A Natural Case of Aujeszky’s Disease in the Cat in Japan

Motonobu HARA, Takehiko SHIMIZU, Seichi NEMOTO, Masafumi FUKUYAMA, Teruo IKEDA, Akio KIUCHI, Kiyoshi TABUCHI, Yasuo NOMURA1, Kinji SHIROTA1, Yumi UNE2, and Ryotaro ISHIKAKI2
1Department of Veterinary Microbiology, 2Veterinary Pathology, Azabu University, 1-17-71 Fuchinobe, Sagamihara-shi, Kanagawa-ken 229, and 3Department of Molecular Oncology, Nippon Veterinary and Animal Science University, 1-396 Kosugi-cho, Nakahara-ku, Kawasaki-shi, Kanagawa-ken 221, Japan
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Since Aujeszky’s disease (AD) was first recognized in swine in Japan in 1981 [6], it has spread to Tohoku and Kantou districts, and has also been reported to occur in several animals other than swine. Aujeszky’s disease virus (ADV) infection has already been reported in cattle [5] and a dog [3] but not in a cat in Japan. This report describes ADV infection in a companion cat which was discovered in Chigasaki City, Kanagawa Prefecture in January 1990.

Clinical history: The cat was a 6-year-old female mongrel weighing 4.0 kg. It was kept in an ordinary home at an urban district and was never given raw hog meat for food. Within a radius of 4 km from this home, there was no pig farm. The clinical symptoms at first consisted of vomiting, ataxia and anorexia. These were followed by repeated manifestation of excessive salivation, exaggerated movement and depression. The cat developed severe pruritus of the body on the fifth day, but did not show self mutilation and died on the sixth day (Fig. 1).

The isolation of virus: The procedures of the viral isolation, examination by electron microscope for the isolate and identification by the immunofluorescent antibody technique were attempted as described previously [3]. Isolation of ADV from various organs was tried using CRFK cells of feline kidney cell line. It was isolated from the cervical and thoracic regions of the spinal cord (Table 1). Herpes viral particles were confirmed by electron microscopic observation (Fig. 2). These viral particles were found to be 125–220 nm in diameter. The infected cells were positive to fluorescent antibody staining (Fig. 3). The conjugate was kindly supplied by the National Institute of Animal Health, Ibaraki. The isolated virus was thus identified as ADV.

Microscopic and histopathological examination: Histopathological examination revealed the presence of intranuclear inclusion bodies in the nerve cells of the medulla oblongata and the cervical, thoracic and lumbar regions of the spinal cord (Fig. 4), and demonstrated acute inflammation, chromatolysis and perivascular cell infiltration (Fig. 5). Small hemorrhagic foci were found in the medulla oblongata and thoracic vertebra. The nerve
Fig. 3. Specific fluorescence in cytoplasm of CRFK cells 23 hr after the infection (×400).

Fig. 4. Intraneuronal inclusion body in neuronal cell in medulla oblongata (×400).

Fig. 5. Perivascular cell infiltration in thoracic cord (×100).
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ganglia of the peritoneal cavity and the nerve plexus of the digestive tract showed the same changes. Focal necrosis was found in the liver. There was follicular necrosis in the tonsil and pharyngeal lymph nodes. By immunohistochemistry, viral antigen was confirmed in the lumbar ganglia and intestinal tract.

The development of clinical signs in the cat was very similar to the previous report [1], and the exaggerated movement, emesis, excessive salivation and pruritus were demonstrated, but in some natural cases [7] and experimental infected cats [2] pruritus was consistently absent. The isolation of virus in the present case was limited to the cervical and thoracic cord. In natural cases, Dow, C. et al. (1963) isolated the virus from the mesencephalon, medulla, cerebellum and the pharyngeal mucosa, and the virus was not isolated from any other organ [1]. While in the experimental infection in the cats, Hagemoser, W. A. et al. (1980) showed that the virus was isolated from nonnervous tissues such as pharyngeal mucosa, tonsil, tubinates, medial retropharyngeal lymph node, mesenteric lymph node, adrenal gland, salivary gland and lung, but the virus was not isolated from kidney, liver, spleen, jejunum-ileum, stomach, colon, heart and colon contents [2].

Pathological findings of the brain, stem and spinal cord of the present cat were similar to previous natural case [1]. Dow, C. et al. showed that the severe neuronal degeneration accompanied by inclusions was present in the semilunar ganglion in the cat. Nervous lesions consisted of multifocal to diffuse microgliosis, mononuclear perivascular cuffing and a mononuclear inflammatory cell infiltration with a variable number of neutrophils occasionally forming microabscesses [1].

In the present case, a direct relation to hog meat could not be established by the owner of the cat. In foreign countries, ADV infection in cats in urban areas has been reported to be caused by eating hog meat thrown by restaurants [4], and the care should be taken to control the potential infectious sources such as waste hog meat and rats feeding on them.

REFERENCES