

# CANINE DISTEMPER REVISITED

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Canine distemper is a highly contagious, debilitating, multisystemic viral disease of carnivores including dogs, ferrets, raccoons, lions and ocelots. The virus is an RNA Morbillivirus loosely related to the human measles virus and the rinderpest virus of ruminants.

The **distribution** is worldwide and domestic dogs are considered important reservoirs. The highest incidence of disease is found in young (2-6 months old) unvaccinated dogs. All body secretions and excretions of infected animals are infectious. **Transmission** is primarily via inhalation of aerosolized body secretions or contact with fomites. Transplacental infection may occur rarely. Recovered animals may shed the canine distemper virus (CDV) for 1-2 weeks post recovery but rarely 60-90 days post recovery. The virus has a survival time of a few hours to a few days in the environment and is easily destroyed by desiccation, common disinfectants such as phenols and quaternary ammonium compounds and lipid solvents.

Viral strain, dose and host immunity all affect the severity of disease. Infection with CDV follows the stages below:

## DAY 1

Viral inhalation → infection of tissue macrophages of the upper respiratory tract



## DAYS 2 - 4

Infection → the tonsils, retropharyngeal and bronchial lymph nodes



## DAYS 4 - 6

Systemic lymphoid involvement of the liver, spleen and abdomen. A transient fever spike and lymphopenia accompanies this.



## DAYS 6 - 8

Viremia



## DAYS 8 - 9

Viral spread to epithelia tissues and the central nervous system CNS



## DAYS 9 - 14

Recovery, severe multisystemic or CNS disease depending on host immune response.

**Clinical signs** observed are variable and may be general/non-specific, respiratory, gastrointestinal, ocular and neurological. Nonspecific general clinical signs include malaise, anorexia, pyrexia (diphasic) and dehydration. Serous to mucopurulent naso-ocular discharge, tachypnea, dyspnea and productive cough may be observed. Acute gastroenteritis results in vomiting and diarrhea. Keratoconjunctivitis (manifesting as serous to mucopurulent discharge), chorioretinitis (resulting in hyper-reflective fundic lesions) and optic neuritis or serous retinal detachment (leading to blindness) may be observed.



**Mucopurulent nasal discharge**

Development of neurological signs cannot be foreseen and may occur without the above signs, simultaneously with the other signs described above or be delayed 1-3 weeks or months after apparent recovery because of chronic progressive demyelination.

“Chewing gum” seizures (jaw champing), pacing, circling and behaviours changes are observed in the acute phase of the CNS manifestation. Ataxia, other gait disturbances, abnormal spinal reflexes, paresis, abnormal proprioception and muscle twitches may also be observed.

Infection before permanent tooth eruption results in enamel hypoplasia. Dogs with CNS disease frequently demonstrate “hard pad disease” (nasodigital hyperkeratosis). Abdominal pustular dermatitis has also been described.



**Hard pad disease**



**Nasal hyperkeratosis**



**Enamel hypoplasia**

**Diagnosis** may be attempted via:

1. A complete blood count (lymphopenia followed by leucocytosis due a neutrophilia)
2. Cerebrospinal fluid (CSF) analysis (presence of a CDV specific antibody in CSF in concentrations greater than in serum is diagnostic).
3. Thoracic radiography (diffuse interstitial pneumonia initially, with air bronchograms and diffuse alveolar and bronchial patterns seen later on in the disease)
4. Serology (recent infection is suggested by a rising serum neutralising antibody titre or a CDV specific IgM titre)
5. Virus isolation
6. Immunohistochemistry
7. Fluorescent antibody techniques
8. Reverse transcriptase polymerase chain reaction assays

Differential diagnoses of CDV include infectious canine hepatitis and leptospirosis.

While no specific therapy exists **treatment** is attempted via supportive and symptomatic care. Broad spectrum antibiotics, intravenous fluid therapy, antiemetics, antidiarrheals, antipyretics, anticonvulsants and general nursing care are indicated.

Mortality is highest in young puppies with severe pyrexia and multisystemic involvement. Neurological complications are often progressive and irreversible leading to a guarded to poor prognosis.

**Prevention** is best done through vaccination of young pups from 6 weeks until 16 weeks of age with the CDV modified live virus and yearly boosters for adults. Vaccination of adults for periods longer than a year is considered extra-label usage.

## REFERENCES

Saunders Manual of Small Animal Medicine, 3<sup>rd</sup> Edition

The Merck Veterinary Manual 2012