Clinicopathology of Meningoventriculitis due to *Streptococcus bovis* Infection in Neonatal Calves

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ABSTRACT. Three neonatal calves ranging in age from 4 to 14 days were examined pathologically and bacteriologically. The calves showed depression, anorexia, pyrexia, and difficulty or inability to stand followed by cloudiness of the ocular aqueous humor or cornea. Autopsy revealed congestion, petechiae, and cloudy areas in the meninges. Histologically, the central nervous system (CNS) lesions were prominent and limited to the meninges where fibrinous exudate and infiltrations of neutrophils, macrophages, and lymphocytes were present. There were mild or slight degrees of choroid plexitis and ependymitis. Endophthalmitis was seen as a concurrent lesion in all cases. Fibrinous or fibrinopurulent changes were found in the peritoneum and epicardium as well as in several other organs. Numerous Gram-positive cocci were detected in affected areas of the whole body. Bacteriologically, *Streptococcus bovis* was isolated from all examined materials consisting of the brain, cerebrospinal fluid, ocular aqueous humor, and several other organs. These results suggest that the lesions were associated with infection of the organism and that the present cases were in the process of septicemia.—KEY WORDS: endophthalmitis, meningoventriculitis, neonatal calf, *Streptococcus bovis*.

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Bacterial meningoventriculitis in neonatal ungulates is characterized by a peculiarly surface-related inflammation of the meninges, choroid plexuses, and ventricular walls with sparing of the underlying central nervous system (CNS) parenchyma [3]. The disease in neonatal ruminants is most often caused by *Escherichia coli* [2, 8, 11, 14], and occasionally by *Pasteurella* [7, 12] or *Streptococcus pneumoniae* [13]. However, it is believed that there are few reports on meningoventriculitis due to *Streptococcus bovis* infection. The purpose of the present report is to describe clinicopathological findings of 3 neonatal calves with meningoventriculitis associated with *S. bovis* infection.

MATERIALS AND METHODS

Three neonatal calves (Nos. 1–3) from 2 farms, 2 Nippon Short Horn and one Holstein-Friesian breeds, were examined in Iwate Prefecture in 1987 and 1990. All calves were born in March. Their ages were 4, 7 or 14 days at euthanasia (Nos. 1 and 2) or death (No. 3).

Pathological examination: At autopsy, specimens of the organs and tissues from the whole body were fixed in 10% neutral formalin. Paraffin sections were cut and stained with hematoxylin and eosin (HE), phosphotungustic acid-hematoxylin (PTAH), and a series of Gram staining.

Bacteriological examination: The liver, spleen, kidneys, heart, lungs, subcutaneous lymph nodes, brain, cerebrospinal fluid, and ocular aqueous humor were obtained at autopsy, and 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. Isolates were identified according to Bergey's Manual.

RESULTS

Clinical signs: Two calves (Nos. 1 and 2) made a little or no attempt to suck colostrum from their dams, and were depressed and anorectic since the birth. They showed pyrexia of about 40°C, coarse hair coat, difficulty or inability to stand at the age of 2 or 3 days, and were followed by bilateral cloudiness of the ocular aqueous humor on the next day. Calf No. 3 ingested colostrum at the birth, but showed similar clinical abnormalities to the others at the age of 11 days and bilateral corneal opacity at the age of 13 days. Lameness was not apparent in any case. The affected animals were fatal or died 2 to 4 days after the onset of the disease.

Pathological findings: Macroscopically, cerebrospinal fluid was cloudy and increased in amount.

There were congestion, petechiae, and cloudiness of the meninges. On the cut surface of eye balls, aqueous humors were cloudy and fibrinous exudate adhered to the surface of the ciliary body, iris or lens (Fig. 1). The cornea of one case (No. 3) was thickened and cloudy. Various amounts of fibrinous exudate was present on the peritoneal surface of all, and the pleural, epicardial, and endocardial surface of one case (No. 3). There were dark reddish lesions in the lungs (Nos. 1 and 3), hemorrhagic erosion in the rumen (Nos. 1 and 2), multiple necrotic foci in the liver (No. 1), and omphalitis (No. 1). Enlargement of the thyroid glands was found in 2 (Nos. 1 and 2) and atrophic thymus in all animals.

