Opis przypadku *Case report*

Atypical manifestation of a multifocal myocardial necrosis associated with selenium deficiency in a dromedary (*Camelus dromedarius*) in Europe: A case report

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Summary

Various species of camelids are gaining popularity as domesticated animals in Europe, including Poland. Due to different environmental conditions, they may be subject to atypical diseases, including infectious diseases and nutritional deficiencies. This paper describes a case of an eight-year-old female dromedary camel (*Camelus dromedarius*) presenting with signs of right-sided heart failure. The clinical diagnosis and treatment were accompanied with cardiac pathological and histopathological examination. Multifocal myocardial necrosis, most probably secondary to selenium deficiency, was diagnosed. This is the first description of such an atypical picture in a dromedary camel.

Keywords: cardiomyopathy, myocardium, myocardial necrosis, nutritional deficiencies, selenium

For centuries, camels have served as domesticated animals in Asia and Africa. In recent years, they have also been gaining popularity as domesticated animals in several European countries. Due to the migration, they may suffer from various atypical diseases, including infectious diseases and nutritional deficiencies (12). As they are a species not native to European countries, the diagnostic and therapeutic process is often challenging.

The current paper describes a case of a dromedary (*Camelus dromedarius*) presenting with signs of severe right-sided heart failure combined with selenium deficiency.

Case description

An 8-year-old female dromedary (*Camelus dromedarius*) was referred to a veterinary clinic because of a 2-3-week history of appetite loss, apathy and decreased activity. Previously, she had had an episode of bloating that had resolved after the administration of spasmolytic drugs and received antiparasitic treatment (fenbendazole) four weeks before admission.

Clinical examination revealed wet, pink mucous membranes, a normal, strong pulse and normal intestinal peristalsis. Oral examination showed a slightly abnormal dental development requiring correction. Auscultation revealed grade II/VI systolic heart murmur over the left side of the chest.

The animal was admitted to the hospital and blood samples were collected for laboratory tests. The results of CBCs and blood chemistry tests are shown in Tables 1-2.

As the laboratory did not provide reference values for the parameters tested, the reference ranges were calculated on the basis of literature data (1, 3, 12, 19, 24, 26) according to the following formula: mean ± 2 × standard deviation. Because blood parameters in camels show significant variation depending on the geographical location of the animals

Tab. 1. The results of complete blood count examination in a camel with an interval of five days between blood collections

Parameter	1st examination	2nd examination	Normal value
RBC	7.66 T/l	**7.24 T/l**	6.11-6.71 T/l (3) 7.61-12.93 T/l (19)
PCV	**21.0%**	**19.7%**	24.48-25.8% (3) 21.66-34.06% (19)
Hb	**9.50 g/dl**	**9.00 g/dl**	10.29-11.05 g/dl (3)
MCV	27.4 fl	27.2 fl	38.47-41.71 fl (3) 23.75-30.63 fl (19)
MCH	**12.4 pg**	**12.4 pg**	16.13-17.85 pg (3)
MCHC	**45.2 g/dl**	**45.7 g/dl**	41.23-43.75 g/dl (3)
Leukocytes	23.3 G/l	20.0 G/l	

Parameter	1st examination	2nd examination	Normal value
Neutrophils	21.7 G/l **93.2%**	18.5 G/l **92.6%**	29.33-35.65% (3)
Lymphocytes	0.71 G/l **3%**	0.80 G/l **4%**	53.1-59.38% (3)
Monocytes	0.58 G/l **2.5%**	0.40 G/l **2%**	3.56-5.24% (3)
Eosinophils	0.20 G/l **0.9%**	0.19 G/l **1%**	4.96-7.12% (3)
Basophils	0.09 G/l 0.4%	0.08 G/l 0.4%	0.32-0.56% (3)
Platelets	228.0 G/l	**619.0 G/l**	93.81-293.81 G/l (19)

Explanations: No reference values were provided by the laboratory, so the reference values were calculated on the basis of literature data according to the following formula: normal value = mean ± 2 × standard deviation. Since the literature data are based on a statistically small numbers of animals, more than one normal range was considered where possible. RBC – red blood cells; PCV – packed cell volume; Hb – haemoglobin; MCV – mean corpuscular volume; MCH – mean corpuscular haemoglobin; MCHC – mean corpuscular haemoglobin concentration. Values below or above the available reference ranges are marked in bold.

