

Acute pancreatitis in parrots

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Acute pancreatitis was diagnosed in three parrots. Antemortem diagnosis of pancreatitis in the avian patient is rarely documented, and should be suspected in birds showing clinical signs attributable to abdominal pain or gastro-intestinal dysfunction. However, not all birds with confirmed pancreatitis display these signs. A serum amylase level greater than 1,500 U/L is suggestive of pancreatitis and pancreatic biopsy can be used to confirm a diagnosis. The aetiology of acute pancreatitis in parrots is discussed and a suggested treatment protocol is described.

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BID	Twice daily
IM	Intramuscular
LRS	Lactated Ringers Solution
N-3 FA's	Omega 3 Fatty Acids
N-6 FA's	Omega 6 Fatty Acids
PMV III	Paramyxovirus Type III
PO	per os
SC	Subcutaneous
SID	Once daily
TID	Three times daily

Although acute pancreatitis is a commonly diagnosed condition in mammalian medicine, its ante-mortem diagnosis and treatment in birds are relatively poorly documented.¹ Birds are frequently presented to veterinarians with clinical signs referable to gastro-intestinal dysfunction or abdominal pain, but little work has been done linking these signs with pancreatitis.

This paper presents three case reports and discusses the clinical signs, diagnosis, aetiology and treatment of acute pancreatitis in parrots. The uses of serum amylase determination and pancreatic biopsy to achieve a diagnosis are highlighted.

Case report 1

An 8-year-old female Red-Collared Lorikeet (*Trichoglossus haematodus rubritorquis*) was presented for vomiting. On examination, her crop was flaccid and distended, and she was markedly underweight at 101 g. (On previous examinations this bird had been slightly obese at 158 g.) A Gram stain of crop contents showed high numbers of Gram-negative bacilli, but no yeasts were detected. The bird was treated with IM enrofloxacin (15 mg/kg SID), metoclopramide (0.5 mg/kg SID) and SC LRS. After 2 days with little improvement a biochemistry panel was performed in-house on a Reflotron[®] dry chemistry analyser. The only abnormal finding was an elevated serum amylase concentration (> 1,800 U/L). A provisional diagnosis of pancreatitis was made. Treatment was continued with enrofloxacin and cisapride (1 mg/kg PO, BID). After 1 week secondary candidiasis was detected, the enrofloxacin suspended and oral amphotericin B (100 mg/kg BID) started. The bird gained weight, but was still dull and occasionally vomited. Serum amylase was still elevated (>1,800 U/L) 2 weeks after initial presentation. After 2 days of intensive SC LRS therapy serum amylase remained elevated, and so exploratory coeliotomy was performed.

The bird was anaesthetised with 5% isoflurane, and maintained at 3% via a facemask. With the bird in dorsal recumbency, the skin between the sternum and cloaca was plucked and disinfected for surgery. The skin and linea alba were incised for 1 to 2 cms in a cranio-caudal direction midway between the sternum and cloaca. Care was taken when entering the abdomen not to damage underlying viscera. The duodenal loop, found on the right side of the abdominal cavity, was gently exteriorised. No gross abnormalities were seen in the pancreas. The distal edge of the ventral lobe of the pancreas, at the apex of the duodenal loop, was carefully reflected to reveal underlying vasculature. Once these blood vessels were located and avoided, the end of the ventral lobe was removed with iris scissors and fixed in formalin. Minimal bleeding resulted. The duodenum was then replaced into the abdomen, and the skin and muscle closed with 4-0 braided polyglactin 910 (Vicryl). The whole procedure was accomplished in less than 15 minutes. The biopsy sample was submitted to Veterinary Pathology Services (now VPS-Idexx) in Brisbane. There it was processed and stained with Haematoxylin and Eosin.

