Aujeszky’s Disease in a Dog
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Aujeszky's disease (AD), first recognized as a fatal disease of cattle and the dog in 1902 [1], is caused by a herpesvirus. Natural infections of domestic and wild animals have been reported in various countries. In Japan, the first outbreak of AD in swine was reported in 1981 [6] and the incidence increased year by year. After that, three cases of cattle naturally infected with AD virus (ADV) were found in 1985 [8].

The purpose of this report is to record ADV isolation from a dog and the pathological findings in Japan. An affected and dead canine was brought on March, 1986 from a veterinarian in Saitama prefecture for diagnosis of the disease. Clinically it showed depression, salivation and pruritus. The dog’s condition was rapidly deteriorated during a few hours and died next morning.

The dog was necropsied systematically. The materials were fixed with buffered 10% formalin, embedded in paraffin and stained with hematoxylin and eosin. The sections, in which detected intranuclear inclusion bodies, were stained by the immunoperoxidase method [9].

Virus was isolated in cloned porcine kidney (CPK) cells cultured on coverslips or in tubes. The brain, spinal cord, tonsil, lung, thymus, liver, kidney, heart and cutis were collected and a 10% suspension of each sample was inoculated in 0.2-ml quantities in these cultured cells after filtration through membrane filter of 0.45-μm pore size. Coverslip cultures were taken out in 24 to 48 hr, fixed with acetone and stained with fluorescent antibody against ADV. Tube cultures were observed daily for 7 days under an inverted microscope for cytopathic effects (CPE).

Macroscopically, no gross lesion was found on the surface of the body, but bloody spots were scattered on subcutis. The parasitism of Filaria immitis was observed from the right ventricle of the heart to the pulmonary artery. No other characteristic gross lesion was observed.

Microscopically, encephalomyelitis and ganglionitis characterized by neuronal degeneration, neuronophagia and perivascular cuffing were observed in the medulla oblongata, thoracic cord and their ganglia. Intranuclear inclusion bodies were present in the affected neuronal and glial cells in the thoracic cord, their ganglia and autonomic ganglia of the heart (Fig. 1). Coincident with the intranuclear inclusion bodies, ADV antigen was detected in the cytoplasm of neuronal and glial cells by immunoperoxidase staining (Fig. 2). Moreover, microfilaries were found in the capillary vessels throughout the body.

Fig. 1. Intranuclear inclusion body observed in the autonomic ganglia of the heart (a: ×191, b: ×957).

Fig. 2. Immunoperoxidase staining of the thoracic spinal cord showing positive reaction in the neuron (×383).
Viruses were isolated from the cerebellum, medulla oblongata, cervical and thoracic cord, heart and thymus. The CPE characterized by rounding was observed about 18 to 24 hr after inoculation of the sample and intranuclear inclusions were also observed in the affected cells by May-Grünwald-Giemsa staining. The isolated viruses were identified as ADV by the neutralization test and fluorescent antibody staining.

The dog was diagnosed as AD from the characteristic clinical signs, histopathologic findings and virus isolation. The pathologic findings in the central nervous system were similar to those previously reported in swine [2, 6, 7], cattle [8], and dogs [3, 4, 5]. The distribution of ADV antigen examined by immunoperoxidase staining was coincident with the pathologic findings and virus isolation. Moreover, the heart characterized by intranuclear inclusion body and virus isolation suggest hematogenous and neuronal disseminations of the virus in the dog. The transmission of the virus to the dog from the swine herds adjacent to the kennel was considered. Under natural conditions, infected swine may play an important role in the transmission of ADV to other animal populations, and care should be taken to control this potential source of infection.

REFERENCES


要約

犬のオーエスキー病の発生例（短報）：松岡俊和・飯島雄二・桜井健一・鴻巣 泰・田宮和枝・沖 三雄・新井則雄・神田幹雄（埼玉県大宮家畜保健衛生所 1）——病理組織学的検査及びウイルス分離により、犬のオーエスキー病を確認された（1986年3月8日）。主な臨床症状は、沈む、流涎および搔痒であった。病理組織検査では、中枢神経系において、脳脊髄炎と神経節炎が観察され、神経細胞およびグリア細胞の核内に尿素体が認められた。免疫凝集抗体法により、核内尿素体に一致して、オーエスキー病ウイルス抗原が検出され、脳、胸腺、心および肺からオーエスキー病ウイルスが分離された。