Right Atrioventricular Valve Dysplasia in a Scarlet Macaw.

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An18 month old hand-raised Scarlet Macaw hen was presented on the 13/09/06. She had been purchased one year earlier along with an unrelated cock bird. The birds had been housed together for several months prior to purchase and were well bonded. The hen's parents were unrelated imported birds.

The bird had been noticed depressed, fluffed and inappetent for two days prior to presentation. She weighed 712 grams and was depressed, fluffed but still aware. Her contour feathers displayed some wear and tear but generally were within normal limits. The upper beak showed a slight deviation to the RHS. She had good bodily condition and her abdomen palpated within normal limits. Her faeces were malaenic with last stool containing frank blood (Figure 1). The urates were white.

Isoflurane mask induction was performed at 2% to facilitate examination.

On auscultation there were raspy respiratory and pounding heart sounds. No murmur was heard. A wet smear of the faeces showed intact red blood cells but no other evident abnormalities. Faecal Gram stains showed Gram positive cocci (GPC 4+) and Gram positive rods (GPR +, Large GPR +).

Radiographs were performed. They showed hepatomegaly and an enlarged heart shadow. (Addendum1). Bloods were taken for a CBC and an MBA (Addendum 2). A cloacal swab was taken for culture and sensitivity (Addendum3). She was wormed with 0.8 ml moxidectin/praziquantal.

At this point a tentative diagnosis of gastro enteritis was made and therapy instigated. This was thought to be secondary to cardiac or hepatic disease or possibly the cardiac signs were due to toxaemia from the gastrointestinal infection.

Therapy was commenced with lincospectin 0.08 ml IM bid and baytril 15 mg/kg IM sid. She showed a steady response to therapy with the intestinal haemorrhage resolving after four days and her appetite returning to normal after five days.

The cloacal culture failed to grow any pathogens and she was discharged after seven days. A revisit examination to assess cardiac function and for repeat CBC/MBA was recommended for four weeks.

She represented on the 3/11/06 with a one day history of depression. No information was available on her appetite or stools. There was significant facial flushing that failed to abate after 20 minutes rest.

Examination was kept to a minimum. Her weight was up to 800g. Auscultation produced normal respiratory sounds but with a pounding heart beat. Her faeces were semi formed and bright green with white urates. Her abdomen was concave on palpation. Mask induction with 2% isoflurane was again performed for full examination and blood collection.

An oral examination was NAD but there was increased erythema proximal to the glottis and on the cranial margin of the glottis. She was then intubated for radiographs, blood collection and fluid therapy. Blood was collected for a repeat CBC/MBA (Addendum 4) and for protein electrophoresis (Addendum 5) and a *Chlamydophila* antibody test if required. There was a pounding jugular pulse on blood collection. A washing machine heart murmur was audible on auscultation. Heart rate was over 200 bpm.

Radiographs showed a very round enlarged heart shadow. A pericardial effusion was suspected. (Addendum 6). There was mild dehydration present so 20 mls of 0.45% glucose saline given s/c as jugular had blown. A cardiac ultrasound was requested as soon as possible.

Therapy was instigated as below:

- Baytril 15 mg/kg i/m sid = 0.24 ml
- Noroclav 150 mg/kg = 0.6 ml i/m sid
- Frudix 1 mg/kg bid = 0.02 ml i/m
- Metacam 0.4 mg/kg sid = 0.06 ml

There was no improvement in her condition over the next four days until electrocardiography could be performed. Echocardiography demonstrated the presence of severe right atrioventricular valve regurgitation and subsequent right ventricular and right atrial eccentric hypertrophy (report - Addendum 7) This finding is most consistent with valvular dysplasia. Images iof the valvular lesion are displayed (Addendum 8).

After discussions with the owners, euthanasia was performed.

