Case Report

Abdominal Hernia in a Common Mynah (*Acridotheres tristis*) Associated With Hepatic Lipidosis and Concurrent Respiratory Aspergillosis

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ABSTRACT

Hepatic lipidosis and hernia are problems that can occur in pet birds due to different causes. Aspergillosis is also an important disease of pet birds. This article reports an interesting case which, to our knowledge, is the first one in Iran. A 7-year-old male common mynah was presented to the clinic of avian diseases at the University of Tehran, Tehran City, Iran, with a history of severe dyspnea, anorexia, feeding with a high-energy diet, and recently been in the northern parts of Iran. Unfortunately, the bird died. Necropsy was done to diagnose the cause of death. Tissue samples from abdominal viscera, lungs, and air sacs were collected and fixed in 10% buffered formalin and stained with hematoxylin and eosin. Bacterial cultures from the liver and bone marrow were performed on blood and MacConkey agars. In the external carcass examination, the abdominal swelling, hernia, and yellow discoloration under the skin were obvious. At necropsy, the liver was enlarged with yellowish discoloration. There was also a high amount of fat around the abdominal viscera. Microscopically, fatty change, hepatocellular necrosis, and hemorrhages were seen in the liver. There were also vegetative forms of Aspergillus on the lung's parenchyma and air sacs. No bacterial growth was observed. Pet birds are usually restricted to a cage by minimal contact with other birds; thus, diseases related to management are common among these birds. So keeping in suitable conditions with an appropriate, balanced diet is important for their health.

Keywords: Abdominal hernia, Aspergillosis, Common mynah, Hepatic lipidosis, Necropsy

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1. Introduction

A 7-year-old male common mynah was presented to the clinic of avian diseases at the University of Tehran, Tehran City, Iran, with severe signs of dyspnea. The owner had not noticed the onset of dyspnea, and his main complaint was an enlargement in the abdominal area. Based on the owner's information, the bird was fed a high-energy diet (Broiler Finisher diet, Pars Animal and Poultry Feed Ltd, Iran). There was also a history of travel to the northern parts of Iran 6 days before the presentation. The bird was off-feed for 2 days, and its droppings were watery green. There was also no history of drug use by the owner.

Clinical presentation

The bird was perch-off and had severe dyspnea with open-mouth breathing and tail bobbing. The wings were dropped, and an obvious hernia and enlargement were seen in the abdominal area (Figure 1a). The yellow discoloration was present beneath the skin of the area, which was an obstacle to the observation of abdominal viscera. Radiography and ultrasonography were proposed to the owner as an aid for diagnosis. Due to the emergency condition of the bird, we first decided to nebulize it with aminophylline, but before any intervention, the bird died after seizure-like movement. After death, the bird was weighed, and its weight was 126g.

The cage of the bird was also unsuitable. It was small, and the feeder had much feed. The cage floor was dirty and unsanitary, and the perch was made of plastic.

Diagnostic testing

Necropsy was done to diagnose the cause of death. The liver was enlarged with round borders and yellow discoloration (Figure 1b). There were copious amounts of fat around the intestine, proventriculus, gizzard, and some parts of the lungs and kidneys (Figure 1c).

Tissue samples of the liver, duodenum, proventriculus, gizzard, lung, air sacs, and kidneys were taken for histopathology. The samples were fixed in 10% neutral buffered formalin and routinely processed, dehydrated, embedded in paraffin wax, sectioned at 5 µm in thickness, and stained with hematoxylin and eosin.

Microscopically, fatty change, hepatocellular necrosis, and hemorrhages were seen in the liver. The fatty change was characterized by numerous small to large intracytoplasmic vacuoles distorting the hepatic cords. Fatty changes were also observed in the kidneys. A massive development of the vegetative forms of the fungus, containing a large number of conidiophores, hyphae, and spores, was observed in the lung's parenchyma and air sacs in H&E staining (Figure 2a & 2b). Cultures from the liver and bone marrow were also performed on blood and MacConkey agar, but there was no bacterial growth.

Assessment

Hepatic lipidosis is a metabolic disorder occurring in various avian species (Schmidt et al., 2015). It is not a specific disease entity but can occur due to a disturbance in normal lipid metabolism (Hochleithner et al., 2006). In this condition, lipids accumulate in the liver. Acidosis is common in adult and hand-fed neonates (Hochleithner et al., 2006). There are different etiologies for hepatic lipidosis in adult birds (Beaufrere and Taylor, 2013; Vali et al., 2020; Hochleithner et al., 2006). A low-protein, high-fat diet, thyroid dysfunction, and restricted exercise are some of them (Beaufrere and Taylor, 2013). Increased lipogenesis, which can be a sequel of estrogen activity, diabetes mellitus, stress, and estrogen-like activity of pesticides, are other causes (Beaufrere and Taylor, 2013; Hochleithner et al., 2006). Another probable cause can be nutritional deficiencies, such as essential fatty acids, sulfur amino acids, and vitamins like biotin, B1, B6, and B12 (Beaufrere and Taylor, 2013; Hochleithner et al., 2006). Acute release of fatty acids from adipose tissues may have a role in this disorder (Beaufrere and Taylor, 2013; Vali et al., 2020). Some references have named mycotoxins and drugs (which impair lipoprotein secretion) as probable causes (Hochleithner et al., 2006).

