



Title	CATARRHAL PROVENTRICULITIS ASSOCIATED WITH A FILAMENTOUS ORGANISM IN PET BIRDS
Author(s)	TSAI, Shinn-Shyong; PARK, Jae-Hak; HIRAI, Katsuya; ITAKURA, Chitoshi
Citation	Japanese Journal of Veterinary Research, 40(4), 143-148
Issue Date	1992-12-28
DOI	10.14943/jjvr.40.4.143
Doc URL	http://hdl.handle.net/2115/2405
Type	bulletin (article)
File Information	KJ00002377609.pdf



[Instructions for use](#)

CATARRHAL PROVENTRICULITIS ASSOCIATED WITH A FILAMENTOUS ORGANISM IN PET BIRDS

Shinn-Shyong TSAI¹, Jae-Hak PARK¹, Katsuya HIRAI²
and Chitoshi ITAKURA¹

(Accepted for publication: Sep. 19, 1992)

ABSTRACT

Catarrhal proventriculitis due to infection by an unidentified organism was diagnosed in 79 of 534 pet birds examined histologically. It was more prevalent in domestic birds (70 cases) than in imported ones (9 cases). A high incidence of the disease was encountered in budgerigars (*Melopsittacus undulatus*) and it was occasionally found in finches (*Poephila gouldiae gouldiae*), parakeets (*Psittacula krameri manillensis*), Amazona parrots (*Amazona aestiva aestiva*) and cockatiels (*Nymphicus hollandicus*). The agent was a large filamentous rod, and was stained positively with Gram, GMS and PAS methods. Histologically, it induced a mild to moderate exudative or proliferative inflammation in the proventriculus. All the cases had an erosion in the gizzard. Ultrastructurally, the organism had a eukaryotic nucleus and three cell-wall layers.

Concurrent infections were very common, including adenoviruses (37 cases), giardiasis (31 cases), candidiasis (13 cases), papovaviruses (11 cases) and knemidocoptic mites (11 cases).

Key words: proventriculitis, filamentous organism, pet bird, histopathology.

INTRODUCTION

Pathologic lesions of avian proventriculus attributable to infectious agents have rarely been encountered³⁾. A recent report indicated that a large, rod-shaped, Gram-positive, non-spore forming bacterium caused proventriculitis in canaries, usually with a clear mucus on the mucosal surface¹²⁾. This paper describes a similar disease, although it might be caused by a different etiological agent, in imported and domestic pet birds.

¹ Department of Comparative Pathology, Faculty of Veterinary Medicine, Hokkaido University, Sapporo 060, Japan

² Department of Veterinary Microbiology, Faculty of Agriculture, Gifu University, Yanagido, Gifu 501-11, Japan

MATERIALS AND METHODS

We histologically examined 293 budgerigars (*Melopsittacus undulatus*) collected from pet bird stores in Japan and 241 birds imported from several other countries. The imported birds consisted of 67 parakeets (*Psittacula krameri manillensis*), 52 cockatiels (*Nymphicus hollandicus*), 29 parrots (19 *Amazona aestiva aestiva* and 10 *Psittacus erithacus erithacus*), 47 budgerigars (*Melopsittacus undulatus*), 18 lorries (*Trichoglossus haematodus*), 8 lovebirds (*Agapornis roseicollis*), 2 rosellas (*Platycercus emimius*) and 18 finches (*Poephila gouldiae gouldiae*) which died within 2 weeks of quarantine in a bird shop.

For histopathological examination, tissues were fixed in 10% buffered formalin, embedded in paraffin, sectioned and stained with hematoxylin and eosin (HE), periodic acid-Schiff (PAS), Grocott's methenamine silver-nitrate (GMS) and Gram methods.

For electron microscopic examination, formalin-fixed tissues were post-fixed in 1% osmium tetroxide and embedded in Quetol 812. Ultrathin sections were stained with uranyl acetate and lead citrate, and examined in a JEOL electron microscope, model JEM-100SX.

