Pulmonary Artery Aneurysm and Polycythemia with Respiratory Hypersensitivity in a Blue and Gold Macaw (*Ara ararauna*)

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A diagnosis of respiratory hypersensitivity was made in a 9-year-old blue and gold macaw due to radiographic changes and clinical pathology to rule out other etiologies. Therapy included corticosteroids and antibiotics in crises, antihistamines, and air quality management over a four year period. The patient presented acutely ill and died during evaluation. Radiographic findings showed cor pulmonale. Gross and histopathologic findings encompassed hemorrhage, a ruptured pulmonary artery aneurysm, and chronic pulmonary inflammation and thickening of the pulmonary arterioles.

"Brutus," a 9-year-old female blue and gold macaw (*Ara ararauna*) was presented August 23, 1988 for acute onset of respiratory distress. Previous medical history included a normal physical exam and hemogram six months prior to the visit, no serious medical problems for the previous seven years. In 1981, the senior author treated the patient successfully for acute severe chlamydiosis occurring soon after importation. The owners housed Brutus at night in a home "kennel" bank of built in cage units, with another macaw and below a pair of moluccan cockatoos (*Cacatua moluccensis*). Abnormal physical findings including "panting" at rest before handling, severely hyperemic tracheal epithelium, and "wet" airway sounds noted aurally.

The owners admitted the patient for diagnosis and treatment. A blood panel was drawn and a fecal Chlamydia antigen ELISA (IDEIA®-Novo-UK) was collected. Anesthesia was induced by masked with isoflurane/oxygen. An airsac tube was placed in the left abdominal airsac, secured with forceps, and attached to the non-rebreathing isoflurane system. Whole body radiographs were made. The trachea was examined endoscopically before the performance of a tracheal wash, achieved by placing the patient in an upright position and inserting a 12 fr urinary catheter intratracheally to the point of bifurcation. Twenty milliliters of warm Lactated Ringers were rapidly instilled into the tube. The syringe was immediately aspirated, then the tube was pulled out. Cytology and culture were performed.

Tracheal wash cytology and microbiology rendered unremarkable findings. The leukocyte count was >80,000/mm3, with a toxic heterophilia, and the packed cell volume (PCV) was elevated. No radiographic lesions were noted and the chlamydia test was negative. A tentative diagnosis of cardiac disease associated with chronic chlamydiosis was considered. Treatment consisted of IM doxycycline (Vibramycin IV, Pfizer-Rotterdam) @ 75mg/kg weekly for six treatments. Within a few days of therapy commencement, Brutus appeared clinically normal and stronger. A recheck on October 13, 1988 revealed a normal physical examination and hemogram.

Two weeks later, the patient presented again with a repeat of the dyspneic signs. The hemogram was unremarkable and the radiograph showed bronchiolar thickening. A tentative diagnosis of respiratory hypersensitivity was made. Therapy began using with hydroxyzine @ 1.0ml per 120ml drinking water daily.

No further problems were reported until September 16, 1989, when the patient again presented with dyspnea, especially during handling. The face patch appeared cyanotic and the blood sample very dark. A blood panel and gram stain, and a radiograph taken. The chemistries and pharyngeal gram stain were unremarkable. The hemogram was normal except for the PCV of 71%. The patient received one intramuscular injection Dexamethasone @0.3mg/kg. Clinical response happened rapidly and dramatically. Upon discharge, the owners administered enrofloxacin (Baytril-Bayer solution, FRG) @15mg/kg PO twice daily for 10 days. A recheck two weeks later found a mild leukocytosis, toxic heterophilia, and an elevated PCV. The enrofloxacin was continued for two more weeks. The owner followed an instruction to move the pair of cockatoos to the far end of the house, in order to minimize exposure to the powder down. The owner designed and installed an electrostatic

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whole house air filtering system (Honeywell) which ran parallel to the central air conditioning system. Additional and specialized filters were added to the central air system.

The patient stabilized with no further signs of dyspnea until July 9, 1990 when she presented with severe dyspnea and collapse. Blood and culture samples were taken, dexamethasone (0.01 mg/kg) and piperacillin (Piperacil-Lederle) @ 100mg/kg) was given and the patient was placed in an oxygen-rich environment. Over the next 24 hours, the patient improved. Enterobacter amnigenus was isolated from the pharynx; treatment with piperacillin continued. On the following day, dyspea returned. The patient was anesthetized with isoflurane/oxygen for radiography. These and previous films were reviewed by the radiologist (Dr. Sam Silverman), who noted that the cardiac silhouette had increased slightly in size from previous studies. The pulmonary arteries appeared enlarged. The tentative diagnosis was reactive airway disease consistent with hypersensitivity.

