犬ジステンパー生ワクチン接種に起因したレッサーパンダのジステンパー感染

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BRIEF NOTE

Distemper Infection in Lesser Pandas Due to Administration of a Canine Distemper Live Vaccine

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Canine distemper (CD) virus has become universally recognized as the cause of a highly contagious, usually fatal disease in a variety of species other than the domestic dog [4, 6, 9]. The raccoon and lesser panda of the family *Procyonidae* are known to be highly susceptible to the disease [2, 3, 6]. A natural affection with CD was reported in 7 pandas (*Ailurus fulgens*) in Germany [6]. Accordingly, it is said that all *Procyonidae* should be immunized with CD vaccines in the zoo, and it is recommended that in the lesser panda inactivated vaccines should be used to be safe [4].

Live or inactivated vaccines are available for the prevention of viral diseases. In general, live vaccines carry potential hazards, while inactivated vaccines do relatively few hazards [8]. An infections of typical distemper due to administration of a CD live vaccine was in 3 lesser pandas in a zoo. This article describes the occurrence aspects and pathologic findings of the disease.

Occurrence aspects: One 5-year-old male and three 3-year-old female lesser pandas were raised in this zoo. Three (1 male and 2 females) of the four animals were administered 1/2 vial (0.7 ml)/animal of CD-infectious canine hepatitis combined vaccine (Philips-Duphar Comp.) by the subcutaneous route. All the three animals vaccinated revealed clinical signs, which were anorexia, diarrhea, fever and nasal discharge, from 9 to 12 days postinoculation. After that, all of them died from 14 to 20 days postvaccination.

Macroscopic findings: One male dead 15 days postvaccination and one female dead 20 days postinoculation were examined pathologically. Focal catarrhal pneumonia and catarrhal enteritis were noticed in the two cases, although macroscopic examination was not thoroughly done.

Light microscopic findings: Essential lesions observed in the two animals were almost the same, so that the findings are described in the lump. Only the organs mentioned below were histologically examined.

Main lesions in the lung were acute catarrhal pneumonia and appearance of eosinophilic cytoplasmic and intranuclear inclusion bodies in the epithelial cells. The former consisted of serous exudation with some macrophages in alveoli, congestion, edema and migration of some neutrophils in interalveolar septa, and swelling of some

alveolar cells. Such pneumonia was extensive, associated with compensatory alveolar emphysema and collapse.

In all the intrapulmonary bronchi and bronchioles epithelial cells were swollen, hyperplastic, sometimes with loss of cilia, and desquamative. Small to large and irregular-shaped to round cytoplasmic inclusions were frequent in the epithelial cells (Fig. 1). One to a few inclusions were recognized in one cell and many of them located in the perinuclear areas. Intranuclear inclusions, on the other hand, occurred very rarely in such cells. These were minute and round, and one or two inclusions were seen in one nucleus. The cytoplasmic inclusions were also demonstrated in a few macrophages in alveoli and bronchial glandular cells in the submucosa of large bronchi.

Lining epithelial cells in the trachea were swollen, hyperplastic, sometimes with loss of cilia, and desquamative in some areas. The same cytoplasmic inclusions as in the lung were frequent in these cells.

In the stomach minute eosinophilic cytoplasmic inclusions were dotted in cells of the glandular body, sometimes with degeneration of the cytoplasm.

In the small intestine acute catarrhal enteritis was present, which consisted of marked desquamation of epithelial cells and an increase in goblet cells. Minute eosinophilic cytoplasmic inclusions were scattered in cells of intestinal glands of Lieberkühn, sometimes with degeneration of the cytoplasm (Fig. 2). In the female both isolated and aggregated lymphatic nodules were very small in size, in which there were marked lymphocytic depletion and scattered karyopycnosis of lymphocytes (Fig. 3). Swelling and moderate proliferation of reticular cells were seen in some of the nodules and a few cells showing hyperchromatosis of the nuclear membrane contained minute eosinophilic intranuclear inclusions.

All the lymphatic nodules in the spleen were very small in size, revealing marked depletion of lymphocytes with slightly swollen reticular cells (Fig. 4). Occasionally, such nodules were associated with edema and fibrinous exudation and karyopycnosis of lymphocytes in the female. Reticuloendothelial cells in the red pulp of the male were diffusely swollen, frequently showing the morphology of macrophages, and some of them contained minute eosinophilic cytoplasmic and intranuclear inclusions.