RESULTS

On histological examination, catarrhal proventriculitis due to infection by a filamentous organism occurred in 79 of 534 birds examined. The incidence was higher in the domestic birds than in the imported ones (70 : 9). The affected birds consisted of 72 budgerigars (91.1%), 3 finches (3.8%), 2 parakeets (2.5%), 1 Amazona parrot (1.3%) and 1 cockatiel (1.3%). The caudal portion of the proventriculus and the proventricular-gizzard junction were the target sites for this organism.

The organism was often localized in the lumina of superficial glands of the proventriculus, while it did not invade either the mucosal layer or the common duct (Figs. 1, 2). The tissue reaction to the organism was not so severe, although some proliferative and exudative changes were present (Table 1).

Most of the cases showed mild to moderate epithelial hyperplasia in the superficial glands, and collecting ducts of the deep glands (Fig. 1). This alteration was often associated with an inflammatory cell reaction in the lamina propria consisting mainly of lymphocytes, plasma cells and fibroblasts. Occasionally, foreign-body giant cells around cystic dilations were seen in the deep glands. An excess of mucus accumulated in the proventricular lumen or the sulcus, often forming a mixture with the desquamated epithelial cells of the superficial glands (Fig. 2). Infiltration of heterophils and edema occurred in the lamina propria, although it was mild in degree.

The gizzards of all the cases had erosions without cell reaction in the superficial portion of the koilin layer.

The organism was a large, filamentous, non-spore forming, and non-branching rod.

Table 1. Proventricular tissue changes in proventriculitis due to a filamentous organism infection

Tissue change	Severity of lesion in each case ^a			Total
	+	++	+++	
Exudative change in lamina propria				
Mucus	46 ^b	20	13	79
Edema	24	8	2	34
Heterophil	15	1	0	16
Proliferative change in lamina propria				
Lymphocyte	47	25	3	75
Macrophage	22	8	0	30
Plasma cell	31	11	1	43
Fibroblast	23	20	5	48
Foreign body giant cell	7	3	0	10
Glandular epithelium				
Epithelial hyperplasia	44	13	3	60
Cystic dilation	3	1	0	4

^a + ; mild, ++ ; moderate, and +++ ; severe.

^b No. of case.



Fig. 1. Many long, unbranched, Gram-positive filamentous organisms (arrow-heads) inhabit the mucosal surface of the proventriculus and sulci of the superficial glands, inducing epithelial necrosis and desquamation. Gram stain. $\times 350$.

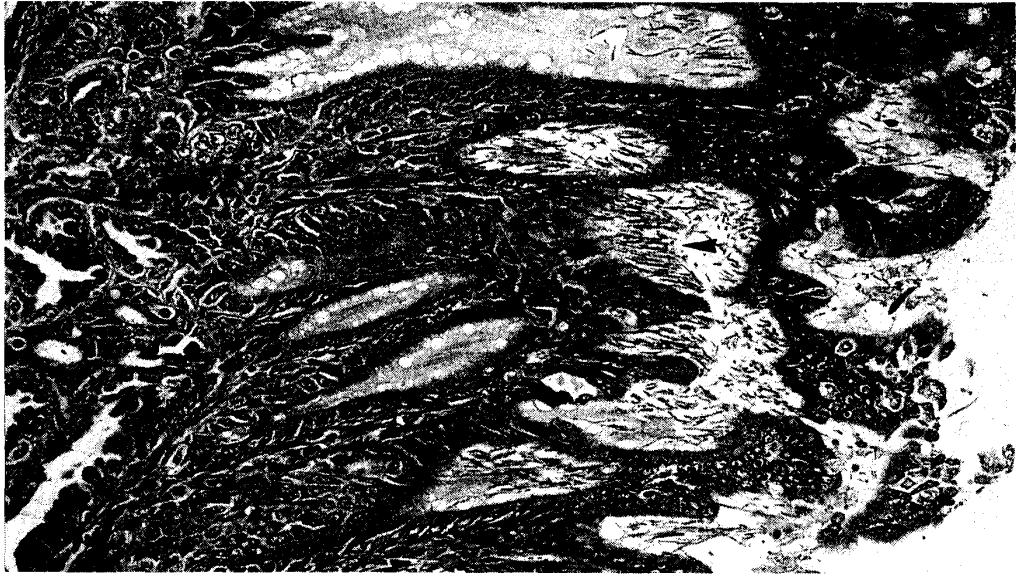


Fig. 2. Many filamentous organisms (arrowhead) invade the sulci of the superficial glands, causing catarrhal proventriculitis. HE stain. $\times 230$.

