Intestinal Obstruction in a Rainbow Lorikeet

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A four-year old male rainbow lorikeet *Trichoglossus haematodus* was presented for acute vomiting. It was depressed with an empty crop and no faeces in the droppings. Crop wash displayed a mixed overgrowth of Gram-positive cocci and Gram-negative rods. Antibiotic therapy was initiated consisting of enrofloxacin (Baytril, Bayer) 30mg/kg IM SID and amoxycillin/clavulanic acid (Clavulox Drops, Pfizer) 125mg/kg SC BID.

Radiographs revealed gross distension of the proventriculus. It vomited immediately after the initial radiographs were taken what appeared to be faecal material with a few particles of grit. Repeat radiographs displayed a normal-sized proventriculus. There was a delayed passage of barium from gizzard into the duodenum until 90 minutes after administration. The barium then passed a reasonable distance into the intestines, which later became much distended. There appeared to be an ileus, either primary or secondary to an underlying obstruction which had now moved along the intestines. Metoclopromide (Metomide, Ceva) 1mg/kg SC TID was given as both an anti-emetic and prokinetic medication in addition to the antibiotic therapy.

The bird later passed bloody diarrhoea droppings. A Gram stain of the faeces revealed a heavy growth of Gram-negative rods and little other bacteria, however a suspicion of a clostridial infection was still considered possible based on the distended intestines and bloody faeces.

The rainbow lorikeet again vomited overnight but following day was brighter and eating. However it further vomited in the evening. Despite no metal particles evident on the radiographs, the owner had discovered a toy bell which appeared to be chewed in the cage at home. Blood tests were declined. A course of Calcium EDTA injections (Calsenate Injection, Parnell Laboratories) 45mg/kg SC BID for three days was also commenced.

Over the following days the bird was treated with a combination of calcium EDTA injections, antibiotics, metoclopromide and fluid therapy (Hartmanns Solution and 5% glucose). There was some improvement in appetite but it would often vomit food a few hours later. Of the scant faecal material passed this was bloody and mucoid. Sucralfate (Carafate, Axcan Scandipharm Inc.) 25mg/kg PO TID was also given for gastrointestinal ulceration. The lorikeet did not improve in general and repeat radiographs continued to show a distended proventriculus and grossly distended intestines. After the bird vomited the proventriculus would return to a normal size but intestines remained distended. Repeated barium studies did not define a foreign body but dilated intestines of reasonable length. The barium did not however pass completely and it was vomited several hours later.

The owner declined exploratory celiotomy for a suspected foreign body for financial reasons and guarded prognosis. Medical management, also with a guarded prognosis, was continued with the antibiotics changed to oral dosing, and the addition of ranitidine (Zantac, GlaxoSmithKline) 2mg/kg SC BID and itraconazole (compounded by local pharmacy) 10mg/kg PO BID. Repeated crop wash and

faecal examination showed minimal bacteria and no gram negatives or fungi. The lorikeet continued to vomit several hours after eating and was passing very little faeces which still contained blood and mucus. The appetite of the bird started to decline for a further three days, as did it its general demeanor and body condition.

Eight days after the bird was first presented it started to improve. It passed a very large sloppy dropping with a small amount of blood and mucus. There was no evidence of a foreign material in the faeces, however there were some structures which looked like branches of budding yeasts and capsulated. These were not seen again. The appetite improved significantly and it continued to improve in its general demeanor. The vomiting appeared more as regurgitation, where it just dropped its head and a small amount of fluid was expelled, and became much less frequent and smaller volumes. The bird continued to rapidly improve and was discharged from hospital 11 days following initial admission with oral medications which included enrofloxacin, amoxycillin/clavulanic acid, sucralfate and itraconazole.

Four days after discharge, despite continued improvement in both appetite and bodyweight, the bird started to vomit again and would 'cry' when passing droppings. Metronidazole (Flagyl Suspension, Sanofi-Aventis) 10mg/kg PO BID was added to the treatment as there was no evidence of gastrointestinal bleeding now and the owners were reluctant to hospitalize the bird again. The bird then continued to improve with only the very occasional vomit/regurgitation. He returned to his previous body weight. Within two weeks after discharge only the metronidazole and itraconazole were continued and the vomiting had ceased altogether.

The patient was re-presented five weeks after discharge from hospital for reoccurrence of inappetance and vomiting. He had been deteriorating rapidly over the previous 5 days. He was also polydipsic and polyuric. Clinically the bird had lost a significant amount of body condition since seen 10 days earlier. It was dyspnoeic with a grossly distended abdomen which was fluid filled. Further work-up and hospitalisation was declined by the owner and abdominocentesis was not performed due to the high risk that it may be due to distension of intestines as was previously seen. Other differential diagnoses included other organomegaly and peritonitis.

The bird failed to respond to medical management and was euthanased four days later. Post-mortem revealed massively distended fluid-filled small intestines and proventriculus. There was intestinal obstruction at the caeco-colic junction and extending into the proximal colon 15mm. It appeared grossly to consist of hard caseous material but no gross evidence of a foreign body and not adherent to the intestinal wall. There was no gross thickening or devitalisation of the intestinal wall at any part.

DISCUSSION

As a review the basic anatomy of the avian intestinal tract is as follows. The avian duodenum emerges from the ventricular pylorus and forms a loop which encompasses the tri-lobed pancreas. In general, the jejunum is thought to begin just distal to where the ascending duodenal loop begins to turn back on itself, where the jejunal branches of the mesenteric artery begin. The ilium is thought to begin at the vitelline (Meckel's) diverticulum and end at the recto-caecal junction (Taylor, 2000). Nectivorous and insectivorous birds have shorter intestinal tracts than the granivorous and herbivorous species. This is due to the higher digestibility of their diets (Gartrell, 2000). The caeca arise at the ileocolic junction and are absent or rudimentary in parrots. The avian rectum or colon is very short and straight and similarly structured to the small intestine. In parrots, the rectum enters the coprodaeum from

the left side (Gelis, 2006).

There is no doubt that severe generalised ileus was present in this patient. However, the initiating cause may have been one or a combination of several reasons. Physical obstructions suspected in this case included evidence of a grit particles in the early vomit, thought to have been from the cement perch in the cage; although not seen, other foreign material may also have been ingested when the bird was free-flighted. The caseous material seen on post mortem may have been associated with the initial problem rather than a complication later. Neurogenic (or paralyitic) causes of ileus in this lorikeet include bacterial enteritis and the possibility of ileus secondary to metal toxicity.

The heavy growth of Gram-negative bacteria seen on the early Gram stain might be considered both as a primary pathogen or as a result of bacterial overgrowth secondary to the ileus.

Ingestion of foreign material is not uncommon in companion birds (Rosenwax and Peacock, 2007) and intestinal obstruction may result if the object is small enough to pass through the pylorus. However, this patient was unusual in that the obstruction was located at the caeco-colic junction. Most reports both in the literature and personal communication describe the obstruction in the duodenum and some in the jejunum. The radiographs and barium studies support the theory that the obstruction, either physical or paralytic, was very distal along the length of the intestinal tract even in the early stages.

Another unusual feature in this clinical case is the extended period of apparent recovery between the initial ileus and final obstruction. At post-mortem the obstruction at the caeco-colic junction was completely occluding the intestinal tract and did not contain any foreign ingested material on gross examination. It is possible that the hard caseous material may have resulted from an auto-obstruction due to a necrotic intestinal slough.

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