A post mortem was performed and samples collected for histological examination. At post mortem the bird was in thin condition. Feathering was within normal limits. There were no discharges from any orifices. On dissection there was no free fluid in the abdominal space to suggest ascites. The heart and liver were enlarged but of normal colour. There was no evidence of any pericardial effusion. Lungs, air sacs, kidneys and spleen appeared within normal limits. The bowel loops contained dark fluid. The heart is pictured with a galah heart for comparative sizing (Figure 2). Histological results are documented (Addendum 9).

It appears that this lesion is congenital in origin but there is nothing to suggest that it is hereditary. Four remaining siblings up to 4 years old are clinically normal but have not undergone any veterinary assessment.

A literature search shows that the prevalence cardiac disease in psittacine birds occurs at a similar percentage to that of other companion animals. Lesions consistent with right sided congestive heart failure predominate in poultry. The incidence in psittacine birds appears similar to this. Right atrioventricular valve dysplasia appears in psittacine birds with no other specific cases found.

This case highlights the need for aviculturists to request a post purchase examination of their birds to enhance to detection of underlying disease.

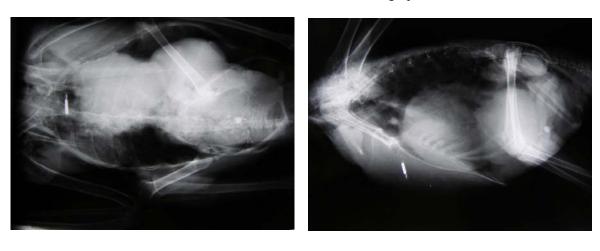
Reference

1. Oglesbee BL and Oglesbee MJ (1998). Results of postmortem examination of psittacine birds with cardiac disease: 26 cases (1991-1995). *J Am Vet Med Assoc*. **212**:1737-1742



Figure 1: Frank blood in macaw faeces.

Addendum1: Ventrodorsal and lateral radiographs 13/09/06.



Ventrodorsal Lateral

Addendum 2: CBC and MBA 13th September 2006

Haematology				
	Value	Reference Range		
Haematocrit	0.44 L/L	0.42-0.54		
PCV	0.44 L/L	0.42-0.54		
Thrombocytes	Normal			
WCC	18.4 x 10 ⁹ /L	10.0-20.0		
Heterophil	95 (50-75)	17.5 x 10 ⁹ /L		
Lymphocyte	5 (23-53)	0.9 x 10 ⁹ /L		
Monocyte	0% (<2)	< 0.1 x 10 ⁹ /L		
Eosinophil	0%	< 0.1 x 10 ⁹ /L		
Basophil	0% (<2)	< 0.1 x 10 ⁹ /L		
Plasma appearance	Normal	·		
Red cell and white cell m	orphology normal.			
	Biochemistry			
Serum glucose	15.3 mmol/L	11.7-20.0		
Urea	1.1 mmol/L	0.3-3.3		
Calcium	1.8 mmol/L	2.1-3.0		
Albumen	3 g/L	3-24		
Globulin	7 g/L	21-38		
AST	52 L IU/L	65-168		
СК	444 H IU/L	88-361		
Cholesterol	1.2 L mmol/L	2.5-6.8		
Amylase	450 IU/L	239-564		
GLDH	7 H IU/L	<2		
Bile Acids Postprandial	5 l umol/L	7-100		
Uric acid	0.56 mmol/L	0.11-0.71		
Sample appearance	Normal			

Comment: HCT and thrombocytes are adequate. Relative heterophilia, but the absolute counts are within published reference ranges. Low calcium may reflect the low normal albumin, but suggest monitor. Low protein, hypocholesterolaemia may reflect gastrointestinal losses/malassimilation with this history. Elevated GLDH without elevated of other liver indicators is difficult to interpret. Consider primary gastrointestinal/ cloacal disease (including IPD, PDD?), lead toxicity.

Addendum 3: Culture and Sensitivity

Specimen: Faeces

Gram Stain: Occasional Gram positive cocci

Culture: Salmonella sp not detected from selective cultures.

Yersinia sp not detected from selective cultures.

No anaerobes isolated.

No significant bacteria isolated from aerobic culture.

