



Avian Liver Disease

Liver disease is a frequent finding in pet birds, and may have one of a list of causes. It may be part of a systemic disease process, such as heart disease complex, viral infection or gastrointestinal problems, or the liver itself may be the causative agent. Supportive care is often required to pull a bird through the acute phase of the disease, while home nursing is necessary to maintain a bird with chronic liver dysfunction.

Normal Avian Liver Anatomy and Physiology

The avian liver has a right and left lobe. Generally the right lobe is larger than the left. The caudal border of the right lobe extends to the edge of the sternum or slightly caudal to the sternum. Liver enlargement may sometimes be diagnosed by visualizing an abnormally extended right lobe through the skin and abdominal wall of neonates or small birds such as budgerigars, finches, and canaries.

The avian liver has much less connective tissue than the mammalian liver and it is larger than in mammals of equal body size. The avian liver receives blood from the caudal region of the body via the caudal mesenteric vein, part of the renal portal system. Medications given in the leg area may end up shunted via the renal portal system predominantly into the caudal mesenteric vein and directly into the liver. This is clinically significant as medications that are metabolized by the liver may be less effective because they are metabolized before they enter systemic circulation. As well, hepatotoxic drugs administered in the leg area may compromise the condition of the liver.

The avian liver has the same functions as it does in mammals: to store excess fat and sugar, produce bile, metabolize and synthesize certain proteins, process medications and food products, cleanse blood of bacteria, and excrete waste products. Birds do not manufacture urea in the liver, as do mammals, but instead produces most of the uric acid. A clinically significant difference between the birds and mammals is that birds synthesize almost all their fatty acids in the liver, with very little synthesis in fat tissue.

Liver Disease

Primary liver disease may be caused by metabolic disorders such as hepatic lipidosis, hemochromatosis, malnutrition or amyloidosis. Congenital defects can cause significant liver-related problems. Toxin exposure such as aflatoxins or rapeseed poisoning can cause primary liver disease as well as heavy metal exposure such as lead or zinc or household drugs such as antidepressants or pain relievers. Traumatic injuries can induce liver dysfunction with vascular

disruption or hematomas. Bacterial hepatitis can be caused by many species of bacteria as well as mycobacteria (tuberculosis). Aspergillus, Nocardia, or Candida may cause mycotic (fungal) hepatitis. Many protozoan parasites affect the liver, such as Atoxoplasma, Trichomonas, Histomonas, Plasmodia, and Leucocytozoan. Reovirus and adenovirus target the liver. The liver may also be affected by neoplasia (cancer) or severe scarring (cirrhosis).

Liver disease may also be seen as part of a multi systemic disease. Chlamydiosis, Pacheco's Disease, pox, polyomavirus, Psittacine Beak and Feather Disease, and bacterial septicemias often involve the liver. Cirrhosis of the liver can also be found as part of chronic congestive heart failure.

Fatty liver syndrome is being found with increasing frequency. Excessive fat deposition and storage in the liver can be due to three mechanisms: increased lipogenesis, decreased transport of lipids from the liver, or decreased deposition of lipid in adipose. Increased lipogenesis occurs with overfeeding a high carbohydrate diet. Such a situation may arise at weaning when some handfeeders force babies to eat more than they are willing to take in. It also occurs when birds being fed free choice choose to overeat. Deficiencies in linoleic and arachidonic acids also lead to increased lipogenesis. Decreased transport of lipids from the liver occurs with deficiencies of choline, inositol, methionine, folic acid, or vitamin B₁₂. These deficiencies result in decreased production of phospholipids, required for transport of lipid from the liver. Physiologic stress may inhibit deposition of lipids from lipoprotein to adipose tissue due to an increase in cAMP, which causes a reduction in lipoprotein lipase.

Hemochromatosis, or Iron Storage Disease, primarily affects toucans and birds of paradise, but has been found in other species of pet birds. A commonly held belief is that this syndrome is caused by excess dietary iron and feeding diets with less than 100 ppm iron will prevent the disease. This theory cannot be supported when prevalence of the disease is investigated. Out of the total number of birds fed diets in excess of 250 to 300 ppm iron, a very small percentage develops iron storage disease. If the diet were the cause, this percentage should be quite high. Other predisposing factors need to be investigated. One study showed increased iron storage in psittacines associated with lead toxicosis. In mammalian species, genetics and physiologic stresses appear to predispose individuals to hemochromatosis.

