



Case Report

Dilated cardiomyopathy in a green-winged saltator (*Saltator similis*)

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Abstract

Cardiovascular diseases are rarely reported in birds. Among pet birds, they are most documented in psittacine birds. Dilated cardiomyopathy is a myocardial disease frequently found in poultry but with few reports in both pet and wild birds. An elderly male green-winged saltator (*Saltator similis*) died suddenly after presenting intermittent clinical signs of dyspnea, weakness, and vocalization changes. A general increase in cardiac size with a bulging apex was observed at necropsy. The heart exhibited an enlarged left ventricular space and flaccid musculature on the cut section. The liver was diffusely enlarged with rounded edges. No other gross lesions were observed. Samples were collected in 10% formalin and routinely processed. Histologically, bundles of myocardial fibers were irregularly arranged, with markedly elongated myofibrils and irregular cell contours. Additionally, multifocal areas of disruption were observed between the myofibrils. Diffuse hepatic congestion of sinusoids and portal veins was observed, and diffuse edema in the interstitium and para bronchi was present in the lungs. The clinical signs observed were nonspecific and can be found in several diseases of the cardiovascular and respiratory systems of birds. The gross and histological findings are compatible with what has been described for dilated cardiomyopathy in birds; however, left ventricular dilatation is uncommon in avian patients. Heart diseases in pet birds are still underdiagnosed, and reports of cardiomyopathies are limited to psittacine birds. To the authors' knowledge, this is the first report of a dilated cardiomyopathy in a passerine bird.

Keywords: heart disease, passerines, thraupidae, pet birds, tournament bird.

Introduction

Reports of cardiovascular diseases in birds are scarce and are mainly reported in poultry, followed by pet and wild birds. Among pet birds, these diseases are most documented in psittacine birds (3, 13, 17).

Heart diseases in avian species include ventricular septal defects, persistent truncus arteriosus, aortic hypoplasia, vegetative valvular endocarditis, valvular dysplasia, infectious myocarditis, amyloid deposition, hypoplastic left heart syndrome and cardiomyopathies (13, 26). Additionally, these conditions can also occur in association with diseases of the great vessels of the heart (12).

Dilated cardiomyopathy (DCM) is a myocardial disease characterized by left and/or right ventricle dilatation, resulting in systolic and diastolic dysfunction. DCM has a multifactorial etiology, and the mechanism behind disease development is not entirely understood (6, 18). In birds, DCM has been observed in broilers chickens, domestic and wild turkeys, a red-tailed hawk (*Buteo jamaicensis*), a whooper swan (*Cygnus cygnus*), a harris hawk (*Parabuteo unicinctus*), as well as in both pet and zoo psittacine birds (1, 7, 10-12, 14, 20, 30).

The aim of this work was to describe the pathological aspects of a case of dilated cardiomyopathy in a green-winged saltator (*Saltator similis*) in the state of Paraíba, Northeastern Brazil.

Case Description

An elderly male green-winged saltator (*S. similis*), kept as a singing competition bird, died suddenly and was referred to necropsy at the Veterinary Pathology Laboratory of the Veterinary Hospital at the Federal University of Paraíba (UFPB). The owner reported that the animal had a 3-month history of changes in vocalization and a decrease in singing intensity, both at home and during singing competitions. Two weeks before death, the animal was taken to a singing tournament, where it exhibited difficulty breathing and drooping wings. These clinical signs ceased within a few days, and the owner did not observe them until the day of the bird's death.

At necropsy, a general increase in cardiac size with a bulging apex was observed (Fig. 1A), indicating a possible ventricular dilation of the heart. On the cut section, the heart exhibited an enlarged left ventricular space and flaccid musculature (Fig. 1B), which was better visualized after the organ was fixed in formalin (Fig. 1C). Additionally, the liver was diffusely enlarged with rounded edges. No other gross lesions were observed. Samples from virtually all organs were collected and fixed in 10% formalin. Tissues were trimmed, routinely processed, and embedded in paraffin. Slide sections with 5 µm thickness were prepared and stained with hematoxylin and eosin (H&E).