Histological examination of the biopsied tissue showed a local area of acinar disruption, associated with marked infiltration by lymphoid cells, heterophils and mild fibroplasia, confirming the diagnosis of pancreatitis. Omega 6 and Omega 3 fatty acids (N-6 and N-3 FA's) were added to the treatment (0.11 mL/kg PO, BID of a 5:1 ratio supplement), and, with budding yeasts still detectable on a faecal Gram stain, amphotericin B was replaced with fluconazole (5 mg/kg PO, BID). Pancreatic extract in powder form (Enzyplex[®], Nature Vet) was added to the bird's diet at a rate of 1 teaspoon per 100 mLs nectar. The candidiasis cleared, the vomiting stopped and the bird gradually gained weight, until 2 months after the initial presentation it had returned to its previous weight of 158 g.

Case report 2

A 5-year-old male Greater Sulphur Crested Cockatoo (*Cacatua galerita galerita*) was presented for feather picking and mild lethargy. On physical examination the bird was slightly overweight (870 g) and was feather picking across its back and dorsal wings. A biochemical profile revealed an elevated serum amylase (5,600 U/L) as the only significant abnormality. Faecal and crop Gram stains were normal. A pancreatic biopsy was performed (anaesthesia and surgery as previously described). Histological examination (VPS-Idexx, Brisbane) showed occasional foci of acinar degeneration associated with mixed leucocytic infiltrates, amongst which lymphocytes, histiocytes and heterophils were identified. This infiltrate was particularly noticeable in periductal locations.

The bird was treated with enrofloxacin and N-6 and N-3 FA's as described previously. It was converted to a pelleted diet (Parrot Maintenance Diet, Vetafarm, Wagga Wagga), seed was discontinued completely, and vegetables and fruit added. Although the bird stopped feather picking, the amylase remained elevated for greater than 1 month, eventually declining to 900 U/L by 6 weeks after presentation. During

treatment the bird's bodyweight dropped to 790 g, but gradually increased to 840 g and has remained stable at that weight for 12 months.

Case report 3

A 6-month-old female Black Capped Lory (*Lorius lory*) was presented with polyphagia and a wide-legged stance. On physical examination the bird was underweight at 120 g (normal for this species 200 g), but no other abnormalities were detected. No skeletal abnormalities were detected on palpation. Faecal and crop smears were negative for parasites, and Gram stains were normal. Serum biochemical and haematological values were normal except for elevated serum amylase levels (2,260 U/L) and a mildly elevated white cell count (16.6×10^9 , normal 8 to 13×10^9).³ A provisional diagnosis of pancreatitis was made, and a pancreatic biopsy was performed as previously described. Histological examination (VPS-Idexx, Brisbane) showed multifocal areas of oedema, slight haemorrhage and exocrine acinar degeneration associated with minimal mixed leucocytic infiltration, suggesting an acute to sub-acute pancreatic necrosis. The bird was treated with oral enrofloxacin (30 mg/kg SID) and pancreatic enzyme extract (Enzyplex®, Nature Vet). The bird's normal diet, a high fat nectar mix, was restricted to a small meal twice daily, and the amount of fruit and vegetables offered was increased. Unfortunately this bird was accidentally killed 4 days after biopsy. However, the owner reported that the bird was alert and eating well prior to its demise.

Discussion

The avian pancreas consists of three lobes. The dorsal and ventral lobes are supported and separated by the pancreatic artery within the duodenal loop, and the splenic lobe runs more laterally up to the spleen, as an extension of the ventral lobe. The pancreas has both endocrine and exocrine functions. While the amount of endocrine tissue is proportionally greater than that of mammals,² over 99% of the pancreatic mass has an exocrine function.³ The exocrine pancreas consists of compound tubuloacinar glands divided into lobules. These glands secrete amylase, lipase, proteolytic enzymes and sodium bicarbonate into the ascending duodenum via pancreatic ducts.⁴ Pancreatic secretion, which is at a higher rate than that of mammals, is controlled by both neural and hormonal mechanisms. Immediately a bird starts eating, pancreatic secretion begins, apparently via a vagal reflex. Distension of the proventriculus stimulates a hormonal response involving vasoactive intestinal polypeptide that results in pancreatic secretion. Diet can also affect the rate of secretion, with diets high in fat and carbohydrates increasing the activity of amylase and lipase.⁴

