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Congestive heart failure in a veiled chameleon (*Chamaeleo calyptratus*): A case report

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Abstract: A two-year-old male veiled chameleon (*Chamaeleo calyptratus*) was referred for a gular oedema and bilateral blepharoedema. The echocardiography revealed a ventricular hypertrophy, pericardial effusion, and valvular regurgitation of the right atrioventricular valve. Treatment with hydrochlorothiazide, enalapril, and carvedilol was commenced. Within 3 weeks of treatment, the valvular regurgitation was noticeably decreased. In the 4th week of treatment, the echocardiography revealed a reduction in the myocardium hypertrophy. After an additional month of home treatment, the patient was presented with anorexia and decreased activity. Despite the supportive care, the patient died. The histopathology revealed mild to moderate fibrosis of the epicardium. Moderate to severe fibrosis, degeneration of the myofibrils, fatty atrophy, interstitial oedema and mild calcification was seen in the atria. The tunica intima, media and adventitia of the major cardiac vessels were moderately fibrotic, swollen and interfused by myxoedema. The kidney histopathology revealed moderate sclerosis and atrophy of the glomeruli, vacuolation of the tubular epithelium, fibrosis, and infiltration of the leucocytes in the interstitium. The therapeutic protocol with hydrochlorothiazide, ACE inhibitor enalapril and β-blocker carvedilol reduced the myocardium hypertrophy and the valvular regurgitation; however, the prolonged use of diuretics jeopardized the renal function in our patient. Frequent blood analyses are necessary using diuretics in reptile patients.

Keywords: ace inhibitors; β-blockers; diuretics; echocardiography; reptile cardiology

New information dealing with the laboratory diagnosis, emerging infectious diseases and clinical techniques have recently been published on reptiles (Manire et al. 2018; Divers and Stahl 2019; Jacobson and Garner 2021a; Jacobson and Garner 2021b), including several papers upon cardiac diseases and clinical cardiology in reptiles (Schilliger et al. 2010a; Schilliger et al. 2010b; Schilliger et al. 2016). However, detailed reports of echocardiography and the treatment of cardiac illnesses in reptiles are still scarce (Mitchell 2009; Simone-Freilicher et al. 2015; Silverman et al. 2016; Schilliger and Girling 2019).

This case report describes a diagnostic approach and the treatment protocol of a congestive heart failure in a veiled chameleon (*Chamaeleo calyptratus*).

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Case presentation

A 2-year-old male veiled chameleon (*Chamaeleo calyptratus*), 290 g in body weight, was referred to the clinic for a gular oedema and bilateral blepharoedema.

The clinical examination revealed a gular oedema, swelling of the helmet, and bilateral blepharoedema (Figure 1). Both eyes were open, and the vision was not compromised. The skin in the lower part of the eyelids showed a deep abrasion, caused by the friction of the swollen tissue on the zygomatic arch. The differential diagnosis included hyperplasia of the lacrimal glands, neoplasia, and a cardiogenic oedema. Vitamin A insufficiency was not included to the differential diagnosis, for the anamnesis revealed adequate vitamin and mineral integration. A blood sample for haematology and plasma chemistry was taken from the ventral coccygeal vein. The plasma activities of the aspartate aminotransferase (AST) and creatine kinase (CK) were extremely high (Table 1). Dorsoventral and

Figure 1. Bilateral blepharoedema
Chamaeleo calyptratus, bilateral blepharoedema and oedema of the helmet

Table 1. Blood profile of the male veiled chameleon with congestive heart failure

Parameters	Units	A	B	Gibbons et al. (2019)	Coufalova (2013)
WBC	10^9/l	9.50	17.00	6.30 (1.20–21.0)	–
RBC	10^{12}/l	1.09	0.81	–	–
PCV	l/l	0.32	0.24	0.24 (0.12–0.37)	–
Heterophils	10^9/l	3.32	10.54	2.35 (0.50–8.32)	–
Lymphocytes	10^9/l	3.32	1.70	2.18 (0.07–10.80)	–
Monocytes	10^9/l	0.19	0	–	–
Eosinophils	10^9/l	0	0	–	–
Basophils	10^9/l	0.48	0.17	–	–
Azurophils	10^9/l	2.19	4.59	0.5 (0–2.75)	–
Total protein	g/l	63.90	69.10	64.00 (35.00 –120.00)	59.58 ± 11.87
Albumin	g/l	31.70	33.00	31.00 (14.00–42.00)	33.37 ± 6.95
Glucose	mmol/l	16.1	17.5	14.88 (6.89–24.46)	13.03 ± 0.71
Uric acid	µmol/l	164.00	6 276.00	333.10 (0 – 1302.61)	105.92 ± 64.71
ALP	µkat/l	0.23	< 0.08	–	–
ALT	µkat/l	< 0.02	< 0.02	–	–
AST	µkat/l	10.36	12.96	6.63 (1.55–16.15)	4.03 ± 0.91
CK	µkat/l	265.43	107.37	31.28 (0.08 –148.71)	23.30 ± 8.60
Ca	mmol/l	3.33	4.63	2.97 (2.17–3.62)	3.77 ± 1.91
P	mmol/l	2.00	2.78	2.71 (1.42–5.20)	3.47 ± 0.62
Bile acids	µmol/l	< 0.50	2.00	–	–

