

Exocrine pancreatic insufficiency in a sulphur crested cockatoo

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Case report

A 20+ year old female sulphur crested cockatoo with marked weight loss, and weakness was presented for diagnosis and treatment. On clinical examination, the bird was depressed, very thin and had voluminous pale coloured faeces at the bottom of its travel cage. Upon questioning, the owner had noticed the bird's poor feather coat for several weeks, and had seen the bird scrounging for seed on the bottom of its cage more than normal for the last two to four weeks. The bird's poor body condition had not been appreciated until the owner picked the bird up, and this precipitated the visit to the clinic. Exocrine pancreatic insufficiency was suspected based on the clinical signs and the bird was admitted to the clinic for further work up.

A blood sample was taken but some biochemical and enzyme readings were too high for the in-house laboratory equipment to determine. The sample was therefore sent to an outside laboratory for biochemical analysis. The PCV was 41% (normal for a sulphur crested cockatoo: 41 to 60%)¹, total protein 2.4g/dL (normal for a sulphur crested cockatoo 2.2 to 6.5g/dL)¹, serum amylase was elevated to 1818 IU/L (normal range for parrots 150-645 IU/L)², aspartate aminotransferase was mildly elevated at 817 IU/L (normal for sulphur crested cockatoos 167-660IU/L)¹ and serum uric acid was elevated at 1018 µmol/L (normal range 208 to 624 µmol/L)².

The bird was weighed at 574 grams (average body weight for a sulphur-crested cockatoo 875grams)³. The pectoral muscles had atrophied significantly, and a very prominent keel bone was palpable.

A Sudan III stain of the bird's faeces showed the presence of an increased amount of lipid. There was also a small increase in the faecal yeast population demonstrated on a faecal Gram stain.

Due to the bird's poor body condition, supportive therapy was initiated. The bird was given a 25ml intravenous bolus of warm Hartmann's solution via the jugular and an intramuscular injection of piperacillin (Pipril, Lederle Laboratories) and multivitamins (Multivitamin injection, Novartis Animal Health Australasia Pty. Ltd.). The cockatoo was fed a 20ml mixture of an oral nutrient supplement (Polyaid, Vetafarm Pty. Ltd.) and Farex (Farex Australia) by crop tube. One 325mg tablet of a pancreatic enzyme supplement (Viokase, Wyeth Ayerst) was crushed and fed with the Polyaid/Farex mixture. The bird was given a poor prognosis for recovery based on its poor body condition and the high serum uric acid.

The next day the cockatoo looked brighter and was eating seed. It had gained an extra 6 grams. Its droppings at this stage of were slightly smaller volume and a more normal colour. Again it received piperacillin intramuscularly and 0.25ml of multivitamin. The farex/polyaid mixture was fed again by

crop tube along with one crushed tablet of Viokase. This treatment was repeated for the following two days. After four days in hospital the bird went home. It was given an intramuscular injection of doxycycline (Psittavet, Vetafarm Pty. Ltd) before leaving to provide long acting antibiotic cover.

The owners were instructed to give one tablet of Viokase a day to the bird. This tablet was to be crushed and sprinkled in the bird's seed. They were also asked to monitor the bird's faeces and body weight.

The bird recovered well on the pancreatic enzyme supplement. Seven weeks after the bird was discharged, its body weight was 650grams. Its droppings were more normal in colour and of smaller volume, although still larger than normal cockatoo droppings. Fourteen weeks after leaving hospital the cockatoo was still doing well. The owner had stopped measuring its weight but reported that the bird looked and felt heavier than at seven weeks after discharge. Its faeces were still of larger volume than normal. The cause of the exocrine pancreatic insufficiency was not determined.

Discussion

Exocrine pancreatic insufficiency is uncommon in birds. It has been reported in several bird species including amazons⁷, macaws⁹, poultry⁴, budgerigars⁵, sulphur-crested cockatoos⁵ and galahs¹². Although the condition can very often be diagnosed on clinical signs, history and response to treatment, the underlying cause of the loss of function of the exocrine pancreas is difficult to determine without an extensive invasive work-up (biopsy, laparotomy) or post mortem examination.