Pancreatitis develops when there is activation of the digestive enzymes (trypsin, protease and phospholipase amongst others) within the gland, with resultant pancreatic autodigestion. Damage to the pancreatic cell walls allows the release of these enzymes into the intracellular space and ducts, and this in turn causes the production of unopposed free radicals, which cause even more damage. This again releases more enzymes, and the cycle continues. Plasma protease inhibitors are vital in inhibiting this cascade.⁵

As with mammals, the initiating aetiology is often difficult to pinpoint. Possible aetiological agents and contributing factors include obesity, often when combined with high fat diets or fatty meals,^{1,5,6} toxicity, particularly zinc,^{1,3,7} mycotoxins⁸ and selenium,⁹ trauma,⁵ viral infection (including PMV III,¹⁰ adeno-

virus,^{11,12} avian influenza A,^{13,14} infectious bronchitis,¹⁵ and herpesvirus¹⁶), *Chlamydophila* infection,¹⁷ bacterial infection,⁵ egg yolk peritonitis,^{1,3} and neoplasia.^{1,3,5,18-20}

The birds described in the case reports above were on high fat diets and two of the birds (Case Reports 1 & 2) had been overweight prior to, or at the time of, the onset of clinical signs. These may have been predisposing factors in the development of pancreatitis in these birds.

Clinical signs shown by birds with pancreatitis usually reflect gastrointestinal dysfunction and pain, and include vomiting, diarrhoea and ileus, as seen in the bird described in Case Report 1. Speer¹ lists clinical signs including anorexia, lethargy, 'colic' or signs of abdominal pain and discomfort, weight loss, polyuria, polydipsia and abdominal distension. Abdominal pain is often a feature of pancreatitis in mammals, and may well be present in birds. Indications of abdominal pain in birds include kicking, falling off a perch, picking towards the abdomen, feather picking, sudden flight attempts, aggression, and obsessive chewing on the cage and other items.²¹ It is possible that abdominal pain or discomfort may have accounted for the feather picking displayed by the cockatoo described above (Case report 2) and the wide-legged stance of the third bird. These birds showed no other signs obviously attributable to pancreatitis. This indicates that not all birds with pancreatitis will show 'classical' signs of pancreatitis, a point also emphasised by Speer.¹ The reason for the polyphagia in the third bird was unclear, as histological examination revealed no evidence of pancreatic insufficiency.

Determination of serum amylase and lipase levels is frequently used in mammalian medicine to diagnose pancreatitis. Amylase is secreted in saliva, intestinal fluid and pancreatic juices.⁴ In mammals and birds, pancreas-derived amylase makes up only a small part of serum amylase, but with acute pancreatitis and leakage of pancreas-derived amylase, total serum amylase levels rise significantly. Other causes of hyperamylasaemia include renal disease, small intestinal obstruction, other alimentary disorders, and glucocorticoid administration.²² However, the rise in serum amylase in these conditions is usually more moderate, in the range of a two- to three-fold increase.^{5,8} Fudge³ described birds with hyperamylasaemia but no pancreatic lesions. These birds did have histological evidence of intestinal disease, thus indicating that not all birds with hyperamylasaemia have pancreatic disease. However, he did not comment on the magnitude of the rise in serum amylase. Clinicians need to interpret serum amylase levels with care. In one study on birds affected with suspected PMV III,¹ there were inconsistencies between serum amylase levels and the severity and type of pancreatic lesions. However, both Speer¹ and Fudge³ agree that serum amylase levels greater than 1,000 U/L are elevated. The author's experience is that levels above 1,500 U/L appear to be associated with pancreatitis. Therefore, it would appear that significant rises in serum amylase should lead the clinician to consider acute pancreatitis in birds showing signs consistent with gastrointestinal dysfunction or abdominal pain. Concurrent evaluation of serum uric acid levels can be used to evaluate renal disease as a possible cause of elevated serum amylase.

If pancreatic disease is suspected, the diagnosis can be confirmed by a pancreatic biopsy. This can be done via a coeliotomy, or endoscopically through the right thoracic air sac.²³ Coeliotomy and pancreatic biopsy are simple procedures with minimal risk or complications. Histological evaluation of the pancreas allows the clinician not only to

confirm a diagnosis, but also more accurately to evaluate the likely prognosis and required treatment.

