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CASE REPORT
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Hepatic Cirrhosis in *Nymphicus hollandicus* (Cockatiel)

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ABSTRACT

***Background*:** The order *Psittaciformes* comprises over 350 bird species, including cockatiels (*Nymphicus hollandicus*), whose beauty and docility make them popular pets. Despite the availability of commercial feeds designed to meet their nutritional needs, many owners provide high-energy seed-based diets, which unfortunately cause metabolic disorders such as hepatic steatosis and cirrhosis. These conditions result from chronic hepatocyte injury and fibrosis, but documented cases of these problems in avian species are limited. This paper reports a case of hepatic cirrhosis in *Nymphicus hollandicus*.
***Case*:** An 11-year-old female cockatiel was subjected to necropsy after being found dead. The owner reported a month-long history of lethargy, progressing to yellowish feces, regurgitation, and dyspnea. The diet consisted primarily of commercial seed mix, rice, and occasional bread and biscuits. Water was chlorinated and unfiltered, and vitamin supplementation was sporadic. The bird lived with three other cockatiels in a cage exposed to natural light during the day and housed indoors at night. No previous health issues were noted. Gross examination revealed no external abnormalities apart from chronic hyperkeratosis on one digit. Internally, ascites was evident, with a firm, multinodular, pale yellow liver exhibiting structural loss. The lungs showed diffuse congestion and edema, but other organs showed no significant changes. Histopathology confirmed hepatic cirrhosis, with fibrous connective tissue surrounding hyperplastic hepatocyte nodules, bile duct proliferation, lymphoplasmacytic infiltration, and hepatocellular fatty degeneration. The lungs showed severe vascular congestion, while the heart exhibited no relevant alterations.
***Discussion*:** Cirrhosis, which is rarely reported in birds, typically arises from chronic inflammation and poor dietary management. Unlike mammals, avian species lack an extensive lymphatic system, making their livers more susceptible to lipid accumulation from high-fat diets. Seed-based diets, deficient in proteins, vitamins, and minerals, exacerbate triglyceride buildup in hepatocytes, leading to hepatic steatosis and eventually, cirrhosis. Similar cases in *Amazona aestiva* and *Psittacus erithacus* reinforce the association between poor diet and liver pathology. The bird's dyspnea was likely due to ascites-related respiratory distress, a common sequela of hepatic failure in birds, attributed to portal hypertension and decreased colloid osmotic pressure from hypoproteinemia. The presence of biliverdin-stained feces further supported hepatobiliary dysfunction. Diagnosis of hepatic diseases in birds is challenging because enzyme alterations are often nonspecific. While radiography, endoscopy, and biopsy aid in diagnosis, they are mostly available in specialized centers. Management of avian hepatic cirrhosis focuses on diet correction and hepatoprotective therapies, including plant extracts and probiotics, which show promise in modulating the progression of liver disease. Despite advances in avian nutrition, many pet cockatiels continue to receive imbalanced diets due to owner misinformation and the affordability of seeds. Nutritional mismanagement remains a primary factor in psittacine metabolic disorders, necessitating increased awareness and dietary adjustments to prevent conditions like steatosis and cirrhosis. This case underscores the consequences of inadequate nutrition, ultimately leading to fatal hepatic cirrhosis in *Nymphicus hollandicus*.

Keywords: necrosis, fibrosis, steatosis, ascites, diet.

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INTRODUCTION

The order Psittaciformes includes over 350 bird species, such as parrots, macaws, cockatoos, and cockatiels [13]. Native to Oceania, cockatiels (*Nymphicus hollandicus*) are known for their curved beaks and thick tongues, primarily consuming insects, fruits, and seeds [1]. The 1st importation of this species into Brazil dates to 1970, and since 1998, it has been classified as domestic fauna [19].

Due to the popularity of cockatiels as pets for their beauty and docile nature [26], commercial feeds have been developed to meet their nutritional needs. However, owners frequently offer sunflower seeds, millet, canaryseed, rice, corn, and wheat as primary foods, which, due to their high energy content, can lead to hepatic and metabolic diseases and obesity over time [29].

