Left Atrioventricular Valve Insufficiency in a Juvenile Scarlet Macaw (*Ara macao*)

Tim Oldfield, BVSc, MACVSc (Avian Health)
Christina Leslie BVSc, DMU (Cardiac)

791 Welshpool Road East, Wattle Grove WA 6107(Oldfield)
PO Box 2165, Clarkson, WA 6030 (Leslie)

A variety of cardiac disorders, both congenital and acquired, has been recognized in psittacine birds. This presentation discusses the clinical signs, diagnosis, treatment and outcome of a juvenile scarlet macaw with a left atrioventricular valve defect.

A six week old scarlet macaw was presented with an apparently sudden onset of abdominal enlargement. The bird was one of a clutch of two, incubator hatched and hand-reared, in a facility that had experienced no significant infectious disease for over five years. Both birds had experienced similar growth rates to this point. On general examination the bird was found to be bright, alert and active. Heart rate was approximately 220 bpm, there were no audible respiratory sounds or detectable cardiac murmur. The abdominal distension was soft and fluidy.

Twenty mls of pale straw coloured fluid was drained from the abdominal cavity, subjective assessment suggested that this was perhaps a third of the total volume present. A blood sample was taken as well as a combined swab of conjunctiva, choana and cloaca for chlamydophila immunoflourescence. X-ray under general anaesthesia was considered an unacceptable risk. The bird was started on frusemide 2 mg/kg bid pending results of the tests.

Peritoneal Fluid	Chlamydia IF	Haematology	Biochemistry	Electrolytes
cell count 1.0 x 10^9/l	Negative	wcc 27 x 10^9/l	AST 309 U/l	Na 139 mmol/l
protein 5 g/l		pcv 40%	CK 306 U/l	K 3.0 mmol/l
S.G. 1.014		aniso 2+	UA 0.185 mmol/l	Cl 101 mmol/l
no bacteria		tsp 20g/l	GLU 13.8 mmol/l	
	heterophilia, hypoproteinemia, bland transudate			

Table 1: Results of samples taken at first presentation

Based on these results an ultrasound exam was scheduled. Seven days after first presentation the bird continued to appear relatively unaffected. It had gained weight, although slightly less than its cohort. Free abdominal fluid was still palpable. A conscious ultrasound was performed using a Mylab Vet 30 with 5 and 7 MHz linear probes via a ventromedian approach through the liver. At the same time the

unaffected cohort was examined for comparison.

Two dimensional echocardiography detected severe ascites and a small amount of pericardial effusion.

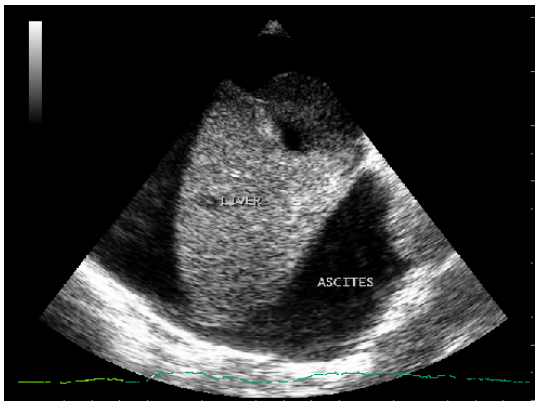

Figure 1: Ascites

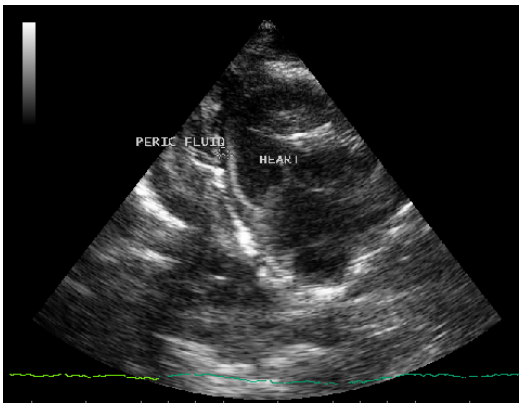

Figure 2: Pericadial effusion

In comparison with the normal bird there was left atrial dilation and a hyperechoic anterior left AV valve leaflet. Colour-flow Doppler revealed a large jet of left AV valve regurgitation (peak velocity 2.28m/sec and peak gradient 20.8mm Hg).

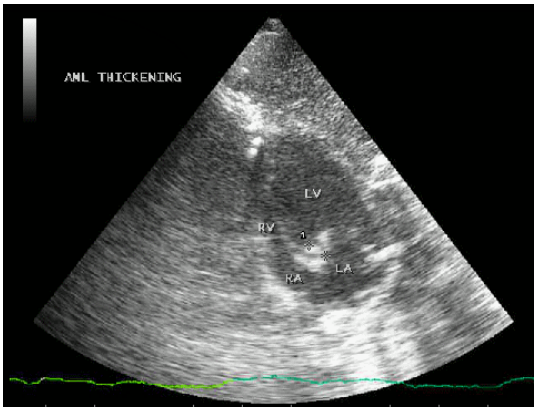

Figure 3: Left AV valve hypertrophy

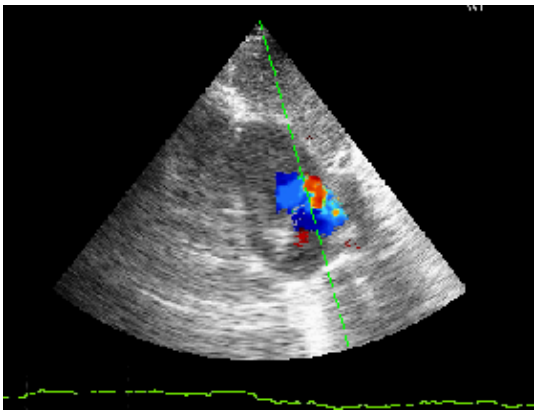

Figure 4: Left AV valve regurgitation

There was also a very small right to left flow just below the aortic valve. This was interpreted as a possible ventricular septal defect although it could not be reliably demonstrated on spectral Doppler. M-mode measurements indicated similar fractional shortening percentage in both birds suggesting a primary valvular disorder rather than a cardiomyopathy.

