular hypertrophy, proventricular dilatation of macaws or psittacines, and proventricular dilatation disease.[1,5,7-17] Still other names have reflected the pathologic features of the disease, including neuropathic gastric dilatation of psittaciformes, myenteric ganglioneuritis, proventricular and ventricular myositis, psittacine encephalomyelitis, and infiltrative splanchnic neuropathy.[4,5,7,18] Various case reports have demonstrated lymphoplasmacytic inflammation in both central and peripheral nervous tissues, especially of the proventriculus and other digestive organs including the crop, ventriculus, and small intestine. Additionally, the possibility of sequelae other than proventricular dilatation, such as serositis and central nervous system (CNS) involvement without gastric neuropathy, has been reported.[1,14,15] Myocarditis also has been observed.[1,2,4,5,7,10,14,15,17,19,20] Furthermore, the syndrome has been observed in psittaciformes other than those in the family Psittacidae (Table 1).

To date, the nomenclature does not accurately reflect the entire spectrum of lesions found in birds with proventricular dilatation syndrome. The most correct designation of this syndrome, based on histopathologic findings, would be lymphoplasmacytic ganglioneuritis and encephalomyelitis. Although we recognize the limitations of the current nomenclature, we will continue to refer to this disease as proventricular dilatation syndrome for the purpose of this review until a cause is found.

SPECIES AND SIGNALMENT

Proventricular dilatation syndrome has been reported in more than 50 species of Psittaciformes, including members of the families Cacatuidae (ie, cockatoos and cockatiels) and Psittacidae (ie, lovebirds, macaws, parakeets, parrots, Amazon parrots, and conures). Pacific, South American, and Afro-Asian species have been described with characteristic lesions.[1,3,4,6,7-26] Some species are commonly affected (eg, blue and gold macaws, *Ara ararauna*; African grey parrots, *Psittacus erithacus*), but this may reflect a population bias rather than a species predisposition to proventricular dilatation syndrome. Suggestive lesions have also been reported in two Canada geese *(Branta canadensis)*.[27] Other nonpsittacine birds may prove to be susceptible as improved diagnostic tests are developed.

A review of available literature suggests that a preponderance of adults over juveniles (3:1) is affected with proventricular dilatation syndrome. Both sexes are equally affected. In a retrospective study of 10,640 pet, exotic, and wild birds necropsied over a 10-year period, 127 (1.2%) had diagnostic histopathologic lesions consistent with proventricular dilatation syndrome.[20] Of these 127, birds of known age ranged from 10 weeks to 17 years (mean age, 3.8 years; median age, two years). Gender was determined in 89 of these, with a ratio of 35 males to 54 females (0.6:1).[20] At the University of Georgia, in a retrospective study of 35 birds with proventricular dilatation syn-

drome, the ratio of adults to juveniles (3.6:1) and males to females (1.2:1) approximates that reported in the literature.[a]

CLINICAL SIGNS

The most common clinical signs of proventricular dilatation syndrome include depression, weight loss (with or without poorer appetite), constant or intermittent regurgitation, and passage of undigested seeds in the feces indicating a malabsorptive or maldigestive disorder.[1,5-12,14,15,17-19,28] Proventricular impaction,[7,12] muscle atrophy,[7,10,14] abdominal enlargement,[6] lethargy,[11,14,15,17] weakness,[12,14,28] polyuria,[9,10] diarrhea,[9,15] scant feces,[9] and hypotension[14] have also been reported.

Concomitant CNS signs may include ataxia, abnormal head movements, seizures, and proprioceptive or motor deficits.[1,6,8,10,14,15,19,28] Some affected birds exhibit only CNS signs.[1,14,15] Of 221 birds described in the literature as having proventricular dilatation syndrome, 89 had histologic lesions in the proventriculus. Of these 89, 77 (86.5%) showed one or more of the four most common clinical signs.

