A 7-week-old 60-kg (132-lb) female dromedary camel (Camelus dromedarius) calf was submitted to the veterinary pathology unit of the veterinary university hospital of Lodi, Italy, for postmortem examination following sudden death without premonitory signs. The owner reported no changes in the camel's behavior or signs of illness during the preceding days.

**Clinical and Gross Findings**

The camel was in good body condition, with no evidence of dehydration. Postmortem examination revealed severe hydrothorax, severe and diffuse pulmonary edema, and diffuse hyperemia. Severe hydropericardium was also present, and the pericardial sac was filled with abundant yellow fluid admixed with scant whitish fibrinous material. The myocardium was pale and characterized by large, irregular, multifocal whitish areas and streaks visible beneath the epicardium (Figure 1). Sections from the atrial and ventricular free walls and interventricular septum revealed severe white discoloration of the myocardium, mainly evident in the left ventricular free wall. Congenital patent ductus arteriosus was identified as an additional heart anomaly. Multifocal small areas of whitish discoloration of skeletal muscle were evident in the hind limb muscles. No other macroscopic abnormalities were found.

Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page →
Histopathologic Findings

Sections of samples from the myocardium were stained for histologic examination (Figure 2) with H&E stain, Von Kossa stain (to assess calcium deposition), and the van Gieson method (to highlight collagen fibers). Up to 60% of the myocardium was affected by severe polyphasic necrotizing and degenerative changes with multifocal to coalescing areas of myofiber loss, mainly localized in the left ventricular free wall. Affected cardiomyocytes were often fragmented and hypereosinophilic with loss of cross striations and had pyknotic or karyorrhectic nuclei. Other cardiomyocytes were swollen with vacuolated sarcoplasm. Necrotic myofibers often contained deposition of granular to crystalline basophilic material (dystrophic mineralization). Scant numbers of fibroblasts and collagen fiber depositions admixed with moderate numbers of macrophages, lymphocytes, and occasional plasma cells were present within areas of necrosis. Von Kossa staining of myocardial tissue sections revealed the presence of abundant multifocal to coalescing black granular material that replaced the myocardial fibers, compatible with severe myocardial mineralization. Staining via the van Gieson method revealed a fine layer of collagen bundles and scant fibroblasts between myofibers, with no evidence of myocardial fibrosis. Mild degenerative changes were also present multifocally in skeletal muscle, mainly in the hind limb musculature.

Additional Laboratory Findings

Samples of the liver, heart, and skeletal muscle from the forelimbs and hind limbs were frozen and subsequently processed for selenium concentration analysis by means of inductively coupled plasma mass spectrometry. In healthy camels, selenium concentrations range from 0.921 to 1.500 mg/kg (0.419 to 0.682 mg/lb) in liver tissue and from 0.351 to 0.640 mg/kg (0.1560 to 0.291 mg/lb) in muscle tissue. In the present case, selenium concentrations were 0.136 mg/kg (0.062 mg/lb) in liver tissue, 0.128 mg/kg (0.058 mg/lb) in heart tissue, 0.186 mg/kg (0.085 mg/lb) in forelimb muscle tissues, and 0.190 mg/kg (0.086 mg/lb) in hind limb muscle tissues.

Morphologic Diagnosis and Case Summary

Morphologic diagnosis: acute to subacute multifocal to coalescing necrosis of the myocardium.

Case summary: sudden death of a dromedary camel calf due to myocardial necrosis.

Comments

Camelids are found worldwide, and in recent years, research has greatly expanded clinical knowledge regarding members of the family Camelidae. In the southern hemisphere, camelids are a main source of milk, meat, wool, transportation,

Figure 2—Photomicrographs of sections of the myocardium obtained from the camel calf in Figure 1. A—Severe polyphasic necrotizing and degenerative changes with multifocal to coalescing areas of mineralization are evident. H&E stain; bar = 750 µm. B—High numbers of macrophages, lymphocytes, and plasma cells are present within the areas of necrosis and surrounding myocardial fibers. H&E stain; bar = 150 µm. C—Within necrotic myocardial fibers, there are abundant multifocal to coalescing areas of black material consistent with calcium salt deposition. Von Kossa stain; bar = 1.5 mm. D—Deposition of abundant black granular material has replaced the cytoplasm of myocardial fibers, a finding compatible with mineralization. Von Kossa stain; bar = 75 µm.
and labor. In the northern part of the world, they are mainly kept as companion animals or found in circuses or zoological parks, although breeding of camelds for wool production has recently been increasing in Europe.1,2 Diseases of camels are a focus of investigation, and the present report describes the macroscopic, microscopic, and histochemical features of a case of sudden death attributable to myocardial necrosis in a young camel calf.

