

## Listeric Septicemia with Meningitis in a Neonatal Calf

Yukio SEIMIYA, Kan-ichi OHSHIMA<sup>1)</sup>, Hiroshi ITOH, and Ryu-koh MURAKAMI

Morioka Livestock Hygiene Service Center, Takizawa-mura, Iwate-gun, Iwate 020-01 and <sup>1)</sup>Iwate Veterinary Medical Association, Morioka 020, Japan

(Received 26 May 1992/Accepted 25 July 1992)

**ABSTRACT.** A 9-days-old calf which had exhibited depression and difficulty to stand and walk was examined pathologically and bacteriologically. The primary pathological changes consisted of multifocal necrosis in several visceral organs and fibrinopurulent meningitis. The necrotic lesions were most frequently found in the liver, and accompanied with mononuclear cell infiltration and Gram-positive small bacilli. The organisms were also present in the foci of mononuclear cells at the central gray matter of the mesencephalon. *Listeria monocytogenes* was isolated from the brain and other organs of the whole body.—**KEY WORDS:** listeric septicemia, meningitis, neonatal calf.

—J. Vet. Med. Sci. 54(6): 1205–1207, 1992

Listeriosis is of worldwide distribution, possibly excepting the tropics [19]. The causal organism, *Listeria monocytogenes*, has been isolated from a wide range of diseased animals and birds as well as man [5]. The disease behaves as three separate syndromes [19]; infection of the pregnant uterus with subsequent abortion [15–17, 24], septicemia with miliary visceral abscesses in fetuses or neonates up to about 2 weeks of age [4, 6, 8, 23], and encephalitis apparently in older animals than neonates [1, 2, 10, 14, 20–22]. Listeric septicemia in neonates may be a continuation of intrauterine infection in most cases, since the visceral infection of the organisms in neonates is also the pattern in affected fetuses [19]. Invasion of the organisms into the central nervous system (CNS) causing meningitis [3, 4, 11] or meningoencephalitis [12] occasionally occurs in neonates with septicemia. In Japan, there are several reports on listeric encephalitis in goats [10, 20], sheep [18, 21, 22], and cattle [14], and on the septicemia in a piglet [9] and a bovine stillborn fetus [7]. Furthermore, an occurrence of the septicemia with meningitis was also observed in a neonatal piglet [4]. However, there are few reports on the septicemic cases with meningitis in neonatal calves. The purpose of this report is to describe pathological and bacteriological findings in a neonatal calf suffering from listeric septicemia with meningitis.

A calf of the Nippon Short Horn breed was born at full-term after dystocia in February, 1987. The animal had exhibited depression, anorexia, difficulty to stand and walk, and subcutaneous edema of the whole body since the birth. The clinical condition was getting worse day by day. The animal became comatose at the age of 8 days and died on the following day. No medical treatment had been tried during the disease course, apart from topical application of iodine tincture to the umbilical cord. The dam had fed on corn silage and rice straw since the fall of the previous year, and repeated vaginocoele within a one-month period before the parturition. The subsequent estrus and pregnancy were normal.

The primary macroscopical changes consisted of systemic circulatory disturbance, subcutaneous edema, and miliary necrotic lesions in the liver and heart. The lesions ranging from 1 to 3 mm in diameter were disseminated throughout the hepatic parenchyma, and sporadically

found in the myocardium. Both sides of the cardiac ventricles and atria were prominently dilated, and the myocardium was flaccid. There was an increase of moderately cloudy peritoneal, pleural, pericardial, and cerebrospinal fluids. Petechiae and cloudy areas were present in the meninges. Purulent omphalitis was observed.

