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Investigating Gastrointestinal Dysfunction in an Eclectus Parrot

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Introduction

The unique gastrointestinal anatomy and physiology of birds includes a two-chambered stomach; a glandular proventriculus with a similar function to the mammalian stomach, and a muscular ventriculus (or gizzard) that grinds ingested food (, Klasing, 1999; Dennison et al., 2008). Gastrointestinal diseases are common in psittacine birds but may be challenging to diagnose (De Voe et al., 2003). Clinical signs associated with proventricular disease in parrots include regurgitation or vomiting, anorexia, and the passage of undigested food in the faeces (Dennison et al., 2009) as well as non-specific signs such as lethargy and the classic 'sick bird look.' In this report, we describe the investigation and management of a case of gastrointestinal dysfunction in an Eclectus parrot (*Eclectus roratus*).

Case Report

A six-month old, home-bred, male Eclectus parrot was referred for persistent regurgitation and malodorous faeces of approximately fourteen days duration. The owner reported that the patient had been unwell for four weeks prior to presentation, with a fluffed appearance, an abnormal posture with the tail down, and had been calling for food excessively. Prior to presentation, a course of oral nystatin (Nilstat oral drops, 100,000 IU/mL, Aspen Pharma Pty, 34-36 Chandos St, St Leonards, NSW 2065) at 300,000 IU/kg BID had been administered. An initial improvement was noted but after three days the clinical signs recurred.

The bird had been parent-reared for the first three weeks of life after which it was removed from the nest due to parental aggression. Subsequently, it had been hand-reared on a commercial formula, and once weaned fed a homemade diet comprising brown rice, quinoa and cooked vegetables, with *ad libitum* fresh fruit and vegetables plus a small amount of seed. Since the bird had become unwell, the owner had restarted hand feeding with the hand-rearing formula. The owner described the bird as the 'runt' of a clutch of three chicks and was one of five Eclectus parrots in the household; the other birds were unaffected.

Physical examination revealed multiple stress bars on the contour feathers but was otherwise unremarkable; weight was 342g and body condition score 5/10. Differential diagnoses based on clinical history and examination findings included an infectious aetiology, a gastrointestinal foreign body, a congenital abnormality affecting oropharyngeal or proventricular outflow, nutritional disease, heavy metal toxicity, metabolic or endocrine disease, and chlamydiosis.

A crop wash wet smear and DiffQuick preparation was examined cytologically. The sample was negative for protozoan organisms, but contained moderate numbers of a mixed population of bacterial rods and cocci, with no single dominant morph evident. A faecal wet smear and Gram stain examination were negative on cytology for protozoan organisms, *Macrorhabdus ornithogaster* and nematode, tapeworm, coccidian and cryptosporidian oocysts. The faecal Gram stain contained moderate numbers of a mixed bacterial population of Gram-positive rods and cocci (~95%), with low numbers of Gram-negative rods (~5%). Occasional non-budding *Candida albicans* were noted in both crop and faecal cytology. The *Candida* and bacteria identified were considered to be a normal part of the gastrointestinal flora rather than indicating a pathological process.

General anaesthesia with isoflurane (Delvet Isoflurane, 100%, CEVA Animal Health Pty Ltd, 11-15 Moores Rd, Glenorie, NSW 2157, Australia) was administered (3% in oxygen for induction; reduced to 2% for maintenance) to facilitate jugular venepuncture for a complete blood count (CBC) and plasma biochemistry, Chlamydia antibody testing, and obtain survey radiographs. The blood sample was sent to an external laboratory for analysis whilst the *Chlamydia* antibody test was run in-house. Lateral and ventrodorsal radiographs were taken. These showed an enlargement of the proventriculus and ventriculus (Figure 1), with a proventricular-diameter: keel-height ratio of 0.91. Recovery from anaesthesia was uneventful. The patient began empirical nystatin (Nilstat oral drops; 300,000 IU/kg TID PO) and amoxicillin-clavulanic acid (Curam Duo powder for suspension, 400mg+57mg/5mL, Sandoz Pty Ltd, 19 Harris St, Pyrmont, NSW, 2009), 125mg/kg PO BID therapy pending the results of the blood-work.

