Hepatic Lipidosis in a Common Mynah (*Acridotheres tristis*) Associated with Pododermatitis and Consumption of Broiler Pelleted Feed

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Abstract:

An adult common mynah (*Acridotheres tristis*) was presented with severe signs of progressive depression, anorexia, debilitation, and also a history of moderate pododermatitis for the last two months. Hepatomegally was diagnosed clinically and radiologically. The bird died a couple of hours after referral. Postmortem examination revealed typical liver enlargement with yellowish brown discoloration. Diffused hepatocytes fatty degeneration was demonstrated in histopathology and hepatic lipidosis was diagnosed as the main lesion in the liver. Providing a very small enclosure and a single low quality perch with minimal activity opportunity led to pododermatitis. Subsequent further activity restriction and feeding a high energy diet to the bird were possibly the triggers of this metabolic disorder. Regarding the bird species, providing appropriate diet and enclosure are essential for having healthy pet birds, and is often ignored by negligent or uninformed owners.

Case History

An adult mynah was presented with severe signs of progressive depression, and debilitation. The bird had been anorexic for the last three days and had been referred two months earlier because of weight bearing problem on its right leg. Former clinical situation had been diagnosed as moderate pododermatitis with limited involvement of plantar skin and erythema due to the bird's small cage, and a single rough and hard plastic bar as a perch. The moderate lameness was healed by exchanging the perch with tree branches. The owner had been advised to prepare a larger cage for the bird. As the bird had been on a high energy pelleted broiler diet (Broiler Finisher diet, Pars Animal and Poultry Feed Ltd, Iran), the new appropriate recipe consisting of fresh fruits (except berries, cherries, and citrus), cooked egg albumen, meal worm, rice, and bread had been prescribed. The lameness and the plantar erythema were cured completely almost two weeks after the perch substitution. Two months after the initial referral, just two wooden perches were installed in a bigger but still inappropriate sized cage, and the diet had not yet been changed .

Clinical Presentation

In clinical examination the bird was obviously alert, but in a perch-off situation with ruffled feathers. The droppings were black tinged with lime bronze urate deposits. The bird was mildly dehydrated with slightly sunken eyes and dried shrunken skin over the keel. The pale yellow to orange organ resembling an enlarged liver was seen below the abdominal skin approximately 15 mm beyond the caudal margin of the sternum. Duodenal loops and pancreas were seen more caudally than normal. As hepatomegally had

been assumed, diagnostic radiography was recommended.

Diagnostic Testing

Two standard avian radiologic views including ventrodorsal and laterolateral radiographs were taken. Hepatomegally was confirmed radiographically (Figure 1). The soft tissue density was seen in the coelomic cavity, which apparently pushed up gizzard, proventriculus, and spleen dorsally (Figure 2).

Initial therapy was conducted by subcutaneous injection of 2 mL of sterile Dextrose-Saline isotonic solution (Shahid Ghazi Pharmaceutical Co., Tabriz, Iran), and intramuscular injection of 0.05 mL Vitamin B complex (Darou Pakhsh, Tehran, Iran). Force feeding of baby cereal was prescribed and an appointment for further investigation of hepatic problems was scheduled, but the bird died a couple of hours later with a sudden course of seizure-like attack.

At necropsy the overall body condition of the mynah was almost normal. No obvious subcutaneous adipose tissue was seen, but copious amount of coronary and abdominal fat were conspicuous. The liver lobes were apparently enlarged, and swollen with pale yellow to brown color. A hyperemic red band was seen on the liver surface, probably the result of swollen liver and the consequent pressure of sternum ridge on it (Figure 3). The gizzard was almost empty with some very dark mucoid fluid and the koilin was tinged green, probably due to bile reflux. The small intestine was filled with orange to dark brown, even black mucoid content resembling hemorrhagic diathesis caused by long term anorexia and shock. The kidneys were mildly pale and swollen. No other macroscopic lesion was revealed at necropsy. Pieces of liver, kidney, heart, duodenum, spleen, lung, proventricle and gizzard, and thigh muscle were collected in 10% buffered formalin for histological investigation. The organs were processed by common histotechnique procedure and stained with hematoxylin and eosin (H&E) method.