Tab. 2. The results of biochemical blood examination in a camel with an interval of five days between blood collections

Parameter	1st examination	2nd examination	Normal value
Urea	9.77 mmol/l 58.91 mg/dl	11.0 mmol/l 66.33 mg/dl	10.7-11.1 mmol/l (26) 5.06-6.26 mmol/l (3) 2.9-12.9 mmol/l (24) 0.4-59.6 mg/dl (12)
Creatinine	141.7 µmol/l	140.4 µmol/l	170-188 µmol/l (26) 65.42-83.1 µmol/l (3) 44.2-221 µmol/l (24)
AST	**1450.4 U/l**	**2555.7 U/l**	9.8-20.2 U/l (26) 28.8-118.8 U/l (24) 19.5-76.7 U/l (12)
GGTP	13.3 U/l	14.6 U/l	9.5-25.9 U/l (24)) 0-21.7 U/l (12)
LDH	**3676.0 U/l**	**4264.2 U/l**	1634-1726 U/l (26)
Total bilirubin	**1.44 µmol/l**	**1.20 µmol/l**	4.3-6.3 µmol/l (24)
GLDH	21.4 U/l	9.10 U/l	0-27.4 U/l (12)
ALP	54.5 U/l	54.2 U/l	0-675.6 U/l (24)
Total protein	5.14 g/dl	**4.94 g/dl**	4. 98-5.62 g/dl (26) 8.27-8.59 g/dl (3) 5.0-7.4 g/dl (24) 5.7-8.14 g/dl (12)
Albumins	**16.3 g/l**	**14.0 g/l**	30.7-32.7 g/l (3) 26-42 g/l(24) 27-45.8 g/l (12)
Globulins	35.1 g/l	35.4 g/l	50.5-66.7 g/l (3) 22.5-42.9 g/l (12)
Glucose	**13.5 mmol/l** **243 mg/dl**	**17.3 mmol/l** **311.4 mg/dl**	4.6-5.4 mmol/l (26) 3.05-3.57 mmol/l (3) 4.7-9.1 mmol/l (24) 86.6-135.4 mg/dl (12)
CK	**6064.0 U/l**	**5573.0 U/l**	186.6-209.4 U/l (26) 74-310 U/l (24)
Alpha-amylase	**1274.3 U/l**	**1468.1 U/l**	641.2-742.8 U/l (26)

Parameter	1st examination	2nd examination	Normal value
Lipase	7.40 U/l	7.00 U/l	
Lactic acid	1.43 mmol/l (12.87 mg/dl)	1.35 mmol/l (12.15 mg/dl)	
Bile acids	0.66 µmol/l	3.62 µmol/l	
Cholesterol	1.45 mmol/l	1.41 mmol/l	1.1-1.5 mmol/l (26) 0.4-1.68 mmol/l (24)
Triglycerides	0.43 mmol/l **37.72 mg/dl**	0.49 mmol/l **42.98 mg/dl**	0-0.7 mmol/l (24) 31.14-37.34 mg/dl (3)
BHB	0.01 mmol/l		
FFA	0.31 mmol/l		0-0.45 mmol/l (12)
Fibrinogen	4.59 g/l	4.66 g/l	0.94-4.92 g/l (1)
Sodium	**146.8 mmol/l**	143.8 mmol/l	141.2-144.8 mmol/l (26)
Potassium	**3.98 mmol/l**	5.23 mmol/l	5.0-5.4 mmol/l (26)
Calcium	**2.1 mmol/l** 8.44 mg/dl	2.04 mmol/l 8.16 mg/dl	2.4-2.8 mmol/l (26) 1.99-2.07 mmol/l (3) 2.4-3.2 mmol/l (24) 0-23.2 mg/dl (12)
Magnesium	0.70 mmol/l **1.71 mg/dl**	0.78 mmol/l **1.9 mg/dl**	1.3-1.7 mmol/l (26) 0.7-1.1 mmol/l (24) 2-3.2 mg/dl (12)
Chlorides	108.1 mmol/l	110.9 mmol/l	93.3-103.7 mmol/l (26) 102.8-130.4 mmol/l (24)
Phosphorus	1.42 mmol/l	1.99 mmol/l	1.7-2.1 mmol/l (26) 1.88-2.0 mmol/l (3) 0.9-8.1 mmol/l (24)
Iron	**1.93 µmol/l**	**7.92 µmol/l**	14.9-23.7 µmol/l (26)
Zinc	0.19 mg/l		0.19-0.5 mg/l (12)
Copper	**7.22 µmol/l** 45.87 µg/dl		12.8-17.2 µmol/l (26) 25-105.8 µg/dl (12)
Selenium	**15.7 ng/ml**		55-220 ng/ml

Explanations: Except for the selenium level, no reference values were provided by the laboratory, so the reference values were calculated on the basis of literature data according to the following formula: normal value = mean ± 2 × standard deviation. Since the literature data are based on a statistically small numbers of animals, more than one normal range was considered where possible. AST – aspartate aminotransferase; GGTP – gamma-glutamyl transpeptidase; LDH – lactate dehydrogenase; GLDH – glutamate dehydrogenase; ALP – alkaline phosphatise; CK – creatine kinase; BHB – beta-hydroxybutyrate; FFA – free fatty acids. Values below or above the available reference ranges are marked in bold.

examined (12) and the season (3), where possible, all available reference data were considered. Given that the clinical signs started in February, values for the dry season were used, where available (3).