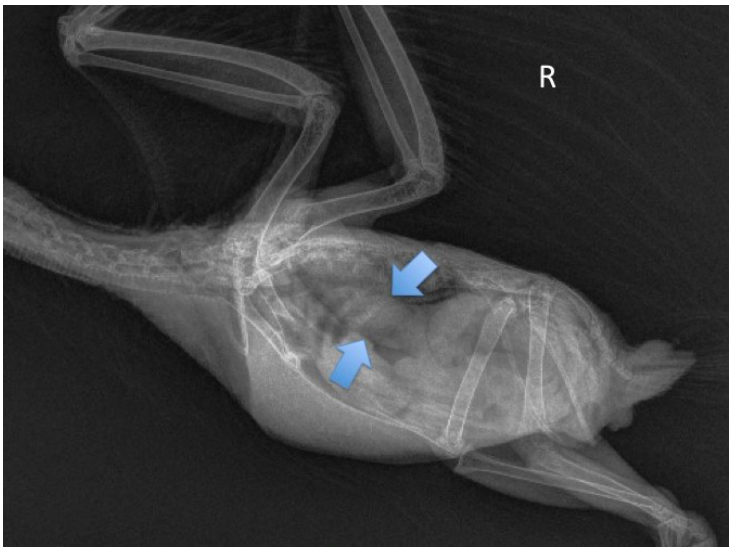

Figure 1: Right lateral radiograph demonstrating an enlarged proventriculus (arrows).