Histologically, the same enlargement of the left ventricular space was observed (Fig. 1D). Additionally, bundles of myocardial fibers were irregularly arranged, with markedly elongated myofibrils and irregular cell contours (Fig. 1E). In approximately 80% of the chamber, multifocal areas of disruption were observed between the myofibrils (Figs. 1E-F) along with a loss of sarcoplasmic striations. Multiple cardiomyocytes showed pyknosis and/or nuclear karyolysis (necrotic cardiomyocytes), and a moderate to intense amount of red blood cells (cardiac hemorrhage) was observed between the fibers of the endocardium and myocardium (Fig. 1F). In the liver, marked diffuse congestion of sinusoids was observed. Moderate diffuse edema in the interstitium and parabronchi was observed in the lungs.

Discussion

Alterations in vocalization, difficulty in breathing (dyspnea), and drooping wings (weakness) observed in our case are commonly reported in broiler chickens and pet birds with heart diseases (3, 17, 26). However, the intermittent presentation of these signs, as seen in our case, is not commonly observed in avian species. This clinical presentation is nonspecific and can be found in any heart disease that leads to congestive failure, as well as infectious and non-infectious respiratory diseases of the paranasal sinuses, larynx, trachea, lungs, and air sacs (8, 25, 28). Furthermore, as seen in our case, sudden death has been reported in a Rio Grande wild turkey (*Meleagris gallopavo intermedia*), a harris hawk

(*Parabuteo unicinctus*), and in broiler chickens presenting DCM (1, 10, 17, 26).

The left ventricular dilation observed in the present case is compatible with DCM reported in poultry (17, 18). However, in most birds and mammals, the usual presentation involves enlargement of the right ventricle or both right and left ventricles (3, 27), similar to what has been reported in a Rio Grande wild turkey (*M. gallopavo intermedia*), a red-tailed hawk (*Buteo jamaicensis*), a whooper swan (*Cygnus cygnus*), a harris hawk (*Parabuteo unicinctus*) and pet and zoo psittacine birds (1, 7, 10-12, 20). Thus, left ventricular dilation, as seen in our case, is an uncommon presentation for pets and wild birds.

The histopathological cardiac changes, mainly irregular and elongated myocardial fibers, disruption of the fibers, and loss of sarcoplasmic striations, are consistent with those observed in other cases of dilated cardiomyopathy in poultry and wild birds (1, 8, 12, 18). These changes are typically seen in advanced cases of the disease (18), confirming the clinical history of a chronic condition.

Cardiomyopathies in pet birds are commonly reported in psittacine birds (12, 19). Krautwald-Junghanns et al. (12) found dilation or hypertrophy of the ventricular myocardium in 15% of 107 psittacine birds evaluated. On the other hand, cardiomyopathies in passerine birds have not been reported to date, and heart diseases reported in this group are restricted to congenital heart malformations caused by environmental exposure to polychlorinated biphenyls during embryonic development, which results in alterations in the size and shape of the heart (2, 4).

The pulmonary edema observed in the present case is commonly found in broiler chickens and wild birds with DCM (1, 7, 10, 17), the major extracardiac manifestation in cases of left-sided failure (21, 23, 26).

Liver congestion in birds and mammals is commonly associated with right-sided heart failure due to congestion in the caudal vena cava (21, 26). In our case, it may have resulted from a cardiogenic shock due to a decreased cardiac output, leading to vascular hypotension and a decrease in hepatic blood flow and, consequently, hepatic blood accumulation (22, 31). Furthermore, the cardiovascular system is a closed circuit; therefore, a chronic failure of one ventricle eventually leads to the failure of the other, and the pulmonary arterial hypertension associated can lead to right ventricular pressure overload, culminating in a generalized congestive heart failure (21, 26).

In avian medicine, diagnostic imaging can detect and diagnose heart diseases (19). Nevertheless, in some cases, differences in size across bird species, lack of a specific clinical presentation, some non-standardized techniques, and the absence of established reference values make the ante-mortem diagnosis difficult. Consequently, in most cases, definitive diagnoses are only possible through post-mortem examination (3, 19). In the present case, the bird was not submitted to any imaging diagnosis tests before its death.

