Cardiac disease in the Spix Macaw (Cyanopsitta spixii): two cases

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Cardiovascular disease in avian species, other than poultry, is being increasingly reported. In psittacine birds, atherosclerosis and congestive heart failure are the leading cardiovascular diseases, often resulting in multiorgan dysfunction and demise. The Spix’s macaw (Cyanopsitta spixii) is arguably the most endangered psittacine species worldwide. We aimed to describe the gross and microscopic findings in two adult Spix’s macaws wherein severe cardiovascular pathology resulted in sudden death. Bird 1 had pathologic findings consistent with fibrinoheterophilic vegetative pulmonic valvular endocarditis with luminal obliterator thrombosis, myocarditis and epicarditis, myocardial fibrofatty infiltration and cardiomyocyte loss, as well as generalized septicaemia. Microbiological analysis yielded Pantoea septica from the intestines and Acinetobacter baylyi from the cerebrum. Bird 2 had changes suggestive of right brachiocephalic coarctation-like obliterator arteriopathy. The latter is a novel cardiovascular pathology in avian species, and its severity and extent likely led to acute decompensation of pre-existing cardiac disease. These results add to the body of knowledge on avian cardiovascular pathology and may aid in veterinary medical decisions on caged birds, including those part of ex situ conservation efforts.

Keywords avian pathology; cardiovascular disease; coarctation; obliterator arteriopathy

Abbreviations CHD, congenital heart disease; CHF, congestive heart failure; CoA, coarctation of the aorta


Cardiovascular disease is common in caged birds. Atherosclerosis and congestive heart failure (CHF), which often result in multiorgan dysfunction and death, are the leading reported cardiovascular diseases in psittacine birds (order Psittaciformes). Atherosclerosis is particularly prevalent in aged and female birds of genera Psittacus, Amazona and Nympicus. The brachiocephalic trunks and ascending aorta are typically most severely affected.5,6 Primary myocardial, great vessel and coronary disease are common causes of CHF, whereas secondary myocardial disease and pericardial disease are less prevalent.1,3 CHF due to cardiomyopathy of unknown aetiology was described in young grey parrots (Psittacus erithacus).7

The Spix’s macaw (Cyanopsitta spixii) is regarded as the most endangered psittacine species in the world, primarily due to capture for illegal trade, and is considered to have become extinct in the wild since 2000.8 Captive breeding and reintroduction to its natural habitat is deemed the last hope for the species.9 Therefore, an ambitious, multidisciplinary Action Plan for the Conservation of the Spix’s Macaw with international collaborations and an active involvement of the local community was established and led by The Chico Mendes Institute for the Conservation of Biodiversity (ICMBio) since 2013. Successful captive breeding correlates with medical knowledge of the species. Nonetheless, there is a paucity of information in the literature on the causes of morbidity and mortality in captive Spix’s macaws. Herein, we describe the gross and microscopic findings in two adults male Spix’s macaws wherein severe cardiovascular pathology resulted in sudden death.

Medical histories

Bird 1 was a 244 g (normal weight range: 288–318 g), approximately 40-year-old (life expectancy in the wild: 20–30 years), male Spix’s macaw in emaciated body condition that was found dead in the cage. The clinical history did not indicate cardiomyopathy. Bird 2 was a 330 g, 27-year-old male Spix’s macaw in moderate body condition that had been historically diagnosed with cardiomyopathy and was being treated with enalapril (0.16 mg/kg/48 h). The bird was found dead in the cage. The birds were housed in different facilities, and death occurred approximately 1 year apart. The diet of both birds consisted of extruded feed for parrots, fresh fruits (guava, banana, papaya, orange, apple), vegetables (broccoli, carrot, green peas) and some nuts. The facilities housed other parrots’ species, but without direct contact with the Spix’s macaws. The birds were not related. Both birds were submitted for postmortem examinations between 2 and 5 h after the animals were found dead.