Avian hepatic steatosis is linked to high-lipid diets [16] combined with low protein intake, contributing to dyslipidemia. This condition is marked by cellular injury, degeneration, inflammation, and a tendency toward fibrosis and cirrhosis [40].

Hepatic cirrhosis in birds, usually a multifactorial and chronic inflammatory condition, results from hepatocyte death and fibrotic tissue repair [38]. In view of the paucity of literature on hepatic cirrhosis in avian species, this paper aims to report a case in *Nymphicus hollandicus*.

CASE

An 11-year-old female cockatiel (*Nymphicus hollandicus*), found deceased by its owner, was taken to the Avian Medicine Laboratory (LMA) of the State University of Londrina (UEL) for necropsy. The owner reported lethargy a month prior, which progressed to digestive symptoms, including yellowish feces, regurgitation, and dyspnea on the day before death. The diet consisted primarily of a commercial seed mix and rice, and the bird showed little interest in fruits and leafy greens, so bread and biscuits were provided as treats. The bird drank chlorinated, unfiltered water and was occasionally supplemented with multivitamins.

The cockatiel shared a cage with 3 other birds of the same species. The cage was placed outdoors in the morning for natural light exposure and brought indoors at night. No prior health issues were noted.

At necropsy, external examination revealed no abnormalities except for a focal, chronic hyperkeratosis on the 3rd digit of the left pelvic limb (Figure 1). The internal examination revealed the presence of a translucent, yellowish, gelatinous fluid in the coelomic cavity, indicating ascites (Figure 2). The liver was multinodular, firm, diffusely pale and yellow, with loss of lobar structure (Figure 3). The lungs showed diffuse congestion and edema (Figure 4), but no abnormalities were noted in other organs.

Samples of liver, heart, and lungs were collected and fixed in a 10% formalin solution. The samples were subjected to histological procedures, embedded in paraffin, sectioned, and stained with hematoxylin and eosin[1] (Certistain®) and Masson's Trichrome[1].

Histological examination of the liver revealed continuous bundles of fibrous connective tissue surrounding hyperplastic hepatocyte nodules (Figure 5), along with lymphocyte, plasma cell, and occasional

Figure 1. Gross findings in *Nymphicus hollandicus*. Focal hyper-keratosis on the third digit of the left pelvic limb (asterisk).

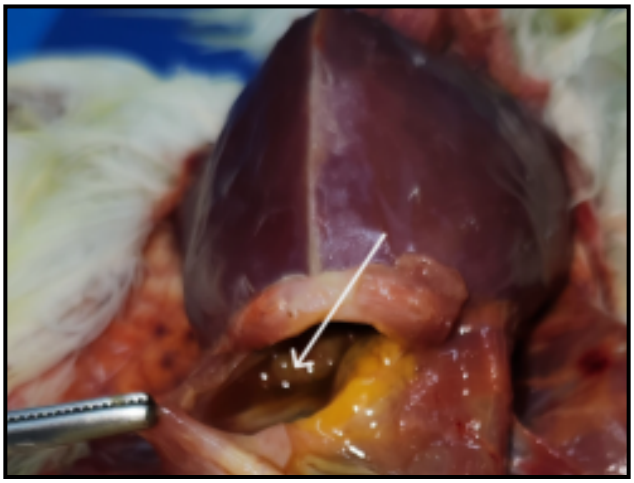

Figure 2. Gross finding in *Nymphicus hollandicus*. Translucent, yellow intracavitary fluid (white arrow).

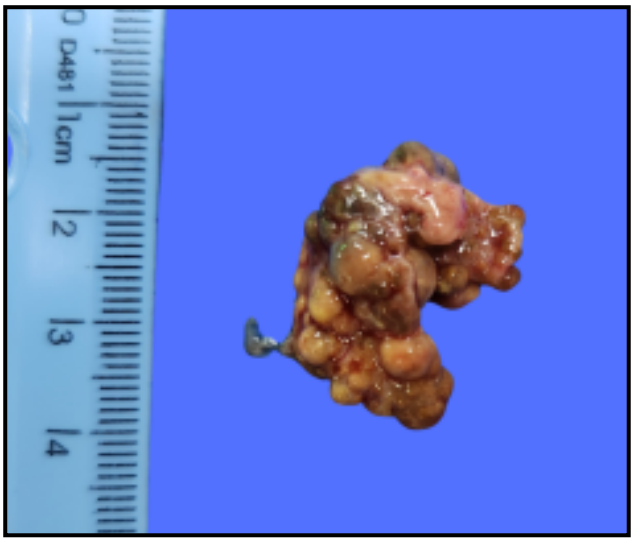

Figure 3. Gross findings in the *Nymphicus hollandicus*. Liver: multifocal to coalescent white, well-delimited, firm nodules with diffuse loss of lobar structure.