In chicks, the most common cause is overfeeding with high-energy formula (Beaufrere and Taylor, 2013). It was claimed that in neonates, high levels of avidin in yolk sac remnants might reduce available biotin, reducing hepatic gluconeogenesis (Davies, 2000). Based on published information, some parrots (like Amazon parrots, galah cockatoos, budgerigar, and lorikeets) are more susceptible to hepatic lipidosis (Beaufrere and Taylor, 2013).

History and clinical signs, clinical pathology, diagnostic imaging, and hepatic biopsy are some methods for diagnosis in live birds. Post-mortem gross lesions in the liver and sampling for histopathology can make a definitive diagnosis (Grunkemeyer, 2010).

In general, treatment includes cool oxygenation in cases of dyspnea, non-lactated fluid therapy to rehydrate the bird and detoxify the body, treatment of secondary infections, nutritional balance, using metabolic aids like silymarin, and preparing conditions for body exercise (Hochleithner et al., 2006).

The etiology of abdominal hernias in birds is unknown, but generally, they can be congenital or acquired (Bennett and Harrison, 1994; Anderson et al., 2018). Some studies consider them a separation in the aponeurosis of abdominal musculature (Bennett and Harrison, 1994; Macwhirter, 1994). Other studies have proposed that they are due to the absence of an opening in muscular aponeurosis, so they are not true hernias (Anderson et al., 2018).

Some predisposing factors have been described to the hernia, such as hyperestrogenism, lack of exercise, malnutrition, trauma, and egg-laying behavior (Bennett and Harrison, 1994; Macwhirter, 1994; Amer et al., 2018). Besides, some hernias were associated with hepatic lipidosis, reproductive tract problems (like egg yolk peritonitis), peritoneal cyst (Bennett and Harrison, 1994; Macwhirter, 1994), and intraabdominal lipoma (Razmyar et al., 2005). Nevertheless, if the hernia grows large, it can cause skin ulcers and hemorrhage (Bennett and Harrison, 1994; Macwhirter, 1994; Amer et al., 2018).

Treatment may not be the same in different cases. In some cases, especially when trauma is present, surgical repair is recommended (Murphy et al., 2018). In some cases, like hepatic lipidosis, treatment can reduce organ size, which may eliminate hernia without surgery (Macwhirter, 1994; Barboza et al., 2018).

Aspergillosis is a fungal, noncontagious disease of wild and caged birds (Nardoni et al., 2006; Carrasco and Forbes, 2016; Melo et al., 2020; Leishangthem et al., 2015). Infection is usually caused by inhalation of conidia or spores of pathogenic species of the genus Aspergillus, especially A. fumigatus. (Nardoni et al., 2006; Carrasco and Forbes, 2016). They are ubiquitous, and all birds are susceptible to infection (Nardoni et al., 2006). The source of infection is usually contaminated feed, litter, and soil (Carrasco and Forbes, 2016). The disease is primarily restricted to the respiratory system but can be systemic and involve other organs (Nardoni et al., 2006; Carrasco and Forbes, 2016).

There are some characteristics in avian species that predispose them to the disease. For example, the unique anatomy of the respiratory tract and higher body temperature accelerates fungal growth. Other factors include vitamin deficiency, especially vitamin A, chronic stress, overcrowding, unsanitary condition, malnutrition, treatment with corticosteroids, and respiratory irritants (Nardoni et al., 2006; Leishangthem et al., 2015; Krautwald-junghanns et al., 2015).

The disease can be acute or chronic. The acute form is common in chicks and results from inhaling many spores. It can develop in less than a week. Clinical signs of this form are not specific and can be confusing. Dyspnea, cyanosis, anorexia, polydipsia, and fetid diarrhea are some of the probable clinical signs (Leishangthem et al., 2015; Fischer and Lierz, 2015).

The chronic form is more common in older birds and takes weeks or months to develop. Clinical signs may vary depending on the site of infection. Different references have reported different clinical signs in the respiratory system, skin, eyes, GI tract, nervous system, bones, joints, and abdominal viscera (Leishangthem et al., 2015; Fischer and Lierz, 2015).