Case report 1

A standard necropsy was performed and selected tissues were collected (trachea, thyroid, oesophagus, crop, lungs, pro-ventricle, ventricle, skin, skeletal muscle, heart, liver, intestines, kidneys, spleen and pancreas) and fixed in 10% neutral buffered formalin. The tissues were trimmed, routinely processed and embedded in paraffin-wax. Thin tissue sections (5 μm) were cut and stained with

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haematoxylin and eosin. Brachiocephalic arteries were additionally stained by Masson’s trichrome (for collagen) and Verhoeff (for elastic) techniques. For immunohistochemistry, 3 μm sections of right (rBa) and left (lBa) brachiocephalic arteries were incubated with primary antibodies specific for smooth muscle actin (monoclonal 1A4; 1 in 1000 dilution; Thermo Scientific Lab Vision; Rockford, IL, USA), vimentin (monoclonal V9; 1 in 3000 dilution; Dako, Carpinteria, CA, USA) and desmin (monoclonal D33; 1 in 3000 dilution; Dako). Antigen–antibody binding was detected by UltraVisionTM LP Detection System HRP Polymer (Thermo Scientific Lab Vision) and ‘visualized’ by use of 3, 3’-diaminobenzidine chromogen (Sigma D5637, St Louis, MO, USA). Positive control tissues included Spix’s macaw brachiocephalic artery and human appendix. As negative controls, primary antibodies were replaced by homologous non-immune serum.

Cerebrum, intestines, kidneys and cardiac blood were sampled for bacteriological analysis and cultured for 24 h under aerobic and anaerobic conditions at 37°C. Identification of isolates was performed by matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS).

On necropsy, this bird had cardiomegaly with multifocal, poorly demarcated, transmural, pale foci throughout the ventricles, atria and auricles (Figure 1). Microscopically, there was fibrinoheterophilic vegetative pulmonic valvar endocarditis (Figure 2) with necrosis and luminal obliterative thrombosis, myocarditis and epicarditis. Myocardial fibrofatty infiltration and cardiomyocyte loss were also noted. Additional relevant findings indicated septicaemia and included: necroheterophilic tubulointerstitial nephritis, ureteritis and polyarthritis; hepatic necrosis; pulmonary congestion, oedema and haemorrhage; and coelomic and abdominal air sac haemorrhage. Microbiological analysis yielded Acinetobacter baylyi (++; pure growth) in the cerebrum and Pantoea septica (++; polybacterial growth) in the intestines. No bacterial growth was observed from the kidneys and cardiac blood samples.

Figure 1. The heart of a Spix’s macaw that died of septicaemia. Frontal view of the heart (ex situ). There are multifocal, poorly demarcated, pale tan foci in the myocardium of the right and left ventricles. Bar, 1 cm. Inset: Detail of pale tan myocardial foci in the left ventricle. Bar, 0.5 cm. Ao, aorta; Lba, left brachiocephalic artery; Lv, left ventricle; Rba, right brachiocephalic artery; Rv, right ventricle.

Case report 2

A standard necropsy was performed and selected tissues were collected as described for bird 1. In bird 2, spanning from the aortic valve to the right brachiocephalic artery (rBa), there was an 8 mm-long segmental aneurysmal dilatation (up to 3.5 mm in diameter; the unaffected left Ba was up to 2.5 mm in diameter). A 4 × 2 × 2 mm obliterative intraluminal mass was located at 3 mm from the aortic valve. Distal to this segment, the arterial lumen was markedly reduced to 1–2 mm in diameter and had transmural haemorrhage (Figure 3). The rBa wall was up to 0.6 mm thick at the coarctation (obliteration, abrupt narrowing of the lumen) point while the left Ba was up to 0.3 mm thick at the same segment level. The intimad and adventitial surfaces at the aneurysmal segment were pale tan to white and smooth. The heart was diffusely enlarged with dilated ventricles and atria. On cut section, the free walls were subjectively thickened and flaccid (hypertrophic eccentric cardiomyopathy). Diffusely, the cardiac walls were brown with multifocal, poorly demarcated, pale tan foci. Additional gross findings consisted of chronic hepatopathy and pulmonary congestion with emphysema.