Addendum 4: Repeat CBC and MBA. 4th November 2006

Haematology				
	Value	Reference Range		
Haematocrit	0.65 H L/L	0.42-0.54		
PCV	0.65 H L/L	0.42-0.54		
Thrombocytes	Normal			
WCC	14.0 x 10 ⁹ /L	10.0-20.0		
Heterophil	90 (50-75)	12.6 x 10 ⁹ /L		
Lymphocyte	8 (23-53)	1.1 x 10 ⁹ /L		
Monocyte	0% (<2)	< 0.1 x 10 ⁹ /L		
Eosinophil	1%	0.1 x 10 ⁹ /L		
Basophil	1% (<2)	0.1 x 10 ⁹ /L		
Plasma appearance	Normal			
Red cell and white cell morphology normal.				
	Biochemistry			
Serum glucose	14.6 L mmol/L	11.7-20.0		
Urea	2.1 L mmol/L	0.3-3.3		
Calcium	1.6 L mmol/L	2.1-3.0		
Protein, Total	16 L g/L	24-44		
Albumen	5 g/L	3-24		
Globulin	11 L g/L	21-38		
AST	50 L IU/L	65-168		
CK	215 H IU/L	88-361		
Cholesterol	2.7 L mmol/L	2.5-6.8		
Amylase	330 IU/L	239-564		
GLDH	3 H IU/L	<2		
Bile Acids Random	2 L umol/L	7-100		
Uric acid	1.01 mmol/L	0.11-0.71		
Sample appearance	Normal	Normal		

Low protein: effusion perhaps given reported clinical suspicion. Apparent haemoconcentration. Leukogram confirmed.

Addendum 5: Protein Electrophoresis. 8th November 2006

Total Protein	16 L g/L	24-44
Albumen, EPP	9 L g/L	12-32
Globulin, EPP	7 g/L	
Globul α 1	>1 g/L	<9
Globulin, α 2	>1 g/L	<9
Globulin, β 1	<1 g/L	<7
Globulin, β 2	<1 g/L	
Globulin, γ 1	2 g/L	
Sample Appearance	Normal	

General Interpretive Guidelines

α globulins:

mainly acute phase proteins (including fibrinogen and C-reactive protein) which rise
2 - 5 days after tissue injury

β globulins:

- include some acute phase proteins
- may include some immunoglobulins, especially during and IGM response

γ globulins:

- immunoglobulins
- account for most marked increase in total globulins

Monoclonal gammopathy:

- may reflect neoplasia of plasma cells or B lymphocytes
- may occasionally be seen with chronic inflammation (including erlichiosis and rarely FIP)

Polyclonal gammopathy:

- reflects inflammation, which may be due to infection, autoimmune disease, allergic disease or neoplasia (non-lymphoid or lymphoid)
- mainly gamma globulins but may extend into the beta region.

Reduced gamma globulins:

• may reflect congenital or acquired immunodeficiency (or failure of passive colostral transfer in a neonate), especially if accompanied by a normal albumin.

Comment: Hypoproteinaemia - may be secondary to high protein effusion, blood loss.

Addendum 6: VD and lateral radiographs 03/11/06.





Ventrodorsal Lateral

Addendum 7: Cardiologist's Report.

Presenting Signs/history

GI, renal and respiratory signs. Overt increase in cardiac silhouette on radiographs. Heart murmur present.

Cardiovascular Examination

General: responsive, but weak Heart Rate: 250 bpm+ Heart Rhythm: regular

Cardiac Auscultation: grade II/VI systolic murmur.

Echocardiographic Findings

The right atrioventricular valve is thickened and demonstrates restricted coaptation, consistent with right atrioventricular valve dysplasia. Marked right ventricular, and right atrial eccentric hypertrophy/dilation is present. Severe right atrioventricular valve regurgitation is present on colour Doppler assessment. Right ventricular diameter in diastole is 29 mm. The peak tricuspid regurgitant jet gradient of 16 mmHg is consistent with the expected normal pulmonary pressure range.