Blood tests for blood parasites and *Mycoplasma* sp. were negative. The faecal examination was positive for parasites – *Trichostrongylidae* (2075 eggs per gram [epg]) and *Capillaria* sp. (80 epg) – which suggests that the previous treatment was unsuccessful because of parasite resistance, or the patient suffered reinvasion after the last treatment.

The animal was treated with a glucose, calcium salt and magnesium salt supplement (Propical, VetAgro, Poland), antiparasitic drugs (levamisole: Levamol 7.5%, Vetoquinol Biowet, Poland; and fenbendazole: Fenbenat, Vetos-Farma, Poland), vitamins (vitamin A, B complex, D3, E), ornithine, a selenium supplement and a preparation for normalising rumen motility (Rumen Go Gel, Ankor, Poland). Levamisole was used because of suspected fenbendazole-resistant *Trichostrongylidae* and fenbendazole over three days for *Capillaria* invasion. Camelidae are pseudoruminants, which means that they have forestomachs functionally similar to those of ruminants, but anatomically different. They consist of three compartments (C1, C2, C3) of which C1 is the largest and functionally corresponds to the rumen. C1 and the rumen are inhabited by the same microorganisms, and it is beneficial to use preparations dedicated to the treatment of ruminal motility disorders. C3 is similar to the horse stomach, and only the last one fifth of its length produces gastric acid.

After five days of treatment, another blood collection was performed along with the examination of urine and faecal samples. The blood test results are presented in Tables 1-2. Urinalysis showed substantial bacterial flora with no other abnormalities. The faecal examination was negative for parasites (*Coccydia* sp., *Eimeria macusaniensis*, *Nematodirus* sp., *Trichostrongylidae*, *Trichuris* sp., *Capilaria* sp., *Strongylus* sp., *Moniesia* sp.).

Additionally, abdominal ultrasound and echocardiogram were performed.

The abdominal ultrasound (performed both transabdominally and transrectally) revealed the presence of ascites with a small amount of intestinal content and no other abnormalities. After the examination, the treatment was continued with the addition of oxytetracycline (Engemycin 10%, Intervet, Poland), flunixin (Vetaflunix, Vet-Agro, Poland) and heparin (25000 U/ml).

An echocardiogram was performed with an Esaote MyLab 30 Gold. Cardiac images were obtained using the right-sided caudal long-axis four-chamber view, the right-sided short-axis views and the left-sided four-chamber view, in accordance with the literature (32). At least three measurements of each parameter were taken, and the mean value was calculated. The examination revealed a smaller left ventricle as compared to the literature data, volume overload of the right ventricle (assessed as the ratio of the left ventricular diameter to the right ventricular diameter) and the right atrium, tricuspid insufficiency, mild pulmonary insufficiency and mild systolic dysfunction of the left ventricle without left-ventricular enlargement (Tab. 3, Fig. 1, Supplementary videos 1-3).

Tab. 3. The results of echocardiographic examination in a camel

Parameter	Value	Normal value
RVDd	**4.81 cm**	2.9-7.7 cm (32)
IVSd	**1.49 cm**	1.5-2.7 cm (32)
IVSs	**1.64 cm**	1.4-4.2 cm (32)
LVDd	**7.69 cm**	8.6-15.0 cm (32)
LVDs	**6.23 cm**	7.0-9.4 cm (32)
PWd	**1.54 cm**	2.0-3.6 cm (32)
PWs	**2.09 cm**	1.1-2.7 cm (32)
EF	38	
FS	19%	
Ao	**4.75 cm**	5.4-8.6 cm (32)
LA	**6.89 cm**	4.6-6.6 cm (32)
LA/Ao	1.45	
AoV	0.67 m/s 1.8 mmHg	
PV	0.49 m/s 1.0 mmHg	
TR	1.98 m/s 25.7 mmHg	
PR	0.5 m/s 1.0 mmHg	

Explanations: RVD – right ventricular diameter; IVS – interventricular septum thickness; LVD – left ventricular diameter; PW – posterior wall thickness; EF – ejection fraction; FS – fractional shortening; Ao – aortic diameter; LA – left atrial diameter; AoV – aortic valve velocity; PV – pulmonary valve velocity; TR – tricuspid regurgitation; PR – pulmonary valve regurgitation; d – diastole; s – systole. Values below or above the available normal range are marked in bold.