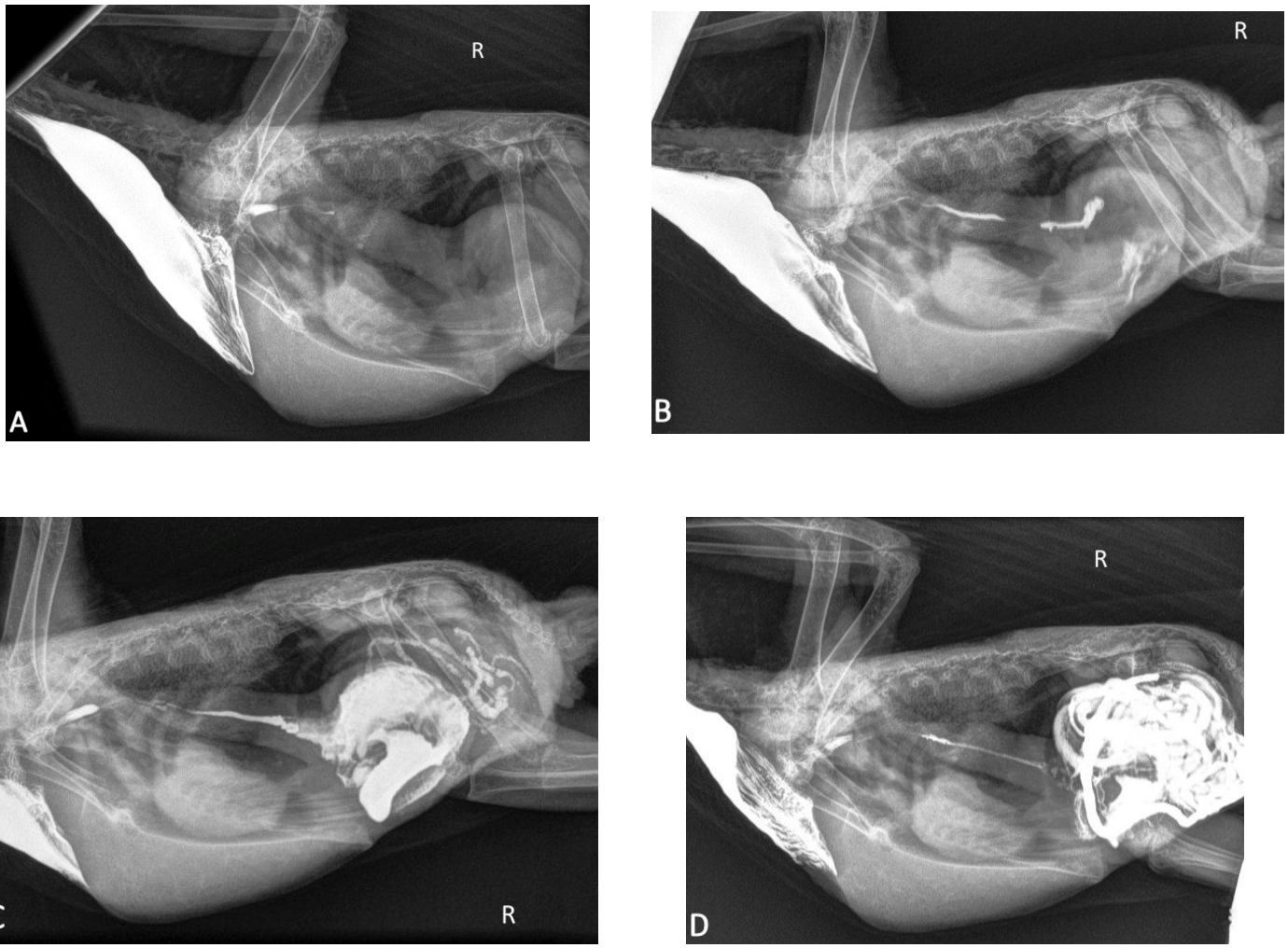

Figure 2: Right lateral barium contrast study.
5 (A), 15 (B), 90 (C) and 240 (D) minutes after administration of barium sulphate solution.

Chlamydia testing was negative. Results of the CBC revealed a low normal PCV (43; 42-55 x 10^9/L) and a normal total white cell count (11.4; 9.0-15.0 x 10^9/L) with a heteropaenia (5.1 x 10^9/L (45%)); mild toxic change was observed on a blood smear. Results of the biochemical analysis showed moderately elevated cholesterol (10.3; 2.6-6.8 mmol/L) and creatinine kinase (915; 132-410 IU/L) and mild decreases in amylase (458; 562-684 IU/L) and uric acid (0.10; 0.12-0.65 mmol/L). All other CBC and plasma biochemical parameters were within reference ranges. The changes were interpreted as consistent with inflammation associated with the gastrointestinal tract and a potential malabsorption syndrome.

The following day a commercial barium sulphate preparation was administered by crop gavage, and radiographs taken at 5, 20, 60, 90 and 240 minutes post ingestion. These showed an apparent hypertrophy of the wall of the proventriculus and corresponding narrowing of the lumen, a loss of definition at the isthmus region between the proventriculus and ventriculus, enlargement of the ventriculus and a delayed gastrointestinal transit time (Figures 2a-d). An invasive infection of the proventricular wall was suspected.

Therapeutically, itraconazole (Sporonox 100mg/10mL, Janssen-Cilag Pty Ltd, 1-5 Khartoum Rd, Macquarie Park, NSW 2113) at 10mg/kg PO SID replaced nystatin; the amoxicillin-clavulanic acid course was continued. The bird gained weight and the regurgitation stopped whilst in hospital, so was discharged on medications two days later. The owner was advised to change the brand of the hand-rearing formula and begin a gradual conversion onto a commercial pelleted diet as long as the bird remained stable.

The bird was re-examined seven days post discharge, weighing 358g. The owner reported the bird was much improved in demeanour and the regurgitation had resolved. At ten days post discharge, the bird continued to improve in demeanour and appetite, but had dropped weight to 326g. Intermittent regurgitation was reported. The owner declined further investigations but agreed to trial treatment with sucralfate (Carafate 1g/tablet, Aspen Pharma Pty, 34-36 Chandos St, St Leonards, NSW 2065) 25mg/kg PO TID in addition to the other medications.

Four weeks following discharge the patient was presented due to a decline in condition and demeanour. Having increased weight to 370g, the bird now weighed 338g and the owner reported an intermittent dribbling of liquid food from the beak. The bird was admitted to the hospital for stabilisation. Barium radiographs were repeated; no additional abnormalities were detected. Three days after admission, weighing 314g, the bird was premedicated with 1mg/kg midazolam (Hypnovel, 10mg/2mL, Roche Products Pty Ltd, 4-10 Inman Rd, Dee Why, NSW 2099) and 1mg/kg butorphanol (Ilium Butorgesic, 10mg/mL, Troy Laboratories Australia Pty Ltd, 35 Glendenning Rd, Glendenning, NSW 2761, Australia) intramuscularly. General anaesthesia with 3% isoflurane in oxygen was induced by mask and a 2mm diameter uncuffed endotracheal tube placed. The bird was maintained on 2% isoflurane in oxygen with IPPV at 4 breaths per minute. Subcutaneous Hartmanns solution (Plasma-lyte 148 Replacement IV Solution, Baxter Healthcare Pty Ltd, 1 Baxter Drive, Old Toongabbie, NSW 2146, Australia) at 3% bodyweight was administered into the precrural fold.