Preliminary diagnosis of exocrine pancreatic insufficiency is done on clinical signs, faecal examination and history. Voluminous pale coloured faeces in a bird that is losing weight but has an increased appetite is almost pathognomonic for exocrine pancreatic dysfunction, but other causes should also be considered. Malabsorption due to primary small intestinal disease presents almost identically to exocrine pancreatic insufficiency in small animals.

An examination of the three components of the droppings, the urine, urates and faeces, is often helpful in determining the cause of the increased faecal bulk. In exocrine pancreatic insufficiency there is often an increase in the fat content of the faeces (false negatives and positives can occur)⁶ because the digestive system is deficient in the enzyme lipase. This can be determined by microscopically examining faeces stained with Sudan III. Faecal amylase activity can be similarly qualitatively measured by staining the faeces with Lugol's iodine. There will be more starch present in the droppings of birds with exocrine pancreatic insufficiency. Faecal trypsin levels can be qualitatively measured, and absence of trypsin in the faeces can be suggestive of exocrine pancreatic insufficiency. These tests are all qualitative and are influenced by factors such as the bird's diet and bacterial populations in the small intestine. As a result false negative and positive results can occur⁶. Therefore they should not be considered alone when trying to make a diagnosis. Faecal examination will also help diagnose other causes of diarrhoea or increased faecal mass. Abnormal bacterial populations or increased numbers of yeast and fungi in the faeces can suggest bacterial or fungal enteritis. Undigested seed in the faeces is suggestive of proventricular dysfunction. Green urates are suggestive of liver disease.

Other tests to determine pancreatic disease in birds include the triglyceride tolerance test performed in the absence of, and then again in the presence of, pancreatic enzyme supplements⁷. Corn oil is fed to the bird and then a serum sample taken two hours later to measure the triglyceride levels. If serum triglyceride levels remain low in the absence of pancreatic enzymes but are then elevated when the test is repeated after supplementing the bird with pancreatic enzymes, this is suggestive of exocrine pancreatic insufficiency. Again this test can have false positive or false negative results. A response to treatment with pancreatic enzyme supplements is another empirical test, which can be performed to assist the diagnosis of exocrine pancreatic insufficiency.

In birds, unlike small animals, it is not possible to measure serum trypsin-like immunoreactivity (TLI) as the test is species specific and there is no commercially available test specifically for use in birds. The measurement of serum amylase and lipase to detect pancreatic disease can be unreliable in birds, mainly due to the little research, which has gone into this topic. It is thought that amylase levels greater than 1500IU/L occur in acute pancreatitis.²²

Exocrine pancreatic insufficiency has been reported in birds with pancreatic atrophy⁹, zinc toxicosis¹⁰, and pancreatic neoplasia⁷. It is also conceivable that exocrine pancreatic insufficiency could develop from other conditions that nonspecifically affect the exocrine pancreas, but it is not the usual clinical presentation. These conditions include acute pancreatic necrosis¹¹, pancreatitis^{10,11}, lipidosis⁴, haemochromatosis⁴, chronic chlamydiosis²¹, and viral infections⁴ including adenovirus¹¹, herpesvirus⁴, polyomavirus⁴ and paramyxovirus⁴.

Pancreatic atrophy has been reported in galahs¹², cockatoos¹² macaws⁹ and poultry⁴. In most species the cause of the exocrine pancreatic atrophy is unknown. In poultry, pancreatic atrophy has been associated with selenium deficiency. Exocrine pancreatic atrophy is also seen in the runting and stunting syndrome of poultry the cause of which is speculated to be a virus¹³

Histologically, exocrine pancreatic atrophy in birds is similar to that seen in dogs⁹. It may then be speculated that, as in dogs, particularly German Shepherds, there may be a hereditary or immune mediated component in the aetiology of pancreatic atrophy in birds.