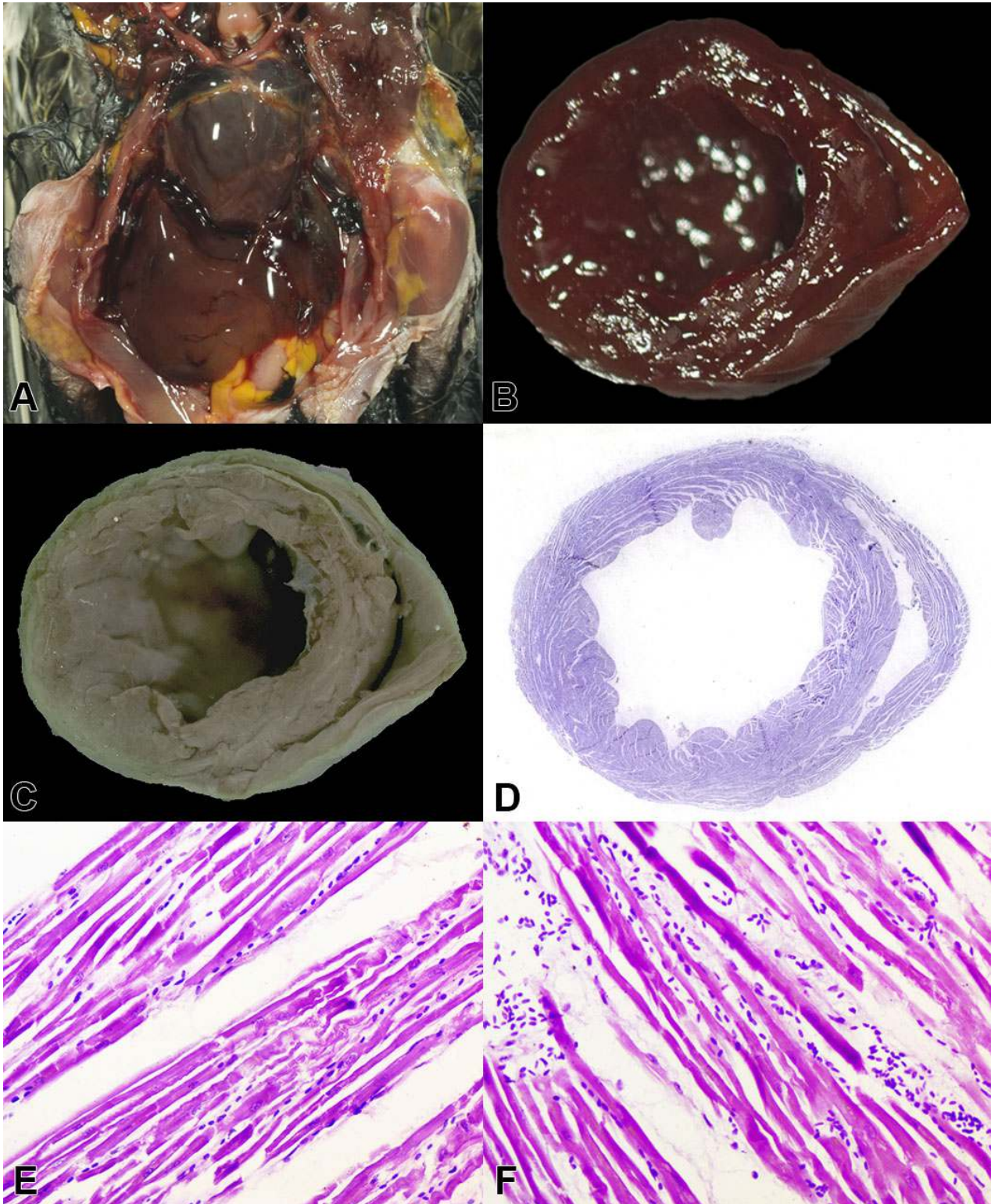


Figure 1. Anatomopathological findings of heart disease in *Saltator similis*. A- Heart in the coelomic cavity with enlargement and bulged apex. B- Cross-section of the heart displaying an increased left ventricular space and flaccid musculature. C- Cross-section of the heart after fixation, evidencing an increased left ventricular space. D- Photomicrograph of the heart with increased left ventricular space. H&E staining. Submacroscopic image. E- Photomicrograph of the heart. Markedly elongated, irregularly arranged myocardial fibers with irregular cell contours, multifocal areas of disruption, and deformed nuclei. H&E staining. Obj. 40x. F- Photomicrograph of the heart. Presence of red blood cells (several without cytoplasm) between the myocardial fibers, especially in the areas of fiber rupture. H&E staining. Obj. 40x.

For pet birds, predisposing factors of heart disease include exercise restriction due to the limited space of cages, as well as hypercaloric and inappropriate diets, as in vitamin E and selenium deficiency (12, 26). Moreover, specific factors contributing to the development of cardiomyopathy in poultry and other avian species include genetic, infectious, metabolic, or toxic etiologies (9, 14, 15, 24). We were unable to identify the cause of the cardiomyopathy in the present case.