In addition, there were a slight degree of congestion, diffuse fatty degeneration and granular change in the liver. A mild degree of tubulonephrosis was present in the kidney of the male. In the kidney of the female there were marked tubulonephrosis with dilation of urinary tubules and retention of proteinic fluid, fibrosis of some glomeruli and multiple focal aggregations of lymphocytes and plasma cells in the interstitium. It was thought that the kidney lesions had nothing to do with this disease. Granular change was observed in muscle fibers of the heart. No significant lesions were in the pancreas.

Electron microscopic findings: To improve observation of the inclusion bodies in the lung, small portions were removed from the tissue paraffin block of the male and examined electron microscopically. The cytoplasmic inclusions, corresponding in size, shape and intracellular location to those seen by light microscopy, were demonstrated in the bronchial and bronchiolar epithelial cells (Fig. 5). They consisted of tangled aggregates of numerous tubular strands, namely viral nucleocapsids (Fig. 6). Each strand was surrounded by a granular coating. Complete virions associated with

the cell membrane of the infected cell were not present.

From the above findings, the animals were typical of CD. Above all, the following alterations result from the viral infection, which are well known to be characteristic of CD; appearance of eosinophilic cytoplasmic and intranuclear inclusion bodies in the epithelial cells and reticuloendothelial cells, lymphocytic depletion from the lymphatic tissues and tangled aggregates of viral nucleocapsids in the cytoplasm of the bronchial epithels. The ultrastructure of cytoplasmic and intranuclear inclusion bodies seen by light microscopy in CD consists of viral nucleocapsids or nucleoprotein strands [1, 5, 7, 10]. While these are commonly found, complete virions are not [7].

Lymphocytic destruction resulting in lymphocytic depletion and atrophy of lymphoid organs and tissues is one of the characteristic lesions in early stages of CD [1, 5, 10]. Tajima et al. [10] described that the lymphoid tissue injury resulted from the direct interaction of CD virus and host cells in the mink. In CD of young pups the primary lymphoid organs (thymus and gut-associated lymphoid tissue) underwent atrophy accompanying immunologic deficits [1]. Unfortunately, no pathologic examination on the lymph node and thymus was performed in the present study. However, lymphocytic depletion from lymphatic nodules was marked in the spleen and small intestine and karyopycnosis which seemed to be in the process of lymphocytic destruction was recognized.

All the present animals vaccinated died in 20 days postvaccination. This fact indicates that the infection has occurred due to administration of CD live vaccine. As one of potential hazards of live vaccines, Rweyemamu [8] pointed out risks of causing severe reactions in some vaccinates. From this incidence, it was reaffirmed that the lesser panda was highly susceptible to CD virus. To immunize the species against this disease, live vaccine should be avoided.

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要　約

犬ジステンパー生ワクチン接種に起因したレッサーパンダのジステンパー感染（短報）：板倉智敏・中村菊保・中塚順子・五藤精知（鳥取大学農学部家畜病理学教室，同電子顕微鏡室）――犬ジステンパー（CD）生ワクチンを接種された3頭のレッサーパンダが，定型的な CD 症状と病変を示した．特徴病変は，上皮細胞，細網内皮細胞における酸好性細胞質ならびに核内封入体形成と，リンパ組織からの著明なリンパ球減数であった．電顕下で，細胞質封入体は nucleocapsid から構成されていた．以上の所見は，本動物が CD に対し高い感受性を持つこと，CD 予防に際し生ワクチン使用は極めて危険であることを示唆する．

Explanation of Figures

Figures 1 through 4 are light micrographs, and Figures 5 and 6 electron micrographs.

Fig. 1. Bronchiolar epithelial cells showing marked swelling and hyperplasia with numbers of small to large cytoplasmic inclusion bodies. Male panda. Epon section and Paragon multiple stain. ×740.

