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Case Report

Systemic arterial hypertension secondary to chronic kidney disease in two captive-born large felids

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Abstract Systemic arterial hypertension (SHT) has been widely described in the domestic cat (*Felis catus*). In these feline patients, SHT is considered as the most common vascular disorder of middle-aged to older animals, and secondary SHT related to chronic kidney disease (CKD) represents the most common form of the disease.

We describe here the first two cases of spontaneous SHT in large felids, i.e. one 18-year old, 34.4 kg, male North-Chinese leopard (*Panthera pardus japonensis*, case #1) and one 20-year old, 28.7 kg, female snow leopard (*Panthera uncia*, case #2), both captive-bred and previously diagnosed with CKD. Both animals underwent complete echocardiographic examination under general anesthesia due to abnormal cardiac auscultation (heart murmur and/or gallop sound), and recurrent lethargy in case #1. The combination of left ventricular remodeling with moderate aortic regurgitation of high velocity was highly suggestive of SHT, which was confirmed by indirect blood pressure measurement (systolic arterial blood pressure of 183 mmHg for case #1 and 180 mmHg for case #2). Amlodipine was prescribed (0.35—0.70 mg/kg/day orally) for 31 and 6 months respectively after the initial diagnosis. In case #1, concurrent amlodipine and benazepril treatment was

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associated with decreased heart murmur grade and reduced aortic insufficiency severity. These reports illustrate that, similarly to domestic cats, SHT should be suspected in old large felids with CKD and that amlodipine is a well-tolerated antihypertensive drug in these species.

Abbreviations

ACEI	angiotensin-converting enzyme inhibitor
BP	arterial blood pressure
CKD	chronic kidney disease
DBP	diastolic arterial blood pressure
IVS	interventricular septum
LV	left ventricle
SBP	systolic arterial blood pressure
SHT	systemic arterial hypertension
TOD	target organ damage

Case 1

An 18-year old, 34.4 kg, captive-born male North-Chinese leopard (*Panthera pardus japonensis*) was referred for echocardiography owing to recurrent lethargy, a heart murmur, and a gallop sound on cardiac auscultation. The animal was monitored for chronic kidney disease (CKD) over the 6 previous months and was regularly anesthetized for renal function monitoring and intravenous fluid therapy. The initial diagnosis of CKD was based on high plasma urea and creatinine values (50 mmol/L and 430 μmol/L; reference range 5.2—16.0 mmol/L and 48—295 μmol/L, respectively) [1], clinical signs (e.g. dehydration, sublingual, and palatine ulcerations), abnormal renal ultrasonography (hyperechoic cortex and medulla with lack of cortico-medullary differentiation), and isosthenuria (urine specific gravity of 1.0125), without any remarkable finding on dipstick analysis. No abnormalities on funduscopic examination were noticed during these interventions. Plasma urea and creatinine levels remained high throughout the 6 months of monitoring, and oral angiotensin-converting enzyme inhibitor (ACEI) treatment (benazepril[d]; 0.29 mg/kg/day) was started one week prior to echocardiography.

The animal was anesthetized using medetomidine[e] (49 μg/kg IM) and ketamine[f] injected 20 min later (3.8 mg/kg IM). Cardiac auscultation confirmed a grade 4/6 left basal diastolic heart murmur with a gallop sound. The animal was placed in lateral recumbency and transthoracic echocardiographic examination with continuous electrocardiogram monitoring performed using an ultrasound unit[g] equipped with 5S (2.0—5.0 MHz) and 7S (2.4—8.0 MHz) phased-array transducers, in accordance with international recommendations and as previously described in large felids [2,3].

Echocardiographic and Doppler variables are presented in Table 1. Two-dimensional examination showed hyperechoic focal myocardial lesions of the left ventricle (LV), more pronounced for the interventricular septum (IVS) than the LV free wall. A mild bulge of the sub-aortic IVS was also observed (Fig. 1A), without any secondary LV outflow tract obstruction. Additionally, two-dimensional and color-flow Doppler modes revealed a prominent aortic base with a ratio between the proximal ascending aorta diameter to the aortic annulus diameter of 1.28, which was associated with aortic insufficiency of important colorimetric extension (Fig. 1B and C). The aortic regurgitation was characterized by a vena contracta width (i.e. the smallest flow diameter immediately below the flow convergence region) of 5.3 mm, a pressure half-time of 235 ms and a regurgitant jet height to outflow tract width (at the junction of the LV outflow tract and aortic annulus) of 59%. According to the recommendations from the European Association of Cardiovascular Imaging for humans, these findings were consistent with a moderate aortic regurgitation [4]. The aortic regurgitation was also characterized by an early-diastolic peak velocity of 5.34 m/s as assessed by continuous-wave Doppler mode (Fig. 1D), with a corresponding estimated mean arterial pressure of at least 114 mmHg using the modified Bernoulli equation ($4 \times 5.34^2 = 114$ mmHg). The aortic

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[e] Medetor, Virbac France, 06517 Carros, France.
[f] Ketamine 1000, Virbac France, 06517 Carros, France.
[g] Vivid i, General Electric medical system, Waukesha, WI, USA.