Clinical Signs

Clinical signs of liver disease may include anorexia and weight loss, depression, PU/PD, regurgitation, yellow urates, and feather, beak, nail and skin abnormalities. Neurological signs, such as incoordination or seizures, may be seen with hepatic encephalopathy.

Diagnosis

Because the clinical signs of liver disease are mostly non-specific, a diagnostic workup should include a CBC and blood chemistry panel, and radiographs. Liver disease may be suspected if four-fold elevations are seen in SOD, LDH, and AP. Elevated cholesterol levels may also be an indication of liver disease. Caution must be exercised in evaluating these chemistries, as studies have shown that significant liver damage did not result in a significant rise

in these values and SGOT and LDH are not liver specific. Significant amounts of SGOT and LDH are found in the heart and skeletal muscle. Hypoproteinemia is also associated with chronic liver disease. BUN is not an accurate indicator of liver disease in birds because they produce very little urea and the liver is not responsible for this process. Studies have shown that elevated bile acid levels appear to be a valuable diagnostic tool for liver disease in birds. Survey radiographs showing an enlarged or reduced liver shadow may also raise suspicion of liver disease.

More definitive diagnosis of liver disease and its cause may require biopsy or laparoscopy. Pansystemic diseases, which affect the liver, should be tested for especially if the CBC indicated viral, chlamydial, bacterial, mycotic or parasitic infection.

Treatment

If possible, specific treatment of the underlying cause of the liver disease must be instituted. In addition, supportive care measures should be employed during the recovery process. Impaired liver function results in poor ability to metabolize nitrogen and glucose, reduced tolerance of volatile fatty acids, and increased need for certain vitamins and minerals due to increased demands and decreased nutrient intake.

Lactulose syrup may be administered to help reduce blood ammonia levels that rise due to the impaired nitrogen metabolism. A specialized diet that addresses the special needs of the bird with liver disease is also important. A low protein diet with balanced amino acid concentrations, balanced to the best of our current knowledge of avian metabolism is needed. The diet should have increased levels of trace minerals and vitamins to compensate for the increased demands and reduced intake and/or digestion. No simple carbohydrates should be used to protect the liver from sudden influxes of glucose after meals. The diet should encourage a slow rate of passage of food through the intestines, encouraging digestion and providing a more constant flow of nutrients from the intestine to the hepatic circulation. The diet should also be gentle to the intestinal lining, resulting in reduced cell turnover and protein loss. Roudybush Formula AL provides these dietary changes. Formula AL is available as a gavage formula, crumble and pelleted for birds that present emaciated or anorexic.

If the bird is already accustomed to a pellet or crumble, switching to Formula AL should be a simple process. Advise the owner to clean the bird's cage at the time of the switch, using paper as the substrate, and observe the size and color of the droppings to determine if the bird is accepting the new diet. If the bird is on a seed diet and is debilitated, it would be advisable to hospitalize the bird during the transition process to closely monitor the bird's well-being and body weight. It will be safer to switch the bird to Formula AL gradually. Begin by offering the bird a low protein diet that it will readily accept. Seed mixtures are generally 12.9% protein, so choose food items that are 8% or less. Some possible choices are: cracked corn, dried fruits, pearl or proso millet, potato, rice, white wheat grain, cracked wheat bread, macadamia nuts, cooked or dry macaroni, immature peas, sweet potato, tapioca, yams, and zwieback. Mix $\frac{1}{4}$ Formula AL with $\frac{3}{4}$ low protein diet. After about 3-4 days, increase the proportion of Formula AL to $\frac{1}{3}$. Continue to increase the proportion of Formula AL every 3-4 days until the diet is 100% Formula AL. Weigh the bird twice weekly. If the bird loses more than 1% of its body

weight between weighings, put it back on a diet that it will maintain, or gain, weight on for one week. It is not advisable to attempt long term maintenance on the low protein food items mentioned above. Extended use will likely result in nutritional imbalances. These are best limited to the transition period. If the owner is able to observe the bird's droppings and well-being daily and weigh it twice weekly, such a transition could be undertaken at home.

Roudybush Formula AL should be fed until the liver function has returned to normal. In the case of chronic liver disease, such as cirrhosis, the bird may need to be maintained on Formula AL for the rest of its life.