Microscopically, there were numerous focal necroses at the peripheral to midzonal areas in the hepatic lobules. Fibrinous thrombi at the interlobular tissue, which were clearly demonstrated by phosphotungstic acid-hematoxylin staining, were occasionally associated with the necroses. The lesions were characterized by necrotic hepatic cells and karyorrhexic mononuclear cell infiltration with a few neutrophils (Fig. 1). Numerous Gram-positive small bacilli were scattered and clumped in the lesions. The larger lesions were sometimes formed by coalescence of several foci. Some lesions consisted of

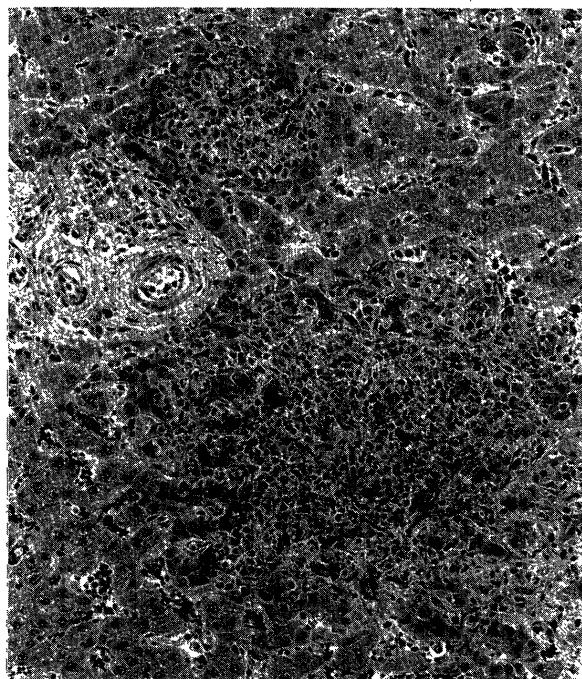


Fig. 1. Necroses with mononuclear cell infiltration at the peripheral area of the hepatic lobule. HE stain.  $\times 40$ .



Fig. 2. Congestion, fibrinopurulent exudate, and mononuclear cell infiltration are observed in the mesencephalic meninges. HE stain.  $\times 100$ .

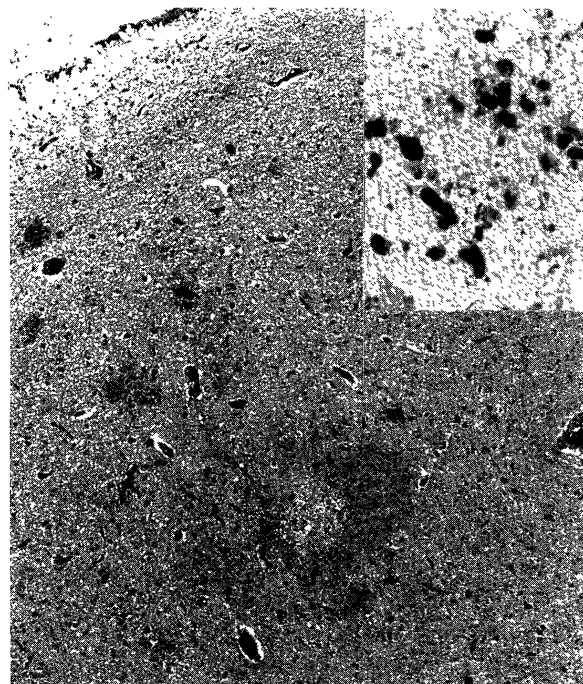


Fig. 3. Several cellular foci with mild loosening of neuropil are found in the central gray matter of the mesencephalon. Edematous changes are seen in the subependymal areas of the aqueduct. HE stain.  $\times 40$ . Inset shows Gram-positive small bacilli in the lesion. Gram stain.  $\times 400$ .

several degenerated hepatic cells and clumps of Gram-positive small bacilli. Lesions similar to those in the liver were frequently found in the myocardium, spleen, lungs, wall of the digestive tract, and several lymph nodes, and occasionally in the kidneys, pancreas, thyroid glands, and urinary bladder. There were focal embolic glomerulonephritis, fibrinopurulent peritonitis, epicarditis, and pleuritis. Serofibrinous exudate and diffuse mononuclear cell infiltration were recognized in the subcutis. Fibrinous thrombi or vasculitis characterized by mononuclear cell infiltration with numerous Gram-positive small bacilli on the vascular wall were often associated with the lesions.