Mild to moderate tubular necrosis in the kidney mirrored the bird dehydration. Intestinal villi tips were broken and some necroses and/or autolyses were seen in the intestinal epithelial cells. The intestinal lumen was filled with amorphous cell debris. The most prominent histopathologic signs were in the liver where massive diffuse vacuolization and necrosis of hepatocytes resembling severe fatty change was obvious (Figure 4). Few, if any, gold/brown pigments were seen in the liver and spleen, so hepatomegally due to iron storage disease was ruled out histologically. The definitive diagnosis of hepatic lipidosis was made based on macroscopic and microscopic findings.

Assessment

Common mynah (*Acridotheres tristis*) is a popular pet bird in Iran. Most captive mynahs are wild-caught. In the past, natural geographic distribution of this species was exclusively limited to the southern and south-eastern parts of Iran, but presently their wild populations are seen in most parts of big central cities like Tehran, probably resulting from their escape from captivity and repopulation in urban and suburban areas (Mansoori, 2008). Despite their popularity in Iran, our knowledge about their captive biology, management, and diseases is really quite sparse. Recording and reporting the diseases of common mynahs can improve their practical medicine and help veterinarians to become familiar with their health issues. In this article, we presented a case of metabolic disorder in common mynah due to mismanagement and malnutrition.

We presented a case of hepatic lipidosis in a mynah bird which had suffered from mismanagement and malnutrition. The bird had been on a high energy diet, but had also been kept in a very small enclosure with restricted activity. Hypothetically, the mentioned conditions led to imbalance of energy and fat metabolism which were likely the trigger for fat storage and steatosis in the liver. Finally the bird died because of the severe hepatic insufficiency and its clinicopathological consequences including dehydration, hypoglycemia, encephalopathy, etc.

Hepatic lipidosis or fatty liver is a condition found in a wide variety of bird species and may occur in both hand-fed neonates and adult birds on high-fat and energy concentrated feed (Hochleithner et al., 2005). The disease is most commonly reported from many psittacine birds, particularly Amazons (Schmidt et al., 2003; Hochleithner et al., 2005). This is also

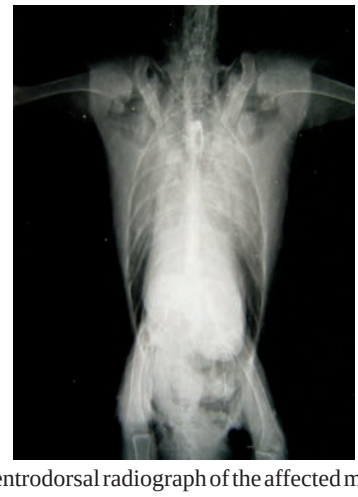

Figure 1. Ventrodorsal radiograph of the affected mynah. Broad hepatic silhouette.

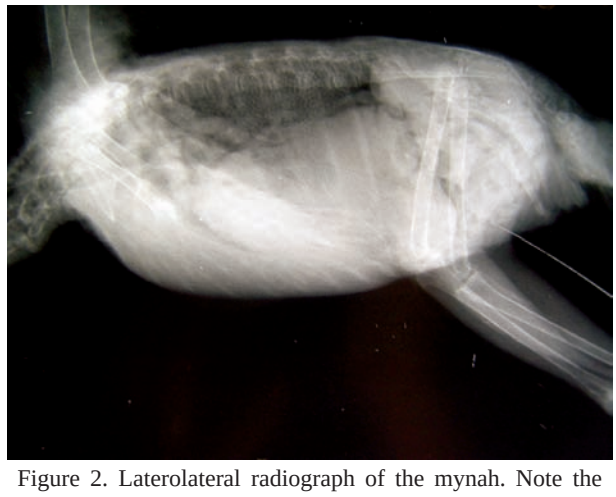

Figure 2. Laterolateral radiograph of the mynah. Note the caudodorsal displacement of intestines.

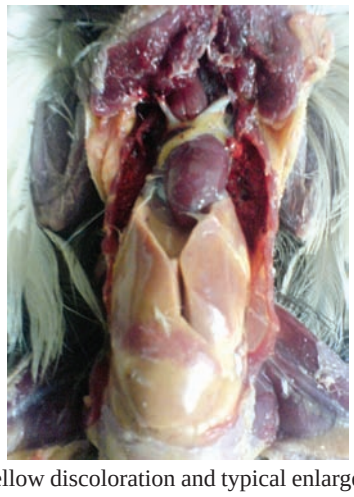

Figure 3. Yellow discoloration and typical enlargement of the liver. Note the fat deposition in coronary groove indicating the predisposing obesity of the bird before death.

