J. Vet. Malaysia (1999) 11(2): 99-102

CASE REPORT

NUTRITIONAL MUSCULAR DEGENERATION IN CATTLE IN MALAYSIA: REPORT OF TWO CASES

M.M. Noordin, J. Shokri and A.R. Sheikh-Omar

Faculty of Veterinary Medicine,
Universiti Putra Malaysia,
43400 Serdang, Selangor, Malaysia

SUMMARY

Nutritional muscular degeneration is a muscular disorder due to selenium and vitamin E deficiency. Although claimed to be widespread, it has not been reported in Malaysia. This paper describes the finding of this disease in two Kedah-Kelantan calves based on gross and microscopic findings of myocyte degeneration and/or necrosis and on low blood glutathione peroxidase activity.

Keywords: Nutritional muscular degeneration, selenium, vitamin E

INTRODUCTION

Nutritional muscular degeneration (NMD) (white muscle disease, nutritional myopathy or nutritional muscular dystrophy) is a nutritional disease in young farm animals associated with the deficiency of selenium (Se), vitamin E or both (Muth *et al.*, 1958). It is characterised by weakness of rear leg, buckling of fetlocks and frequently generalised shaking or quivering of muscles. The clinical signs seen are usually associated with the degeneration and/or necrosis of the hindlimb muscles, diaphragm and the heart (McGavin, 1995). This paper reports the findings in two calves with NMD and is believed to be the first documentation in Malaysia.

CASE HISTORY

Two 10-12 months old Kedah-Kelantan (KK) bull calves were brought in for slaughter at the Universiti Putra Malaysia abattoir when it was found that they were unable to walk. In one of the cases, this happened two days after the calf was herded into the holding yard from the paddock. The calves were reported to have been the presumably lush pasture for a week after being on the previous overgrazed paddock for almost a month.However, in both cases, the main reason for them being brought in for slaughter was their extremely temperamental behaviour.

Although it had been claimed that they were not able to walk, the *ante-mortem* examination revealed that these animals were able to stand and walk for about a minute albeit with an unsteady due to the flexion of the fetlock and hock joints. Prior to slaughter, blood sample were taken from both calves via jugular venopuncture into heparinised tubes for blood glutathione peroxidase (GSH-Px) determination according to the direct method using dinitro-bi-nitrobernzoic acid (DTNB) as described by Zhang *et al.* (1997).

POST-MORTEM FINDINGS

After exsanguination and dressing of the carcass, almost similar lesions were observed in both animals. The semi-tendinosus, semi-membranosus, bicep femoris, gluteobiceps and the gastrocnemius muscles were all pale resembling the appearance of fish flesh. These bilateral symmetrical lesions extended deep into the muscles. The musculatures of the heart, intercostal and diaphragm were grossly normal. Samples of the above mentioned muscles along with that of the heart, intercostal and the diaphragm were fixed in 10% formalin for histopathologic examination. In addition, samples of the kidney were also taken for microscopic examination since capture myopathy is also a differential in this case.

Histologically, it was found that the mentioned hind limb muscles and the gastrocnemius were undergoing degeneration and necrosis at different stages. This includes hyalinization, vacuolar and floccular degeneration of the myocytes along with calcification (Fig. 1). No significant abnormalities were seen in the musculatures of the heart, intercostal and the diaphragm.

The microscopic feature of the kidneys was within normal limits without any evidence of myoglobinuric nephropathy nor hyaline cast.

BLOOD GSH-Px ACTIVITY

The concentrations of blood GSH-Px in both calves were between 10 and 12 U/mL whole blood respectively.

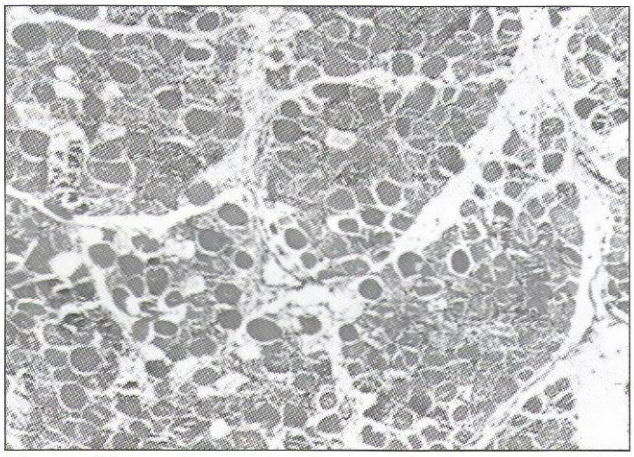

Fig. 1. Photomicrograph of a muscle bundle depicting evidence of hyalinisation, vacuolar and floccular degeneration and calcification. The intersititum, however, remain unchanged. HE x125

DISCUSSION

Two features of NMD has been described in ruminants (McDowell *et al.*, 1993). Firstly, the congenital form which is extremely rare whereby the calves are stillborn or die within a few days of birth. Secondly, dubbed as the delayed form (skeletal type) is more commonly seen in calves between the age of 1 to 4 months or older. This latter form is the one that is being described in the case presented here, which usually leads to weakness and recumbency.