A – Results from the day before the treatment had been started; B – Results received 48 h before the patient died
ALP = alkaline phosphatase; ALT = alanine aminotransferase; AST = aspartate aminotransferase; Ca = calcium; CK = creatine kinase; P = phosphorus; PCV = packed cell volume; RBC = red blood cells; WBC = white blood cells

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laterolateral radiographic examinations were performed, with unremarkable results.

An echocardiography was performed from both the right and left axillary windows (Gustavsen et al. 2014; Silverman et al. 2016), using an Aloka Prosound Alpha 7 ultrasound machine (Hitachi Aloka Medical, Tokyo, Japan) equipped with a phased array 2–9 MHz cardio probe. From the modified long-axis view of the left atrioventricular junction, the myocardium showed increased echogenicity and decreased contractility. The thicknesses of *cavum pulmonale* and *cavum venosum* walls were 4.3 and 3.7 mm, respectively (Figure 2). A 2 mm film of anechoic fluid was detected between the myocardium and pericardium. On the right trans-arterial long axis view, a turbulent Doppler flow (mosaic pattern) was detected at the level of the right atrioventricular valve, confirming atrioventricular valve regurgitation (Figures 3 and 4). The echocardiography revealed congestive heart failure, ventricular hypertrophy and valvular regurgitation (Figures 2–4).

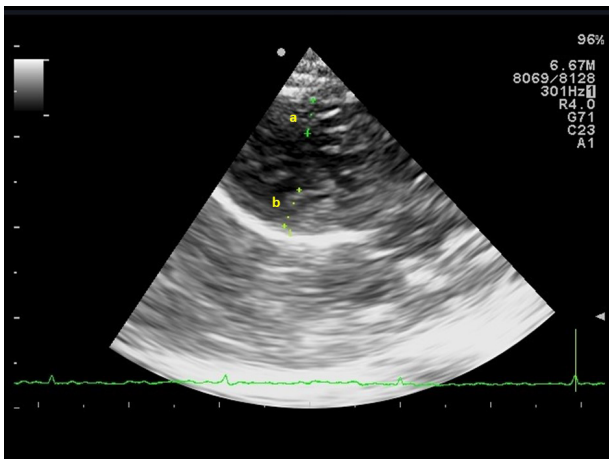

Figure 2. Ventricular hypertrophy
Modified long-axis view of the left atrioventricular junction. The myocardium showed increased echogenicity and decreased contractility; a – the wall of the *cavum venosum* was 3.7 mm; b – the wall of the *cavum pulmonale* was 4.3 mm

The patient was stabilised over a period of one month at the clinic, using carnitine (Vigosine Liq; Ceva Animal Health Slovakia, Bratislava, Slovakia), a multivitamin compound with vitamin A (CombiSol-V; Neospark, Telangna, India) and a non-specific immunostimulant (Imunoglukan PH4 sirup; Pleuran s.r.o., Bratislava, Slovakia), combined with regular assisted feeding. The congestive heart failure, ventricular hypertrophy and valvular regurgitation were treated using the diuretic hydrochlorothiazide (1 mg/kg p.o. q72 h, Hydrochlorthiazid, Zentiva, Prague, Czech Republic) in order to decrease the oedema and pericardial effusion, and supporting the ventricular filling using the ACE inhibitor enalapril, (0.5 mg/kg p.o. q48 h; Enap, Krka, Slovenia), combined with the β-blocker carvedilol (0.2 mg p.o. q48 h; Coryol, Krka, Slovenia). Within three weeks of treatment, the valvular regurgitation was noticeably decreased. In the fourth week of treatment, the echocardiography revealed a reduction in the myocardium hy-