Zinc toxicosis is relatively common in birds due to the use of galvanized wire in bird cages^{15,16} and the common use of zinc in everyday items^{16,17}. Zinc is an essential trace element used by the body in RNA and protein synthesis. It is a constituent of many metalloenzymes and a co-factor for an array of enzyme systems¹⁹. Excretion of zinc in pancreatic juices is the major means by which mammalian and avian species remove zinc from the body¹⁸. The pancreas also synthesizes a large quantity of proteins. As a result, the pancreas accumulates zinc when a high level of zinc is ingested¹⁷. High levels of zinc cause apoptosis and degeneration of pancreatic acinar cells in birds^{16,20} as in other species. The pancreas is not the only organ affected by high zinc levels however, and zinc toxicity may manifest itself as an organ system failure or just generalized metabolic failure. Acute zinc poisoning can present as depression, anorexia, lethargy, lack of motor coordination and diarrhoea¹⁶ (or constipation)¹⁷. In acute zinc toxicity, many organs such as liver, kidneys, gizzard and pancreas show signs of zinc toxicity but generalized metabolic disruption is the probable cause of the clinical signs seen. In chronic zinc toxicity lethargy, depression, diarrhoea and weight loss is seen. Death due to chronic zinc poisoning is probably due to renal failure¹⁵.

It has been reported that pancreatic neoplasia has resulted in exocrine pancreatic insufficiency⁷. It is conceivable that neoplasia could lead to exocrine pancreatic insufficiency in two ways. Firstly by occluding or destroying the pancreatic ducts leading to the duodenum, or secondly by destroying a large proportion of the exocrine portion of the pancreas resulting in production of an inadequate amount of digestive enzymes. A pancreatic adenocarcinoma has been reported to cause exocrine pancreatic insufficiency by destruction of exocrine pancreatic parenchyma⁷. This tumour also destroyed endocrine pancreatic tissue, but diabetes mellitus was not seen. It is also feasible that a biliary adenocarcinoma could cause exocrine pancreatic insufficiency in a similar fashion. They occur more commonly than pancreatic adenocarcinomas and have been known to invade the pancreas⁷.

Treatment of exocrine pancreatic insufficiency in psittacines is similar to that in small animals. Initially, the bird should be stabilized. Intravenous fluids and nutrient supplements in the presence of a pancreatic enzyme supplement should be given if the bird is severely malnourished. Multivitamins are recommended in the undernourished bird, as the bird may be deficient in some vitamins or trace elements as a result of decreased food digestion.

Antibiotic therapy is given because studies have shown that, both in birds and mammals, abnormal bacterial overgrowth in the small intestinal lumen occurs in the absence of pancreatic enzymes^{9,14}. In mammals, the use of antibiotics in all cases of exocrine pancreatic insufficiency is questionable as the overgrowth is often (but not always) corrected once enzyme replacement therapy is begun¹⁴. In this case, the antibiotics were probably of benefit to the cockatoo to help it fight any infections it may develop in its weakened state.

In the long term, a deficiency of endogenous pancreatic digestive enzymes can be corrected with regular dietary supplements. These can be given with the bird's food each day. One crushed tablet per day delivers enough enzymes to the duodenum for appropriate digestion of food to occur. The bird's diet should be varied to include a mixture of seed, fruit and vegetables with occasional meat and dairy products. Food should be provided ad lib, especially if the bird is underweight. A varied diet will ensure that the bird receives its proper energy, protein, vitamin and mineral requirement.

Although the cause of the problem in this case was not determined, the signs and clinical findings were typical of exocrine pancreatic insufficiency in birds. The cockatoo responded well to treatment for this condition and was reported to be doing well by the owner some fourteen weeks after the cockatoo had been released from hospital. The clinical signs and good response to treatment suggest that a progressive disease was not the cause of the exocrine pancreatic insufficiency. However the presence of a slowly growing neoplasm or a smoldering pancreatitis (possibly supported by the elevated serum amylase) could not be definitively excluded.

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