An exploratory laparotomy was performed. In right lateral recumbency, an incision was made in the left paravertebral space using an Ellman radiosurgical unit (Ellmann Surgitron, Ellmann International Mfg, Hewlett, NY, USA). The coelomic cavity was visually inspected using a rigid endoscope (Hopkins 2.7mm Forward Oblique Endoscope, Karl Storz GmbH & Co, Mittelstr. 8, 78532 Tuttlingen, Germany). The testicles, kidneys, intestines and liver appeared grossly normal. The external appearance of the proventriculus and ventriculus was grossly normal. Stay sutures were placed into the proventriculus with 3-0 polydioxanone (PDS II, Ethicon, EBOS Healthcare Australia, 2/109 Vanessa St, Kingsgrove, NSW 2208, Australia), and a full thickness biopsy taken. The inner proventriculus was examined endoscopically, and had a pale, green-tinged appearance with multiple raised ridge-like areas. No foreign material or ingesta were identified. The biopsy site was closed with 4-0 polydioxanone (PDS II, Ethicon, EBOS Healthcare Australia, 2/109 Vanessa St, Kingsgrove, NSW 2208, Australia) in a simple interrupted pattern, and the proventriculus leak tested with sterile saline. The coelomic muscle layers were closed with 4-0 polydioxanone in a simple interrupted pattern, and the skin was closed with 3-0 polyglactin 910 (Vicryl, Ethicon, EBOS Healthcare Australia, 2/109 Vanessa St, Kingsgrove, NSW 2208, Australia) in a simple continuous pattern. Recovery from general anaesthesia and surgery was uneventful, and the patient had oral meloxicam (Ilium meloxicam, 0.5mg/mL, Troy Laboratories Australia Pty Ltd, 35 Glendenning Rd, Glendenning, NSW 2761, Australia) at 1mg/kg PO BID administered in the immediate post-operative period. A small amount of the biopsied tissue was submitted for bacterial and fungal aerobic and anaerobic culture and sensitivity. An impression smear was made with the remaining tissue, which was subsequently placed into formalin and submitted for histopathology.

In house examination of the impression smear revealed very low numbers of Gram-positive cocci in the background of erythrocytes and activated heterophils. No overt fungal elements were identified. The biopsy tissue was negative on bacterial and fungal culture. Histopathology revealed that the tissue had a disrupted koilin layer at the surface and a well differentiated epithelial component extending to the muscle layers, consistent with the appearance of normal ventricular tissue (see discussion). There was mild distortion of the surface of the koilin but

it was unclear whether this was a sampling artefact. No abnormalities in the myenteric plexus' were identified. No distinct fungal or yeast elements were identified in PAS stained sections. An open diagnosis was given.

The patient was discharged three days post-surgery on meloxicam, itraconazole, sucralfate and amoxicillin-clavulanic acid. Itraconazole and amoxicillin-clavulanic acid were stopped four days post-surgery once negative bacterial and fungal culture results had been received. The owner reported that at this time, the bird was doing well and body weight had increased to 326g. Fifteen days post-surgery the owner reported a weight of 324g and that the bird was deteriorating; euthanasia was considered. After this, contact was lost until four months post-surgery, when the owner reported the bird was much improved and all medication had ceased. Unfortunately subsequent to this the bird's owner has become very ill and the patient has been lost to follow up.

Discussion

Inappetance, regurgitation, delayed crop emptying and weight loss despite a ravenous appetite are signs that may be associated with gastrointestinal disease in parrots, but determining the underlying aetiology of these symptoms can be complicated since dysfunctions associated with the crop, oesophagus, proventriculus, ventriculus and intestines often have a similar clinical presentation (Morrisey, 1999). Numerous diagnostic tests are available to aid in the diagnosis of gastrointestinal disease in psittacine birds; a thorough review with helpful algorithms is provided by Rupley (1999).

Proventricular disease should be suspected in any psittacine bird presenting with regurgitation or vomiting, anorexia or passing undigested food in faecal matter (Dennison et al., 2009). However non-specific signs such as fluffing or lethargy can be indicative of gastrointestinal disease (De Voe et al., 2003). Dennison et al. (2009) determined that calculating the proventricular-diameter: keel-height ratio is an objective method for differentiating between a normal and enlarged proventriculus in psittacine birds (Dennison et al., 2008, 2009). A proventricular-diameter: keel-height ratio of ≥ 0.522 is indicative of proventricular enlargement. In this instance, a proventricular-diameter: keel-height ratio of 0.91 was calculated, hence investigations focused on the proventriculus as the cause of the gastrointestinal dysfunction in this patient. Aetiology of proventricular disease may include an infectious agent (bacterial, fungal, viral or parasitic in origin), foreign body ingestion, a toxic proventriculitis, papillomatosis, or neoplasia (De Voe et al., 2003; Dennison et al., 2009). Hand-fed chicks may also have an enlarged proventriculus until approximately one year of age (Rupley, 1999). In this case, given the age of the patient, a congenital abnormality causing a mechanical obstruction in the oropharynx, crop or proventriculus was also considered.

