HYPERTROPHIC CARDIOMYOPATHY IN A DROMEDARY CAMEL

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INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is hypertrophy of the left ventricle in absence of causative systemic or cardiac disease. In animals with HCM there is a decreased cardiac output. Usually the HCM affects mature males, but subclinical disease is very common[1]. In these animals only syncope and/or exercise intolerance is observed. Sudden death may occur[1].

In small animals, HCM is more common in the cat and rare in the dog[1]. However in large animals, the primary disease has been reported in the pig[2] and two cases in the cow[3]. To the author´s knowledge, HCM has not been reported in the horse and the present case would be the first description in the camel.

CASE REPORT

The patient was a dromedary camel (Camelus dromedarius), castrated male, 9 years old and was a part of a tourist farm placed in Gran Canaria. The dromedary no history of previous diseases had been noted. In the previous six months, several episodes of syncope had been observed after moderate exercise and at rest. In the prior week to presentation, the animal had five episodes of syncope which had a sudden onset and lasted 30 to 45 secs. The camel presented unconscious, lateral recumbency, Cheyne-Stokes breathing and slight convulsions with pale mucosae. Sphincter anal relaxation or micturition during syncopes was not observed. The animal recovered quickly after a brief period in a daze.

On physical examination, the animal without syncopal signs was in good body condition, had normal pale pink mucous membranes, rectal temperature of 37,4° C, respiratory rate 8 breaths/min. and heart rate 50 beats/min. (normal data: 5-10 breaths/min and 35-50 beats/min[4]). The pulse, taken on femoral artery, was weak and irregular. The camel did not exhibit respiratory distress. The first heart sound was louder and a grade III/VI mitral murmur was auscultated on 4th intercostal space in the middle line from shoulder to elbow. Neurological, abdominal and muscle-skeletal explorations resulted seemingly normal and oedemas were not noted.

Light decrease on appetite or water intakes were observed in the patient several weeks previous to the euthanasy.

In relation to normal ECG values obtained in dromedaries[3], sinus arrhythmia with electrical activity in QRS complex, and QRS axis of 10° were observed. The echocardiogram showed an enlargement of the left ventricular free wall (75 mm in systole and 69 mm in diastole), an enlargement of the interventricular septal (71 mm in systole and 65 mm in diastole) and fractional shortening was 24%. Echocardiographical values obtained in 4 adult healthy dromedaries were: left ventricular free wall (41 mm in systole and 28 mm in diastole), interventricular septal (32 mm in systole and 22 mm in diastole) and fractional shortening, 42%.

The haematological findings were: WBC, 8.300/µL; RBC, 7.600.000/µL; hemoglobin, 13.6 g/dL; PCV, 32%; platelets, 280.000/µL; serum total proteins, 75 g/L; serum urea, 2.8 mmol/L; creatinine, 132 µmol/L; glucose, 6.3 mmol/L; calcium, 1.9 mmol/L; potassium, 4.87 mmol/L; sodium 140 mmol/L; alkaline phosphatase, 57 U/L; AST, 28 U/L; ALT, 25 U/L; GGT, 30 U/L; LDH, 195 U/L and gluthatione peroxidase activity, 183 IU/g Hb. These values are included within normal ranges for camels[4,6,7]. Trypanosoma evansi investigation using the serological CATT/T.evansi (Card direct agglutination test, Institute of Molecular Biology and Institute of Tropical Medicine, Belgium) and stained blood smears by Giemsa were negative.

The tentative diagnosis was idiopathic HCM and the owner elected to euthanize the camel.

Fig. 1. Cross-sectional slice of the dromedary heart showing proportionate hypertrophy of the ventricle.

On necropsy the heart had a globose shape and was firm on palpation. On cross-section the left ventricular free wall and the interventricular septum were thickened with a decreased left ventricular lumen size (Fig. 1). The left ventricular free wall measured 78 mm at anterior papillary muscle level and 69 mm at base level. The interventricular septum measured 64 mm. The left atrium was slight dilated with scattered white patches areas. The coronary arteries were grossly normal and no congenital malformations were seen. Histologic examination demonstrated hypertrophy of myocardial fibers with vesicular nuclei and diffuse interstitial fibrosis. However, disorganization of cardiac myocytes was minimal. Intramural arteries showed thick walls and narrowed lumen due mainly to proliferation of smooth muscle and fibrous tissue in the intima (Fig 2). The valves and ventricular wall adjacent to valves showed fibrosis and calcification at endocardial level.