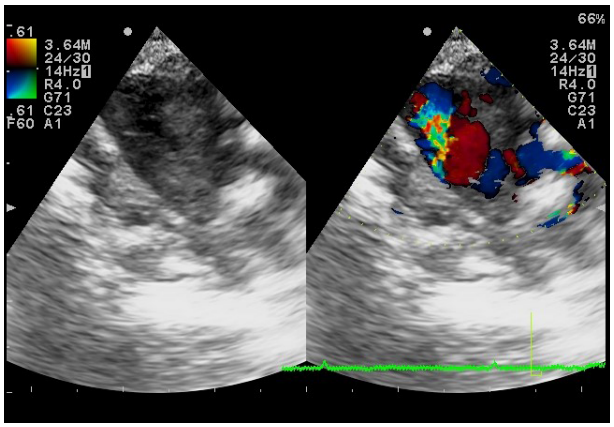

Figure 3. Turbulent Doppler flow in the right atrioventricular valve
Modified long-axis view of the right atrioventricular junction. Turbulent Doppler flow (mosaic pattern) is visible at the level of the right atrioventricular valve and *cavum venosum*, the extent of the turbulence was considered abnormal

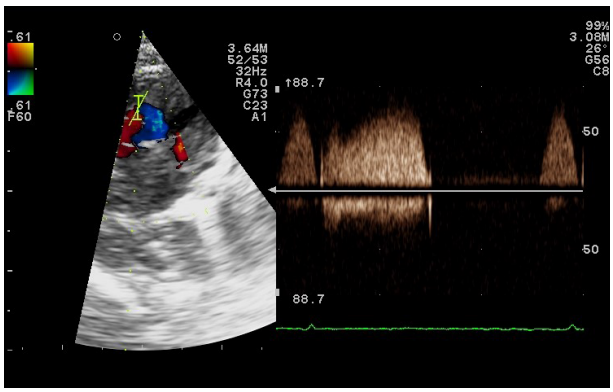

Figure 4. Continuous wave Doppler of the right atrioventricular valve
Modified long-axis section of the right atrioventricular junction. Continuous wave Doppler of the right atrioventricular valve at the moment of the first echocardiography interrogation. The upper systolic flow (after the QRS complex of the ECG) shows flow movement from the *cavum venosum* to the single atria demonstrating valve insufficiency

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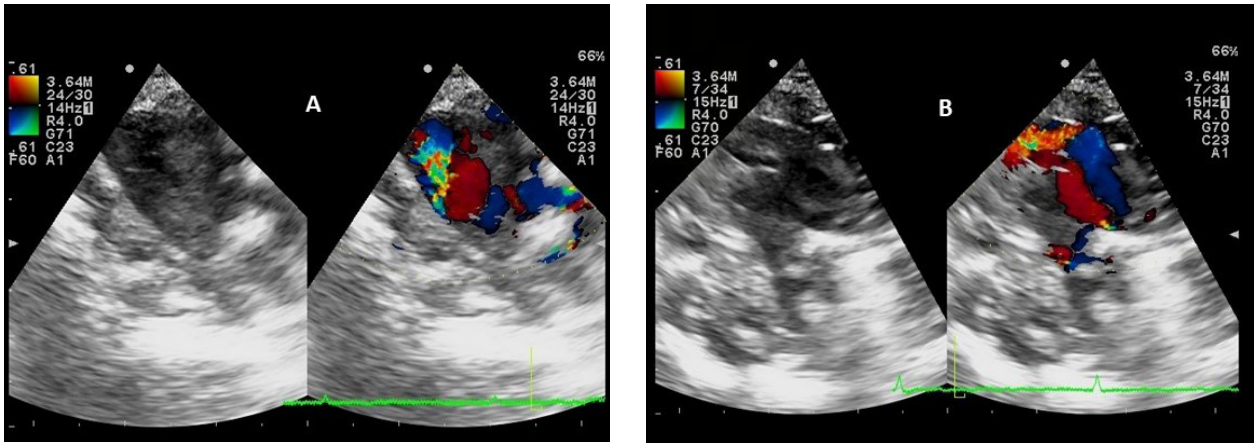

Figure 5. Turbulent Doppler flows recorded at the beginning and after one month of therapy
Modified long-axis view of the left atrioventricular junction. Comparison between the turbulent Doppler flows recorded at the beginning and after one month of therapy. (A) Same image as in Figure 3. (B) Colour Doppler of the ventricle, modified long-axis view of the left atrioventricular junction. Notice how the turbulent Doppler flow (mosaic pattern) is decreased in (B)

