Short Paper

Canine Distemper Virus Infection in a Masked Palm Civet (Paguma larvata)

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Summary

A free-living masked palm civet (Paguma larvata) died after exhibiting signs of canine distemper (CD). The microscopic lesions consisted of cytoplasmic and intranuclear eosinophilic inclusion bodies, bronchointerstitial pneumonia, non-purulent encephalitis accompanied by demyelination and lymphocytic depletion in various lymphoid tissues. CD virus-specific antigens were demonstrated immunohistochemically in intracellular eosinophilic inclusions, which were ultrastructurally confirmed to be viral nucleocapsids. From these findings, the present case was diagnosed as CD virus infection in a masked palm civet.

Introduction

Canine distemper (CD) occurs naturally in all members of the Canidae and Mustelidae families and certain members of the Procyonidae family (Budd, 1981; Dungworth, 1985). Among the Viverridae family only the binturong (Arctictis binturong) has been reported to be susceptible to canine distemper virus (CDV) (Hamerton, 1937; Goss, 1948). Recently, we encountered a spontaneous case of CD infection in a masked palm civet (Paguma larvata), a member of the Viverridae family that lives in south-east Asia, including Japan, and this report describes the pathological findings.

Case Report

A young female masked palm civet, weighing 3.6 kg (Fig. 1), was found lying by a tree root in a forest in the vicinity of Tokyo (about 50 km distant from the centre of the metropolis) and was carried to a local veterinarian. She was markedly depressed with clinical signs that included severe dehydration, dyspnoea, serous oculonasal discharge, diarrhoea, local alopecia and convulsions. Antibiotic treatment and fluid therapy did not lead to improvement and the animal died the following day.

Necropsy was performed shortly after death. Macroscopic lesions were present in the skin, lung and gastrointestinal tract. A vesiculopustular dermatitis was present on the skin of the abdomen and inner aspects of the thighs. All lung lobes had various sized reddish spots and patches with some congestion and consolidation and greyish zones of emphysema along their margins. The
stomach and small and large intestines exhibited mild to moderate catarrh and congestion.

Histopathologically, pulmonary lesions were characterized by diffuse bronchointerstitial pneumonia, the predominant feature of which was proliferation of alveolar and bronchiolar epithelial lining cells resulting in syncytial giant cell formation (Fig. 2A). Interstitial accumulation of lymphocytes and neutrophils appeared to be concurrent in such lesions. These tissue responses were especially dominant in the peribronchiolar area. Cytoplasmic and, less frequently, intranuclear eosinophilic inclusion bodies were found in the linings of the alveolar and bronchiolar epithelium (Fig. 2B), occasionally in the macrophages in alveoli and rarely in the bronchial epithelium. Similar cytoplasmic and intranuclear inclusion bodies were recognized in epithelial cells of the skin, tongue and cornea, and the lining cells of oesophagus, stomach, small intestine, large intestine, gall bladder, bile duct, pancreatic duct (Fig. 3A), renal tubules, renal pelvis, urinary bladder, uterus and salivary gland. In addition, they were found in the glia of the retina and optic nerve. The cells containing inclusions were often swollen, hydropic and desquamative.

Lesions in the central nervous system consisted of focal areas of mild to moderate demyelination with spongy appearance of the tissue and with proliferation of astrocytes in the white matter of the cerebrum, cerebellum and midbrain. Eosinophilic cytoplasmic and intranuclear inclusions were present in astrocytes and neurones not only in and around the demyelinating lesions, but also in areas of the white matter which lacked detectable lesions (Fig. 4).

Lymphoid tissues in the mucosa of the digestive and respiratory tract and in the spleen and lymph nodes were depleted of lymphocytes with hyperplasia of reticular cells and cytoplasmic and intranuclear inclusions being formed in reticular cells and lymphocytes (Fig. 5).

For immunohistochemistry, an avidin-biotin-complex immunoperoxidase technique (Vector Laboratories, CA, U.S.A.) was employed on paraffin-wax sections of all the organs and tissues collected, using mouse monoclonal antibodies directed against CDV (courtesy of Drs H. Oh-ishi and M. Senda, National Veterinary Assay Laboratory). CDV-specific antigens were demonstrated in epithelial cells of various organs and tissues (Fig. 3B), neurones and astrocytes in the brain and reticular cells and lymphocytes in various lymphoid tissues, corresponding with the presence of eosinophilic inclusion bodies in the HE-stained specimens. Ultrastructurally, cytoplasmic and intranuclear inclusions seen by light microscopy were recognized as aggregates of viral nucleocapsids (Fig. 6).

**Discussion**

The clinical signs and histopathological lesions were compatible with those that have been shown to be characteristic of CD in dogs (Jones and Hunt, 1983; Dungworth, 1985). Furthermore, the demonstration of CDV antigen and the ultrastructural features of the inclusion bodies provide very strong evidence for an aetiological role of CDV as the cause of death. There has been no previous
CDV in a Civet

Fig. 1. Gross appearance of a masked palm civet (*Paguma larvata*) at necropsy.

Fig. 2. (A) Bronchointerstitial pneumonia with hyperplasia of alveolar and bronchiolar epithelial cells. HE $\times 136$. (B) Higher magnification of bronchiolar linings with fine granular and droplet cytoplasmic and intranuclear inclusion bodies. HE $\times 272$.

Fig. 3. Pancreatic duct. (A) Cytoplasmic and intranuclear inclusion bodies in degenerating epithelial cells. HE $\times 302$. (B) Canine distemper virus antigen in the linings. Immunoperoxidase staining $\times 193$.

Fig. 4. Intranuclear (arrow) and cytoplasmic (arrowhead) inclusion bodies in a neurone and an astrocyte of the cerebral cortex. HE $\times 678$. 
report of CD in masked palm civets and this description is the first of CDV infection in this host species.

The pathogenicity of CDV for masked palm civets, a member of the *Viverridae* family, is still unknown. There have been many accounts of spontaneous and experimental CDV infection in the *Canidae, Mustelidae* and *Procyonidae* families. However, a review of the literature shows surprisingly little precise information regarding CD in the *Viverridae* (Hamerton, 1937; Goss, 1948). From an evolutionary standpoint, the *Viverridae* is a unique family in that it descends from the superfamily *Aeluroidea*, despite the fact that the *Canidae, Mustelidae* and *Procyonidae* families have a common ancestor, the superfamily *Arctoidea* (Goss, 1948). These facts may indicate that members of the *Viverridae* have lower susceptibility to CDV. Therefore, the condition of the host or virulence of the strain of the CDV may influence CDV infection in masked palm civets.

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**References**

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