Exercise intolerance is typically observed in birds and mammals with heart failure. However, in pet birds, the observation of this clinical sign is directly linked to the size of the cage provided for the animal to fly, and if the size is not sufficient, this sign will not be observed (3), which might explain the non-observation of this clinical sign in the present case.

The exact age of the bird was not determined, but it was an elderly animal, which may have contributed to the development of the DCM. Once birds age, they become more predisposed to the development of heart disorders (27, 29). Correspondingly, in other wild birds reported with DCM, a specific age predisposition was not identified. However, the animals were adults as observed in *Meleagris gallopavo intermedia*, *Buteo jamaicensis*, *Cygnus cygnus* and *Parabuteo unicinctus* (1, 7, 10, 11). On the other hand, in domestic poultry, this disease has been observed between 1 and 4 weeks of age, and it is correlated to the rapid growth that the birds undergo (14, 17).

Notably, in the present case, the bird was male, and in mammals, such as dogs and humans, males are more predisposed to develop DCM (5, 16). However, there is no reported correlation between the occurrence of DCM and gender for birds.

In conclusion, heart diseases in pet birds are still underdiagnosed, and the occurrence of cardiomyopathies in passerine birds is still obscure. To the authors' knowledge, this is the first report of dilated cardiomyopathy in a passerine bird featuring an uncommon left ventricular dilation. DCM-associated heart failure must be investigated and included as a differential diagnosis for passerine birds exhibiting nonspecific signs such as weakness, dyspnea, and sudden death.

Conflict of Interest

The authors declare no competing interests.

References

- Brandao J, Reynolds CA, Beaufre H, Serio J, Blair RV, Gaschen L, Johnson JGI, Del Piero F, Barker SA, Nevarez JG, Tully TN. Cardiomyopathy in a Harris hawk (*Parabuteo unicinctus*). *J Am Vet Med Assoc*. 2016;249(2):221-7.
- Carro T, Walker MK, Dean KM, Ottinger MA. Effects of in ovo exposure to 3,3',4,4'-tetrachlorobiphenyl (PCB 77) on heart development in tree swallow (*Tachycineta bicolor*). *Environ Toxicol Chem*. 2018;37(1):116-125.
- Cornelia K, Krautwald-Junghanns ME. Heart Disease in Pet Birds - Diagnostic Options. *Vet Clin North Am Exot Anim Pract*. 2022;25(2):409-433.
- DeWitt JC, Millsap DS, Yeager RL, Heise SS, Sparks DW, Henshel DS. External heart deformities in passerine birds exposed to environmental mixtures of polychlorinated biphenyls during development. *Environ Toxicol Chem*. 2006;25(2):541-51.
- Fairweather D, Beetler DJ, Musigk N, Heidecker B, Lyle MA, Cooper LT, Jr., Bruno KA. Sex and gender differences in myocarditis and dilated cardiomyopathy: An update. *Front Cardiovasc Med*. 2023;10:1129348.
- Fatkin D, Graham RM. Molecular mechanisms of inherited cardiomyopathies. *Physiol Rev*. 2002;82(4):945-80.
- Fischer I, Christen C, Scharf G, Hatt JM. Cardiomegaly in a whooper swan (*Cygnus cygnus*). *Vet Rec*. 2005;156(6):178-82.
- Fitzgerald BC, Beaufre H. Cardiology. In: Speer BL, editor. *Current Therapy in Avian Medicine and Surgery*. St. Louis, Missouri 63043: Elsevier; 2016. Chapter 6; p. 252-328.
- Frame DD, Hooge DM, Cutler R. Interactive effects of dietary sodium and chloride on the incidence of spontaneous cardiomyopathy (round heart) in turkeys. *Poult Sci*. 2001;80(11):1572-7.
- Frame DD, Kelly EJ, Van Wettene A. Dilated Cardiomyopathy in a Rio Grande Wild Turkey (*Meleagris gallopavo intermedia*) in Southern Utah, USA, 2013. *J Wildl Dis*. 2015;51(3):790-2.
- Knafo SE, Rapoport G, Williams J, Brainard B, Driskell E, Uhl E, Crochik S, Divers SJ. Cardiomyopathy and right-sided congestive heart failure in a red-tailed hawk (*Buteo jamaicensis*). *J Avian Med Surg*. 2011;25(1):32-9.
- Krautwald-Junghanns M-E, Braun S, Pees M, Straub J, Valerius H-P. Research on the Anatomy and Pathology of the Psittacine Heart. *Journal of Avian Medicine and Surgery*. 2004;18(1):2-11.
- Kubale V, Merry K, Miller G, Diaz MR, Rutland CS. Avian cardiovascular disease characteristics, causes and genomics. In: Liu X, editor. *Application of Genetics and Genomics in Poultry Science*. IntechOpen; 2018. p. 141-162.
- Lin KC, Gyenai K, Pyle RL, Geng T, Xu J, Smith EJ. Candidate gene expression analysis of toxin-induced dilated cardiomyopathy in the turkey (*Meleagris gallopavo*). *Poult Sci*. 2006;85(12):2216-21.
- Lin KC, Xu J, Kamara D, Geng T, Gyenai K, Reed KM, Smith EJ. DNA sequence and haplotype variation in two candidate genes for dilated cardiomyopathy in the turkey *Meleagris gallopavo*. *Genome*. 2007;50(5):463-9.
- O'Grady MR, O'Sullivan ML. Dilated cardiomyopathy: an update. *Vet Clin North Am Small Anim Pract*. 2004;34(5):1187-207.