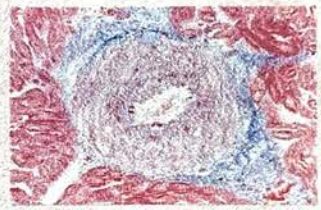

Fig.2. Abnormal intramural coronary artery showing thick walls and narowed lumen.

No other organs showed either macroscopic or microscopic alterations.

Histological findings in HCM revealed substancial morphologic variability among the species[8]. The hipertrophy of myocardial fibers and interstitial fibrosis represent a diffuse cardiomyopathic process. Endocardial fibrosis and calcification are described in a high percent of human and bovine HCM patients[9,3]. Maron et al (1986) postulated that the abnormal intramural coronary arteries might constitute an independent marker of HCM and a component of the cardiomyopathic process.

Hypertrophy of the left ventricular free wall and/or interventricular septum causes diastolic dysfunction. Increased myocardial stiffness and decreased lumen size due to hypertrophy, mitral regurgitation and systolic anterior motion of the mitral valve are contributing to the obstructive cardiac syncope[1]. From the initial episode of syncope, six months previously, the camel only showed new syncope without other clinical sings.

In HCM, the electrocardiogram may be normal or conduction disturbances and arrhythmias can be noted. The echocardiographic findings were consistent with a clinical diagnosis of HMC. The necropsy confirmed the clinical findings and showed a cardiac symmetric hypertrophy. Finally, the dromedary was euthanized and only the occult phase of HMC could be appreciated.

REFERENCES

1. Meurs KM. Myocardial diseases. In: Morgan RV, editor. *Handbook of small animal practice*. 3rd. Saunders, Philadelphia, 1997; 103-112.
2. Chiu YT, Liu SK, Teau HL, et al. Pathological studies on hypertrophic cardiomyopathy in pigs. J Chinese Soc Vet Sci 1994; 20: 126-133.
3. Machida N, Kiryu K, Nakamura T, et al. Two necropsy cases of hypertrophic cardiomyopathy in Holstein cattle. *J Vet Med Sci* 1996; 58: 929-932.
4. Higgins AJ, Kock RA. A guide to the clinical examination, chemical restraint and medication of the camel. In: Higgins AJ, editor. *The camel in health and disease*. Bailliere Tindall, London, 1986; 21-40.
5 Rzezkhas A, Szabuniewicz M. The electrocardiogram of the camel (Camelus dromedarius). Zbl Vet Med A 1977; 24: 277-286.
6. Bengoumi M, Faye B, Farge F, et al. Clinical enzymology in the dromedary camel (Camelus dromedarius). Part 1. Enzyme activities and distribution of AST, ALT, GGT, AP and LDH in liver, kidney, muscle, myocardium and blood. *J Camel Pract Res* 1997; 4: 19-23.
7. Nyang´Ao JWN, Olaho-Mukani W, Maribet JM, et al. A study of some haematological and biochemical parameters of the normal dromedary camel in Kenya. *J Camel Pract Res* 1997; 4: 31-33.
8. Liu SK, Roberts WC, Maron BJ. Comparison of morphologic findings in spontaneously occurring hypertrophic cardiomyopathy in humans, cats and dogs. *Am J Cardiol* 1993; 72: 944-951.
9. Davies MJ, Pomerance A, Teare RD. Pathological features of hypertrophic obstructive cardiomyopathy. *J Clin Pathol* 1974; 27: 529-535.
10. Maron BJ, Wolfson JK, Epstein SE, Roberts WC. Intramural ("small vessel") coronary artery disease in hypertrophic cardiomyopathy. *J Am Coll Cardiol*, 1986 8: 545-557.