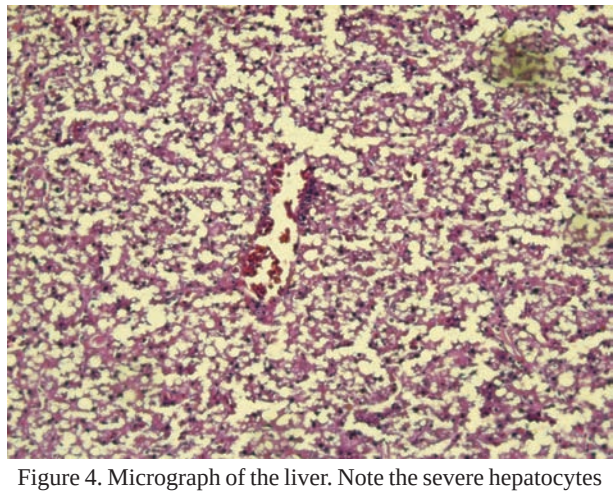

Figure 4. Micrograph of the liver. Note the severe hepatocytes vacuolization and degeneration, and deterioration of normal histologic architecture of the liver. H&E staining. 40 x.

common in young cockatoos suspected to be caused by overfeeding hand feeding formula, or feeding a high fat formula (Schmidt et al., 2003). There is a report of ventral abdominal hernia associated with hepatic lipidosis in a red lory (*Eos bornea*) (Langlois and Jones, 2001). Hepatic lipidosis has been reported for many years in hummingbirds (Davis, 2000). However, FLKS in young chickens appears to be prevented by adequate dietary biotin. It is postulated that day-old chicks may contain high levels of avidin in their yolk sac remnants, and this may reduce the available biotin, leading to reduced hepatic gluconeogenesis (Davis, 2000). Fatty livers are occasionally seen in Estrilid finches (zebra finches, parrot finches and star finches) and may be associated with inadequate exercise and high-energy diets such as soft foods and mealworms (Macwhirter, 1994). There has been a single report of hepatic lipidosis concurrent with iron storage disease in a Hill mynah (*Gracula* spp.) (Young et al., 2008).

Hepatic lipidosis in birds is most commonly due to the feeding of a high-fat-low-protein diet, where the fat becomes the major source of calories. In a normal bird, albumin carries circulating fatty acids to the liver where they are oxidized for energy or incorporated into triglycerides. If the amount of circulating fatty acids exceeds the liver's metabolic capacity, the excess lipid will be stored in the liver, resulting in lipidosis. Therefore Hepatic lipidosis can occur either due to the consumption of excessive fatty acids (in the form of dietary fat, especially seed); increased lipolysis (e.g., diabetes mellitus or egg-laying activity), decreased fatty acid oxidation in the liver; or decreased ability of the liver to secrete

processed fatty acids back into the circulation. Dietary deficiencies of lipotrophic factors such as choline, biotin, and methionine may decrease the transportation of lipids from the liver (Doneley, 2004). Hepatic lipidosis is also seen as a consequence of increased fat mobilization. In female birds, estrogen-controlled lipogenesis during egg laying period increases the fat content of the liver (Davis, 2000).

Widening of the hepatic silhouette on the ventrodorsal view may be suggestive of hepatomegaly which is a common finding in avian radiologic investigations (Krawtwald-Junghanns et al., 2001). The liver should not extend past a line drawn from the coracoid to the acetabulum. In many cases a gastrointestinal contrast examination is needed to identify the cause of the abnormal hepatic silhouette. The lateral view can help identify which organ is enlarged. The liver does not usually extend beyond the sternum. Hepatomegaly can cause dorsal displacement of the ventriculus (Krautwald-Junghanns et al., 2001).

At necropsy the liver is swollen, pale-yellow and may float in formalin (Macwhirter, 1994; Schmidt et al., 2003). Hepatic lipidosis produces cytologic specimens that appear "greasy" on gross examination. The stained smears reveal enlarged hepatocytes that contain round, cytoplasmic vacuoles. The background material also contains these round vacuoles suggestive of lipid material (Campbell, 1994).

