Lameness in a Sulphur -Crested Cockatoo Associated with Hepatic Iron Accumulation

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CASE HISTORY

A 26 year old obese (1119g) male sulphur-crested cockatoo (*Cacatua galerita)* was presented for listlessness and partial weight bearing on the right leg of five days duration. The bird was fed on many treats including cheese, biscuits, bread, walnuts and occasionally other nuts. The main diet consisted of sunflower seeds as well as a variety of vegetables.

On physical examination the right hock was thickened and swollen. There was moderate to marked inflammation of the left ventral foot. The abdomen was concave with no palpable organ swellings or lesions.

Radiographs revealed an enlarged cardio-hepatic silhouette. The joints appeared normal. Abnormalities on biochemistry included globulin 25 (13-24 g/L), AST 254 (140-360 IU/L), CK 496 (147-418 IU/L), cholesterol 43.2 (3.5-7.3 mmol/L), gldh 72 (1-4 IU/L) and bile acids 362 (0-81 umol/L). Hepatic lipidosis with secondary pressure sores on the hock, related to obesity, was initially considered the most likely cause of the clinical signs. Other differentials included hepatic mycotoxins, chlamydia, bacterial hepatitis and hepatic fibrosis.

The bird was prescribed Chlorhexidine 2% baths to the hock BID 10 days, amoxycillin/clavulanic acid 125mg BID 10 days PO and lactulose with milk thistle (90mg Silybin in 13.5mg lactulose) 0.5ml BID PO. The bird did not improve and was hospitalised five days later for subcutaneous Hartmann's 20ml BID and crop tube feeding with Polyaid 20ml BID for two days. Enrofloxacin 25mg/ml 0.5ml and Fungilin® Lozenges in solution (Amphotericin-B 10mg/6ml sterile water solution) 0.25ml BID 10 days PO were prescribed for home treatment. A Chlamydia Immunocomb antibody test and an in-house lead Leadcare test were both negative. One month later the bird had not improved significantly and the abnormal liver analytes included cholesterol 31.4 mmol/L, gldh 9 IU/L and bile acids 336 umol/L. Under isoflourane general anaesthetic, a biopsy of the liver was performed. The liver was enlarged and friable. The tissue was fixed in 10% buffered neutral formalin and was submitted to the Elizabeth Macarthur Agricultural Institute for histopathological evaluation. Haematology was performed at the time of biopsy. The results were PCV, 40 % (40-48%), leukocytes 32 x 10^3/ul (5-11), heterophils 93% (55-80%), lymphocytes 7% (20-45%). The platelets and red blood cells were normal. No parasites were noted.

RESULTS

On histopathology there was extensive fatty change in the liver, with abundant golden haem globular

pigment in portal and sinusoidal areas within macrophages and Kuppfer cells. No circulating iron-laden macrophages or siderocytes, were noted. The capsule was not thickened. Perles Prussian blue stain revealed clumps of cells and individuals with dark blue granular globular cytoplasmic masses - some were in Kuppfer cells but most were in the portal macrophages. The hepatocytes had finer pale blue granules only sparsely seen in the periacinar regions. The conclusion was hepatopathy with iron accumulation.

TREATMENT

Six mls of whole blood was removed from the ulnar vein weekly for the first two weeks. On the first occasion phlebotomy was performed under isoflourane general anaesthetic with 10ml of Hartmann's SC given subsequently. Six to seven mls of blood were removed from the un-anaesthetised bird via the jugular weekly for a further 14 weeks. After 16 weeks of treatment the bird was not lame and the swelling of the hock had disappeared. The biochemistry and haematology results were now unremarkable. A repeat liver biopsy was performed at 16 weeks under isoflourane anaesthetic. There was a marked improvement in the appearance of the liver. Multifocal groups of vacuoles were noted within the parenchyma. There were occasional clusters of heterophils and lymphocytes as well as occasional small clusters of brown pigmented material within parenchyma. Four subsequent biochemistry and haematology samples performed over two years were unremarkable aside from one spike (42 IU/L) in GLDH one year post surgery. This led to a strict enforcement of a dietary change to pelleted food, vegetables, native plants and no fatty treats. There has been no significant change in weight at its two annual health checks post the original presentation,m it has been bright and happy since the initial 16 week treatment, and there have been no further joint swellings.

DISCUSSION

Hepatic iron accumulation is an uncommon finding in Sulphur crested cockatoos and the treatment regimes have rarely been described for large seed eating psittacine birds. Iron storage has been reported in several species including passerine birds, toucans (Worrell, 1994) and lories (West et al., 2001), as well as several other psittacine species (Pereira et al., 2010). Joint swelling as the primary presenting sign is unusual in cases of hepatic iron accumulation in birds. In humans, joint swelling and lameness combined with listlessness are the three most common presentations in cases of haemochromatosis. This is not the case in birds where lameness is not considered a typical clinical sign of hepatic iron accumulation.

The response to 16 weeks of phlebotomy in this case was similar to that of European starlings *(Sturnus vulgaris*) with haemosiderosis (Olsen et al., 2006). The other treatment option available is the use of the iron chelating capacity of desferrioxamine. The owner's preference was for weekly phlebotomy. Previous treatment regimes have also explored the use of inositol and tannic acid as dietary additives (Olsen et al., 2006). The dietary additives reduce iron absorption rather than remove the iron from the liver. As there was no history of excess iron in the diet, no dietary supplements were used.

The lack of dietary iron suggests the hepatic iron accumulation was the result of a different cause or disease aetiology rather than from dietary iron overload. The distribution of the iron in liver is more consistent with chronic disease rather than dietary iron accumulation. This excess hepatic iron is possibly a reflection of increased iron metabolism associated with increased turnover of tissue iron (Cork, 2000). A common cause of hepatic iron accumulation is fibrosis. This is the most likely scenario

in an obese mature cockatoo on a high fat diet, with fatty changes present in the liver.
In hepatic iron accumulation, biochemical response does not always equate to clinical or histopathological change. In this case the histopathological and clinical improvement post the removal of iron from the liver was related to the return of hepatic biochemical tests to the normal range. This may be a reflection of the improvement in the liver once the iron was removed. Plasma iron levels were not measured as these are not considered a useful indicator of hepatic iron levels (Phalen et al., 2005). No hepatic samples were collected to measure the iron content of the liver. This would have been a very useful method of monitoring the total iron levels present rather than relying on histopathology alone.

There is often the question of whether hepatic iron accumulation is clinically significant or simply an incidental histopathological finding. Hepatic iron accumulation does not always induce clinical signs. In this case the presence of excessive iron accumulation in the liver with fatty depositions combined with the clinical signs is consistent with iron accumulation secondary to chronic fatty liver changes. Clinically, in humans, iron accumulation and hepatic fibrosis/lipidosis may cause listlessness, arthritis and joint swelling (Bacon, 2011). The lameness and joint swelling may simply have been a result of pressure sores and arthritis in an obese bird. However, the bird's response to phlebotomy without significant dietary change, suggests that the hepatopathy and iron accumulation were related to the clinical signs. The joint swellings and listlessness only resolved post-phlebotomy despite there being no significant weight loss or dietary change in the first year post treatment and no response to any prior treatments.

CONCLUSION

Lameness and listlessness in an overweight lame sulphur crested cockatoo related to iron accumulation is an unusual diagnosis. This case shows the value of liver biopsy when there are swollen joints and serially increased liver biochemical analytes. Phlebotomy over 16 weeks was successful in removing the iron accumulation in the liver and relieving the clinical signs of listlessness and a swollen hock in a psittacine bird.

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