Eosinophilic Meningitis in Two Cows

Kazuyuki UCHIDA, Susumu TATEYAMA, Ryoji YAMAGUCHI, Mina ROSTAMI, Kimie KADOTA, Takashi HASEGAWA[1)], Hiroyuki OGAWA[2)], and Akihiro KUROKI[3)]

Departments of Veterinary Pathology, [1)]Veterinary Internal Medicine, and [2)]Veterinary Surgery, Faculty of Agriculture, Miyazaki University, Miyazaki 889-21, and [3)]Miyazaki Central Livestock Hygiene Center, Miyazaki 880, Japan

(Received 10 March 1993/Accepted 5 August 1993)

ABSTRACT. A neurological disorder was noted in 5 cows on a farm. Histopathological examination in 2 of these 5 animals revealed an infiltration of eosinophils with some macrophages in the meninges and around arterioles of the cerebral cortex. The endothelial cells of the reactive blood vessels were swollen. From these observations, the brain lesions were diagnosed as "eosinophilic meningitis". In one case, pulmonary arterioles were also infiltrated with eosinophils and macrophages in a granulomatous tissue. No bacteria, fungi, protozoa, or parasitic worms were detected in the present cases.—KEY WORDS: cattle, eosinophilic meningitis.

J. Vet. Med. Sci. 55(6): 1071–1072, 1993

Eosinophilic meningitis or meningoencephalitis has frequently been observed in cases of swine salt poisoning [2, 3, 6, 7]. Bovine salt poisoning is occasionally responsible for polioencephalomalacia without eosinophilic reaction [7]. Although an eosionophilic inflammatory reaction in the brain of cattle is commonly observed in several parasitic infections and rarely in some poisoning [1, 4, 7], such brain lesions due to causes other than parasites are thought to be very rare in cattle [2]. This paper describes the histopathology of an eosinophilic meningitis in 2 cows.

From April to May 1992, five 13-year-old Japanese black cows, raised on a farm in Miyazaki city showed depression and neurological signs of progressive hyporeflexia. They had been fed on fermented corn silage that smelled of butyric acid. Case No. 5 showed an abnormally high titer of serum creatinin phosphokinase (336 U/L). Serum ion levels such as Ca^{2+}, Na^{+}, K^{+}, and Cl^{-} were within normal range. The cows died or were killed within 5 days after the onset of the clinical symptoms.

Among 3 of the 5 cows autopsied, one showed severe post-mortem changes (case No. 3). In case No. 4, slight hemorrhage in the nasal cavity and small intestines was observed. Case No. 5 exhibited slight congestion and edema in the meninges of cerebral cortex and the lungs. In both cases, no parasitic worms were detected in either the visceral organs or brain. No bacteria was isolated from the cerebrospinal fluid of case No. 5.

For histopathological examination, tissue samples of case Nos. 4 and 5 were fixed in 10% formalin and paraffin sections were stained as follows; hematoxylin and eosin (HE), Hucker-Conn's Gram, May-Grunwald-Giemsa, Grocott's silver impregnation, periodic acid-schiff, and Ziehl Neelsen's acid-fast, Luna's staining for the granules of eosinophils, and avidin-biotin peroxidase complex (ABC) method (ABC kit, Vectastain, Vector Laboratories, Burlingame, CA., U.S.A.) using rabbit antisera against glial fibrillary acidic protein (GFAP, Dako, Capinteria, CA., U.S.A.) and factor VIII related antigen (Dako, Carpinteria, CA., U.S.A.).

In case No. 5, marked infiltration of eosinophils with some macrophages containing cell debris was seen in the meninges and around the arterioles of the cerebral cortex (Fig. 1). The granules of eosinophils were confirmed by Luna's staining. The neuropil in the subleptomeningeal layer exhibited mild edema. Immunostaining with anti-GFAP antibody revealed that a few swollen astrocytes were present in this area. The arterioles exhibiting an eosinophilic inflammation were markedly swollen (Fig. 2), and the endothelial cells stained intensely with the antiserum against factor VIII related antigen. Infiltration of eosinophils around the cortical capillaries was occasionally observed. The brain lesions described above were limited to the cerebral cortex. No bacteria, protozoa, fungi, or parasitic worms were found in each region of the brain examined.

Fig. 1. Massive infiltration of eosinophils and macrophages in the leptomeninges of cerebral cortex. The subleptomeningeal layer exhibits mild edema. HE. × 60.

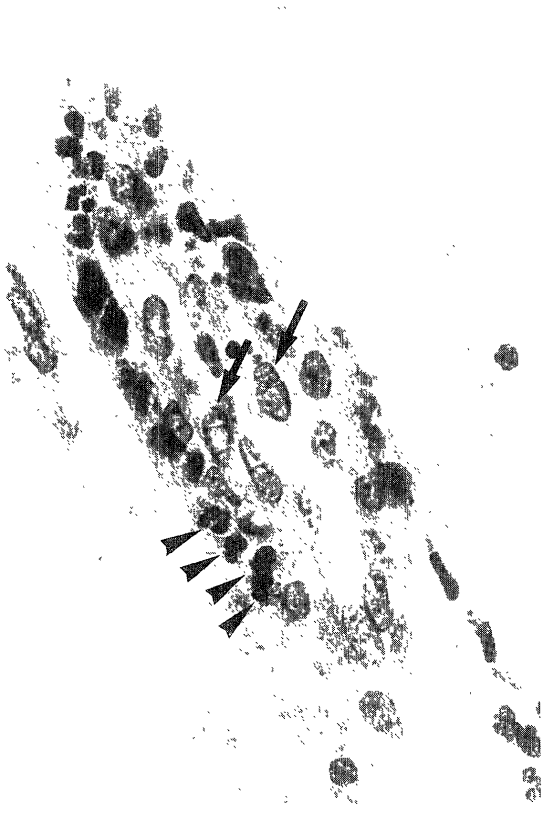

Fig. 2. Perivascualr cuffing with eosinophils (arrow heads) in the cerebral cortex. Endothelial cells (aroows) are markedly swollen. HE. × 480.