Histologically, the CNS lesions were limited to the meninges, choroid plexuses, and ventricular walls, and more prominent in the meninges. Marked meningitis was seen throughout the CNS of all cases. There were congestion, mild perivascular hemorrhages, fibrinous exudate, and infiltrations of neutrophils, macrophages, and lymphocytes (Fig. 2). Fibrinous thrombi were occasionally recognized in the affected meninges (Fig. 3), with an exception of those in the mesencephalon of calf No. 3 in which many vessels had thrombosis. Endovasculitis characterized by intimal cellular infiltration was frequently found throughout the CNS of one case (No. 2).

One or more of the choroid plexuses were mildly affected in all cases. The changes were similar in character to those of the meninges and accompanied by occasional epithelial desquamation. Small amount of fibrinopurulent exudate was trapped among the folds of the choroid plexuses. The lesions of the ventricular wall were also mild or slight in degree, and characterized by sporadic and focal

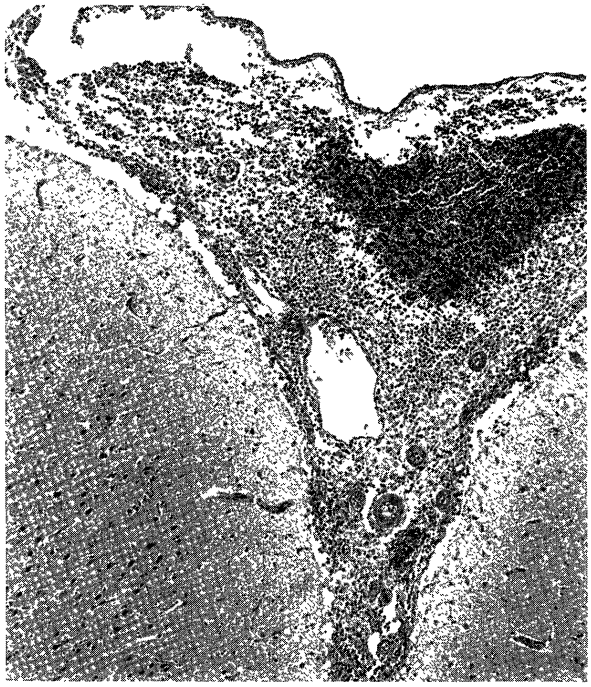

Fig. 2. There is large amount of fibrinopurulent exudate in the leptomeninges of frontal lobe of case No. 3. HE stain. × 40.

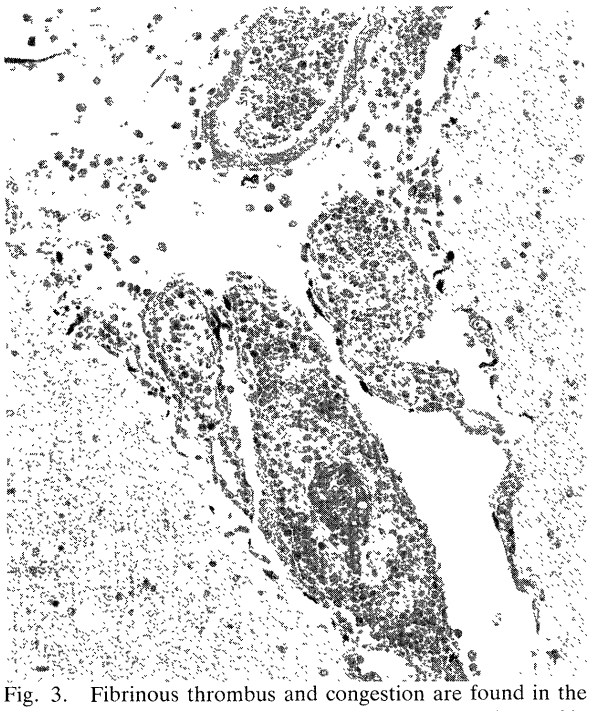

Fig. 3. Fibrinous thrombus and congestion are found in the leptomeninges of cistrial lobe of case No. 2. HE stain. × 100.