Diagnosis of the disease in live birds is very difficult, especially in acute cases (Leishangthem et al., 2015). Some diagnostic tests like biochemistry, hematology, endoscopy, and imaging can be helpful (Fischer and Lierz, 2015; Savelieff et al., 2018). Definitive diagnosis is based on histopathology from post-mortem samples or biopsy (Elad and Segal, 2018; Leishangthem et al., 2015). Fungal culture from suspected organs can also be helpful (Savelieff et al., 2018). Other diagnostic tools like ELISA and agar gel immunodiffusion are not common and should never be used alone (Fischer and Lierz, 2015). Treatment with antifungal drugs is complex and depends on the site and extent of infection (Leishangthem et al., 2015).

In Iran, keeping birds as pets (mainly from psittacines and passerines) has increased recently. All domestic birds do not have the same nutritional and management requirements. Unfortunately, most of the owners are not familiar with these needs. Thus, malnutrition and mismanagement are characteristic findings in most cases presented to avian clinics.

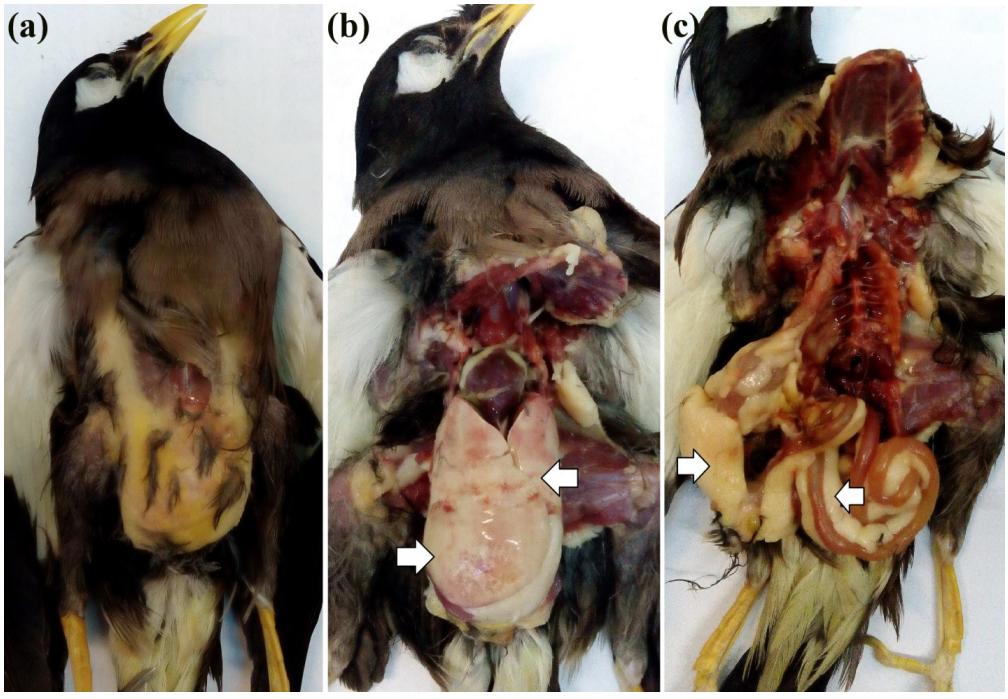

Figure 1. Carcass external examination and necropsy findings

a) Abdominal enlargement and hernia; b) Enlarged yellowish liver with rounded borders; c) Copious amount of fat around abdominal viscera.

In this case, according to the history and clinical signs, it seems that hepatic lipidosis is a result of feeding with a high-energy broiler diet and less activity due to the small size of the cage. Feeding mynah with broiler pellets is usual in Iran because it is similar to mynah's pelleted feed. There is another report of hepatic lipidosis associated with high-energy feed in Iran (Madani et al., 2012).

As described earlier, hepatic lipidosis can make birds prone to hernias. There is a report in a red lory that supports their association (Langlois and Jones, 2001). In this case, the most probable cause of hernia is hepatic lipidosis. This condition causes liver enlargement, which puts the respiratory tract under pressure. This pressure can lead to lower respiratory capacity.

A damp climate, common in northern parts of Iran in summer, encourages fungal growth. The acute form of aspergillosis in birds results from inhaling a high number of spores (Leishangthem et al., 2015). Because the bird had been to the northern part of Iran, its exposure to high numbers of fungal spores may lead to acute aspergillosis. This disease has exacerbated the effect of hepatic enlargement on the respiratory tract. This finding can elucidate the cause of death and premortem clinical signs.