Table 2. The treatment protocol used for the congestive heart failure in the male veiled chameleon

Drug	First month	Second month	Rationale
Hydrochlorothiazide	1 mg/kg p.o. q72 h	1 mg/kg p.o. q72 h	diuretic, decreases the peripheral oedema and the pericardial effusion
Enalapril	0.5 mg/kg p.o. q48 h	0.5 mg/kg p.o. q72 h	ACE inhibitor, decreases the arterial pressure and, therefore, the cardiac load
Carvedilol	0.2 mg p.o. q48 h	0.2 mg p.o. q72 h	β-blocker, decreases the renin secretion, reducing the heart oxygen demand by lowering the extracellular volume and increasing the oxygen-carrying capacity of the blood

pertrophy, and an almost complete resolution of the atrioventricular valve regurgitation (Figure 5). The patient was discharged with enalapril (0.5 mg/kg p.o. q72 h; Enap, Krka, Slovenia) and carvedilol (0.2 mg p.o. q72 h; Coryol, Krka, Slovenia) (Table 2). Weekly controls were scheduled. On each clinical examination, the patient was found to be stable, furthermore, the blepharoedema decreased.

One month later, the patient was presented with apathy and anorexia. The biochemical and haematological analyses revealed leucocytosis with heterophilia and azurophilia. The plasma concentration of the uric acid (UA), as well as the AST and CK activities were extremely high (Table 1). Despite the supportive treatment, the patient died within the following 48 hours. The necropsy revealed a cardiomegaly with pericardial white adhesions (Figure 6), a marbled liver parenchyma, marked distension of the gall bladder and attenuation of the stomach wall. The pleuroperitoneal membrane and surfaces of all the organs were covered with multiple white-yellow deposits. The histopathology of the eyelids revealed moderate orthokeratotic hyperkeratosis with moderate hyperplasia, infiltration of lymphocytes, plasma cells and macrophages in the superficial dermis and myxoedema in the deep dermis. The histopathology of the heart revealed mild to moderate fibrosis of the epicardium, and moderate to severe fibrosis, degeneration of the myofibrils, fatty atrophy, interstitial oedema and mild calcification of the atria. The tunica intima, media and adventitia of the major vessels were moderately fibrotic, moderately swollen and interfused by myxoedema with a mild infiltration of lymphocytes and macrophages. The endocardium of the ventricle was unremarkable. The histopathology of the kidneys revealed moderate sclerosis and mild to moderate atrophy of the

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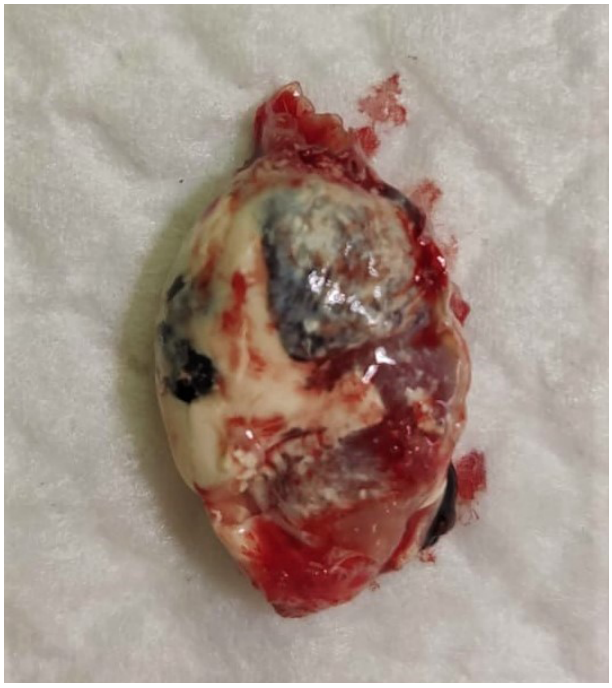

Figure 6. Necropsy
Heart during necropsy revealing cardiomegaly with pericardial white adhesions

glomeruli. The epithelium of the tubuli was moderately vacuolated. The tubular lumen was normal. The interstitial tissue was moderately fibrotic with a mild infiltration of lymphocytes, macrophages and plasma cells.