After the cardiologic examination, the treatment was broadened to include furosemide (1 mg/kg i.v. TID; Furosemid 5% Biowet Drwalew, Poland) and digoxin (3 µg/kg i.v. BID; Digoxin WZF, Polfa SA, Poland).

Most procedures were performed without sedation, including echocardiogram and transabdominal ultrasound, but some, such as oral examination and transrectal ultrasound, required tranquilization. Standing sedation was achieved with xylazine and butorphanol. (Nerfasin vet 100 mg/ml 0.3 mg/kg and Morphasol 10 mg/kg 0.03 mg/kg i.m.).

After two days of the treatment described and thirteen days following the admission to the hospital, the camel suddenly died.

The necropsy revealed approximately 5 l of serous fluid in the abdominal cavity and an increased amount of serosanguineous fluid in the pericardial sac. In the C1 stomach foreign bodies (strings and ropes) were found, and in C3 ulcerative lesions were noted. The lungs showed a pattern of multiple dark areas. The heart was severely enlarged. No other lesions were observed in the internal organs.

A detailed cardiopathological examination showed a very severe dilation of the right ventricle and a mild enlargement of the right atrium (Fig. 2). The heart weighed 1862 g, was 21.7 cm high (the largest longitudinal diameter) and 19.6 cm wide (the largest transverse diameter at the level of the atrioventricular valves). In the cross section below the level of the atrioventricular valves, the right ventricle was severely enlarged; its internal area was 53.99 cm^2, while

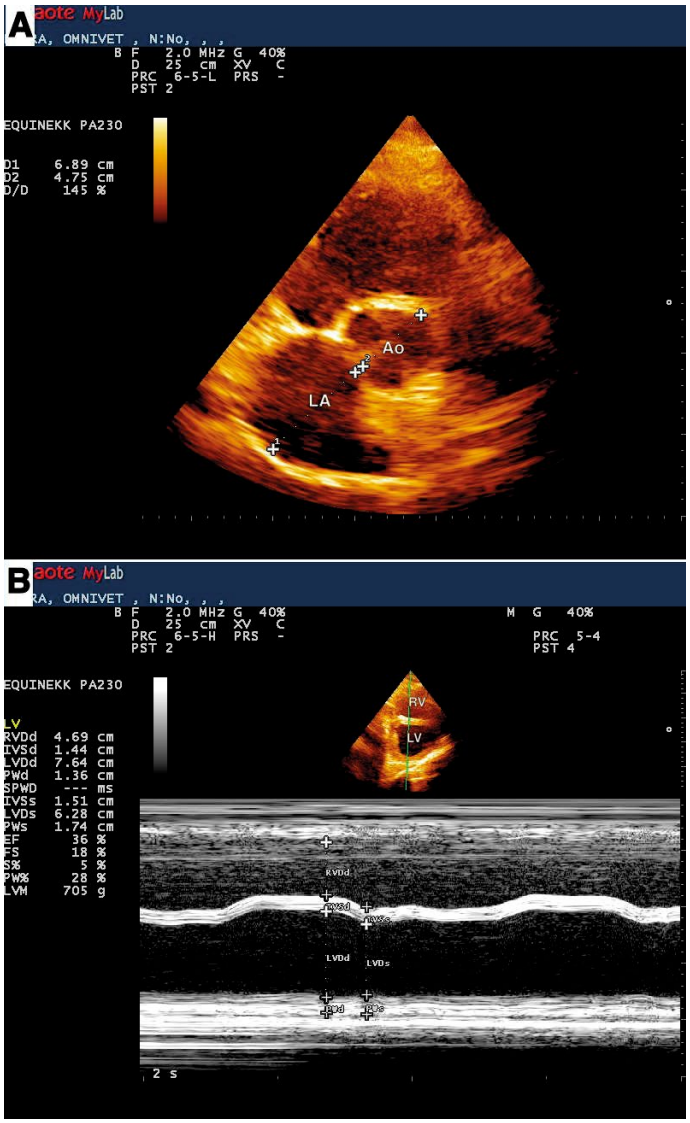

Fig. 1. Echocardiographic examination of a camel: right-sided short-axis views. A: View at the level of the aorta: the left atrium is not enlarged; B: View at the level of the ventricles: the right ventricle shows a marked dilation

Explanations: Ao – aorta, LA – left atrium, LV – left ventricle, RV – right ventricle.

