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CONGENITAL NUTRITIONAL MYODEGENERATION (WHITE MUSCLE DISEASE) IN A GIRAFFE (*GIRAFFA CAMELOPARDALIS*) CALF

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Abstract: It is well known that vitamin E and selenium deficiencies in domestic ruminants can lead to white muscle disease. After a clinically normal gestation period at Ouwehand Zoo in the Netherlands, a newborn giraffe (*Giraffa camelopardalis*) calf showed clinical signs of white muscle disease almost immediately after birth. The calf was rejected by the mother and was euthanized 3 days later because of deterioration of clinical signs. At necropsy, pulmonary edema and pallor of skeletal and heart muscles was noted. Histologically, there was hyaline degeneration of skeletal muscle myocytes and pulmonary edema. Blood concentrations of vitamin E were ≤ 0.7 mg/L. Based on clinical, biochemical, and gross and microscopic pathological findings, congenital nutritional myodegeneration was diagnosed. This case of neonatal white muscle disease is particularly remarkable given that the diet of the dam contained more than the recommended amount of vitamin E.

Key words: *Giraffa camelopardalis,* nutrition, selenium, vitamin E, white muscle disease.

BRIEF COMMUNICATION

Nutritional myodegeneration, also referred to as white muscle disease, typically affects neonatal lambs, foals, goat kids, calves, and piglets, as well as a range of other species of domestic and exotic hoofstock. It is infrequently observed in mature animals.[7,15] It is caused by deficiencies of vitamin E and/or selenium, and both congenital and delayed forms of the condition have been described in ruminants.[8]

Vitamin E is an antioxidant that scavenges free radicals, and selenium protects cellular membranes and organelles from peroxidative damage.[1] The location and mechanism of the antioxidant effects of vitamin E and selenium vary; selenium is a constituent of glutathione peroxidase enzymes, which are mainly active in the cytosol, and vitamin E is incorporated into cell membranes. Deficiency of vitamin E and/or selenium can result in damage to cell components, which can lead to cell death.[3] The primary manifestation of white muscle disease is degeneration of skeletal muscles, although the heart and diaphragm muscles can also be involved.[15] Usually, affected animals have weak leg muscles, with trembling or a staggering gait, and rising from a lying to a standing position requires exceptional effort. If a large part of the heart muscle is involved, arrhythmia, a weak pulse, dyspnea, hypoxia, and tachycardia can occur.[10,15] In these cases, heart failure is ultimately the cause of death. Here we describe congenital nutritional myodegeneration in a newborn giraffe (*Giraffa camelopardalis*). Clinical, biochemical, and pathological findings led to a diagnosis of congenital nutritional myodegeneration caused by vitamin E deficiency.

On 31 January 2015 a male giraffe calf was born in Ouwehand Zoo (Utrecht, Rhenen 3911 AV, Netherlands). The calf was full-term and on first examination, 1 hr after parturition, no clinical abnormalities were noted. Sternal recumbency was achieved within 10 min after birth. The calf stood within 1 hr of being born, but hind-leg weakness was noted and the calf showed knuckling at the pasterns. After several hours it became evident that the calf had mobility problems that resulted in difficulties standing. The calf was weak, showing difficulties rising and standing, periodically trembling, and often lying down after standing for only a short period. The mother showed no interest and immediately rejected the calf. Zookeepers commenced a twice-a-day feeding regimen with frozen-stored bovine colostrum. The following day, blood samples were taken to determine the complete blood count, and vitamin E and selenium concentrations were determined at an external laboratory (Euregio Laboratory Services Stadionplein 46, 6225 XW Maastricht, the Netherlands). The blood for vitamin E quantification was stored in a 2-ml heparin

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Table 1. Vitamin E and selenium concentrations in a giraffe calf (*Giraffa camelopardalis*) with presumptive white muscle disease compared to concentrations in giraffe and other ruminants found in the literature.

Source	This study	Reference 2	Reference 5	Reference 13	Reference 10	Reference 15	Reference 11
Age group and species	1–3-day-old neonate giraffe	Adult giraffe	28 adult giraffe	Adult + subadult giraffe ranging from 2 to 28 years	5-day-old red deer	2-day-old neonate Holstein-Friesian calf	Adult cattle, sheep, and deer
Selenium (ppm)	0.118	–	–		>0.01	0.07–0.106	0.08–0.5
Vitamin E (mg/L)	≤0.7	0.04 –1.6	1.4	0.04–0.42	2.5–5.7	2.93–3.8	–

container and wrapped with aluminum foil, and samples were sent using refrigerated transport. At this time, a prophylactic dose of 5 ml vitamin E and selenium was given intramuscularly (Alfasan per ml 100 mg alpha-tocopheryl acetate and 1 mg sodium selenite, Alfasan Nederland Bv, Kuipersweg 9 3440 ab, Woerden, the Netherlands). Two days after it was born, the condition of the calf deteriorated to the extent that it could no longer drink or stand up. A second batch of blood was collected and the calf was euthanized.