17. Olkowski AA. Pathophysiology of heart failure in broiler chickens: structural, biochemical, and molecular characteristics. *Poult Sci.* 2007;86(5):999-1005.
18. Olkowski AA, Wojnarowicz C, Laarveld B. Pathophysiology and pathological remodelling associated with dilated cardiomyopathy in broiler chickens predisposed to heart pump failure. *Avian Pathol.* 2020;49(5):428-439.
19. Pees M, Krautwald-Junghans ME. Cardiovascular physiology and diseases of pet birds. *Vet Clin North Am Exot Anim Pract.* 2009;12(1):81-97, vi.
20. Phalen DN, Hays HB, Filippich LJ, Silverman S, Walker MJ. Heart failure in a macaw with atherosclerosis of the aorta and brachiocephalic arteries. *1996;209(8):1435-1440.*
21. Robinson WF, Robinson NA. Cardiovascular System. In: Maxie MG, editor. *Jubb, Kennedy, and Palmer's Pathology of Domestic Animals.* 6th ed. Vol 3. St. Louis, Missouri: Elsevier; 2016. Chapter 1; p. 1-101.
22. Samsky MD, Patel CB, DeWald TA, Smith AD, Felker GM, Rogers JG, Hernandez AF. Cardiohepatic interactions in heart failure: an overview and clinical implications. *J Am Coll Cardiol.* 2013;61(24):2397-2405.
23. Shirakabe A, Matsushita M, Shibata Y, Shighihara S, Nishigoori S, Sawatani T, Kiuchi K, Asai K. Organ dysfunction, injury, and failure in cardiogenic shock. *J Intensive Care.* 2023;11(1):26.
24. Stedman N, Brown TJ. Cardiomyopathy in broiler chickens congenitally infected with avian leukosis virus subgroup J. *2002;39(1):161-164.*
25. Stout JD. Common Emergencies in Pet Birds. *Vet Clin North Am Exot Anim Pract.* 2016;19(2):513-41.
26. Strunk A, Wilson GH. Avian cardiology. *Vet Clin North Am Exot Anim Pract.* 2003;6(1):1-28.
27. Welle KR, Lightfoot T, Reavill D. Cardiovascular Disease in Aging Birds. *Association of Avian Veterinarians.* 2011:9-15.
28. Wigle WL. Respiratory diseases of gallinaceous birds. *Vet Clin North Am Exot Anim Pract.* 2000;3(2):403-21.
29. Wilson FD, Magee DL, Jones KH, Baravik-Munsell E, Cummings TS, Wills RW, Pace LW. Morphometric Documentation of a High Prevalence of Left Ventricular Dilated Cardiomyopathy in Both Clinically Normal and Cyanotic Mature Commercial Broiler Breeder Roosters with Comparisons to Market-Age Broilers. *Avian Dis.* 2016;60(3):589-95.
30. Wu DJ, Lin JA, Chiu YT, Cheng CC, Shyu CL, Ueng KC, Huang CY. Pathological and biochemical analysis of dilated cardiomyopathy of broiler chickens--an animal model. *Chin J Physiol.* 2003;46(1):19-26.
31. Xanthopoulos A, Starling RC, Kitai T, Triposkiadis F. Heart Failure and Liver Disease: Cardiohepatic Interactions. *JACC Heart Fail.* 2019;7(2):87-97.