There are no pathogonomic clinical signs of avian liver diseases and the affected bird may be presented with non-specific clinical signs of sudden and severe anorexia, depression, lethargy, obesity, vomiting, regurgitation, dyspnea, and ataxia (Grunkemeyer, 2010). Commonly the birds are obese and most have been on all-seed diets for the major part of their lives. Birds with severe liver disease may demonstrate signs of hepatic encephalopathy. Both hepatic lipidosis and hemochromatosis have been reported to cause clinical signs of depression, ataxia, diminished conscious proprioception and seizures (Bennett, 1994).

Up to 80% of hepatic function must be compromised before the disease will become clinically apparent (Jaensch, 2000). Therefore birds presented with clinical signs associated with liver disease are often in a critical state (Redrobe, 2000). Initially, proper supportive careis essential for a successful outcome (Redrobe, 2000). Treatment requires a reduction of dietary fat and an increase in dietary protein. Anorectic patients may need force feeding with such a diet until their catabolic state is reversed. Vitamin E (as an antioxidant) and B complex vitamins may also be beneficial (Doneley, 2004). Water soluble vitamin supplementation may help the affected bird acquire its normal metabolism (Doneley, 2004).

Malpractices and mismanagement are also usual causes of presentation and referral of pet and aviary birds in veterinary clinics. In the presented case, small enclosure and low quality perch caused the bird to suffer from a typical pododermatitis and lameness which was cured by replacement of the perch and providing an extra perch to encourage the activity of the bird. Pododermatitis or bumble foot can limit the bird activity and, as a result its metabolic energy requirement is reduced. Metabolic and nutritional disorders can be the most important clinical problems of the isolated cage birds, when separating them from other birds limits the probability of the occurrence of infectious diseases. Our knowledge about the practical nutrition of pet birds is in its infant stage of development and most diets which are provided for pet birds like common mynah have been extrapolated from the requirements of commercial poultry, particularly chicken. Feeding commercial broiler diets as pellets to common mynah is a common practice in Iran. As long as declared by their manufacturers, these kinds of feeds are high in energy and fat and almost exclusively formulated for the fast growing broiler chickens, while mature fully grown mynahs do not require such high energy for growth and the restricted activity in a cage environment further reduces their energy requirement. The avian clinicians should always consider their patients' special needs and further investigation of pet birds' health issues can improve our knowledge about their biology, management and veterinary problems.

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لیپیدوز کبدی در یک قطعه مرغ مینا (۱کریدوترس تریستیس) مرتبط با پودو درماتیت و مصرف دان پلت جوجه گوشتی

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چکیده

سابقه: یک قطعه مینای معمولی (۱کریدوترس تریستیس) با علائم شدید دپرسیون پیش رونده، بی اشتهایی و ناتوانی، و با تاریخچه دو ماهه درگیری با پودودرماتیت متوسط ارجاع داده شد. **یافته های بالینی:** بزرگ شدگی کبد به صورت بالینی و با کمک رادیولوژی تشخیص داده شد. ساعاتی پس از مراجعه، پرنده تلف شد. **آزمایشات تشخیصی:** بزرگ شدگی کبد همراه با تغییر رنگ زرد تا قهوه ای آن طی معاینات پس از مرگ مشاهده گردید. پس از مطالعات بافت شناسی، لیپیدوز کبدی به عنوان مهمترین ضایعه تشخیص داده شد. **ارزیابی نهایی:** احتمالاً سوء مدیریت در نگهداری این پرنده شامل تغذیه با جیره دارای انرژی زیاد، همراه با نگهداری پرنده در قفس بسیار کوچک و فراهم نمودن تنها یک چوب زیر پا با کیفیت پایین، در نتیجه محدود نمودن تحرک پرنده و وقوع پودودرماتیت ناشی از این وضعیت، موجب مستعد شدن به این اختلال متابولیک شده است. با توجه به گونه پرنده، فراهم نمودن جیره و محل نگهداری مناسب برای داشتن پرنده خانگی سالم ضروری به نظر می رسد که این مسأله گاهی از سوی صاحبان بی اطلاع یا بی دقت این پرندگان، نادیده گرفته می شود.

واژه های کلیدی: لیپیدوز کبدی، مینای معمولی، ۱کریدوترس تریستیس، پودودرماتیت.

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