CLINICAL PATHOLOGY

Clinical laboratory findings in affected birds are inconsistent. Hypoproteinemia,[9,10,18,19] hypoglycemia,[9] heterophilia,[1,9,14,17,19] and anemia[9,10,17,18] have been reported. Mycotic or bacterial opportunistic infection is common in affected birds and may complicate the laboratory findings.[1,2,9,10,12,14,17]

ANCILLARY DIAGNOSTIC FINDINGS

Survey and contrast radiography are useful diagnostic techniques in birds with proventricular dilatation syndrome.[4,9] Distension of the proventriculus and long transit time of barium are common findings (Figs 1, 2a, and 2b).[1,9,10,12,14,23] Ultrasonic examination may be used to demonstrate dilatation and impaction of the proventriculus.[8] Endoscopic examination may show impaction, ulceration, and dilatation of the proventriculus.[10]

NECROPSY

Emaciation,[1,11,15] pectoral muscle atrophy,[7,10,14] and dilatation of the esophagus,[1,6] proventriculus,[1,6,7,10-12,14,15,17,19] ventriculus,[7,10,12] or small intestine[10,14,18] are common. The proventriculus may appear thin-walled and friable (Figs 3a, and 3b).[6,9,11,15] However, none of the aforementioned physical, laboratory, radiographic, or gross changes are pathognomonic for proventricular dilatation syndrome. Microbial and parasitic infections, gastrointestinal obstructions, neoplasms, trauma, malassimilation disorders, toxin ingestion, or malnutrition may cause similar changes and must also be considered.[5,10,12,19] Presence of characteristic histopathologic lesions in central and peripheral nervous tissues remains the most definitive diagnostic finding.[2]

Figure 1. Ventral/ dorsal survey radiograph of a bird with proventricular dilatation syndrome. Notice the silhouette on the right, representing the distended proventriculus.

HISTOPATHOLOGY

A presumptive diagnosis of proventricular dilatation syndrome is often based on history, clinical signs, and radiographic evidence of proventricular dilatation or dysfunction. Antemortem biopsy of the ventriculus[20,23] or postmortem histologic evaluation of the proventriculus and ventriculus can be used to confirm the diagnosis by demonstrating lymphoplasmacytic ganglioneuritis.[2] However, antemortem techniques are invasive and potentially fatal in sick birds. Also, small biopsy specimens may not contain affected nerve plexuses and may be nondiagnostic.[23] Examination of affected tissues by light microscopy reveals lymphoplasmacytic infiltrates in both central and peripheral nervous tissues. Most commonly affected are the myenteric plexuses of the tunica muscularis in the proventriculus (Figs 4a, 4b) and ventriculus.[1,2,4-7,9-11,14,15,17,19,29] Infiltrates also may be observed in plexuses of the crop, duodenum (Fig 4c), and esophagus. Conduction fibers in the heart also may be involved (Fig

a. Gregory CR, Latimer KS, Ritchie BW, et al. Unpublished data. College of Veterinary Medicine, University of Georgia, 1992-1994.

4d).[1,2,4,5,7,10,14,15,17,19] Perivascular infiltrates may be seen in affected organs. Neural or perivascular cellular infiltrates may extend into surrounding tissue layers. In the brain and spinal cord, lymphoplasmacytic encephalitis (Fig 4e) and myelitis may be present with concomitant perivascular cuffing.[1,2,4-7,9-11,14,15,17,18,22]

Figure 2a and b (following). Ventral/dorsal contrast radiographs of a bird with proventricular dilatation syndrome after oral administration of barium.

