Congenital nutritional muscular dystrophy in a beef calf

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Abstract — A 13-hour-old Aberdeen-Angus was involuntarily recumbent since birth. Congenital nutritional muscular dystrophy was suspected based on clinical findings, increased serum creatine kinase, and decreased serum vitamin E and selenium levels. Recovery followed after supportive therapy and parenteral vitamin E and selenium. Reports of this disease in newborn calves are unusual.

Résumé — Dystrophie musculaire nutritionnelle congénitale chez un veau de boucherie. Un Aberdeen-Angus âgé de 13 heures était en décubitus involontaire depuis sa naissance. Une dystrophie musculaire nutritionnelle congénitale a été soupçonnée sur la base de trouvailles cliniques, d'une créatine kinase sérique augmentée et d'un taux sérique de vitamine E et de sélénium diminués. La guérison s'est effectuée à la suite d'une thérapie de support et de l'administration parentérale de vitamine E et de sélénium. Les comptes rendus de cette maladie chez le veau nouveau-né sont rares.

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A 13-hour-old, Aberdeen Angus female calf was admitted to the Large Animal Clinic at the Western College of Veterinary Medicine with a history of being recumbent and unable to move since birth. The calf was born to a heifer from a 60-cow herd, which was vaccinated against bovine viral diarrhea virus and infectious bovine rhinotracheitis virus. The cows were fed alfalfa hay and pellets, and ground barley, and had access to salt blocks. The owner had noticed signs of parturition in the heifer 9 h before he provided obstetrical assistance; the calf was delivered easily. The heifer was milked 4 h later and the calf was tube-fed 1L of colostrum. The owner reported that the calf had not sucked.

On presentation, the calf was in lateral recumbency and appeared comatose. The extremities were cool and dehydration was about 5%. The sucking reflex was diminished. The body temperature could not be registered with an electronic digital thermometer (< 32°C), the heart rate was 124 beats/min, and the respiratory rate 36 breaths/min. Total serum protein was 61 g/L (reference range, 57 to 81 g/L) and the blood glucose 2.7 mmol/L (reference range, 2.5 to 4.2 mmol/L).

The calf was warmed with heating lamps and warm water bags placed between its legs and around the abdomen. A bolus of 20 mL of 50% dextrose was given, IV, and followed by a total of 6 L of warm lactated Ringer's solution containing 1.4% dextrose over the next 24 h. The calf's temperature increased gradually; next day, the vital signs were normal and she was able to maintain sternal recumbency. However, she did not attempt to stand, even if assisted. The sucking reflex was weak, so she was tube fed milk, 10% of body weight, for the first 48 h. Congenital nutritional muscular dystrophy was suspected.

After 48 h, the calf's condition became stable, but she was still unable to stand. A venous blood sample was submitted for measurement of creatine kinase (CK), aspartate aminotransferase (AST), and vitamin E and selenium. The urine was grossly normal, but on analysis with the use of a urine reagent strip (Chemstrip 9; Roche Diagnostics, Laval, Quebec), it was found to contain blood, hemoglobin, or myoglobin. The markedly high levels of serum CK and AST, and the low serum levels of vitamin E and selenium (Tables 1 and 2) were suggestive of nutritional muscular dystrophy, which would explain the weakness, stiff gait, and the presence of blood (presumably myoglobin) in the urine. The weak sucking reflex may have been due to possible involvement of the tongue muscles. After 272 IU of vitamin E and 6 mg of selenium (Dystosel; Pfizer Canada, Kirkland, Québec; Selenium [as sodium selenite], 3 mg/mL; and vitamin E [dl-alpha tocopherol acetate] 136 IU/mL), had been given SC on day 2, the serum muscle enzymes were monitored over 5 successive days. The decrease in total protein to 48 g/L after hydration suggested either over hydration or failure of passive transfer of immunoglobulins because of failure to suck after birth. Mature cow's blood (800 mL) was transfused, IV, into the calf. A trimethoprim and sulfadoxine combination (Trivetrine; Schering-Plough Animal Health, Pointe-Clair, Quebec), 120 mg and 600 mg, respectively, was given, IV, daily for 5 d to prevent infection and septicemia, which could have occurred in association with the failure of passive transfer of immunoglobulins. The sucking reflex improved 48 h after presentation, but the calf was still given the same amount of milk (10% of body weight) by a nursing bottle. About 72 h after presentation, the calf stood without help but had a stiff gait. Over the next 3 d, the calf continued to improve and was given another dose of 272 IU of vitamin E and 6 mg of selenium, SC. It developed diarrhea that resolved with supportive therapy. The CK and AST levels continued to decrease and become

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Table 1. Serum creatine kinase (CK) and aspartate aminotransferase (AST) levels of a beef calf with congenital muscular dystrophy treated on days 2 and 5 with vitamin E and selenium

Reference range (1)	CK 35–280 U/L	AST 78–132 U/L
Day 2 (before treatment)	29 280	NA
Day 3	6200	299
Day 4	966	198
Day 5	267	145
Day 6	119	97
Day 7	166	78

NA — Not available

Table 2. Serum levels of vitamin E and selenium of a beef calf with congenital muscular dystrophy before, day 2, and after, day 5, treatment with vitamin E and selenium on day 2 and 5

Reference range (2,3)	Vitamin E (ppm) 0.8 – 1.2	Selenium (ppm) 0.08 – 0.3
Day 2	0.31	0.034
Day 5	1.55	0.25

normal (Table 1). Serum levels of vitamin E and selenium also became normal (Table 2). The calf had no difficulty in standing, walking, or running, but it was kept in the clinic to monitor the clinical response and observe the muscle enzymes. Nine days after presentation, the calf was discharged from the clinic. Two months later, the owner reported that the calf was normal and thrifty.