Diagnostic tests included cytology of the crop contents and faecal matter, a CBC and plasma biochemistry, Chlamydia antibody testing, plain and barium radiographs (including calculation of the proventricular-diameter: keel height ratio), and an exploratory laparotomy that included coelomic endoscopy and a biopsy of the proventriculus with samples submitted for bacterial and fungal culture, cytology, and histopathology. Whilst wound dehiscence and leakage of proventricular contents are significant risks associated with proventriculotomy (Speer, 1998), in this instance the patient made a complete recovery post-surgery without evidence for such complications.

In this bird, CBC and plasma biochemistry revealed mild inflammatory changes associated with gastrointestinal inflammation and malabsorption. Plasma lead and zinc titres may have been appropriate in this case, but were not performed as there was no historical evidence of exposure to toxins, and the radiographic findings were not consistent with the typical appearance of birds with heavy metal toxicosis. Metal toxicosis has been associated with ileus, proventricular impaction, and erosions of the koilin (Lumeij, 1994; De Voe et al., 2003).

Mycotic proventriculitis such as that caused by *Macrorhabdus ornithogaster* or *Candida* species and parasitism by coccidian or cryptosporidian organisms may affect the structure of the proventriculus or gizzard lining and koilin layer (Anderson, 1993; De Voe et al., 2003; Ravich et al., 2014). In this patient these conditions were excluded due to lack of evidence based on crop wash and faecal cytology and tissue histopathology. Faecal flotation or PCR testing may have been useful to definitively rule out gastrointestinal parasites prior to histopathology, but were declined. Viral agents such as avian adenovirus and avian bornavirus (ABV) have been shown to affect the structure and function of the gastrointestinal tract (Goodwin, 1993; Hoppes et al., 2013). Viral testing was declined, but again the likelihood of these agents causing the proventricular wall hypertrophy in this patient was considered low since there was no histopathologic evidence for viral inclusion bodies or the pathognomonic lymphoplasmacytic infiltrates in the myenteric plexus associated with ABV infection/proventricular dilatation disease (PDD). In addition, PDD is more likely to cause the distension of a thin-walled proventriculus due to accumulation of food secondary to gastrointestinal hypomobility (Hoppes et al., 2013) rather than hypertrophy of the proventricular wall as demonstrated in this patient.

Since the patient improved with treatment, histopathology was limited to a single biopsy sample. At the time of surgery, the biopsy appeared anatomically to have been taken from the proventriculus although this was not consistent with the histopathological results reported. Rath-

er, histopathological assessment determined the sample to have been consistent with the typical appearance of the ventricular lining. This may have resulted from sampling artefact, misinterpretation of the histopathological findings, or have represented the true appearance of the proventriculus. In addition, as the patient had received antimicrobial and antifungal therapy prior to the biopsy sample being taken, bacterial or fungal agents that may have been present initially may not have been in evidence at the time of histopathological examination.

Nutritional factors may also have an effect on the structure and function of the proventriculus. Diets low in fibre (O'Dell et al., 1959; Riddell, 1976) and protein (Branion, 1963) have been shown to cause proventricular hypertrophy in broiler chickens. In parrots, proventriculitis associated with hypovitaminosis A has been reported (Keymer and Samour, 2000). Proventricular hypertrophy of unknown aetiology has also been reported in some macaw and cockatoo species (Keymer and Samour, 2000). Although this bird was fed on a proprietary hand-rearing formula suitable for psittacine chicks during weaning, it is possible that the nutritional composition of this or the home-made diet may have resulted in hypertrophy of the proventricular wall. This hypothesis is pertinent since the bird appeared to improve following a change in brand of hand-rearing formula and subsequent conversion onto a high quality pelleted diet. Histopathological changes associated with nutritionally related proventricular hypertrophy in chickens included submucosal glandular dilation and degeneration (O'Dell et al., 1959). Definitive diagnosis in this case would likely have relied on post mortem examination and histopathology. This did not happen since in spite of the initial severity and chronicity of the clinical signs, with treatment, diet change and supportive care, the bird made a full recovery.

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