Necropsy was performed at the department of Pathobiology, Pathology, Faculty of Veterinary Medicine, Utrecht University, Yalelaan 1, 3584 CL Utrecht, the Netherlands. Based on fat and muscle reserves, the calf was deemed to be in a moderate to good body condition, and weighed 50 kg. Abnormalities noted on macroscopic inspection included pulmonary edema and pallor of the heart and the skeletal muscles. Tissue samples from heart, lung, kidney, liver, colon, jejunum, duodenum, brain, bone marrow, thymus, abomasum, rumen, cervical and thoracic spinal cord, and five different skeletal muscles were fixed in 10% phosphate-buffered formalin, embedded in paraffin, cut at 4 mm, and stained with hematoxylin and eosin (H&E). Histologically, all the skeletal muscles showed interstitial edema and hyperemia with hyaline degeneration of myocytes. The lungs were edematous with focal fibrin deposits and bacteria without an inflammatory reaction. Edema was also seen in the heart interstitium, and the lumbar spinal cord showed signs of very mild edema. No significant macroscopic lesions were seen in other organs. The main differentials for the clinical signs and the findings of the pathological investigation were white muscle disease and trauma during or after birth.

The complete blood count showed no significant abnormalities. The first blood sample contained a selenium concentration of 0.118 ppm, and vitamin E (tocopherol) concentration was below the minimum detection level of 0.7 mg/L. The second blood sample contained 0.7 mg/L of vitamin E. These low concentrations of vitamin E make white muscle disease likely in this case. There are data available concerning vitamin E concentrations in giraffe, which we used as reference to compare with the calf (Table 1).[2,5] EAZA husbandry and management guidelines for *Giraffa camelopardalis* reviews numerous cases of vitamin E deficiency in captive giraffe.[6] However, there have been no previous reports about nutritional myodegeneration in a neonate giraffe only 3 days old. Serum vitamin E concentrations of other neonate ruminants were used to compare with this report (Table 1).[2,5,10,11,13,15]

Liu et al. described nutritional myodegeneration associated with vitamin E deficiencies (0.3–0.9 mg/L blood) and normal selenium concentrations in over 100 zoo ruminants.[7] Clinically, affected neonatal ruminants were weak, had difficulty rising, were unable to suckle, and died within 1 or 2 days. The giraffe calf had blood vitamin E concentrations below detection levels but selenium concentrations were within the normal reference ranges for other species, suggesting that vitamin E deficiency was solely responsible for the observed clinicopathological picture.[10,11,15] To determine the etiology of the low vitamin E status of the calf, the vitamin E content of the adult giraffe ration fed at Ouwehand Zoo was assessed. The dam's feed consisted of two components, lucerne hay and browser pellets (Kaspers Fauna Food, 3440 AA, Woerden, the Netherlands). The giraffe dam was fed approximately 5.4 kg dry matter (DM) of browser pellets throughout the day, with ad libitum lucerne hay (harvested by a local farmer). It was estimated that the 700-kg female giraffe had a total intake of 9.9 kg DM.[12] The browser pellets contained 725 IU vitamin E/kg DM. The vitamin E concentration of lucerne hay was unknown. The consumed

diet was therefore calculated to contain at least 395.5 IU/kg ([5.4 × 725]/9.9).

Based on the known requirements for domestic ruminants, the recommended dietary concentration of vitamin E for giraffes is 60 IU/kg DM.[12] In a follow-up to the Giraffe Nutrition Workshop, the Ruminant Browser Nutrition Workshop suggests that the dietary vitamin E requirement is 100–150 IU/kg DM.[13]

Although extrapolation of vitamin E requirements from cattle seems logical in the absence of sufficient species-specific data, this case highlights potential problems with this logic that may be related to the many differences between these two species regarding natural feeding, management, and digestive anatomy and physiology. Although much about giraffe digestive physiology is still unknown, there are several accepted differences between browsing and grazing ruminants. Cattle and giraffe are primarily foregut fermenters. The rumen in cattle is highly specialized and compartmentalized, whereas in the digestive tract of a browser like the giraffe, contents tend to pass more quickly from the rumen to the intestines.[12] In 1990, Shin and Owens showed that in cattle, the ruminal degradation of different sources of vitamin E ranged between 35.6% and 52.1%.[14] It is not known whether the bioavailability of vitamin E in giraffes is similar to that reported in cattle, but Dierenfeld accentuates that: "grazers display lower mean vitamin E levels than browsers even when fed diets containing similar vitamin E levels."[4]

Instead of comparing the dietary vitamin E requirement of giraffe with cattle, it is also possible to compare it with small ruminants like goats, sheep, lamas, and alpacas. The National Research Council for Small Ruminants advises a vitamin E intake of 5.6 IU/kg body weight (BW) for pregnant animals.[9] For our giraffe this would correspond with 396 IU/kg DM.

Another factor that could have contributed to the etiology of the nutritional myodegeneration in this case was the inability of the calf to drink colostrum in the all-important first few hours after birth. It is well known that neonatal lambs and calves are susceptible to low vitamin E status because of the limited placental transfer of vitamin E, and that this is partially compensated by the high vitamin E concentration in bovine colostrum (6–7 times higher than in normal lactation milk).[9] Dierenfeld[4] describes many other factors that could contribute to low vitamin E serum concentration: high concentration of polyunsaturated fatty acids in the feed, stress, and seasonal variation. Other factors like heat, humidity, artificial dehydration for forages, increased oxidation due to mixing or grinding of feed, and UV radiation have a direct influence on the vitamin E level in the feed.[8] These factors could very well play a role in any case of white muscle disease.

In conclusion, although the precise cause of this condition in the giraffe calf was not determined, it is noteworthy that this case of congenital nutritional myodegeneration occurred despite the provision of recommended concentrations of vitamin E in the dam's diet.

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