Meningitis was found throughout the CNS, and several cellular foci in the mesencephalic parenchyma. Meningitis was especially severe in the mesencephalon, medulla oblongata, and pons. There were congestion and mild perivascular hemorrhages as well as fibrinous exudate and mononuclear cell infiltration with some neutrophils (Fig. 2). Thrombosis and vasculitis with numerous Gram-positive small bacilli were sometimes seen in the affected areas. Endovasculitis characterized by mononuclear cell infiltration at the intima was present frequently in the meninges of the mesencephalon and pons, and occasionally in the other parts. Mononuclear cell infiltration and occasional epithelial desquamation were found in the choroid plexuses. Subependymal edema and occasional desquamation of ependymal cells with a few mononuclear cells were also observed in the ventricular walls.

Several foci of mononuclear cells with a few neutrophils were located at the central gray matter of the mesencephalon, where congestion, degeneration of neurons, mild loosening of neuropil, and multiplication of Gram-positive small bacilli were accompanied (Fig. 3). Mild perivascular mononuclear cell infiltration with serofibrinous exudate was sometimes present throughout the CNS. There was fibrinopurulent exudate in the meninges covering the hypophysis and perineural tissues of the trigeminal ganglia. Congestion and fibrinous thrombi were recognized in the optical retina and choroidea.

The liver, spleen, kidneys, heart, lungs, subcutaneous lymph nodes, subcutis, and brain were cultured aerobically on tryptic soy agar supplemented with 5% sheep blood, or anaerobically on GAM agar using a gas generating kit (Oxoid Limited, England) at 37°C for 48 hr. Both aerobic and anaerobic cultures yielded *Listeria monocytogenes* from all materials examined.

The primary pathological findings observed here consisted of multifocal necrosis in several visceral organs and fibrinopurulent meningitis. Gram-positive small bacilli were detected in the lesions, and *Listeria monocytogenes* was isolated from the organs and tissues of the whole body including the brain. These results suggest that the lesions were associated with infection of the organism and that the present case was in the process of septicemia. The present case may have been a continuation of intrauterine infection since the clinical abnormalities had been

observed in a short time after birth.

Thrombosis and vasculitis were found in both visceral organs and the CNS of the present case. The topographic pattern of the vascular lesions suggests that the causal organism may have reached the brain by hematogeneous route. The lesions of cerebral parenchyma were limited to several foci of mononuclear cells in the mesencephalon. The lesions are believed to be different from those of typical listeric encephalitis with pathognomonic distribution and lesions which are characterized by microabscess, focal gliosis, and perivascular cuffing in the brain stem, especially in the pons and medulla oblongata [1, 2, 10, 14, 18, 20–22]. The cellular foci as well as meningitis in the present case appear to have resulted from septicemia rather than an affinity to the nervous tissue of the causal organism. Ladds *et al.* [13] described a similar opinion on CNS lesions due to listeric infection in neonatal lambs.

Multifocal necrosis in the visceral organs, especially in the liver, is characteristic of listeric septicemia, but not pathognomonic [13]. The lesions were distributed in various visceral organs in the present case in comparison to those cases reported previously on the neonatal calves [6, 8, 11] or sheep [3, 12, 23] with septicemia. Jeleff and Djurov cited by Ladds *et al.* [13] reported that necrosis in the liver was found in young as well as older ovine fetuses, while the lungs, kidneys, and heart were predominantly involved only in younger animals. The distribution of the lesions in the present case suggests that the infection may have occurred at the early fetal age than the previous cases [6, 8, 11]. It is deeply suspected that the infection with the organism to the dam may have occurred at the time of vaginocoele observed within a one-month period before the parturition, and that an intrauterine infection to the fetus followed.