Microscopically, the rBa had a focal intraluminal mass arising from the interface of the subintima and tunica media that led to complete arterial stenosis (Figures 4 and 5). The mass was composed of densely packed 1A4- and vimentin-positive myofibroblasts admixed with abundant dense collagen (blue with Masson’s trichrome stain) bundles arranged perpendicular to bloodstream (Figure 5). No elastin fibres were detected. Neither recanalization nor hemosiderophages
were observed. Distal to this segment, the rBa had transmural necrosis and haemorrhage; this finding was interpreted as an area of infarction. In the heart, there were scattered foci of myocardial fibrosis associated with cardiomyocyte atrophy and loss, mild myofiber disarray, hypertrophic cardiomyocytes, focal fibrofatty infiltration, lipofuscinosis, congestion, interstitial oedema and rare haemorrhage. Additional relevant microscopic findings associated with pulmonary and systemic hypertension were as follows: pulmonary haemorrhage associated with focal rheoxic (Figure 6), oedema and congestion; intrapulmonary arterial smooth muscle hypertrophy/hyperplasia; chronic glomerulopathy with fibrosis and sclerosis; and renal arterial tunica media hypertrophy/hyperplasia. Based on these findings, a diagnosis of rBa coarctation-like oblitative arteriopathy and acute decompensation of pre-existing cardiac disease was determined.

Discussion

We have presented two cases of severe cardiovascular disease with significant pathology in two Spix’s macaws. Bacterial infection of the heart can result in endocarditis, including valvular endocarditis, myocarditis or epicarditis, and may be the result of haematogenous spread of infection or direct extension from adjacent tissues. In pet and aviary birds, valvular endocarditis is relatively rare, and the left valve is chiefly affected. The most common agents are streptococci, staphylococci, Pasteurella multocida and Escherichia coli. In Bird 1, we found fibrinoheterothetic pulmonic valvular endocarditis, myocarditis and epicarditis together with septicaemia-associated lesions. Although we did not find an obvious route of entry in this case, we propose that vegetative endocarditis and myocarditis may have led to cardiac failure, resulting in a clinicopathological picture that was further aggravated by septicaemia.

In Bird 1, P. septica and A. baylyi were isolated from the intestines and cerebrum, respectively. The genus Pantoea encompasses a diverse group of widely distributed environmental Gram-negative bacilli (Enterobacteriaceae) that are commonly isolated from plants, insects and nonmammalian and mammalian species, including humans. Pantoea spp. are typically regarded as environmental contaminants or are associated with opportunistic or nosocomial infections in humans and animals. Currently, there is much debate as to the pathogenicity of this genus in animals. Several Pantoea species, including P. septica, have been routinely isolated from clinical samples of diseased and nondiseased patients who are immunocompetent or immunocompromised. Many disease processes, including septicaemia, have been ascribed to various Pantoea spp.; however, direct causation for most of these cases has not been demonstrated. Furthermore, P. septica has been isolated from presumably healthy saffron finches (Sicalis flaveola) from São Paulo state, Brazil. To the best of our knowledge, there are no reports of P. septica isolated from diseased avian species or any healthy or diseased Spix’s macaws. A. baylyi is a nonfermentative environmental Gram-negative bacillus that is frequently isolated from soil. Nonetheless, a case series reported this bacterium as responsible for nosocomial infection in immunocompromised humans. To the best of our knowledge, A. baylyi has not been reported in animals. Taken these considerations into account, the pathological relevance of P. septica is uncertain and the possibility of environmental contamination or
opportunistic outgrowth from normal intestinal microbiota cannot be excluded. Analogously, the pathogenic significance of *A. baylyi* in this case remains elusive. Overall, Enterobacteriaceae and non-fermentative Gram-negative bacteria are not part of the microbiota of free living psittacine birds, they are often a consequence of captivity.15

