羊の大脳皮質壊死症の一例

誌名	Japanese journal of veterinary science
ISSN	00215295
著者	清宮, 幸男 伊藤, 博 大島, 寛一
巻/号	51巻5号
掲載ページ	p. 1075-1077
発行年月	1989年10月

A Case of Cerebrocortical Necrosis in a Sheep

Yukio SEIMIYA, Hiroshi ITOH, and Kan-ichi OHSHIMA[1)]

Morioka Livestock Hygiene Service Center, Takizawa-mura, Iwate-gun, Iwate 020-01, and [1)]Department of Veterinary Pathology, School of Veterinary Medicine, Faculty of Agriculture, Iwate University, Morioka 020, Japan

(Received 17 September 1988/Accepted 6 July 1989)

Jpn. J. Vet. Sci. 51(5): 1075–1077, 1989

KEY WORDS: cerebrocortical necrosis, sheep.

Cerebrocortical necrosis (CCN), also called polioencephalomalacia, is a neurological disease of cattle and sheep characterized pathologically by disseminated necrosis throughout the cerebral cortex [1, 5–10, 12, 19, 22–25]. Sheep suffering from the disease range in age from 6 weeks to 7 years or more, but the majority of them are lambs 8 to 16 weeks of age [23]. The exact cause of the disease remains obscure, but affected animals often respond to parenteral administration of thiamine in respect of rapid alleviation of clinical signs [2, 20, 21]. There are a few reports on the disease of sheep [7, 25] as well as cattle [9, 19, 22] in Japan. In the present paper, pathological findings of a case of the disease in a sheep will be described.

Three adult wethers had been fed several kinds of feeds as shown in Table 1 at 2- or 4-week intervals for about 3 months under some feeding experiment since January 19, 1987. On April 8, one of them, a 6-years-old coridale developed sudden clinical abnormalities such as anorexia, inability to stand, diminished vision, and intermittent convulsions, and on the next day showed lateral recumbency and coma preceding to death. Temperature was within the normal range. The other two wethers showed no abnormal symptoms. The feeds having been given were of proper nutritional components for those animals.

At autopsy, there were congestion and scattered petechiae in the leptomeninges. The cerebrospinal fluid increased in quantity without any changes in quality. The surface of the hemispheres was somewhat edematous. Frontal sections of the cerebrum revealed multiple focal areas showing yellowish discoloration throughout the cortex of the hemispheres, predominantly in the parietal and occipital lobes. The ventriculus dexter dilatated in the heart.

After autopsy, tissue specimens from the organs and tissues from the whole body were fixed in 10% neutral formalin solution, and the paraffin-embedded sections were stained with

Table 1. Feeds given to each sheep

Feeding period (1987)	Feed	Quantity/day (kg)
Jan. 19—Feb. 15	Wooden feeds[a)]	0.6
	Hay cube	0.9
Feb. 16—Mar. 15	Silaged rice straw with supplement of 2% urea	0.44
	Corn silage	2.0
Mar. 16—Mar. 30	Silaged wooden feeds	0.8
	Silaged alfalfa	1.8
Mar. 31—Apr. 8	Silaged wooden feeds	0.8
	Silaged orchard grass	1.7

a) Prepared by steamed wooden chips of white birch tree.

Fig. 1. There are neuronal ischemic change, edematous perineuronal and perivascular spaces, and looseness of neuropil in the superficial and deeper cortex of the cerebral parietal lobe. LFBstain. ×40.

hematoxylin and eosin (H-E), luxol fast blue (LFB) and azan.

Histologically, multiple laminar or focal spongy areas were observed in the cerebral cortex. The spongy changes were more widespread in distribution in the parietal and occipital lobes and more severe in degree in the superficial and deeper layers of the cortex.

In the spongy areas, were observed ischemic changes of neurons, edematous perineuronal and perivascular spaces, and loosening of neuropils (Fig. 1). The neurons were shrunken and angular in outline with acidophilic cytoplasms, and their nuclei were shrunken and pyknotic with scarcely recognizable nucleoli. The neurons were stained homogeneously deep blue with LFB stain. Capillary endothelial cells enlarged mildly in degree, and the walls of the several arterioles showed loosening suggestive of edema.

The telencephalic white matter showed congestion and was spongy just beneath the cortex. There were pronounced congestion, scattered small perivascular hemorrhages, and a few small round cells in the leptomeninges.

Several Purkinje cells were swollen and had pale-staining cytoplasms in the cerebellum. Mild perivascular small round cell infiltration and degeneration of a few neurons were seen in the brain stem. No changes were found in any sections of the spinal cord. There were focal mildly fibrotic lesions in the myocardium.

The essential changes observed here were multiple laminar or focal necrosis of the cerebral cortex. The changes closely resembled those described previously in cattle and sheep [5, 7–10, 19, 22, 24, 25], and in particular those of sheep which died within 24 hours of the onset of clinical signs [24]. The changes appeared to express the initial or middle stage of the disease, since the earliest pathological evidence in the affected brain is said to be edematous changes followed by neuronal degeneration [17, 18]. Mild degeneration of Purkinje and granular cells of the cerebellum has been often recognized in the spontaneous cases [5, 7–10, 19], but it has been understood to be nonspecific [5].