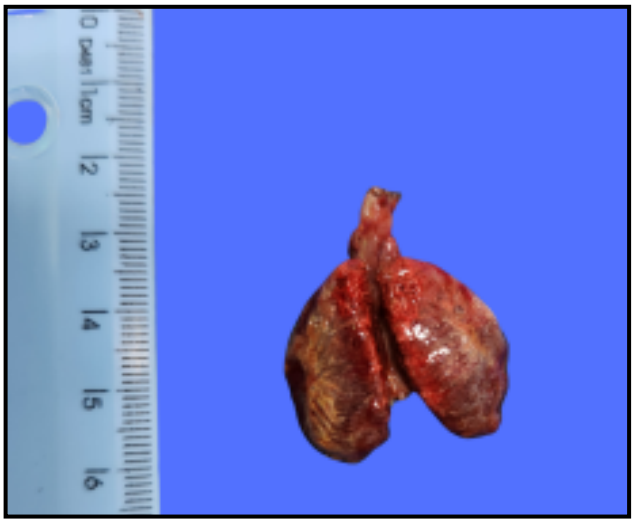

Figure 4. Gross findings in the lung of *Nymphicus hollandicus*. Lung: diffuse congestion.

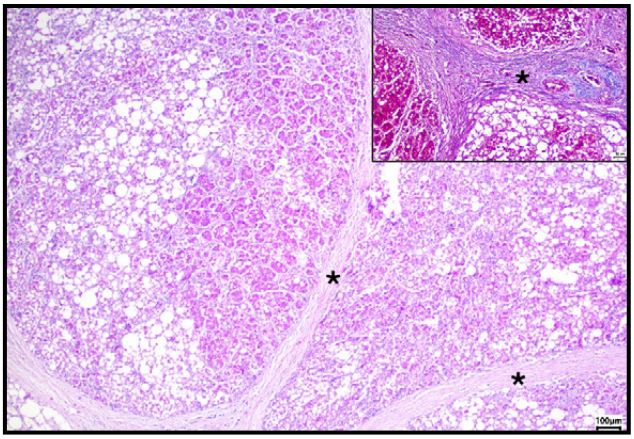

Figure 5. Microscopic lesions in liver of the *Nymphicus hollandicus*. Collagen bundles surrounding hepatocyte islands (asterisks) [HE; obj.4x]. Inset: Collagen bundles (blue) surrounding hepatocytes in Masson's Trichrome special stain (asterisk) [Obj.4x].

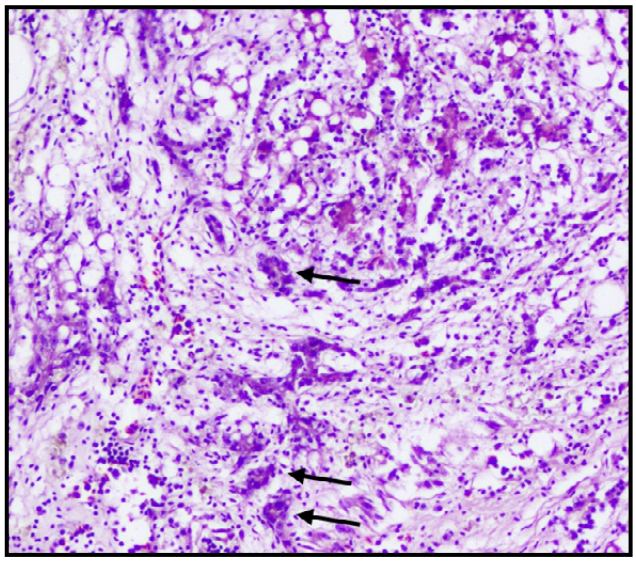

Figure 6. Microscopic lesion in the liver in *Nymphicus hollandicus*. Bile duct proliferation (black arrows) [HE; obj.20x].