In a study of 421 psittaciforme birds submitted for necropsy, 16 (3.8%) had proventricular dilatation.[11] Of these 16 birds, four had lymphoplasmacytic infiltrates in the proventriculus. In another study of seven psittaciforme birds with proventricular dilatation syndrome, all seven had lymphoplasmacytic infiltrates in the proventriculus and five had similar infiltrates in the ventriculus.[7] In addition, all seven had lymphoplasmacytic infiltrates in the pons, medulla, and midbrain, three had infiltrates in the cerebrum, and one had infiltrates in the cerebellum. Two birds had cardiac lesions consisting of focal lymphocytic myocarditis and fibrosis. Of the 221 birds in the literature described as having proventricular dilatation syndrome, 89 had histologic confirmation of the disease. Of these 89 birds, all had lymphoplasmacytic infiltrates in the proventriculus, 80 (89.9%) had proventricular dilatation, and 67 (75.3%) had lymphoplasmacytic infiltrates and perivascular cuffing in the brain. Of the 35 birds studied at the Univer-

Figure 3a and b (below). Necropsy of a bird with proventricular dilatation syndrome.

Figure 2a. Ten minutes after gavage, the contrast medium is present within the upper portion of the proventriculus and outlines a filling defect (food).

Figure 3a. The liver is reflected to show the proventricular dilatation seen at necropsy. Notice the undigested seed visible through the thin-walled proventriculus.

Figure 2b. Minimal movement of the contrast medium is observed 2 hours later.

Figure 3b. Organs removed from the bird in figure 3a. In addition to the changes in the proventriculus, the duodenum is also abnormally large.

Figures 4a-4e (below). Photomicrographs of typical histopathologic findings in birds with proventricular dilatation syndrome.

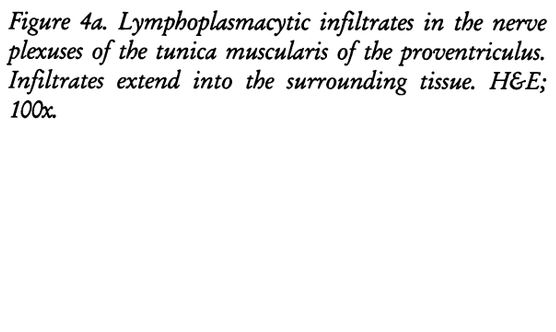

Figure 4a. Lymphoplasmacytic infiltrates in the nerve plexuses of the tunica muscularis of the proventriculus. Infiltrates extend into the surrounding tissue. H&E; 100x.

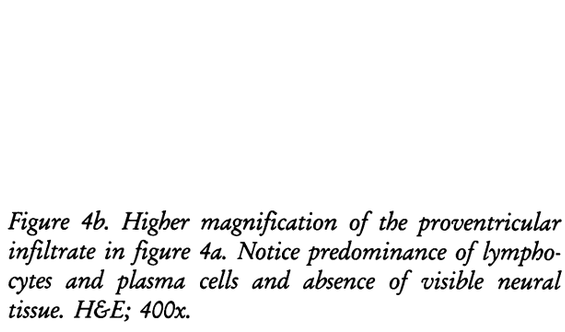

Figure 4b. Higher magnification of the proventricular infiltrate in figure 4a. Notice predominance of lymphocytes and plasma cells and absence of visible neural tissue. H&E; 400x.

Figure 4c. Lymphoplasmacytic infiltrates within ganglia of the small intestine. H&E; 100x.

sity of Georgia, findings on gross and histologic examination of tissues revealed that 26 had lymphoplasmacytic infiltrates in the proventriculus, 24 had infiltrates in the ventriculus, 18 had infiltrates in the brain, and 22 had proventricular dilatation.[a]

ETIOLOGY

The cause and pathogenesis of proventricular dilatation syndrome are unknown. Some findings suggest that the disease is infectious,[1,3,23,30] and if so, transmission of the disease by mechanical or insect vectors, airborne and fecal/oral transmission, and direct contact would all be possible etiologic factors.[1,3] Conversely, it is possible that proventricular dilatation syndrome is a randomly occurring noninfectious disease.