The left ventricular diameter in diastole measures 18 mm, the interventricular septum and left ventricular free wall measures 2.5 mm in diastole, the right ventricular free wall measures 1.5 mm in diastole.

Marked vena cava dilation (6.0 mm) and hepatic vein dilation (4.0 mm) is present, consistent with elevated right atrial pressures. No fluid present within the pericardial space.

Clinical Diagnosis

Lesion consistent with right atrioventricular tricuspid valve dysplasia (TVD), resulting in severe tricuspid regurgitation and hepatic/splanchnic congestion.

Comments

This is likely to be a congenital defect given the valve appearance and patient age. Infectious endocarditis of the right atrioventricular valve is very rare and unlikely to result in an evenly thickened valve. Post-mortem exam is required for a definitive diagnosis. There are no guidelines in terms of suggesting a hereditary mechanism for this lesion.

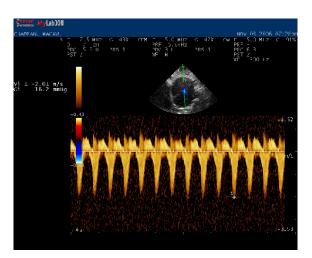
Screening (auscultation and echo) of offspring would be ideal if breeding within this line occurs in the future.

Recommendations

Frusemide may be of some benefit, but is also likely to result in some degree of dehydration, and potentially renal dysfunction and reduced cardiac output.

This lesion may be well-tolerated if CHF can be controlled, however the current status of this bird's general health suggests a poor prognosis.

Addendum 8: Ultrasound images.





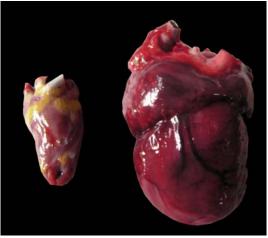


Figure 2: Macaw Heart with Comparative Galah Heart

Addendum 9: Histology Report.

Gross Pathology:.

An entire heart is submitted. The heart weight is 14g. The heart is cylindrical in shape with a uniform width of 31mm height of 47mm and a circumference of 95mm. There is marked dilation of the right atrium and ventricle due to marked dilation of the right A-V ostium.

Cassette A contains a section through the right atrium and two sections through the right ventricle. Cassette B contains a section through the left atrium and two through the left ventricle. Cassette C and D contain sections of liver that is firmer than normal.

Histopathology:.

Sections of right atrium and right ventricle are similar. There is extensive interstitial fibrosis with replacement of myofibres by fibrous connective tissue. Individual myofibre nuclei are moderately anisokaryotic. There is minimal inflammation.

In sections from the left ventricle there is minimal to mild interstitial fibrosis and mild anisokaryosis of ventricular muscle nuclei. Sections of left atrium appear normal.

In sections of liver there are dense bands of fibrous connective tissue extending randomly throughout the liver and often isolating the parenchyma into smaller nodules. In many of the fibrotic areas there is marked dilation of portal veins.

Cardiac dimensions are as follows:

Weight of heart: 13.2g: note this is minus sections taken for histology. Circumference of heart at level of right AV valve (22mm from apex): 91mm

Circumference of left AV valve: 22mm Circumference of right AV valve: 43mm

Left atrium: S - 17mm, T 15mm, H 13mm WT- 0.5 to 1mm Right atrium: S - 25mm, T 18mm, H 19mm WT- 0.5 to 1mm

Left ventricle: S - 6mm, T 23mm, H 9mm WT- 3.5 to 4mm lateral/ 6mm anterior/ 7mm posterior

Right ventricle: S - 18mm, T 23mm, H 12mm WT- 2.5 to 3mm

Interventricular septum: 3 to 4mm thick

Note: S- sagittal plane, T transverse plane, H horizontal plane, WT- wall thickness

Diagnosis:

Cardiomyopathy associated with right ventricular dilation and right A-V dysplasia Hepatic fibrosis

Comments:

Cardiomyopathy is associated with marked right ventricular dilation due to right atrioventricular valve defect. This has resulted in subsequent hepatic fibrosis due to congestive heart failure.