For the next 26 months, Brutus was maintained on hydroxyzine therapy in the water. Occasional bouts of dyspnea were reported. The patient bonded with a cage mate, and the pair produced one baby which was pulled for hand-rearing at 5 weeks of age.

On September 26, 1992 the patient was presented during a busy appointment schedule, after falling off of a high perch. Another fertile egg had been laid in August which the owners pulled for artificial incubation at another site. The owners were concerned about bone fractures. A cursory examination found a possible egg palpated in the abdomen, non-weight bearing in the right leg and normal respiratory rate. The patient seemed bright and alert. The owners were asked to leave the patient for a radiograph, which was performed two hours later. During the Xray procedure (restraint board, no anesthesia), some abnormal respiratory sounds were noted. The patient was returned to a cage. Five minutes later, she collapsed and died.

The antemortem radiograph showed marked rounding of the cardiac silhouette and indistinctness in the caudal thoracic air sac. The post mortem examination found an egg in the oviduct, massive hemorrhage in the right caudal thoracic air sac, blood filled lungs, and a 5 millimeter rupture in the proximal pulmonary artery. The histologic diagnosis (Dr. RE Schmidt) was pulmonary artery aneurysm, pulmonary hemorrhage, and hypersensitivity pneumonitis. The lung was collapsed with diffuse congestion and multifocal hemorrhage. Arteries and arterioles were thickened. There was hypertrophy of atrial muscles, and a diffuse infiltration of plasma cells and lymphocytes was noted. Variable edema was noted in air capillaries and tertiary bronchi.

Discussion

Pulmonary hypersensitivity, pneumonitis, and pulmonary arterial hypertrophy directly contributed to the pulmonary artery aneurysm.[1] Cor pulmonale was present radiographically at the final presentation. Cardiac circulatory physiology of avians follows mammalian principles closely[2]. Electrocardiography was not performed. Changes in the avian ECG with cor pulmonale would likely include a right axis shift.[3]

Polycythemia was found in this patient. Postulated causes in macaws include hypoxia due to poor ventilation.[1,2] Taylor demonstrated reduced pO_2 in several clinical cases[1]. Increased circulating blood viscosity exacerbates cardiac failure. The authors of this report felt that intimate exposure to powder-down producing birds (cockatoos) was the predisposing cause of the hypersensitivity in this case.

Diagnosis of pulmonary hypersensitivity (PHS) should be considered when the following radiographic changes are present: bronchiolar thickening and great vessel enlargement. The following radiographic features suggest a diagnosis of pulmonary hypersensitivity: on the lateral view the bronchiolar walls are thicker or the lumens are smaller. Changes in imaging are due to one of the following: bronchoconstriction, bronchiolar thickening, or endobronchial sectretion. Imaging patterns associated with secretions differ from PHS because some bronchioles will be completely occluded. No complete occlusion occurs with PHS and the lung field lacks evidence of consolidation. The air sac changes in PHS are variable but may include hyperinflation. Abdominal visceral air sac changes are minimal, if any, showing no thickening or mass lesions[4]. Right heart failure in chronic cases can result in variable vascular changes associated with right heart failure.

Polycythemia may also be present. Specific diagnosis of pulmonary hypersensitivity requires a lung biopsy. This can be accomplished via a laparoscopic approach from the ventral abdomen or in the intercostal space dorsocaudal to the scapula.[5]

This case was successfully handled for months at a time using air management and antihistamines. Due to reduced pulmonary clearance secondary bacterial infections will frequently occur. While corticosteroids can be useful during a crisis, chronic use may not be safe. At this time, no therapy has been devised to arrest the progression of this disease.

References

1. Taylor M: Polycythemia in the blue and gold macaw- a report of three cases, in Proceedings First International Conference of Zoological and Avian Medicine, Turtle Bay; Hawaii, 1987 95-104.

2. Sturkie PD:Body Fluids: Blood. In Sturkie, PD (ed), Avian Physiology. New York, Springer-Verlag, 1986. 130-167.

3. Miller M: personal communication, Cardiopet, Floral Park, NY, 1992.

4. Silverman S: personal communication, Veterinary Telerad, Corte Madera, CA 1993.

5. Taylor M: Lung biopsy as a diagnostic tool in avian medicine, Proc AAV.207-211, 1992, New Orleans, LA.

Postscript: this appears to occur primarily in large Ara, particularly Blue and Gold Macaws. Maximum ventilation, HEPA filters are the best prevention.