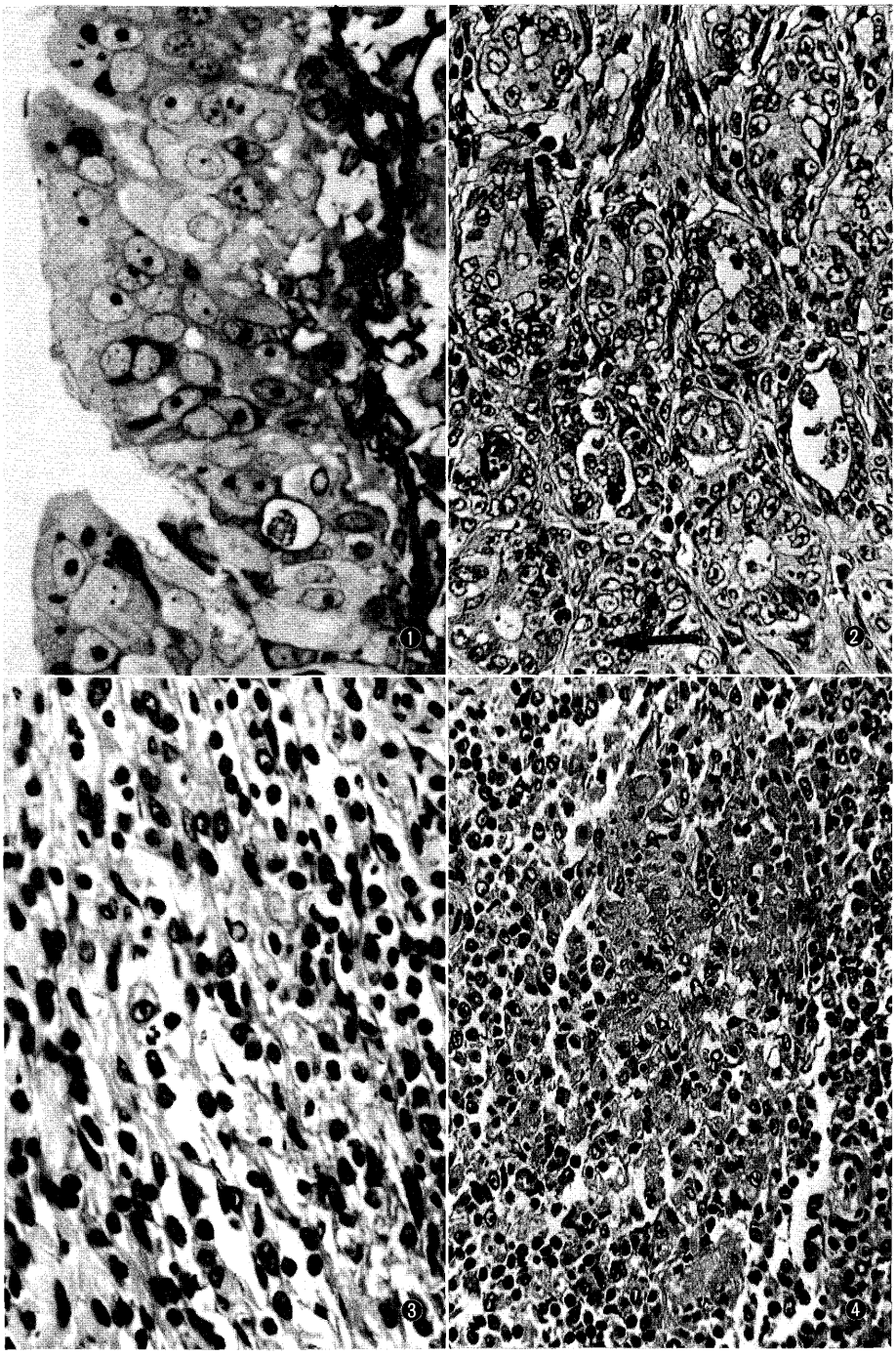
Fig. 2. Intestinal glands of Lieberkühn revealing minute cytoplasmic inclusion bodies (arrows) and degeneration of the cytoplasm of some cells (lower half). Male panda. H-E stain. ×400.

Fig. 3. Isolated lymphatic nodule in the small intestine displaying marked lymphocytic depletion and scattered karyopycnosis with some swollen reticular cells. Female panda. H-E stain. ×560.

Fig. 4. Lymphatic nodule in the spleen showing marked lymphocytic depletion, karyopycnosis of some lymphocytes and slight swelling of reticular cells with edema and fibrinous exudation. Female panda. H-E stain. ×400.

Fig. 5. Bronchiolar epithelial cells revealing many cytoplasmic tangled aggregates of viral nucleocapsids (arrows). Male panda. ×5400.

Fig. 6. Bronchiolar epithelial cell showing a cytoplasmic aggregate of viral nucleocapsids which are cut transversely or obliquely. Male panda. ×38000.


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