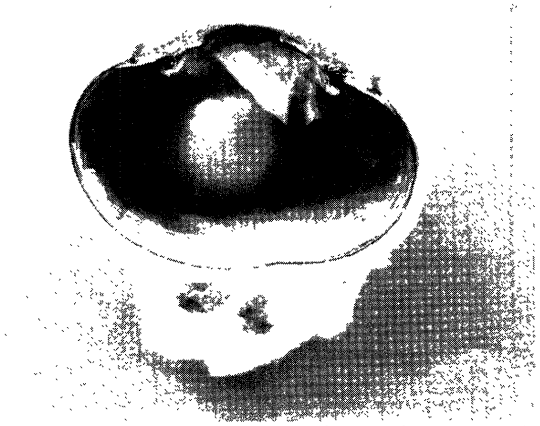

Fig. 1. Gross longitudinal section of eye of case No. 1. Fibrinous exudate adheres to the surface of the iris and lens.

desquamation of the ependymal cells with edema and neutrophil infiltration at the subependymal zone. The parenchymatous lesions in the CNS were limited to the superficial molecular layer and perivascular spaces in the deeper gray and white matters of the cerebrum and cerebellum. There were scattering of several neutrophils in the molecular layer and mild infiltrations of neutrophils and macrophages in the perivascular spaces. Fibrinopurulent exudate was also found in meninges covering the hypophysis and perineural tissues of the trigeminal ganglia.

Bilateral endophthalmitis was seen in all cases. The changes consisted of fibrinopurulent exudate in the ocular cavities, and congestion, edema, and infiltrated neutrophils in the stroma of the iris and ciliary body (Fig. 4). Small amount of neutrophils was also present in the retinal inner layer. There was congestion in the chorioidea. Fibrinopurulent exudate was found in the submeningeal space of the optic nerves and the orbital adipose tissue. The corneal proprial layer of one case (No. 3) was edematous and thickened accompaning by infiltrations of neutrophils and lymphocytes.

Fibrinous or fibrinopurulent lesions were present in the peritoneum of all, in the pleura and epicardium of 2 (Nos. 1 and 3), and in the endocardium of one case (No.3), respectively. Similar fibrinopurulent lesions were seen in several organs such as spleen, renal medulla, myocardium, lungs, parotid glands, and regional lymph nodes associated with the organs. Focal embolic glomerulonephritis was recognized in all cases. Purulent hepatitis with multifocal necrosis was found in one case (No. 1). There were rumenal erosions with thrombosis and vascular degeneration in two cases (Nos. 1 and 2) and mild catarrhal enteritis in all cases. In addition to the above purulent lesions, atrophic thymus was seen in all, and diffuse hyperplastic goiter, which was moderate in degree on the histological criteria previously described by us [10], in the thyroid glands of two cases (Nos. 1 and 2).

Numerous Gram-positive cocci were found in and around macrophages infiltrating into purulent lesions of the whole body (Fig. 5). Accumulations of the cocci were sometimes present in several organs or tissues with purulent lesions.

Bacterial isolation: Both aerobic and anaerobic cultures yielded many *S. bovis* colonies from all examined materials of all cases.

DISCUSSION

S. bovis was isolated from endocarditis of man [4]

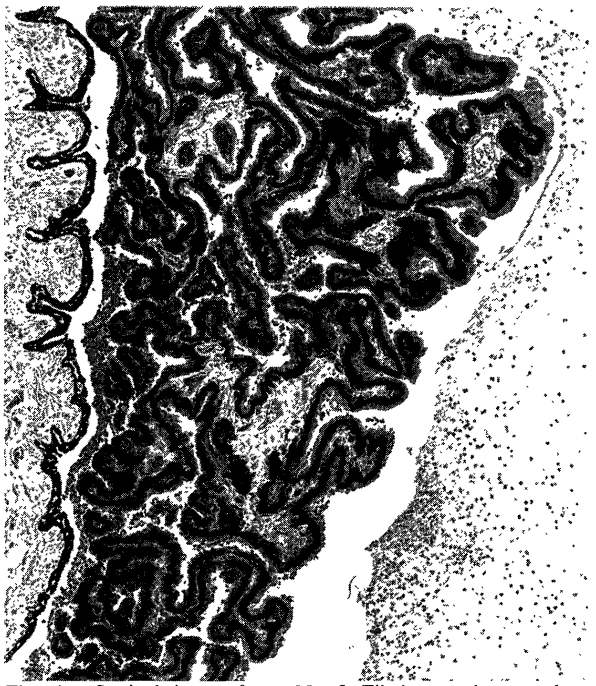

Fig. 4. Optical tissue of case No. 2. Fibrinopurulent exudate was present in the vitreous chamber and trapped among the folds of the ciliary processes. HE stain. × 40.