It has been shown that uncomplicated thiamine deficiency does not produce CCN [3, 11]. The lesions indistinguishable from field cases of CCN have been produced experimentally in calves [15, 16] and sheep [13, 18] by administration of Amprolium, a structural analogue of thiamine and a competitive antagonist. Significant amounts of thiaminase has been demonstrated in the rumen contents of natural cases [4, 14, 21]. Thiaminase activity has been detected in various feeds for bovine or ovine [3]. It has been suggested that CCN may be resulted from substances closely resembling Amprolium produced owing to the action of thiaminase on thiamine in the rumen [3]. The effects of Amprolium on the brain have been prevented by concomitant administration of thiamine [15] and accentuated by thiamine intake kept at a low level [18]. Therefore, occurrence or course of CCN has been suggested to be influenced by the relative quantitative proportion of thiamine to antimetabolite formed [15]. The frequent changes of the feeds within a short period, such as might have been followed by thiamine deficiency due to alteration of bacterial flora in the rumen, might have been involved in the present disease.

REFERENCES

1. Bajmocy, E., Fazekas, B., and Glavits, R. 1987. *Magy. Ao. Lapja* 42: 459–465.
2. Davies, E. T., Pill, A. H., Collings, D. F., and Venn, J. A. J. 1965. *Vet. Rec.* 77: 290.
3. Edwin, E. E., Lewis, G., and Allcroft, R. 1968. *Vet. Rec.* 83: 176–178.
4. Edwin, E. E., Spence, J. B., and Woods, A. J. 1968. *Vet. Rec.* 83: 417.
5. Elliott, G. A. 1958. pp. 75–92. *In*: Veterinary Extention Quarterly, No. 150, Univ. Pennsylvania, Harrisburg.
6. Harris, A. H. 1962. *Vet. Rec.* 74: 307–371.
7. Hosokawa, S., Ohshima, K., Miura, S., and Ito, T. 1971. *J. Fac. Agric, Iwate Univ.* 10: 139–155 (in Japanese with English summary).
8. Howell, J. McC. 1961. *Vet. Rec.* 73: 1165–1168.
9. Ishikawa, S., Ito, T., Uemura, U., Yamamoto, Y., and Nomura, Y. 1986. *J. Jpn. Vet. Med. Assoc.* 39: 233–238 (in Japanese with English summary).
10. Jensen, R., Griner, L. A., and Adams, O. R. 1956. *J. Am. Vet. Med. Assoc.* 129: 311–321.
11. Lewis, G., Terlecki, S., Markson, L. M., Allcroft, R., and Ford, J. E. 1967. *Proc. Nutr. Soc.* 26: xiii-xiv.
12. Little, P. B. and Sorensen, D. K. 1969. *J. Am. Vet. Med. Assoc.* 155: 1892–1903.
13. Loew, F. M. and Dunlop, R. H. 1972. *Am. J. Vet. Res.* 33: 2195–2205.
14. Loew, F. M., Dunlop, R. H., and Christian, R. G. 1970. *Can. Vet. J.* 11: 57–61.
15. Markson, L. M., Lewis, G., Terlecki, S., Edwin,

E. E., and Ford, J. E. 1972. *Br. Vet. J.* 128: 488–498.
16. Markson, L. M., Terlecki, S., and Lewis, G. 1966. *Vet. Rec.* 79: 578–579.
17. Morgan, K. T. 1973. *J. Pathol.* 110: 123–130.
18. Morgan, K. T. 1974. *J. Pathol.* 112: 229–236.
19. Ohshima, K., Sato, T., Yamanome, Y., Miura, S., and Numakunai, S. 1977. *Jpn. J. Vet. Sci.* 39: 415–423.
20. Pill, A. H. 1967. *Vet. Rec.* 81: 178–181.
21. Roberts, G. W. and Boyd, J. W. 1974. *J. Comp. Pathol.* 84: 365–374.
22. Seimiya, Y., Obara, T., Tanaka, S., Kawamukai, N., Ohshima, K., and Okada, K. 1988. *J. Jpn. Vet. Med. Assoc.* 41: 104–107 (in Japanese with English summary).
23. Spence, J. B., Stevens, A. J., and Saunders, C. N. 1961. *Vet. Rec.* 73: 28–34.
24. Terlecki, S. and Markson, L. M. 1961. *Vet. Rec.* 73: 23–27.
25. Yamagiwa, S. and Tajima, M. 1952. *Memoirs Fac. Agric. Hokkaido Univ.* 1: 145–158 (in Japanese with German summary).

要　　約

羊の大脳皮質壊死症の一例(短報)：清宮幸男・伊藤　博・大島寛一[1)](盛岡家畜保健衛生所，[1)]岩手大学農学部)——6歳齢の去勢羊が起立不能，視力障害，間欠性痙攣を示し死亡した．肉眼的に，大脳皮質領域に巣状の帯黄色病巣の多発が両側性に観察された．組織学的に，大脳皮質領域に神経細胞の乏血性変化，血管および神経細胞周囲の水腫性拡張および神経網の疎性化が認められた．発症までの3か月に行われた頻繁な飼料の切り替えが本症発生にかかわっていたのではないかと疑われた．