Psittacine Proventricular Dilatation Syndrome ("Macaw Wasting Disease")

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Introduction

Psittacine proventricular dilatation syndrome (PPDS), a fatal disease of psittacine birds, was first recognised in the USA in the late 1970s and has also been reported in Europe. The first recognised cases occurred in macaws and the condition was originally termed "macaw wasting disease". As far as we are aware, this is the first report of the disease in Australia.

A 6-month-old, female, green-winged macaw spent approximately 7 weeks in quarantine after importation into Australia. Three weeks after its release from quarantine, the bird presented with persistent regurgitation and weight loss. Contrast radiological studies revealed crop stasis. A crop wash failed to reveal *Candida* spp. The bird failed to respond to a 3-week course of nystatin and died shortly afterwards. At necropsy there was dilatation of the distal oesophagus, crop, proventriculus and gizzard with erosion of the gizzard mucosa. The proventriculus and gizzard contained poorly digested feed. Histologic examination revealed the myenteric ganglia and extrinsic and intrinsic nerves of the crop, proventriculus and gizzard to be infiltrated by lymphocytes, plasma cells and macrophages with occasional progression to demyelination. These changes were most prominent in the gizzard where in places the inflammatory infiltrate extended into the muscularis. These microscopic changes are pathognomonic for PPDS.
The aetiology of PPDS is not known but the nature of the lesions is highly suggestive of infection with a neurotropic virus. Electron microscopic studies have identified paramyxovirus-like particles in organs of some affected birds, but repeated attempts to isolate a virus have been unsuccessful to date. There have also been suggestions that the disease is immune mediated, perhaps following viral infection. The incubation period may be quite long and birds in isolated households have developed clinical signs months to years after introduction. However rapidly spreading local outbreaks have also been reported.

Since dilatation of the proventriculus is a consistent finding, psittacine proventricular dilatation syndrome seems the most appropriate and widely accepted name for the disease. More pathologically descriptive synonyms have included myenteric ganglioneuritis, infiltrative splanchnic neuropathy and neuropathic gastric dilatation.

PPDS is seen most often in macaws and cockatoos but has also been reported in African grey parrots, Amazon parrots, eclectus parrots, conures and cockatiels. The clinical signs are quite variable and include depression, weight loss, anorexia, regurgitation, pendulous crop, passage of undigested seeds in the faeces and polydipsia/polyuria. Most birds develop secondary bacterial or fungal infections of the cranial gastrointestinal tract and these may contribute to the severity of the clinical signs. In a significant percentage of cases there are also neurological signs including leg weakness, proprioceptive deficits,

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ataxia, torticollis, opisthotonus, seizures and behavioural changes. Radiology is useful in demonstrating dilatation of the cranial gastrointestinal tract, especially the proventriculus. Barium sulphate passage time is usually increased but may be decreased.

Typical necropsy findings are dilatation of the cranial gastrointestinal tract with thinning of the proventricular wall. There may also be ulceration, impaction and even rupture of the proventriculus and gizzard. Histologically, the non-suppurative inflammation typically seen in the ganglia and nerves of the proventriculus and gizzard may also involve the crop, duodenum, coeliac ganglion, brain, spinal cord, subepicardial ganglia and myocardium.

A strong presumptive diagnosis can be made on the basis of history, clinical signs and radiographic appearance. The differential diagnosis includes fungal, megabacterial or mycobacterial infection of the proventriculus or gizzard, gastrointestinal foreign body, abdominal neoplasia including intestinal papillomatous disease, metabolic dysfunction, lead poisoning and other neurological diseases. Confirmation of the diagnosis requires histologic demonstration of the characteristic neural lesions in the proventriculus or other organs. As an antemortem test, crop biopsy may be of value when there is clinical involvement of this organ. False negative biopsies may be due to the segmental nature of the histologic lesions of PPDS. Proventricular or ventricular biopsies obtained by laparotomy have an unacceptably high rate of post-surgical complications, though they may have a role when the aviculturist needs a diagnosis for aviary management purposes and the potential death of the bird is an acceptable risk.
Case fatality is virtually 100%, though individual birds have been maintained for extended periods by use of intensive supportive care such as providing fluid therapy, tube feeding of liquid diets and eliminating secondary microbial infections. Anti-inflammatory doses of corticosteroids may reduce damage to the inflamed ganglia and nerves.

With its identification in Australia, PPDS becomes an important differential diagnostic consideration in diseases of macaws, cockatoos and other susceptible psittacines.

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