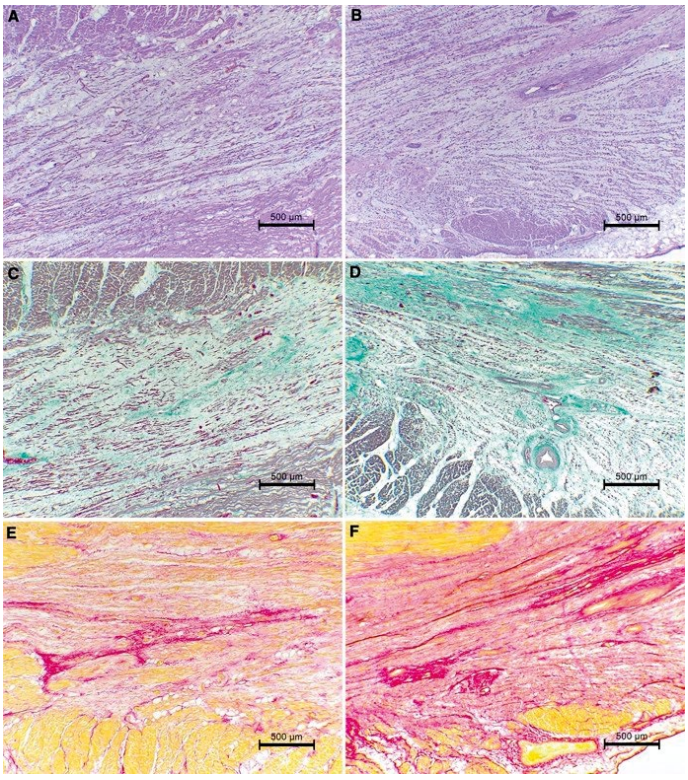

Fig. 3. Histopathological examination of right ventricular specimens. Vast areas of myocardial atrophy with granulation tissue rich in newly formed blood vessels and with proliferation of connective tissue fibres are visible. The remaining cardiomyocytes are degenerated. A, C, E: the anterior wall of the right ventricle; B, D, F: the wall of the right ventricle in the area of the outflow tract. AB: haematoxylin-eosin; CD: Masson-Goldner stain; EF: Picrosirius red stain; magnification: 40 ×

the internal area of the left ventricle was 21.6 cm^2. In both ventricular walls and in the interventricular septum, subendocardial and subepicardial bands of dark-coloured tissue were observed (Fig. 2).

Multiple samples collected from the heart and the lungs were fixed in 4% buffered formalin for 48 h, embedded in paraffin blocks, cut into 6 µm sections, placed on microscopic slides and stained with hematoxylin-eosin, Masson-Goldner trichrome and Picrosirius red stain.

Histological examination revealed lesions throughout the heart (Fig. 3-7). Both ventricular walls and the interventricular septum presented with vast areas of myocardial atrophy, with granulation tissue rich in newly formed blood vessels and with proliferation of connective tissue fibres. The remaining myocardium showed severe degeneration (Fig. 3-4).

In both atrial appendages, severe damage of myocardium with almost total cardiomyocyte atrophy was noted. The myocardial tissue was replaced with connective tissue (Fig. 5). Lesions in the atrial walls were less advanced, with cardiomyocyte degeneration and

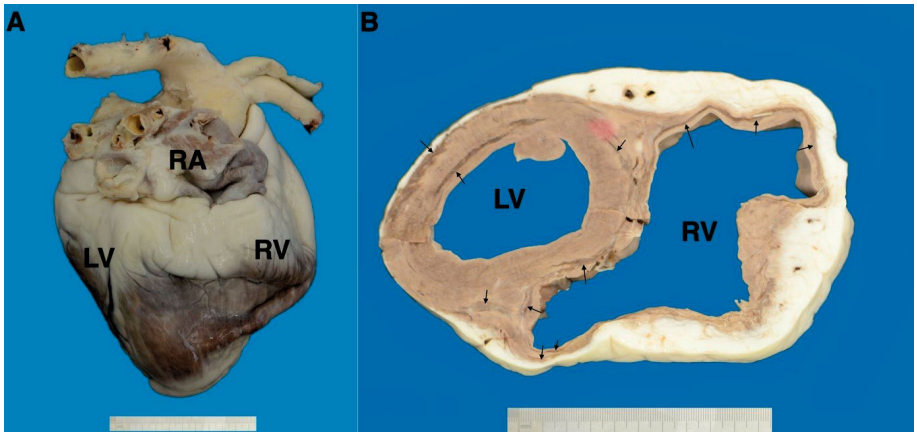

Fig. 2. Cardiopathological gross examination. A. External view of the heart: a dilation of the right ventricle and an enlargement of the right atrium. B. Cross sectional view of the heart below the atrio-ventricular valves: a severe dilation of the right ventricle with segmental thinning of the ventricular wall; multiple subepicardial and subendocardial bands of dark-brown tissue (arrows)

Explanations: LV – left ventricle; RA – right atrium; RV – right ventricle.