macrophage infiltrates. Bile duct proliferation was observed (Figure 6), along with blood vessels within periportal fibrosis. Hepatocytes within nodules displayed clear, well-defined cytoplasm with macrovesicular lipid droplets and pyknotic and karyorrhectic nuclei (Figure 7), leading to a diagnosis of lymphoplasmacytic hepatitis, bile duct hyperplasia, fatty degeneration and cirrhosis. The lungs exhibited pronounced diffuse capillary and vessel engorgement with erythrocytes (Figure 8), supporting a diagnosis of marked diffuse congestion. The heart showed no significant morpho-physiological changes.

DISCUSSION

Information regarding cirrhosis in birds remains scanty, with limited availability of reports in the literature [3,7,20,38]. Cirrhosis arises in response to chronic inflammation, often of undetermined origin [32]. In this case, hepatic cirrhosis was associated with inadequate dietary management, characterized by a high-fat diet.

Unlike mammals, birds possess an underdeveloped lymphatic system, thus, their hepatic portal system transports portomicrons (chylomicrons) from the gastrointestinal tract to the liver, delivering dietary triglycerides and cholesterol to hepatocytes [39]. Excessive triglyceride accumulation in hepatocytes results in hepatic steatosis [25], which, if chronic, can result in cirrhosis, as evidenced in humans [27].

High-energy diets was associated with hepatic steatosis in *Acridotheres tristis* [16]. Seed-rich diets,

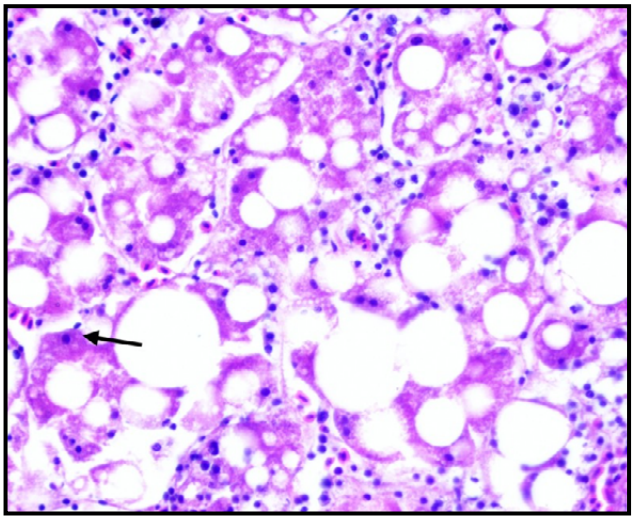

Figure 7. Microscopic lesion in the liver of the *Nymphicus hollandicus*. Liver: Hepatocytes showing well-delimited intracytoplasmic vacuoles, pyknosis (black arrow), and karyorrhexis [HE; obj.40x].

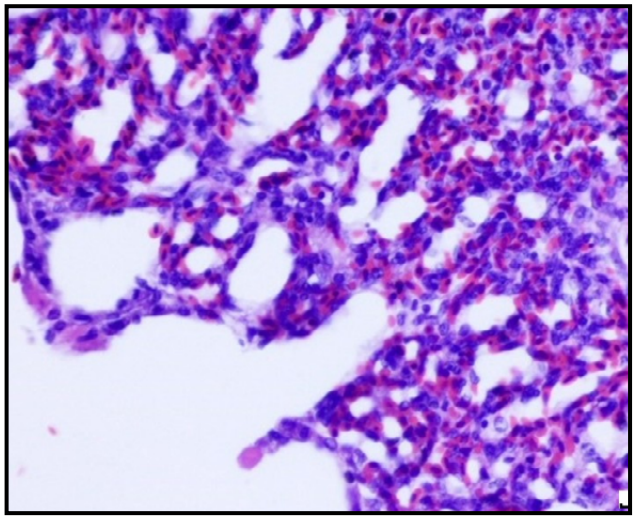

Figure 8. Microscopic lesion in the lungs of *Nymphicus hollandicus*. Lung: Diffuse ngorgement of septal capillaries with erythrocytes [HE; obj.40x].

high in fat and low in protein, minerals, and vitamins (A, D3, and B-complex) [23] increase the risk of hepatocyte triglyceride accumulation [30].