The disease may occur sporadically or may affect several psittacine birds in a group over a brief period.[2,20,29] Seasonal occurrence, with a higher incidence of disease in warmer months, has been described.[2] This may suggest an insect vector. Proventricular dilatation syndrome does not develop in all exposed birds,[1,20] which suggests that some birds have innate resistance, develop a protective immune response, or become carriers. Proventricular dilatation syndrome apparently has subacute, acute, and chronic stages; however, most birds die within a year after developing clinical signs.[1,3,10,14,15,17,18] Histopathologic lesions are most consistent with an inflammatory response to viral infection.[1,4,5,7,18,19,30] Viral isolation has generally been unsuccessful.[2,6,7,11,19,30] Serum antibody titers to paramyxovirus types[1-4, 6, 7,] avian herpesviruses, avian papovaviruses, and avian encephalitis virus have been negative.[7,22]

Results of light and electron microscopy studies vary. Viral particles have not been seen by some investigators,[7,11,12,22] whereas others have observed virus-like inclusion bodies and particles in peripheral and CNS tissues.[4,19,25] Light microscopy has shown intranuclear and intracytoplasmic inclusion bodies in the myenteric plexuses and celiac ganglion of some affected birds. A distinct halo surrounds the intranuclear inclusions.[4]

Electron microscopy has revealed virus-like particles, approximately 100 nm in diameter, in the spinal cords of some affected birds.[25] Mannl and colleagues describe intranuclear inclusion bodies near the nucleoli and intracytoplasmic inclusion bodies near the cell membranes in affected neural tissues. Both types of inclusion bodies had diameters of 1.5 to 3.5 μm and electron-dense, irregular subunits of 15 to 50 nm in diameter. Intranuclear and intracytoplasmic particles, ranging from 30 to 250 nm in diameter, also were observed. Larger particles had a distinct envelope.[4]

Electron-dense, intranuclear inclusion bodies 50 to 200 nm in diameter have also been reported in the columnar epithelium of the proventricular mucosa[19] and intranuclear particles 70 to 80 nm in diameter in renal epithelium of affected birds[24]; the latter may have been an incidental finding to concurrent adenovirus infection. The possibility of an insect vector and the ultrastructural characteristics of proventricular dilatation syndrome suggest a togavirus; however, this virus group does not characteristically produce inclusion bodies in tissue sections or cell culture.

The first reported cases of avian viral serositis were birds from a group of various psittacine species from an aviary with a history of proventricular dilatation syndrome. The only reported association between the two diseases originates from this aviary. In these reports, it is speculated that avian viral serositis and proventricular dilatation syndrome may be different manifestations of the same disease.[26,31,32] However, a direct relationship between the two diseases has not been shown. Birds with lesions suggestive of avian viral serositis and experimentally-infected chicks had lesions in the proventriculus, heart, and CNS that were histologically similar to lesions described in birds with proventricular dilatation syndrome.[26] In addition, hepatocellular and bursal lymphoid necrosis, epicarditis, splenitis, and serofibrinous ascites were present in psittacine birds with avian viral serositis.[26] Initial isolates from the birds with avian viral serositis suggested a togavirus,[26] one which has been speculated to be a member of the eastern encephalitis virus complex.[31] Demonstration of a definitive relationship between proventricular dilatation syndrome and avian viral serositis requires isolation or identification of a togavirus in tissues of psittacine birds affected with proventricular dilatation syndrome.

Figure 4d. Infiltrates of lymphocytes and plasma cells involving myocardial conduction fibers. H&E; 100x.

Figure 4e. Perivascular infiltrates or "cuffing" of lymphocytes and plasma cells in the brain. H&E; 100x.

Past failures to isolate a causative agent of proventricular dilatation syndrome may be explained by loss of a microorganism's viability in tissue specimens, or absence of a microorganism during the chronic stages of the disease. Alternatively, it has been suggested that the neuropathic lesions in birds with proventricular dilatation syndrome might be the result of a viral-induced autoimmune response,[20] similar to the postinfectious sequelae in human patients with measles, in which viral damage induces inflammatory damage of neural tissue.[33] However, demyelination of neural tissue, a sequela commonly reported in humans with autoimmune neuritis, is not observed in tissues from psittacine birds affected with proventricular dilatation syndrome

Table 1. Avian species reported to be affected by proventricular dilatation syndrome.