Nutritional muscular dystrophy (NMD) or white muscle disease is caused by a deficiency of vitamin E, selenium, or both (1). Dietary polyunsaturated fatty acids, unaccustomed exercise, and rapid growth are considered as precipitating factors (1,4). It occurs in all farm animal species, especially rapidly growing calves, lambs, kids, and foals (1). Both vitamin E and selenium are important in the protection of cellular membranes from free radicals, which cause peroxidation of the membrane lipids (4). Vitamin E is an antioxidant that decreases hydroperoxide formation and acts at the extracellular or intracellular level to scavenge free radicals (1,4). Selenium is an important biochemical component of the enzyme glutathione peroxidase, an intracellular enzyme that protects the cellular membranes and organelles from peroxidative damage (1,4). It inhibits and destroys the endogenous peroxides and, in conjunction with vitamin E, maintains the cellular membranes (1). When these mechanisms are inadequate, the cell membranes become physiologically defective, resulting in accumulation of calcium in and injury to the mitochondria (4). Injured mitochondria are then unable to maintain homeostasis, resulting in cell death or segmental necrosis (4). Clinical signs of NMD include stiffness, weakness, and recumbency (1). Diagnosis is usually based on clinical findings; elevated levels of muscle enzymes (CK and AST); low levels of vitamin E and selenium in the diet, tissue, and serum; and muscle degeneration (1,4). At necropsy, affected muscles are usually pale, and histological examination reveals hyaline degeneration and segmental necrosis (1,4). Congenital nutritional muscular dystrophy is rare and not well documented in calves, perhaps because the developing fetal calf is able to sequester sufficient selenium, so that, except under extremely deficient conditions, ample quantities of selenium are normally available at birth to prevent expression of congenital white muscle disease (5). In this case, the clinical findings were suggestive of the disease because of the recumbency and weakness, which may have predisposed to the hypothermia because the calf was unable to stand and suck. The slightly elevated heart and respiratory rates and the weak sucking reflex may have been an indicator of myocardial, diaphragmatic, and tongue involvement. The indication of the presence of blood on the urine dipstick could have been due to myoglobin, hemoglobin, or even intact red blood cells. In the presence of myopathy, it may have been myoglobin. The low levels of vitamin E and selenium in the blood strongly support a diagnosis of congenital muscular dystrophy. A muscle biopsy could have helped to confirm the diagnosis but was not done.

Congenital white muscle disease in the calf has been reported twice before (6,7). One case was diagnosed at necropsy in a calf a few days of age (6). However, no description of any clinical signs or response to the treatment was described. The other case was an 18-hour-old recumbent calf that was euthanized 2 h after presentation, when it developed dyspnea (7). The diagnosis was based on clinical signs, and biochemical and histopathological findings. No attempts at treatment were described. In our case, the calf responded well to supportive fluid therapy and treatment with vitamin E and selenium. The owner was asked to obtain blood samples from the dam of the calf and from a representative sample of calves of the same age for vitamin E and selenium determination, but he didn't comply. Early cases of white muscle disease can be treated with parenteral injection of vitamin E and selenium. It can be prevented by supplementation of vitamin E and selenium in the diet, or by strategic oral and/or parenteral vitamin E and selenium to pregnant dams or young animals on pasture (1). CVJ

References

1. Radostits OM, Gay CC, Blood DC, Hinchcliff KW. Veterinary Medicine: A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats and Horses, 9th ed. London: WB Saunders, 2000:1515–1533, 1819–1822.
2. Puls R. Mineral Levels in Animal Health, 2nd ed. Clearbrook: Sherpa Int, 1994:230–231.
3. Puls R. Vitamin Levels in Animal Health, 2nd ed. Clearbrook: Sherpa Int,1994:98–99.
4. Carlton WW, McGavin MD. Thomson's Special Veterinary Pathology, 2nd ed. St. Louis: Mosby, 1995:417–419.
5. Koller LD, Whitbeck GA, South PJ. Transplacental transfer and colostral concentrations of selenium in beef cattle. Am J Vet Res 1984;45:2507–2510.
6. Lohr JE, Wouden MV. Congenital white muscle disease in a calf. N Z Vet J 1971;19:222–223.
7. Cawley GD, Bradley R. Presumed congenital myodegeneration in a calf. Vet Rec 1979;105:398–399.