It was stained positively with Gram, GMS and PAS methods. Ultrastructurally, the longitudinal section of the organism had a distinct nucleus enclosed by a nuclear membrane (Fig. 3). The organism was surrounded by three layers; the inner layer was thick and had mild indentations into the cytoplasm, the middle one was very thin

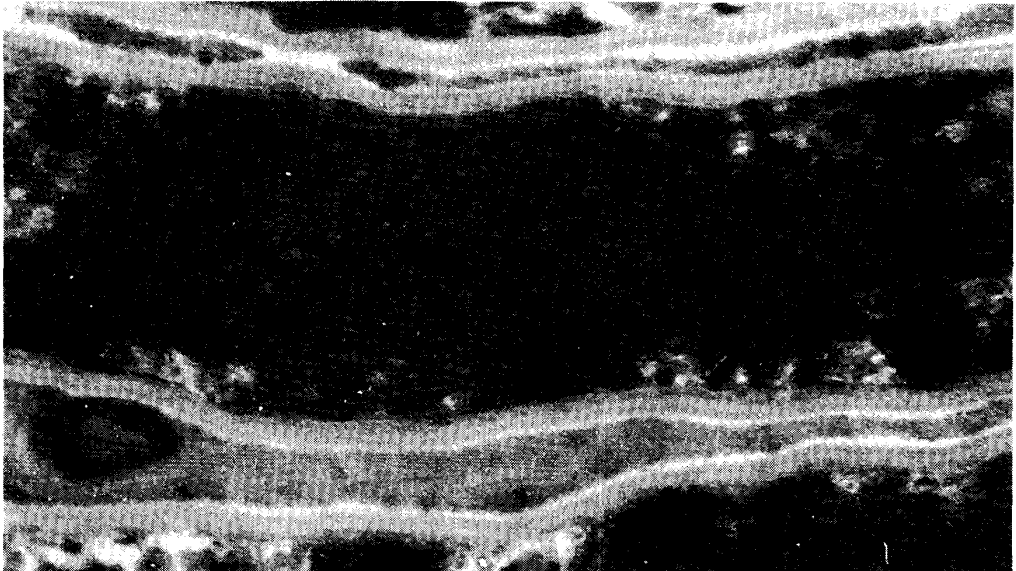


Fig. 3. Electron micrograph of an organism in the proventricular lumen. It has a distinct nucleus (N) enclosed by a nuclear membrane and is surrounded by a three-layered wall. $\times 31,000$.

and more electron-dense, and the outer layer was very thick and more electron-opaque.

Based on the histological examination, most of the cases (67/79) in this study had concurrent infections. The frequency of the occurrence was as follows; adenovirus infection (37 cases), giardiasis (31 cases), candidiasis (13 cases), papovavirus infection (11 cases), knemidocoptic mite (11 cases), cryptosporidiosis (1 case), and sarcocystosis (1 case).

DISCUSSION

Proventriculitis is usually associated with a generalized condition rather than constituting a specific problem by itself¹¹. Proventricular dilatation had been reported in macaws, cockatoos and psittacines with unknown causes⁷. Candidiasis might be the most common agent involving the proventriculus of pet birds⁸. Recently, high morbidity (30%) of bacterial proventriculitis has been reported in canaries from which the agent has not been isolated¹². Electron microscopically, the agent lacked a definite nuclear structure and cytoplasmic granules.

Based on the lesion distribution, and the organism morphology and stainability examined by light microscope, the disease of our cases was identical to the bacterial proventriculitis¹². However, the organism observed here had a eukaryotic nucleus, suggested that it was not a bacterium. Morphologically, blastospores and pseudohyphae typical for *Candida* species⁸ were not demonstrated in our cases. The present study indicated that the agent had a wide host range, including canaries, budgerigars, finches, parakeets, Amazona parrots and cockatiels.