Myocardial necrosis is a common and often fatal disorder in a wide range of animals; calves, lambs, swine, and foals are mainly affected.3,4 Myocardial necrosis can result from several causes, including nutritional deficiencies and chemical and plant toxicoses.5 Nutritional deficiencies include lack of vitamin E and selenium, thiamine, copper, and taurine.6 Toxicoses can be related to ionophore toxicity (eg, monensin in ruminants), rodenticides containing thallium, gossypol, or poisonous plants such as those containing glycoside (eg, oleander).7 Myocardial necrosis is also a typical feature of nutritional myopathy or white muscle disease.8,9

White muscle disease is a metabolic disorder caused by dietary deficiency in vitamin E or selenium.1,2 This type of nutritional myopathy is known to develop across a wide range of domestic, wild, and zoo animals. Young animals are typically affected, and clinical signs, if present, become noticeable between 2 and 12 weeks of age.3 This nutritional myopathy represents a widespread problem in dromedary camel breeding programs, occurring more often in intensive livestock agriculture operations.5,8 In the body, selenium and vitamin E have similar actions and function synergistically, and their tissue concentrations are directly related to dietary intake.2 Selenium and vitamin E are important as antioxidants in tissues, promote the maintenance of good reproductive performance, and prevent muscle damage. Inadequate nutritional management or dietary supplementation of these elements may lead to various degrees of metabolic disorders.1,2,5

Clinical signs of white muscle disease are not specific and may mimic numerous other diseases. Subclinical forms are generally associated with susceptibility to infections and reproductive problems, whereas the severe form results in cardiac and musculoskeletal abnormalities. Diarrhea and hepatic necrosis in affected camels have been reported.3,7 When the disease affects the heart, animals develop respiratory distress, fever, and high heart and respiratory rates. When skeletal muscles are affected, signs vary from mild weakness to difficulty standing or walking.2 Although not well described, recent studies3-7 revealed that in camels, as in other herbivores, selenium deficiency can cause muscular dystrophy, with degenerative myocardial lesions and discoloration of skeletal muscle similar to those observed in selenium-deficient cattle. In the camel calf of this report, postmortem cardiopulmonary findings were consistent with pulmonary edema and congestion, hydrothorax, and hydropericardium and were related to progressive heart failure. Nevertheless, no clinical signs were reported, and the young animal died suddenly without premonitory signs, which is consistent with other reported cases.5

Selenium metabolism in camels is analogous to that of other herbivores. In cases of selenium deficiency and toxicity, camels have similar clinical signs, similar selenium concentrations in serum and organs, and similar selenium excretion kinetics as other affected herbivores. There are few data in the veterinary medical literature concerning selenium concentrations in dromedary camel organs, but according to the results of a previous study, selenium concentrations in the dromedary camel calf of the present report were lower than published values, confirming selenium deficiency. Selenium supplementation should be a common practice in camelid breeding operations to avoid the occurrence of reproductive diseases and myocardial and muscular dystrophy.2,6 However, substantial individual variation in susceptibility to the toxic effects of selenium has been reported among different species.4 In animals, there is a fairly narrow concentration range between conditions of selenium deficiency and selenium toxicosis. In experimental situations, dromedary camels have been shown to have an apparent sensitivity to toxic effects of excessive selenium supplementation. In dromedary camels, severe intoxication developed with selenium supplementation at a concentration a fifth of that which caused similar intoxication in sheep and cattle.2

In general, camel metabolism prevents mineral deficiencies.2 Wild camels are rarely affected by this kind of nutritional problem.2 Their adaptation to arid conditions and poor mineral feeding resources allow them to improve the absorption capacity of trace elements in periods of scarcity. Wild camels have developed a higher storage ability and higher tolerance for excesses in minerals and electrolytes.2

An additional finding in the case described in the present report was patent ductus arteriosus. Congenital malformations of camels are relatively rare, with few reports in the veterinary medical literature. Heart defects are rare in these animals and usually have no relation to other anomalies.8 Ventricular septal defects seem to be the most common congenital anomaly.9,10

As illustrated by the case described in this report, myocardial necrosis should be considered as part of the classical form of white muscle disease in camels, as in other animals. This case emphasizes the lethal potential of selenium deficiency, which sometimes may be underestimated. Selenium deficiency should be included as a differential diagnosis in cases of sudden death among camels. Careful clinical management and dietary supplementation are necessary to avoid this condition. In dromedary camels, evaluation of selenium and vitamin E concentrations in foodstuffs as well as in blood samples may be useful to better define the pathogenesis of myocardial necrosis and plan suitable dietary supplementation.

References