As reported here, the gross and histological lesions are consistent with Se and vitamin deficiency (McDowell *et al.*, 1993). This is further supported by the absence of renal lesions of capture myopathy and the low concentration of whole blood GSH-Px activity. In a state of deficiency to adequacy, the activity of GSH-Px is a sensitive and reliable indicator of Se status. Values of GSH-Px activity between 0-15 U/mL, 15-25 U/mL and more than 25 U/mL whole blood is regarded as deficient, marginal and adequate respectively (Gerloff, 1992). Thus, the concentrations of GSH-Px activity in the two calves are within the deficient range.

Diets deficient in Se and/or vitamin E permit widespread myocyte lipid peroxidation leading to Zenker's necrosis and calcification of muscle fibres (McGavin, 1995). Undoubtedly, the presence of degeneration and/or necrosis of muscles in the two calves signifies NMD. Nevertheless, the lesion in the group of the thigh and that of the gastrocnemius can explain the lack of support leading to flexion of the fetlock and hock joints or the unsteady gait observed in both calves.

It is not suprising for these to be the first NMD cases to be reported in this country since the field of nutritional pathology is one that is most neglected (Noordin, 1996). The authors strongly believed that NMD is widespread albeit more as the subclinical form (illthrift) due to soil condition, management practices and the growth rate of ruminants (especially the local breed).

Soils in this region of Malaysia are often acidic (Kanapathy, 1976) which can lead to a decrease in the bioavailability of Se to animals since low pH decreases the uptake of Se by pastures (Ylaranta, 1990).

Deficiency in vitamin E could have resulted from management practices. The animals in this case were recently shifted from a overgrazed pasture and end stage growth pasture into a lush pasture paddock. Pastures at the end stage of growth in the Malaysian hot climate usually have low vitamin E content (McDowell *et al.*, 1993) while lush pasture has high concentrations of linolenic acid (C18:3) (Noordin, 1986). The high concentrations of C18:3 can be detrimental since linolenic acid has the highest peroxidizability rate compared to other essential fatty acids (McMurray *et al.*, 1980; 1983). In the case presented here, this will enhance peroxidation of muscles that are already deficient in Se and vitamin E.

Although there is a complex interaction between Se and vitamin E such that each can spare or alter the requirement of the other, unfortunately they cannot completely replace each other (McDowell *et al.*, 1993). Likewise in the Malaysian context, this supplementary interaction could never exist since both elements are under deficient state due to the above mentioned conditions.

It seem plausible that the growth rate of the local breed of animals or in general other breeds reared in Malaysia contributed to the rare occurrence of clinical NMD. The clinical disease is usually seen in very young rapidly growing animals (Pehrson, 1993). The KK breed of cattle is known to have a rather slow growth rate (Hilmi and Yahya, 1993) rendering it difficult to see evidence of clinical NMD. Nevertheless, this breed of cattle and possibly other ruminants in this country could have the subclinical form of the disease (Noordin, 1996).

Unaccustomed exercise (Arthur, 1982: Pehrson *et al.*, 1986) and excitable temperaments (McDowell *et al.*, 1993) can accelerate the oxidative process and precipitate the clinical signs. In both cases, the animals were known to be very temperamental and in one this happened following herding which is analogous to unaccustomed exercise. Thus, temperamental behaviour and unaccustomed exercise might have also contributed to the development of clinical NMD in the case presented here.

Although not an objective of this paper, a brief mention on the diagnosis, treatment and prevention of NMD would be meaningful. Diagnosis can be based on

serum creatinine kinase (indicating muscular damage) (Smith *et al.*, 19940, GSH-Px activity (indicating Se status) (Thompson *et al.*, 1981) and the concentration of Se and vitamin E in tissues and feed (Campbell, 1992). Muscle biopsy especially that of the bicep femoris is a reliable diagnostic tool for NMD (McGavin, 1995).

Pehrson (1993) suggested that affected animals should be isolated and that undue muscular exertion should be avoided. Intramuscular or subcutaneous injections of Se and vitamin E should be given and this could be followed by additional vitamin E orally for the next 3 to 4 days.

Depending on the status of the herd in question, supplementation of Se and vitamin E can be given either through parenteral routes or fortification of the diet and salt licks. Liming (Gissel-Nielsen, 1986) and fertilising with Se (Ylaranta, 1990) will help to increase the uptake of Se by pastures. Calves supplemented with α-tocopherol or Se were protected against NMD (McMurray and McEldowney, 1977).