Congenital heart disease (CHD) in avian species, other than poultry, is rarely described.16 The main CHD conditions reported in psittacine birds include ventricular septal defects and congenital aneurysms, mainly in cockatoos (Cacatuidae), cockatiels (*Nymphicus hollandicus*) and African grey parrots.2,7 Abnormal narrowing of cardiac chambers, heart valves or blood vessels lead to forms of CHD that result from obstruction to blood flow. Among them, coarctation of the aorta (CoA) consists of an abrupt narrowing of the lumen of the aorta due to the projection of a shelf-like curtain of aortic medial tissue from the superioposterior wall into the aortic lumen adjacent to the ductus or ligamentum arteriosum.17 Coarctation has been historically used interchangeably with hypoplasia and aplasia, however, they are dissimilar. Hypoplasia defines an arrested development leading to smaller size or immature state of development of the arterial segment. Aplasia, so-called ‘atresia’, denotes absence of the arterial segment. Therefore, hypoplasia and aplasia have diverging pathogeneses and/or genetic bases, which primarily involve intracardiac/aortic blood flow defects, and lack a shelf-like projection of the arterial medial components into the lumen.11,17 In case 2, the rBa presented a focal intraluminal mass composed of myofibroblasts and collagen bundles, which were perpendicularly oriented to the

Figure 5. The heart of a Spix’s macaw that died of heart failure associated with right brachiocephalic (obstructive) arteriopathy. (A) Close-up view of squared area in Figure 4 with intraluminal myofibroblastic and collagenous mass (arrowhead). The mass merges with the adjacent subintima/tunica media interface (asterisks). Haemorrhage is indicated with an arrow. (B) The rBa intraluminal mass (arrowhead) is composed of 1A4- (left inset) and vimentin-positive myofibroblasts (right inset). Immunohistochemistry. (C) The rBa intraluminal mass (arrowhead) is composed of abundant dense collagen (blue). Masson’s trichrome. (D) The rBa intraluminal mass (arrowhead) lacks elastin fibres. Verhoeff stain. Inset: Close-up view of squared area in D. Lu, lumen.
bloodstream, that merged with the overlying intima, subintima and tunica media and led to complete arterial stenosis. These features resembled CoA.18,19 We observed additional findings associated with systemic hypertension likely as a result of rBa coarctation-like arteriopathy.

Although CoA is well-known in humans, this condition is very rare in animals, being only reported in few dogs.19,20 Its pathogenesis is not fully understood, but it is known that CoA results from abnormal development of the embryologic left fourth and sixth aortic arches.18 Often, CoA is associated with other CHD (e.g. bicuspid aortic valve, persistent ductus arteriosus, atrial and ventricular septal defects), aortic stenosis and other disease processes (e.g. Turner syndrome and berry aneurisms of the circle of Willis).18 We do not know whether this bird was born with such defect and evolved over time or whether this represents a secondary phenomenon, as CoA-like reported in humans subsequent to traumatic events.21,22 The lack of early diagnostic imaging data and the fact that the animal died at an advanced age render a congenital condition difficult to prove definitively in this case, however, the absence of recanalization, hemosiderophages, varying degrees of myofibroblastic disarray or any evident pre-existing predisposing disease process for chronic thrombotic event, lends support to a diagnosis of rBa coarctation-like arteriopathy.11,18 Furthermore, CoA is not invariably fatal and human patients may live for a number of years after diagnosis. If uncorrected, most human patients with CoA die before the age of 40 years from heart failure by increased resistance to the ejection of blood (afterload CHF), ruptured aorta or cerebral vessel, or infective endocarditis.23

To the authors’ knowledge, this is the first report of spontaneous rBa oblitative arteriopathy in avian species that recapitulates features of CoA. Neither bird presented gross or microscopic evidence of atherosclerosis. In conclusion, these cases add to the body of knowledge of cardiovascular pathology in avian species and may aid in medical decisions on captive avian collections, including those part of ex situ conservation efforts, and may prove of value for further comparative pathological analysis.

Acknowledgments

JDD is the recipient of a postdoctoral fellowship by the São Paulo Research Foundation (FAPESP; grant #2017/02223-8). ILCD is the recipient of research fellowship from the CNPq (grant # 304999/2018-0).

Conflicts of interest and sources of funding

The authors declare no conflicts of interest or sources of funding for the work presented here.

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(Accepted for publication 22 May 2021)