Very little is known on the epidemiology and pathogenesis of listeriosis [5, 19]. To define them, further studies will be needed for accumulation of the knowledge in the future.

**ACKNOWLEDGEMENTS.** We are grateful to Dr. Masanori Okutomo and Dr. Shūichi Tanaka in Morioka Livestock Hygiene Service Center for their bacteriological examination.

## REFERENCES

1. Charlton, K. M. and Garcia, M. M. 1977. *Vet. Pathol.* 14: 297–313.
2. Cordy, D. R. and Osebold, J. W. 1959. *J. Infect. Dis.* 104: 164–173.
3. Gitter, M. and Terlecki, S. 1965. *Vet. Rec.* 77: 11–15.
4. Goto, M., Itakura, C., and Eguchi, H. 1972. *Jpn. J. Vet. Sci.* 34: 173–178.
5. Gray, M. L. and Killinger, A. H. 1966. *Bacteriol. Rev.* 30: 309–382.
6. Gray, M. L., Lassiter, C. A., Webster, H. D., Huffman, C. F., and Thorp, F. Jr. 1956. *Vet. Med.* 51: 316–319.
7. Hara, F., Ueno, H., Shiraishi, T., Okada, Y., and Ohfuku, S. 1983. *J. Jpn. Vet. Med. Assoc.* 36: 205–209 (in Japanese with English summary).
8. Harbour, H. E. 1941. *Br. Vet. J.* 97: 401–407.
9. Hosoda, T., Asahi, O., Akiyama, Y., and Tubo, T. 1954. *J. Jpn. Vet. Med. Assoc.* 7: 493–497 (in Japanese).
10. Hyogo, H. and Kato, H. 1951. *J. Jpn. Vet. Med. Assoc.* 4: 338–339.
11. Jack, E. J. 1961. *Vet. Rec.* 73: 826 & 846.
12. Kidd, A. R. M. and Terlecki, S. 1966. *Vet. Rec.* 78: 453–454.
13. Ladds, P. W., Dennis, S. M., and Njoku, C. O. 1974. *Vet. Bull.* 44: 67–74.
14. Ohshima, K., Miura, S., Numakunai, S., and Chihaya, Y. 1974. *Jpn. J. Vet. Sci.* 36: 183–185.
15. Olson, C. Jr., Rollins, C. L., Bagdonas, V., Blore, I. C., and Segre, D. 1953. *J. Infect. Dis.* 93: 247–256.
16. Osebold, J. W., Kendrick, J. W., and Njoku-Obi, A. 1960. *J. Am. Vet. Med. Assoc.* 137: 221–226.
17. Osebold, J. W., Kendrick, J. W., and Njoku-Obi, A. 1960. *J. Am. Vet. Med. Assoc.* 137: 227–233.
18. Sugawa, A. and Miyairi, K. 1951. *J. Jpn. Vet. Med. Assoc.* 4: 80–83 (in Japanese).
19. Sullivan, N. D. 1985. pp. 286–288. In: *Pathology of Domestic Animals*, vol. 1, 3rd ed. (Jubb, K. V. F., Kennedy, P. C., and Palmer N. eds.), Academic Press, New York.
20. Tajima, M. 1950. *Jpn. J. Vet. Sci.* 12: 241–245 (in Japanese).
21. Tajima, M., Fujimoto, Y., and Ishiguro, M. 1951. *J. Vet. Med.* 56: 75–77 (in Japanese).
22. Tajima, M. and Nishihara, Y. 1953. *Jpn. J. Vet. Sci.* 15: 301–313 (in Japanese).
23. Watson, W. A. and Hunter, D. 1958. *Vet. Rec.* 70: 1189.
24. Young, S. and Firehammer, B. D. 1958. *J. Am. Vet. Med. Assoc.* 132: 434–438.