REFERENCES

Arthur, J.R. (1982). Nutritional inter-relationship between selenium and vitamin E. *Rep. Rowett Inst.* **38**: 124-135.

Campbell, A.D. (1992). A crtiical survey of hydride generation technique in atomic spectroscopy. *Pure Appl. Chem.* **64**: 227-244.

Gerloff, B.J. (1992). Effect of selenium supplementation on dairy cattle. *J. Animal Sci.* **70**: 3934-3940.

Gissel-Nielsen, G. (1986). Comparisons of selenium treatment of corps in the fields. *Biol. Trace. Elem. Res.* **10**: 209-213.

Hilmi, M. and Yahya, M. (1993). Beef and feedlot production: constraint and prospects. In: Animal Industry in Malaysia. Fatimah, C.T.N.I, Ramlah, A.H. and Bahaman, A.R. (Eds), Faculty of Veterinary Medicine and Animal Sciences, Malaysia. pp 31-40.

Kanapathy, K. (1976). Guide to fertiliser use in Peninsular Malaysia. Ministry of Agriculture, Kuala Lumpur. 141p

McDowell, L.R., Conrad, J.H. and Glen Hembry, F. (1993). Minerals for Grazing Ruminants in Tropical Regions, 2nd Edition, University of Florida, Gainsville. pp78.

McGavin, M.D. (1995). Muscle. In: Thompson's Special Veterinary Pathology. Carlton W.W. and McGavin, M.D. (Eds) 2nd Edition, Mosby-year Book Inc., St. Louis. pp 393-422.

McMurray, C.H. and McEldowney, P.K. (1977). A possible prophylaxis and model for nutritional degenerative myopathy in young cattle. *Br. Vet. J.* **133**: 535-542.

McMurray, C.H., Rice, D.A. and Blanchflower, W.J. (1980). Changes in plasma levels of linoleic and linolenic acids in calves recently introduced to a spring pasture. *Proc. Nutr. Soc.* **39**: 65A.

McMurray, C.H., Rice, D.A. and Kennedy, S. (1983). Nutritional myopathy in cattle: from a clinical problem to experimental models for studying selenium and polyunsaturated fatty acids interactions. *In;* Trace Elements in Animal Production and Veterinary Practice. Occasional Publication No. 7, Brit. Soc. Anim. Prod. pp61-73.

Muth, O.H., Oldfield, J.E., Remmert, L.F. and Schubert, J.R. (1958). Effects of selenium and vitamin E on white muscle disease. *Science* **128:** 1090.

Noordin, M.M. (1986). The plasma fatty acid profiles of Kambing Kacang, DVM Final Year Project Paper, Faculty of Veterinary Medicine and Animal Sciences, Universiti Pertanian Malaysia. 26 p

Noordin, M.M. (1996). The status of trace element research in Malaysia: problems and solution. *ACIAR Proc.* **73**: 72-77.

Pehrson, B. (1993). Diseases and diffuse disorders related to selenium deficiencies in ruminants. *Norwegian J. Agric. Sci. Suppl* **11**: 78-93.

Pehrson, B., Hakkarainen, J. and Tyopponen, J. (1986). Nutritional muscular degeneration in young heifers. *Nord. Vet. Med.* **38**: 26-30.

Smith, G.M., Fury, J.M., Allen, J.G. and Costa N.D. (1994). Plasma indicators of muscle damage in a model of nutritional myopathy in weaner sheep. *Aust. Vet. J.* **71**: 12-17.

Thompson, K.G., Frazer, A.J., Harrop, B.M., Kirk, J.A., Bullians, J. and Cordes, D.O. (1981). Glutathione peroxidase activity and selenium concentrations in bovine blood and liver as an indicator of selenium intake. *N.Z. Vet. J.* **29**: 3-6.

Ylaranta, T. (1990). Effects of liming and addition of sulphate and phosphate on selenium content of Italian rye grass. *Ann. Agric. Fenn.* **29**: 141-149.

Zhang, S.Z., Wang, X.Y. and Qi, Z.M. (1997). Studies on the mechanism and its effects of selenium and vitamin E in the free radicals metabolism of animals with selenium deficiency. *Husb. Vet. Sci.* **28**: 23-27.

RINGKASAN

PENYAHJANAAN OTOT PEMAKANAN PADA LEMBU DI MALAYSIA: LAPORAN DUA KES

Penyahjanaan otot pemakanan ialah suatu gangguan otot disebabkan kekurangan selenium dan vitamin E. Walaupun penyakit ini dikatakan berleluasa, ianya belum dilaporkan di Malaysia. Kertas ini menghuraikan penemuan penyakit ini dalam dua ekor anak lembu Kedah-Kelantan berasaskan penemuan kasar dan mikroskopi penyahjanaan miosit dan/atau nekrosis dan pada aktiviti glutation peroksidase darah.