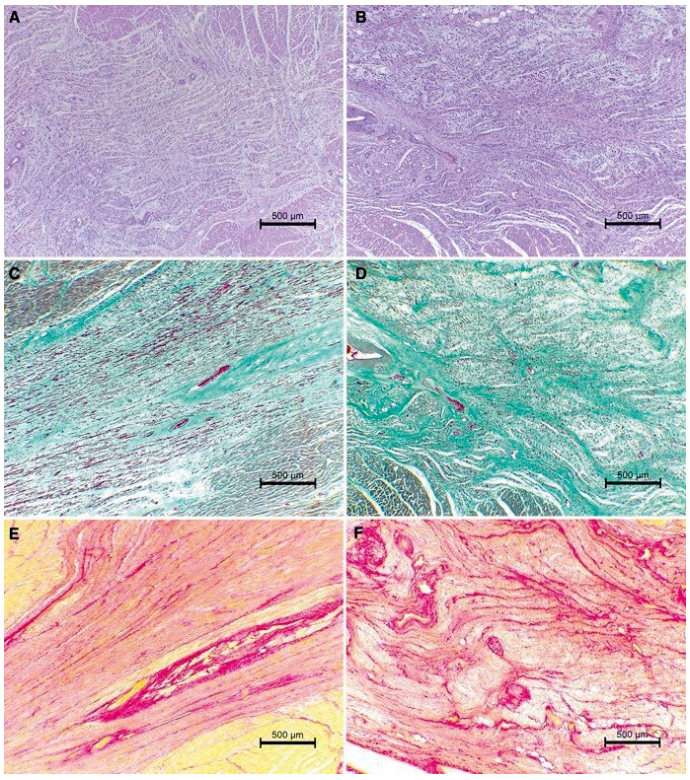

Fig. 4. Histopathological examination of left ventricular specimens. Vast areas of myocardial atrophy with granulation tissue rich in newly formed blood vessels and with proliferation of connective tissue fibres are visible. The remaining cardiomyocytes are degenerated. A, C, E: the posterior wall of the left ventricle; B, D, F: the wall of the left ventricle forming the heart apex. AB: haematoxylin-eosin; CD: Masson-Goldner stain; EF: Picrosirius red stain; magnification: 40 ×

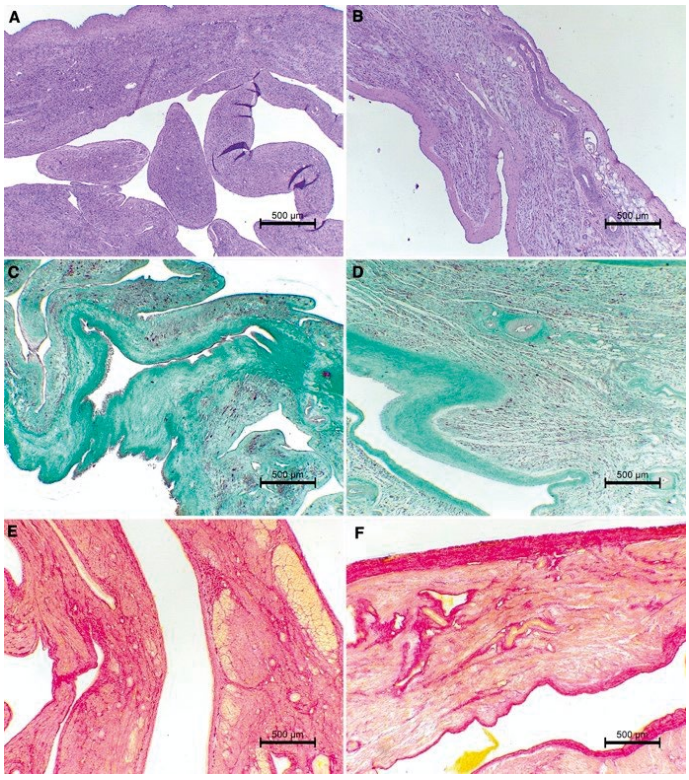

Fig. 5. Histopathological examination of atrial appendage specimens. Severe damage of the myocardium with an almost total atrophy of cardiomyocytes. The myocardial tissue has been replaced with connective tissue, and the remaining cardiomyocytes are degenerated. A, C, E: right atrial appendage; B, D, F: left atrial appendage. AB: haematoxylin-eosin; CD: Masson-Goldner stain; EF: Picrosirius red stain; magnification: 40 ×