Echo Parameter	Affected Bird	Unaffected Nest-mate
left ventricle internal dimension diastole (mm)	7.8	6.5
left ventricle internal dimension systole (mm)	3.3	2.9
left ventricle wall diastole (mm)	2.4	2.3
left ventricular wall systole (mm)	3.3	3.2
interventricular septum diastole (mm)	2.4	2.2
interventricular septum systole (mm)	4.3	4.0
left atrium internal dimension (mm)	13.1	7.1
fractional shortening (%)	58	55
ejection fraction (%)	91	89
	marked left atrial dilation	

Table 2: Echocardiography measurements from first ultrasound examination

The bird was started on enalpril, 0.5mg/kg sid administered as a compounded liquid, and the frusemide dosing maintained. The bird remained stable and after seven days a repeat blood sample was taken. This demonstrated a marked improvement in serum protein and a reduction in the heterophilia. At this time a further 50 mls of transudate, as much as was possible, was removed from the abdominal cavity. Blood was again taken a further 7 days later showing a consistent stabilization of the haemogram and no evidence of diuretic induced potassium depletion. At this time there was no detectable abdominal fluid.

	Haematology	Biochemistry	Electrolytes
2 weeks post presentation	wcc: 19 x 10^9/l pcv: 44 % aniso: occ tsp: 37 g/l	CK : 574 U/l UA: 0.309 mmol/l ALB: 16 g/l GLOB: 21 g/l	Na: 139 mmol/l K: 3.1 mmol/l
3 weeks post presentation	wcc: 20 x 10^9/l pcv: 44 % aniso: occ tsp: 38 g/l		K: 5.6 mmol/l
Results Withn Normal Limits			

Table 3: Results of monitoring blood samples two and three weeks post presentation

Eight weeks after the first presentation a repeat echocardiography was performed. At this time, other than weighing slightly less, the bird was clinically indistinguishable from its nest mate. There was no detectable ascites and the pericardial effusion had resolved. Mild left atrial dilation was subjectively assessed as still being present but unfortunately the patient was considerably less compliant than at the first examination and m-mode measurements were unable to be obtained. The anterior leaflet of the left AV valve had a thickened, hyperechoic and mildly nodular appearance. Colour-flow Doppler again revealed left AV valve regurgitation which was now assessed as mild (velocity 1.21 m/s and gradient 5.8 mmHg). At this examination it was clearly evident that there was a wide jet of aortic valve regurgitation wrapping down the left ventricular free wall. There was no evidence of a septal defect and the right/left flow seen at the initial examination appears to have been due to a small high velocity aortic regurgitation. Cardiac contractility appeared to be normal.

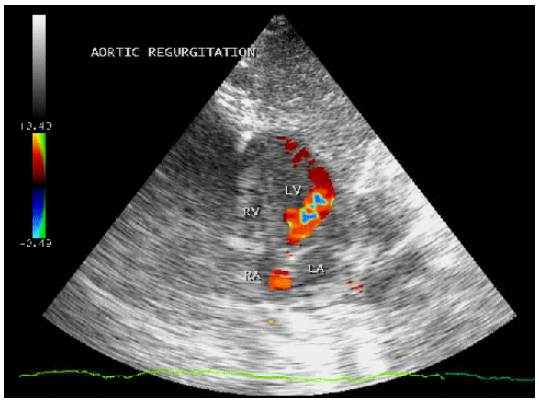

Figure 5: Aortic valve regurgitation

The frusemide was reduced to once daily and then withdrawn after a further four weeks. The enalpril was continued for four months and then withdrawn because of the difficulty of medicating a six month old fledged aviary macaw. At this stage the bird had no clinical or behavioural signs to distinguish it from other fledgling macaws in the same aviary. One month after the withdrawal of medication there had been no recurrence of clinical signs.

Comments

The marked right-sided symptoms this bird initially presented with are interesting in light of the left-sided changes seen on echocardiography. This pattern of disease has been noted previously in birds. Ascites and hepatomegally can be consequential to primary left sided failure once pulmonary hypertension becomes severe enough to increase the right ventricular workload. The rapid improvement in clinical signs once frusemide was commenced may indicate that there was significant pulmonary oedema. However at no time were there any audible respiratory changes. Radiographs may have been informative, particularly at first presentation but understandably the owner was reluctant to permit general anaesthesia. Despite the low number of mammalian type nephrons in the avian kidney experience has shown that diuretics can be effective in relieving fluid accumulations, particularly pulmonary oedema and pericardial effusions. Accumulations in the coelom do not appear to be so responsive.

The development of aortic valve incompetence was considered to be most likely due to the left atrial dilation distorting the proximal aorta.

Both the frusemide and the enalpril appeared to be well tolerated and effective at relatively conservative dose rates.

It is possible that there was a congenital cause for this bird's cardiac abnormalities. Twelve months earlier a chick from the same parent birds and of similar age became ill and died before a diagnosis could be made. Autopsy and histological examination showed ascites, an enlarged liver with perihepatic effusion and centrilobular fibrosis and moderate diffuse pulmonary congestion. No significant histological abnormalities were detected in other organs including the heart and there was no evidence of inflammatory disease or infection. The changes were considered to be consistent with congestive heart failure due to a possible functional or vascular anomaly.

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