BRIEF NOTE

An Incidence of Pseudorabies (Aujeszky’s Disease) in Piglets in Japan

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Pseudorabies (Aujeszky’s disease) has occurred in Europe, Russia, North and South America, China and Taiwan [2]. It has been unknown in Japan. The disease occurs most frequently in pigs, and when suckling piglets are affected, most of the litter mates succumb within a few days, resulting in a serious economic loss [3, 12]. This note describes pathological findings of three piglets affected with pseudorabies on a pig farm in the northern area of Yamagata Prefecture, Japan.

On this farm 9 litters were farrowed during the month of January, 1981. From the middle of this month, 39 of 76 litter mates died successively up to 25 days of age, showing the following symptoms, diarrhea, fever of up to 40.5°C, and tremor or epileptiform convulsions. Three piglets from 3 litters involved in the incidence, died on February 1 at ages 1 (case 3), 3 (case 1) and 25 (case 2) days, respectively, were subjected to this study. The brain, liver, spleen, kidneys, lungs, heart and mesenteric lymph nodes were histologically examined.

Case 1: Non-suppurative meningo-encephalitis was present. Meningitis (Fig. 1) consisted of leptomeningeal infiltration of small to medium-sized lymphocytes with a few plasma cells, macrophages and neutrophils. Meningitis occurred in almost all areas of the cerebrum, being mild to moderate in degree, while it was focal and mild in degree in the brain stem and cerebellum.

Encephalitis (Fig. 1) was prominent in the cerebral cortex, which was composed of degeneration and necrosis of nerve cells, frequently with neuronophagia, focal gliosis, and perivascular cuffing. The nerve cell changes were focal but multiple. Cellular elements appeared in the perivascular areas were the same as those in the meninges. Some lesions were accompanied by rarefaction or slight destruction of the ground substance, neutrophilic and mononuclear cell infiltration, and swelling of astrocytes. Intranuclear inclusion bodies were fairly frequent in nerve cells and astrocytes. Nuclei of nerve cells having the inclusions were swollen with slight margination of the chromatin. Two types of inclusions were observed (Fig. 2): The first was eosinophilic and most commonly single, large, discrete, spherical or reniform bodies, often associated with clear halo. The second was basophilic homogeneous or granular, filling completely the nucleus.

In the cerebral medulla there were scattered glial nodules, perivascular cuffing, eosinophilic intranuclear inclusions in as-
tocytes, and sometimes fragmented swollen axis cylinders. The same lesions with inclusions as those in the cerebral cortex were also marked in some nuclei of the cerebrum, diencephalon and mesencephalon. Slight perivascular cuffing, neuronophagia and glial nodules were dotted in the pons, medulla oblongata and cerebellum. So-called glial shrubbery was occasionally seen in the molecular layer of the cerebellum.

Small portions of a tissue paraffin block of the cerebral cortex were examined electron microscopically. Naked nucleocapsids were seen mainly in the nucleus and occasionally in the cytoplasm of nerve cells and astrocytes (Fig. 3). The capsids were empty or had varying morphology and density of cores. A few enveloped virus particles were demonstrated in the cytoplasm of cells containing the unenveloped capsids (Fig. 4).

Case 2: Only a few, relatively large, glial nodules were in the cerebral medulla and the white matter of the cervical spinal cord. Multiple focal necrosis with numerous nuclear debris and swollen reticuloendothelial system (RES) cells was in the hepatic lobules. Eosinophilic granular inclusions were demonstrated in nuclei of some swollen RES cells within the necrotic foci.

Case 3: Although no lesions were in the brain, multiple focal necrosis with eosinophilic intranuclear inclusions in swollen RES cells was in both red and white pulp of the spleen. The structures of the necrotic foci were quite identical to those in the liver of case 2.

No significant lesions were seen in other organs in all the cases.

The most characteristic lesions of the central nervous system (CNS) in pseudorabies are acute non-suppurative meningoencephalitis with relatively mild myelitis [1, 3, 6, 8, 10, 12]. The essential alterations are neuronal degeneration and necrosis with neuronophagia, perivascular cuffing, and lymphocytic meningitis. In encephalitis of case 1 intense neuronal damage with multiple neuronophagia was present in the cerebral cortex. It is emphasized that the predilection sites of lesions in the CNS are the anterior regions, especially the cerebral cortex, all parts of the CNS being involved [8, 12]. The correlation of sites of involvement in the CNS with the site of primary infection is discussed from the experimental evidence [5, 12].

In suckling piglets affected with this disease, focal necrosis appears to be frequent in the liver [3, 6, 9, 10, 13], although occasional in the spleen and other visceral organs [3, 6]. Intranuclear inclusions were demonstrated in hepatic cells and RES cells within the necrotic foci [6, 9]. It is suggested that focal necrosis in the liver could be pathognomonic of this disorder [13].

Mature and immature virus particles in case 1 would be the first detection in animals naturally affected with pseudorabies, having been observed in many kinds of cells in the spinal or sympathetic ganglia of experimentally induced cases [4, 7, 11].

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References
Explanation of Figures

Fig. 1. Non-suppurative meningo-encephalitis in the cerebrum of case 1. Hematoxylin and eosin (H-E) stain. ×116.  
Fig. 2. Eosinophilic intranuclear inclusion bodies in nerve cells (arrows) of the cerebral cortex of case 1. H-E stain. ×680.  
Fig. 3. Intranuclear inclusion body with unenveloped nucleocapsids, showing margination of the chromatin, in an astrocyte of the cerebral cortex of case 1. ×19,000.  
Fig. 4. Two enveloped virus particles (arrows) in the cytoplasm of a nerve cell of the cerebral cortex of case 1. ×55,000.
