A Case of Bovine Cerebral Mucormycosis

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ABSTRACT. Cerebral mucormycosis was reported in a cow. The lesion was found in the left occipital lobe. Histopathologically, it was subacute thrombo-embolic meningoencephalitis with cortical malacia and medullary demyelination in which there were hyphae revealing the characteristic morphology of the order Mucorales.—KEY WORDS: cerebrum, cow, mucormycosis.

The term "mucormycosis" used here designates infection caused by various species of the order Mucorales [4, 13, 17, 23].

Mucormycosis in the central nervous system (CNS) occurs occasionally and has been reported in humans [1, 4, 10, 12, 20, 21, 25], bovine fetuses [18], calves [2, 6, 8, 14, 16], cattle [7, 11], dogs [9], cats [22], wild animals [19] and laboratory animals [15].

Six hundred and ninety-two cattle aged more than 6 months old were autopsied at the College of Dairying during a decade from April 1975 to March 1985. Out of them, 45 cases were systemic (deep seated) mycosis and only one cow was cerebral mucormycosis. This means the rare occurrence of bovine cerebral mucormycosis (BCM).

Clinical history: A 10-year-old Holstein cow, who had calved on April 12, 1984, showed anorexia and subcutaneous edema in the submandibular area and brisket on postpartum day (PPD) 11, and was diagnosed as traumatic pericarditis. She was routinely treated with antibiotics and transfusions. Anemia (Ht; 12.0-20.0%), leukocytosis (11x10³-32x10³/mm³), neutrophilia (8.5x10³-29x10³/mm³) were observed between PPD 16 and 32. On PPD 34, she was recumbent and unable to stand, and died on PPD 35.

Autopsy: A circumscribed (ca. 2x2 cm) fibrinous thickening of the meninges was found on the dorsal area of the left occipital lobe. Changes other than the brain lesion were chronic traumatic gastro-phrenico-pericarditis, increased fluids in body cavities, pulmonary congestive edema, cachexia, chronic cholangitis due to fascioliasis, a fist-sized encapsulated liver abscess, enlargement of the superficial lymph nodes, and soybean-sized mucosal erosions of the tongue and esophagus.

Histopathology: The gross meningeal lesion consisted of fibrinous thickening with marked neutrophilic and mononuclear cell infiltration and thromboangiitis (Fig. 1). There were hyphae in the thrombi and vascular walls.

In the parenchyma adjacent to the meningeal lesion, a well-demarcated necrotic area extending to the medulla was found (Fig. 1). In the cortical area thromboangiitis with neutrophilic and mononuclear cell infiltration was observed associated with hyphae in the perivascular tissue as well as in the thrombi. Within the lesion irregular malacic foci were also scattered with fat granule
cells, neutrophils, mononuclear cells and hyphae (Fig. 2).

In the outer region of the medulla, demyelination was present accompanied with thromboangiitis having intra- and peri-vascular hyphae and with a few neutrophils

(Fig. 3). In the inner region of the medulla, intravascular hyphal proliferation and marked perivascular neutrophilic cell infiltration were observed.

Hyphae were nonseptate, bulbous and 4–12 μm in diameter, and branched fre-

Fig. 1. Dorsal area of the left cerebral occipital lobe. A circumscribed necrotic lesion in the cortex to the medulla, associated with meningeal fibrinous thickening (arrows). HE. ×3.

Fig. 3. Outer region of the medulla. Thromboangiitis with perivascular hyphae and neutrophils. The parenchyma is rarefied and spongiotic. GMS and HE. ×173.

Fig. 2. Cortical malacic lesion consisting of rarefaction, infiltration of neutrophils, mononuclear cells and fat granule cells, and irregularly proliferated hyphae (arrows). HE. ×340.

Fig. 4. Hyphae in the lesion. Note nonseptate, bulbous and haphazardly or frequent right-angle (arrows, 4a and 4b) branched hyphae positively by GMS. GMS and HE. ×490.
quent in a right-angle direction though haphazardly. Walls of hyphae were thin, unparallel to the opposite wall, and stained positively by Gomori’s methenamine silver method (GMS) (Fig. 4). From the morphological characteristics of the hyphae and cellular reactions to them, the organisms were considered to be a member of the order *Mucorales* [13, 23]. No mycological examination was performed.

The cerebral lesions observed here were diagnosed as thrombo-embolic meningoencephalitis with cortical malacia and medullary demyelination caused by mucormycosis. The necrosis and malacic foci are thought to have been induced by the intravascular hyphal proliferation and thrombosis. The fresh lesions were poor in cellular reaction. These findings are regarded as characteristics of cerebral mucormycosis [6, 8, 14, 23–25].

Most of mucormycosis in the CNS occur via hematogenous route [6, 7, 16, 25]. In the present case, the hyphae were apparently hematogenously metastasized to the brain.

In human cerebro-rhino-orbital mucormycosis, the nasal cavity, paranasal sinus and orbital region have been considered to be the main portals of entry [1, 3–5, 10, 12, 24, 25]. As the portals of entry in BCM, the nasal cavity [8], lung [14], umbilical vein in fetuses [6] and alimentary canal [16] have been suggested. In the present case, however, the portal of entry could not be identified.

Other histopathological changes observed in this case were papular stomatitis in the mucosa of the tongue and esophagus, focal necrosis in the liver and sinus catarrh in all the lymph nodes of the whole body.

As predisposing factors to opportunistic fungal infection, diabetes, leukemia, lymphoma and other disseminated malignant processes, or long-term administration of antibiotics, corticosteroids, cytotoxic drugs and immunosuppressive agents were discussed [20, 24]. In the present case, chronic traumatic pericarditis might be regarded as a predisposing factor.

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REFERENCES

要約

牛の大脳ムコール症の1例（短報）：千早 豊・松川 清・岡田洋之（酪農学園大学家畜病理学教室）——分娩後、創傷性心膜炎を発症し約1ヶ月の経過の後の飼育したホルスタイン種10歳の牛に大脳ムコール症を認めた。病理学的に、大脳皮質における軟化帯、髄質における脱髄を伴った急性性血栓性髄質脳炎の像を示していた。ムコール目（Mucorales）の特徴を示す菌糸の増殖が血栓内、血管壁および実質内に認められた。