Clinical signs of steatosis, such as emesis, polydipsia, diarrhea, ascites, and dyspnea, have been documented in a parrot (*Amazona aestiva*) [36]. Similar signs have also been noted in other *Amazona aestiva* and *Psittacus erithacus* with hepatic lipidosis [30,33], which aligns with findings in this study.

The dyspnea observed prior to death is attributed to ascites, a multifactorial condition that affects both domestic and wild birds [10]. Ascitic syndrome occurs due to factors that favor the extravasation of plasma from the bloodstream into the coelomic cavity, such as vascular permeability, decreased colloid osmotic pressure [22], and portal vein resistance due to fibrosis [12].

In the case reported here, yellowish feces were present, likely due to fecal biliverdin, a coloration indicative of hepatopathy [14]. Unlike mammals, birds do not produce bilirubin; thus, jaundice of mucous membranes does not occur [15].

Decreased colloid osmotic pressure can result from hypoproteinemia caused by injured hepatic tissue or a low-protein diet, given that seeds alone fail to meet the ideal crude protein content (10-14%) [9]. This deficiency lowers serum albumin levels and colloid osmotic pressure, facilitating fluid leakage into the interstitium.

A case of ascites with similar signs, including dyspnea, was reported in a budgerigar (*Melopsittacus undulatus*) receiving a low-protein diet [6]. Here, dyspnea likely resulted from lung and air sac compression due to fluid accumulation in the coelomic cavity, since birds lack a diaphragm [4,31]. We believe that the cockatiel's death resulted from cardiorespiratory failure, as indicated by the observed dyspnea in the final stages.

In cirrhosis, the multinodular appearance of the liver results from the response to chronic injury. Inflammatory processes in the parenchyma stimulate cellular proliferation and structural distortion [21]. In humans, hepatic stellate cells in the space of Disse are central to fibrosis, transforming into myofibroblasts and fibroblasts under the influence of fibrogenic cytokines, thus producing collagen, elastin, and proteoglycans [5,11]. Avian pathogenesis of cirrhosis remains poorly documented, but may mirror human mechanisms.

Cassmann *et al*. [7] described hepatic cirrhosis in a cockatiel with adenovirus infection, presenting hepatomegaly and a greenish brown multinodular liver. Histological findings included fatty degeneration and bile duct hypertrophy, similar to the findings reported here.

The diagnosis of hepatopathy in living birds may be difficult, but changes in alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP), and lactate dehydrogenase (LDH) may be observed [17]. In birds, the aspartate aminotransferase (AST) to creatine kinase (CK) ratio aids in detecting liver injuries, but liver enzyme values may remain unaltered despite significant pathology.

Low serum glucose, protein, uric acid, and high bile acids are associated with liver failure [8,37].

While serological liver profile tests are useful, most of the enzymes in birds are nonspecific, since they are also present in muscle, bone, and kidney tissues [17]. Complementary diagnostics, including radiography, endoscopy and biopsy, are useful in the diagnosis of cirrhosis [17], but are typically available only in specialized avian centers.

Cirrhosis management focuses on treating underlying causes and complications [34]. In birds, studies indicate hepatoprotective effects from extracts of Milk Thistle (*Silybum marianum*), White Acacia (*Moringa oleifera*), Vernonia (*Vernonia amygdalina*), and Artichoke (*Cynara scolymus*) [2,24,28,35]. Probiotic modulation of gut microbiota also shows a promising potential in cirrhosis management, linking the disease severity to the gut-liver axis [18].

Most pet psittacines continue to receive seed-based diets, despite available commercial feeds and options for fruit and vegetable supplementation. Inadequate nutritional management, influenced by seed affordability and owner unawareness, contributes to conditions such as nutritional hyperparathyroidism, steatosis and cirrhosis. In this case, inadequate dietary management led to cirrhosis in *Nymphicus hollandicus*, culminating in the bird's death.

MANUFACTURER

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***Declaration of interest*.** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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