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

Running title: Hepatic lipidosis, hernia & aspergillosis in a mynah

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Abstract

Background: Hepatic lipidosis and hernia, are problems in pet birds that can occur due to different causes. Aspergillosis, is also an important disease of pet birds.

Objectives: This article aims to report an interesting case which by our knowledge is the first one in Iran.

Methods: A 7-year-old male common mynah was presented to the clinic of avian diseases, the University of Tehran with a history of severe dyspnea, anorexia, feeding with a high energy diet, and travel to the northern parts of Iran in its history. 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 method. Bacterial cultures from the liver and bone marrow were performed on Blood and MacConkey agars.

Results: In carcass external examination, 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.

Conclusions: 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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Case history

A 7-year-old male common mynah was presented to the clinic of avian diseases, the University of Tehran with severe signs of dyspnea. The owner had not noticed the onset of dyspnea and his main complaint was enlargement in the abdominal area. Based on the owner's information, the bird was fed with a diet of high energy (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 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 there were obvious hernia and enlargement in the abdominal area (Figure 1.a).

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 with Aminophylline, but before any intervention, the bird died after seizure-like movement. After death, the bird was weighed and its weight was 126 grams.

The cage of the bird was also unsuitable. It was small and the feeder had a lot of 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 (Figure1.b). There were copious amounts of fat around the intestine, proventriculus, gizzard, some parts of the lungs and kidneys. (Figure1.c)

Tissue samples of 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, and embedded in paraffin wax, sectioned at 5 µm in thickness, and stained with hematoxylin and eosin method.

Microscopically, fatty change, hepatocellular necrosis, and hemorrhages were seen in the liver. The fatty change was characterized by numerous, fine 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 were observed in the lung's parenchyma and air sacs in H&E staining (Figure 2.a & b).

Cultures from the liver and bone marrow were also performed on Blood and MacConkey agar, but there was no bacterial growth.

Assessment

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Hepatic lipidosis is a metabolic disorder occurring in a wide variety of avian species (Schmidt et al., 2015). It is not a specific disease entity but can occur due to disturbance in normal lipid metabolism (Hochleithner et al., 2006). In this condition, lipids accumulate in the liver. It is common in both 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). High fat-low protein 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 factors (Beaufrere and Taylor, 2013; Hochleithner et al., 2006). Nutritional deficiencies, as essential fatty acids, sulfur amino acids and vitamins like biotin, B1, B6, and B12 can also be a probable cause (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 secretion of lipoprotein) as a probable cause (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 may reduce available biotin, which can lead to reduced hepatic gluconeogenesis (Davies, 2000). Based on published information, some species of 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 of the methods for diagnosis in live birds. Post mortem gross lesions in the liver and sampling for histopathology can lead to 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).

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The etiology of abdominal hernias in birds is unknown, but in general, they can be congenital and acquired (Bennett and Harrison, 1994; Anderson et al., 2018). Some studies consider them as a separation in the aponeurosis of abdominal musculature (Bennett and Harrison, 1994; Macwhirter, 1994). Other studies have proposed that due to the absence of opening in muscular aponeurosis, they are not a true hernia (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). Anyway, if the hernia becomes larger, it can be a cause of 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 lead to the elimination of hernia without surgery (Macwhirter, 1994; Barboza et al., 2018).

Aspergillosis is a fungal, noncontagious disease of both wild and caged birds (Nardoni et al., 2006; Carrasco and Forbes, 2016; Melo et al., 2020; Leishangthem et al). Infection is usually by inhalation of conidia or spores of pathogenic species of Gnus *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 unique anatomy of the respiratory tract and higher body temperature accelerates fungal growth. Other

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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; Krautwaldjunghanns et al., 2015).

The disease can be acute or chronic. The acute form is more common in chicks and results from inhaling a high number of 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 the 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 never should be used alone (Fischer and Lierz, 2015). Treatment by antifungal drugs is complex and depends on the site and extent of infection (Leishangthem et al., 2015).

In Iran, keeping birds as a pet (mainly from psittacines and passerines) is increasing in recent years. All domestic birds have not the same nutritional and management requirements. Unfortunately, most of the owners are not familiar with these needs. Thus, malnutrition and mismanagement are usual findings in the most cases presented to avian clinics.

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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 pellet is very 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 enlargement of the liver which puts the respiratory tract under pressure. This pressure can lead to lower respiratory capacity.

Damp climate, which is common in northern parts of Iran in summer, encourages fungal growth. The acute form of aspergillosis in birds is a result of inhaling a high amount of spores (Leishangthem et al., 2015). Because the bird had traveled to the north part of Iran, it is possible that had exposure to high numbers of fungal spores which may lead to acute aspergillosis. This disease has exacerbated the effect of hepatic enlargement on the respiratory tract. This can elucidate the cause of death and premortem clinical signs.

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, so reporting these kind of cases could be helpful for developing this field (Pollock et al., 2016). It can also help avian veterinarians and owners to promote their knowledege in captive animals management and potential diseases of domestic birds which are rare in the wild bird populations.

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