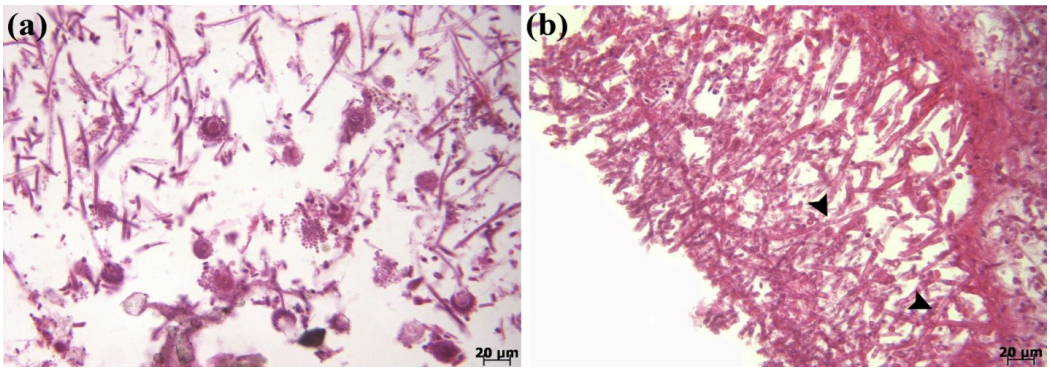

Figure 2. Fungal conidiophores and hyphae

a) Fungal conidiophores in air sac section stained with H & E (40x magnification); b) Fungal hyphae in air sac section stained with H& E (40x magnification)

There is no published report of simultaneous hepatic lipidosis, hernia, and aspergillosis in any domestic bird in Iran. Because avian medicine is a relatively new field in Iran and the world, reporting these cases could help develop this field (Pollock et al., 2016). It can also help avian veterinarians and owners to promote their knowledge of captive animal management and potential diseases of domestic birds, which are rare in wild bird populations.

Ethical Considerations

Compliance with ethical guidelines

All procedures were conducted according to the animal care guideline of the Research Committee of the Faculty of Veterinary Medicine, University of Tehran.

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Authors' contributions

Methodology: Peighambari, Shokrpoor, and Razmyar; Writing–original draft and Writing–review & editing: Khodayari and Asghari Baghkheirati; Shokrpoo, Peighambari, and Razmyar.

Conflict of interest

The authors declared no conflict of interest.

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گزارش موردی

فتق شکمی در یک مینای معمولی مرتبط با لیپیدوز کبدی و همزمانی با آسپرژیلوز تنفسی

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چکیده

لیپیدوز کبدی و فتق مشکلاتی در پرندگان خانگی هستند که می‌توانند به دلایل مختلفی ایجاد شوند. همچنین، آسپرژیلوز یک بیماری مهم در پرندگان خانگی است. هدف این مقاله گزارش یک کیس جالب است که با توجه به اطلاعات ما اولین مورد در ایران است. مینای معمولی ۷ ساله نر با تاریخچه‌ای از سختی تنفس شدید، بی‌اشتهایی، تغذیه با جیره پرانرژی و سفر به مناطق شمالی ایران در تاریخچه‌ی خود، به کلینیک بیماری‌های پرندگان، دانشگاه تهران ارجاع شد. متاسفانه، پرنده تلف شد. کالبدگشایی به منظور تشخیص علت مرگ انجام گرفت. نمونه‌های بافتی از احشای محوطه بطنی، ریه‌ها و کیسه‌های هوایی جمع آوری شده و در بافر فرمالین ۱۰٪ فیکس شده و به روش هماتوکسیلین و ائوزین رنگ‌آمیزی شدند. کشت‌های باکتریایی از کبد و مغز استخوان در Blood agar و MacConkey agar انجام گرفتند. در معاینه بیرونی لاشه، تورم ناحیه شکم، فتق و تغییر رنگ زرد در زیر پوست مشهود بود. در کالبدگشایی، کبد متورم و همراه با تغییر رنگ مایل به زرد بود. همچنین مقادیر زیادی چربی در اطراف احشای شکمی وجود داشت. با استفاده از میکروسکوپ، در کبد، تغییر چربی، نکروز سلول‌های کبدی و خونریزی مشاهده شدند. همچنین اشکال رویشی آسپرژیلوس در پارانشیم ریه و کیسه‌های هوایی وجود داشتند. هیچ رشد باکتریایی مشاهده نشد. معمولاً پرندگان خانگی در قفس محصور بوده و تماس کمی با سایر پرنده‌ها دارند، بنابراین بیماری‌های مرتبط با مدیریت در بین این پرنده‌ها رایج است، بنابراین نگهداری در شرایط مناسب به همراه جیره متعادل و مناسب برای سلامت آنها مهم است.

کلیدواژه‌ها: فتق شکمی، آسپرژیلوز، مینای معمولی، لیپیدوز کبدی، کالبدگشایی

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