Gizzard erosion has been attributed to water deprivation⁴ or a bile acid deficiency¹. It has also been associated with the use of quaternary ammonium compounds as disinfectants in drinking water⁹, deficiencies of sulfur-containing amino acids¹⁰, and B-6 avitaminosis⁵, as well as feeding moldy corn². All the cases in the present study had an erosive lesion in the gizzard. However, the pathogenesis seemed to be different from those mentioned above.

The koilin layer of the gizzard is composed of a scaffolding of interconnecting vertical rods embedded in a horizontal matrix, which contains a carbohydrate-protein complex⁶. The vertical rods are secreted by the gland of the lamina propria and harden within the lumen as filaments. The horizontal matrix is a secretion of the crypt cells and surface epithelium, which is hardened by a fall in its pH as a result of diffusion, through the membrane, of hydrochloric acid from the proventriculus. Bacterial proventriculitis causes an increase in the pH of the proventriculus, from 0.7 – 2.4 in the uninfected stomach to 7.0 – 7.3 in the infected stomach. This change in pH causes poor precipitation of the protein-carbohydrate in the koilin layer of the gizzard, resulting in formation of erosions. This might be the reason why gizzard erosion has been found in all the cases of this study. The identification and pathogenicity of the

organism need to be studied further.

REFERENCES

- 1) ALMQUIST, H. J. & MECCHI, E. (1946): Influence of bile acids, vitamin K and cincophen on erosions of the chick gizzard lining. Proc. Soc. Exp. Biol. Med., **46**, 168-172
- 2) BEASLEY, J. N., BLALOCK, L. D., NELSON, T. S. & TEMPLETON, G. E. (1980): The effect of feeding corn molded with *Penicillium lanosum* to broiler chicks. Poul. Sci., **59**, 708-713
- 3) BICKFORD, A. A., CORSTVED, R. E. & ROSENWALD, A. S. (1978): Pathology of experimental erysipelas in turkeys. Avian Dis., **22**, 503-518
- 4) BIERER, B. W., CARLL, W. T., ELEAZER, T. H. & BARNETT, B. D. (1966): Gizzard erosion and lower intestinal congestion and ulceration due to feed and water deprivation in chickens of various ages. Poul. Sci., **45**, 1408-1411
- 5) DAGHIR, N. J. & HADDAD, K. S. (1981): Vitamin B-6 in the etiology of gizzard erosion in growing chickens. Poul. Sci., **60**, 988-992
- 6) EGLITIS, I. & KNOUFF, R. A. (1962). An histological and histochemical analysis of the inner lining and glandular epithelium of the chicken gizzard. Am. J. Anat., **111**, 49-66
- 7) GERLACH, H. (1984): Survivors of macaw wasting disease in Germany. AAV Newsletter, **2**, 52
- 8) MAESTRINI, N. & GOVONI, S. (1972): Studio histopathologico su di un focolaio di candidosi del pappagallino ondulato. Folia Vet. Lat., **2**, 96-108
- 9) MAYEDA, B. (1968): The toxic effects in turkey poults of a quaternary ammonium compound in drinking water at 150 and 200 ppm. Avian Dis., **12**, 67-74
- 10) MILLER, D., BAUERSFELD, P. E., BIDDLE, G. N. & FORTNER, A (1975): Effect of sulfur-containing dietary supplements on gizzard lining erosions. Poul. Sci., **54**, 428-435
- 11) MINSKY, L. & PETRAC, M. L. (1982): Diseases of the digestive system. pp. 432-448. In: Diseases of Cage and Aviary Birds, 2nd ed. Ed. Patrak, M. L. Philadelphia, Lea & Febiger
- 12) Van HERCK, H., DUJSER, T., ZWART, P., DORRESTEIN, G. M., BUITELAAR, M. & Van der Hage, M. H. (1984): A bacterial proventriculitis in canaries (*Serinus canaria*). Avian Pathol., **13**, 561-572