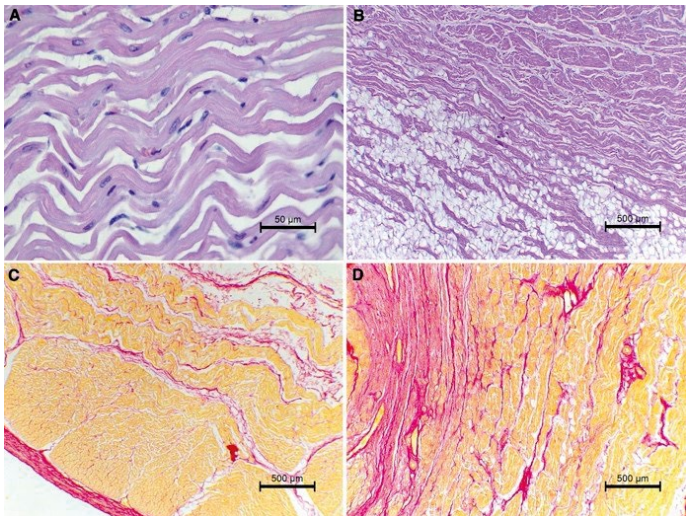

Fig. 6. Histopathological examination of atrial specimens. Cardiomyocyte degeneration and hyperplasia of interstitial connective tissue and fatty tissue (in the left atrium only). A, C: right atrium; B, D: left atrium. AB: haematoxylin-eosin; CD: Picrosirius red stain; magnification: 40 ×

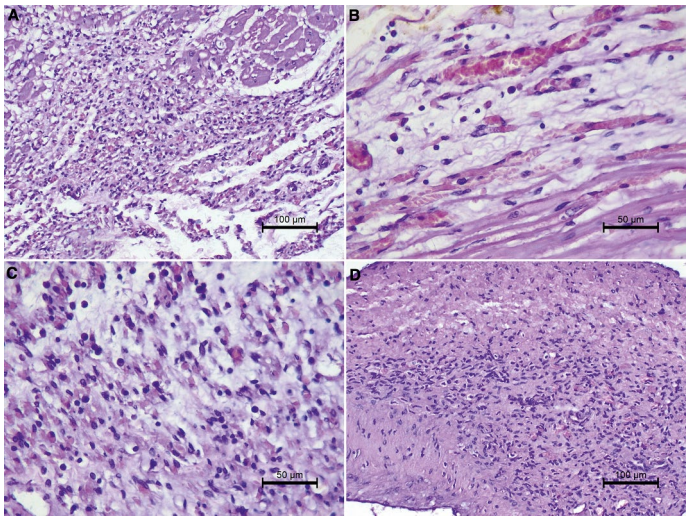

Fig. 7. Histopathological examination of cardiac specimens. Vast areas of myocardial atrophy with granulation tissue rich in newly formed blood vessels. A: the wall of the right ventricle; magnification 200 ×; B: the wall of the right ventricle; magnification 400 ×; C: the wall of the left ventricle; magnification 400 ×; D: the wall of the right atrial appendage; magnification 200 ×. Haematoxylin-eosin stain

hyperplasia of interstitial connective tissue and fatty tissue (in the left atrium only) (Fig. 6).

Additionally, the lungs showed a pattern of interstitial lymphocytic infiltration with areas of pulmonary fibrosis and pulmonary atelectasis. In the tracheobronchial lymph nodes, intense activation of lymphatic tissue was present.

On the basis of the clinical and pathological examination, a diagnosis was made of multifocal myocardial necrosis due to selenium deficiency leading to right ventricular cardiomyopathy.

Discussion

Reports describing cardiac diseases in camels are very limited (14-17, 25, 29). Some of the pathologies reported were a part of complex congenital anomalies (25, 29), while others presented in old animals (15, 17). The authors found no description of generalised cardiomyopathy affecting mainly the right ventricle, either primary or secondary.

In the present case, the right ventricular dimensions did not exceed the reference ranges presented by Tharwat et al. (32), but the left ventricular dimensions were smaller than noted in the abovementioned study. The report cited is the sole attempt at developing the reference values for echocardiographic examination in dromedary camels. Nonetheless, as it studied twenty-two animals, significant differences are possible due to individual variability.