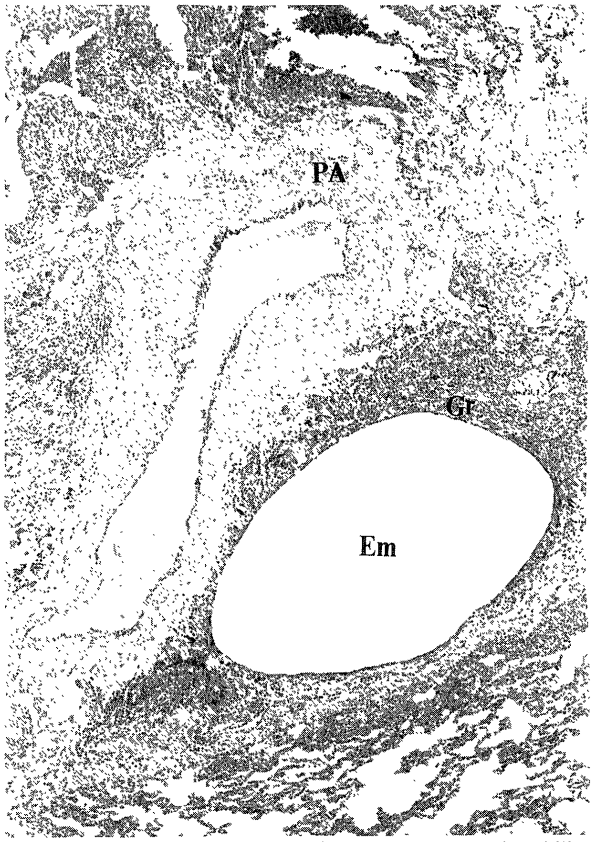

Fig. 3. Pulmonary arteriole (PA) with an eosinophilic inflammatory reaction. Around the vessel, interstitial emphysema (Em) surrounded with granulomatous tissue (Gr) is present. HE. × 60.

In the lung of case No. 5, interstitial emphysema surrounded granulomatous tissues was seen (Fig. 3). The endothelial cells of the arterioles in the lesion were swollen and detached. In addition, infiltration of eosinophils to the endothelial layer and subintimal tissues were noted. The granulomatous tissue consisted of macrophages, multinucleated giant cells and eosinophils. Slight accumulation of neutrophils in the mucosa of the rumen, chronic nephritis, hemosiderosis of the spleen, and slight degeneration of hepatocytes were also observed, respectively.

In case No. 4, infiltration of eosinophils and macrophages was obvious in the meninges and around arterioles of the cerebral cortex. In this case, hemorrhagic foci were scattered in the cerebral cortex. In the lungs, there was purulent aspiration pneumonia, and no eosinophils were recognized.

From these histopathological findings, the brain lesions reported in this paper were diagnosed as "eosinophilic meningitis". Bovine eosinophilic meningoenchephalitis has occasionally been described, although the cause has been unknown as in the present cases [2]. Eosinophilic meningoencephalitis is pathogenomonic in swine salt poisoning [6], but not in bovine salt poisoning [3]. In case No. 5, serum ion levels and percentage of eosinophils were kept within normal range which is not in conformity with those in typical bovine salt poisoning [5].

REFERENCES

1. Baker, J. C. 1987. pp. 137–147. *In*: The Veterinary Clinics of North America: Food Animal Practice, vol. 3, No. 1, Bovine Neurologic Diseases (Baker, J. C. ed.), W. B. Saunders, Philadelphia.
2. Inners, J. R. and Sunders, J. Z. 1962. pp. 700–717. *In*: Comparative Neuropathology (Inners, J. R. and Sunders, J. Z. eds.), Academic Press, New York.
3. Kopcha, M. 1987. pp. 119–135. *In*: The Veterinary Clinics of North America: Food Animal Practice, vol. 3, No. 1, Bovine Neurologic Diseases (Baker, J. C. ed.), W. B. Saunders, Philadelphia.
4. Little, P. B. and Sorencen, D. K. 1969. *J. Am. Vet. Med. Assoc.* 155: 1892–1903.
5. Osweiler, G. D. and Hund, J. W. 1974. *J. Am. Vet. Med. Assoc.* 165: 165–167.
6. Smith, D. L. T. 1957. *Am. J. Vet. Res.* 18: 825–850.
7. Sullivan, N. D. 1985. The nervous system; malacia and malacic diseases. pp. 251–261. *In*: Pathology of Domestic Animals, vol. 1, 3rd ed. (Jubb, K.V.F., Kennedy, P. C., and Palmer, N. eds.), Academic Press, Orland.