Species	Common name	Number of birds in the literature*
Nymphicus hollandicus	cockatiel	7
Cacatua galerita galerita	greater sulphur-crested cockatoo	8
Cacatua alba	umbrella cockatoo	6
Cacatua moluccensis	Moluccan cockatoo	4
Cacatua goffini	Goffin's cockatoo	3
Cacatua sulphurea citronocristata	citron-crested cockatoo	2
Cacatua roseicapillus	rose-breasted cockatoo	2
Cacatua galerita triton	triton cockatoo	1
Cacatua haematuropygia	red-vented cockatoo	1
Cacatua sulphurea sulphurea	lesser sulphur-crested cockatoo	1
Agapornis spp	lovebird	1
Ara ararauna	blue and gold macaw	39
Ara auricollis	yellow-collared macaw	7
Ara rubrogenys	red-fronted macaw	5
Ara macao	scarlet macaw	5
Anodorhyncus hyacinthinus	hyacinth macaw	5
Ara severa	severe macaw	3
Ara nobilis	noble macaw	3
Ara militaris	military macaw	3
Ara spp.	macaw hybrid	2
Ara maracana	Illiger's macaw	1
Ara chloroptera	green-winged macaw	1
Brotogeris pyrrhopterus	grey-cheeked parakeet	1
Psittacula spp	parakeet	1
Psittacus erithacus	African grey parrot	56
Psittacus erithacus timneh	Timneh African grey parrot	4
Poicephalus senegalus	Senegal parrot	4
Deroptyus accipitrinus	hawk-headed parrot	4
Eclectus roratus	eclectus parrot	2
Poicephalus rufiventris	red-bellied parrot	1
Poicephalus meyeri	Meyer's parrot	1
Poicephalus guliemi	Jardine's parrot	1
Pionus senilis	white-capped pionus	1
Pionus menstruus	blue-headed pionus	1
Coracopsis vasa	vasa parrot	1
Rhynchopsitta pachyrhynca	thick-billed parrot	1
Amazona aestiva	blue-fronted Amazon parrot	7
Amazona autumnalis	red-lored Amazon parrot	4
Amazona leucocephala	Cuban Amazon parrot	2
Amazona tucumana	Tucuman Amazon parrot	2
Amazona ochrocephala	yellow-crowned Amazon parrot	1
Amazona albifrons	white-fronted Amazon parrot	1
Amazona xantholora	yellow-lored Amazon parrot	1
Aratinga guarouba	golden conure	5
Aratinga jandaya	jenday conure	3
Nandayus nenday	nanday conure	1
Cyanoliseus patagonus	Patagonian conure	1
Aratinga solstitialis	sun conure	1
Aratinga aurea	peach-fronted conure	1
Aratinga auricapilla	golden-capped conure	1

* Of these 221 birds, only 89 had histologic confirmation of proventricular dilatation syndrome.

THERAPY AND PREVENTION

Currently, there is no specific treatment for proventricular dilatation syndrome. The long-term prognosis remains poor, with death occurring from emaciation, secondary infections, autointoxication, or CNS disturbances.[4] Morbidity is low, but mortality approaches 100%.[4,12,22] The reported survivors have not had histologically confirmed proventricular dilatation syndrome.[8,9,17] Supportive treatment such as fluid replacement, administration of antiemetics and vitamin preparations, tube or handfeeding of small portions of liquid or semisolid diets, and antimicrobial therapy for secondary infection may extend life for a short period of time.[1,6,9,19] Until an infectious agent can be identified, preventive measures such as quarantine of new birds, avoidance of direct or indirect contact between isolated groups of psittacine birds, and appropriate hygiene seem prudent.

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