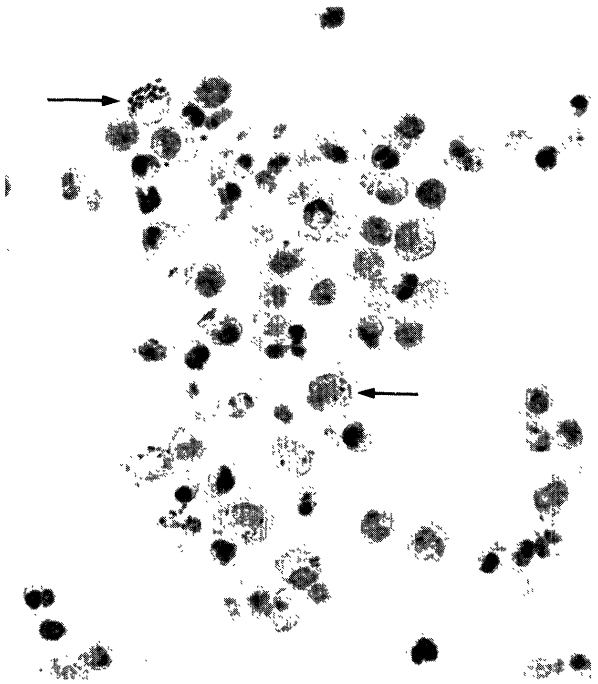

Fig. 5. Gram-positive cocci (arrows) were seen in the cytoplasm of macrophages infiltrated in the leptomeninges of parietal lobe of case No. 1. Gram staining. × 400.

and pigs [1], and from septicemia of piglets [5] and ducks [6]. These results seem to suggest that the organism can cause purulent lesions in various organs or tissues of the affected animals via blood stream. The principal pathological change observed in the present cases was fibrinopurulent meningoventriculitis, and concurrently accompanied by endophthalmitis as well as purulent lesions in several other organs or tissues. Numerous Gram-positive cocci were present in the lesions, and *S. bovis* was isolated from the brain, cerebrospinal fluid, ocular aqueous humor, and other organs. These results suggest that the lesions were associated with infection of the organism and that the present cases were in the process of septicemia.

The CNS changes in the present cases were characterized by fibrinopurulent inflammation in the meninges, choroid plexuses, and ventricular walls. The changes were similar in lesional distribution and character to those in neonatal ungulates [3] or calves [8, 11] with meningoventriculitis due to *E. coli* infection. Necrotic foci in the CNS parenchyma, which were sometimes or frequently seen in *E. coli*-infected neonatal ungulates [3] or calves [11], were not found in the present cases. The existence of thrombosed vessels in the affected meninges was occasional in the present cases, while common in the *E. coli*-infected cases [3, 11]. The absence of necrotic foci in the present CNS parenchyma may have been related to fewer thrombi in the present cases.

It is reported that the *streptococci* and *E. coli* behave differently with respect to production of the ocular lesions in neonatal calves [13]. The former frequently causes endophthalmitis, while the latter seldom does [13]. Endophthalmitis was seen in all of the present cases and corneal inflammation in one case (No. 3). The endophthalmitis seems to have resulted from systemic infection, while the corneal changes may have been attributable to secondary trauma.

The most common portal of entry for the bacterial invasion may be the alimentary tract in meningoventriculitis of neonatal ungulates due to *E. coli* infection [3]. Although the source of infection remains obscure in the present cases, the existence of omphalitis with multifocal necrosis in the liver in one (No. 1) and of rumenal erosion with thrombosis and vascular degeneration in two cases (Nos. 1 and 2) suggest that the umbilicus or rumenal mucosa may have been a possible portal of entry in those cases. Two (Nos. 1 and 2) of the present cases have been detected clinical abnormalities since the birth. The infection might have been a continuation of the intrauterine infection, although the weak calf syndrome-like signs might have been due to congenital diffuse hyperplastic goiter.

Schleifer [9] cited the result of Medrek and Barnes that *S. bovis* has many serotypes. Further study will be needed on the relationship between the pathogenicity and serotype of the organism in neonatal calves.

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