The analysis of blood examination results in camels is difficult, as reference ranges differ across authors (1, 3, 12, 24, 26), and a relatively small number of animals were examined in each paper. Moreover, the reference values depend on the season and the geographical location (3, 12). The levels of AST, LDH, total bilirubin, albumins, glucose, creatinine kinase, alpha-amylase and iron were significantly beyond the reference ranges in the case discussed.

The normal selenium level in camel's blood has not been well established. The normal levels provided vary from 5.3 ng/ml to 288 ng/ml, depending, among others, on the sample type (2, 6, 8, 13, 27). The analytical procedures were not described in all of the cases and could differ between authors. In the present case, the normal range for this parameter was provided by the laboratory, indicating a significant decrease in its level.

Selenium is of crucial importance for many regulatory and metabolic functions in organisms, including the thyroid hormone metabolism, the antioxidant defence systems, the adaptive and acquired immune system and the prevention of certain cancers (9, 18). The most important proteins in the selenoprotein family are glutathione peroxidases (GPx). GPx neutralise the reactive oxygen and reactive nitrogen species (10) and were shown to protect mice from viral myocarditis (7). The thioredoxin (TrxR) system is also involved in the selenoprotein synthesis. TrxR are enzymes that regulate numerous redox processes in the cell, including signalling, cell–cell communication, as well as DNA metabolism and repair. Thioredoxins, especially Trx-1/TrxR1, are known to regulate cardiac functions and are involved in cardiovascular disease processes (4, 9, 22).

Accumulating evidence suggests that selenium is also important for the optimal functioning of the cardiovascular system in humans and animals. Selenium deficiency is the cause of the white muscle disease (WMD) in various animal species, especially in young animals (11, 18, 23, 31). The disease involves hyaline degeneration of muscle cells in various skeletal muscles, including the diaphragm and the heart. Cardiomyocytes show focal areas of non-inflammatory coagulative necrosis with swollen myocardial fibres presenting with granular cytoplasm and loss of striation (13, 34). Electrocardiographic examination indicates sinus tachycardia, an increased P wave amplitude, shorter PR, QT and ST intervals, a narrower QRS complex, a shorter T wave duration, a discretely increased T wave amplitude, supraventricular and ventricular premature beats and atrial fibrillation (20). Mortality from WMD in foals and horses is high (ranging from 30% to 45%) (11). Diagnosis should be supported by a low serum selenium level coupled with low glutathione-peroxidase and vitamin E levels. Troponin I (cTn-I) can be used to diagnose the clinical and subclinical forms of WMD, since cTn-I concentrations increase during the early stages of WMD-mediated myocarditis (5).

In young animals, WMD is accompanied by a decrease in haemoglobin concentration, PCV values and total erythrocyte count, yet, these changes were not observed in the three selenium-deficient adult camels described (13). The case discussed in the current paper showed deviations in the CBC results consistent with the literature, despite the subject being an adult female.

In humans with selenium deficiency, a rapidly progressive cardiomyopathy was observed, resulting in extensive necrosis and fibrosis, known today as Keshan disease (30, 33). Nonetheless, it was noted that selenium deficiency in Keshan disease patients was a conditional predisposing factor rather than a specific or etiologic factor for the occurrence of the disease (33).

The pathological image of Keshan disease consists of generalised multifocal myocardial necrosis combined with fibrosis, mainly in the middle part of the left ventricle, but also in other parts of the heart. The necrosis is not accompanied by inflammatory infiltration or vascular alterations (21, 28). In the present case, the morphological and histological picture of the heart resembled Keshan disease more closely than typical WMD.

In conclusion, the clinical and post-mortem examination presents a case of advanced cardiomyopathy in a camel. Although the reference ranges for selenium have not been well established for camels, the multifocal lesions observed in the myocardium may have been caused by selenium deficiency. The rapid progression of the clinical signs may have been related to the presence of interstitial pneumonia causing the overload of the right side of the heart and the subsequent signs of right-sided heart failure.

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Supplementary materials

Video 1. Echocardiographic examination: right-sided short-axis view at the level of the ventricles. A dilation of the right ventricle and systolic dysfunction is visible.

Video 2. Echocardiographic examination: right-sided long-axis view. A dilation of the right ventricle and the right atrium is visible together with systolic dysfunction.

Video 3. Echocardiographic examination: right-sided long-axis view with colour Doppler imaging. A tricuspid regurgitation is visible.

Link: https://bazawiedzy.upwr.edu.pl/info/researchdata/UPWRb1438d808b5744a8ae93c268edb9bf07/

DOI: https://doi.org/10.57755/4sfh-m270

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