At this time there have been no treatment protocols developed for avian pancreatitis. However, treatment should follow the guidelines used in treating pancreatitis in dogs and cats. Suggested treatment should include oral, subcutaneous, intravenous or intraosseous fluid therapy (such as LRS) to correct dehydration and improve perfusion of the pancreas. Fasting should not be used as a treatment in birds, because of their high metabolic rate and energy requirements. However, conversion to a pelleted diet, where appropriate, is likely to be of benefit. These diets are generally low in fat and provide a consistent balanced diet.

If gastrointestinal ileus develops the use of motility modifiers such as metoclopramide (0.5 mg/kg IM, BID) or cisapride (1 mg/kg PO, BID) may be warranted. Abdominal pain can be treated with analgesics. Butorphanol (3-4 mg/kg IM, TID) and carprofen (2-10 mg/kg IM, SID) have been used in birds for analgesia,²⁴ and may play a role in the treatment of avian pancreatitis. Parenteral antibiotics may be required if bacterial involvement is suspected. Enrofloxacin penetrates the canine pancreas,⁵ and might be expected to penetrate the avian pancreas.

If possible, identify, treat and eliminate inciting causes of pancreatitis. For example, Speer (personal communication) has used carboplatin to resolve pancreatic adenocarcinoma in a Green Wing Macaw (*Ara chloroptera*). Calcium EDTA (10 to 45 mg/kg BID, IM) can be used to chelate zinc in cases of heavy metal toxicity.

Recently attention has been focused on the use of N-6 FA's and N-3 FA's for their anti-inflammatory, lipid stabilising, anti-neoplastic and other potential qualities. They have been shown to reduce hyperlipidaemia in dogs,²⁵ and may also prevent the development of Type II diabetes mellitus in humans.²⁶ Speer (personal communication) uses a 5:1 ratio combination of N-6 and N-3 FA's (0.11 mL/kg SID) for treating glomerulonephritis. The author has used this treatment for birds with acute pancreatitis, but it is difficult to assess its efficacy in clinical cases. Based on work with canine renal disease, treatment could be expected to be required for a minimum of 2 to 4 weeks. The use of N-6 and N-3 FA's in acute pancreatitis in birds represents an area for future research.

Work in dogs has shown that whole blood or plasma transfusions may replace protease inhibitors cleared from the circulation during severe pancreatitis. This may be lifesaving in some cases, inhibiting the cycle of pancreatic cell damage,⁵ and may be of benefit in severe cases of pancreatitis in birds. The use of pancreatic enzyme therapy may decrease the pain that accompanies chronic pancreatitis in human beings, probably by an inhibitory feedback on endogenous pancreatic secretions.⁵ This therapy may be worth pursuing in cases of avian pancreatitis, as chronic pancreatitis may be a sequel to acute bouts.

Birds that survive a bout of acute pancreatitis should be regularly monitored for evidence of pancreatic insufficiency and diabetes mellitus, both well-recognised sequelae to acute pancreatitis in mammals. Regular weight checks and annual blood screening may detect complications before they become life threatening. According to Speer (personal communication), most pancreatic disease is diagnosed as end-stage pancreatic atrophy, usually found at necropsy. There is obviously scope for earlier intervention in these cases.

Acute pancreatitis in birds may be more common than is documented. It should be considered in the differential

diagnosis in birds with gastrointestinal dysfunction and/or abdominal pain. Hyperamylasaemia should alert the clinician to the possibility of pancreatitis, and pancreatic biopsy can be used to confirm a diagnosis. Pancreatitis should not be regarded as a disease state in its own right. Rather, it should be seen as part of a larger clinical picture, with further investigation required to elucidate the originating cause. Treatment should be directed towards eliminating the inciting cause and providing supportive therapy, such as fluids, analgesia and a low fat diet. At a minimum, patients diagnosed with pancreatitis should be examined annually to detect any sequelae such as diabetes mellitus or pancreatic insufficiency.

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