DISCUSSION

A number of causes of gular oedema and blepharoedema have been described in reptiles. In our case, retrobulbar neoplasia and abscesses were excluded radiographically, and the simple hypertrophy of the lacrimal glands could not explain the oedema of the helmet and throat. In lizards, a vitamin A deficiency causes squamous metaplasia of the lacrimal and mucus-secreting epithelium of the eye. The resulting flattened keratinised epithelium continually desquamates accumulating keratin debris. The accumulated keratin blocks the lacrimal, salivary, and mucous glands, and accumulates under the eyelids causing swollen eyes (Miller et al. 2001). In a panther chameleon, a vitamin A deficiency caused gular swelling and blepharospasm (Ferguson et al. 1996). In the present case, however, a vitamin A deficiency was not included in the differential diagnosis due to the anamnestic protocol.

A blepharoedema and gular oedema can result from an inefficient venous flow, causing stasis and increased intravascular pressure leading to the leakage of fluid from the vessels to the tissue. Clinical signs in mammals include a symmetric, non-painful, pitting oedema of the head, neck, and forelimbs (Mulz et al. 2010).

Cardiac related illnesses have been reported to cause oedemas in the head and throat of lizards and snakes (Mitchell 2009). In the present case, the echocardiography revealed congestive heart failure, ventricular hypertrophy and valvular regurgitation. On the right trans-arterial long axis view, a turbulent Doppler flow (mosaic pattern) was detected at the level of the right atrioventricular valve. Despite being a common finding in reptilian hearts (Gustavsen et al. 2014; Silverman et al. 2016), this turbulence was considered abnormal for its intensity and extension. The thickness of the wall of the *cavum pulmonale* and the *cavum venosum* were 4.3 and 3.7 mm, respectively, despite the lack of references, these values were considered pathological by the authors, for they abundantly exceeded those seen in other healthy specimens during routine examinations.

Silverman et al. (2016) reported a moderate to scant amount of pericardial fluid in 62.5% of examined bearded dragons (*Pogona vitticeps*), which is in contrast with the data regarding the green iguana (*Iguana iguana*), where a pericardial effusion is considered pathological (Gustavsen et al. 2014). Reliable data regarding the echocardiographic anatomy of chameleons is still lacking. In the experience of the authors, however, a film of more than 1 mm of pericardial fluid could be considered pathological in adult veiled chameleons (*Chamaeleo calyptratus*), particularly if coupled with other cardiologic symptoms.

In the present case, the congestive heart failure was treated with a combination of hydrochlorothiazide, enalapril and carvedilol (Table 2). Diuretics, in congestive heart failure, are meant to decrease the peripheral oedema and the pericardial effusion. Hydrochlorothiazide is a commonly used diuretic, which inhibits the reabsorption of Na^+ in the distal tubule of the nephron. Furosemide would have been another valid alternative, as significant effects were recently reported in lizards (Parkinson and Mans 2018). Enalapril decreases the level of angiotensin II reducing the blood pressure. The decreased arterial pressure eases the blood flow reducing the myo-

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cardial load, which was the primary cause of the ventricular hypertrophy detected during the echocardiography. Enalapril successfully inhibited the angiotensin I conversion in an American alligator [*Alligator mississippiensis*; Silldorff and Stephens (1992)], and briefly managed congestive heart failure in a spiny-tailed monitor [*Varanus acanthurus*; Schilliger and Girling (2019)]. The effectiveness of β-blockers in reptiles is still unknown. Carvedilol (β-blocker) is advised in the treatment of congestive heart failure (Gordon 2010), and atenolol administration significantly reduced the resting heart rate and the range of the baroreceptor reflex in a juvenile green iguana [*Iguana iguana*; Hernandez et al. (2011)]. B-blockers are mainly indicated in the treatment of hypertrophic cardiomyopathy and supraventricular arrhythmias in small mammals.

The treatment protocol in the present case proved to be effective in reducing the valvular insufficiency and myocardial hypertrophy. However, the severe increase in the uric acid concentration recorded two months after starting the treatment can easily be related to diuretic-induced dehydration. Hyperuricemia seldom ensues before the majority of the renal function has been lost (Divers and Innis 2019). The plasma activities of AST and CK increase in renal failure as well as in cardiac muscle diseases (Divers and Innis 2019; Schilliger and Girling 2019).

In conclusion, the echocardiography proved to be a useful tool for the diagnosis of congestive heart failure in a veiled chameleon. The therapeutic protocol with hydrochlorothiazide, the ACE inhibitor enalapril and β-blocker carvedilol was successful in reducing the myocardium hypertrophy and valvular regurgitation. To prevent the high risk of severe dehydration and chronic kidney disease in patients, frequent clinical controls with regular analyses of biochemistry and haematology is recommended.

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Conflict of interest

The authors declare no conflict of interest.

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