Canine Diseases

Canine Distemper

Canine distemper is a highly contagious, systemic, viral disease of dogs seen worldwide. Clinically, it is characterized by a diphasic fever, leukopenia, GI and respiratory catarrh, and frequently pneumonic and neurologic complications. Its epidemiology is complicated by the large number of species susceptible to infection.

Etiology and Pathogenesis

Canine distemper is caused by a paramyxovirus closely related to the viruses of measles and rinderpest. The fragile, enveloped, single-strand RNA virus is sensitive to lipid solvents, such as ether, and most disinfectants, including phenols and quaternary ammonium compounds. It is relatively unstable outside the host. The main route of infection is via aerosol droplet secretions from infected animals. Some infected dogs may shed virus for several months.

Virus initially replicates in the lymphatic tissue of the respiratory tract. A cell-associated viremia results in infection of all lymphatic tissues, which is followed by infection of respiratory, GI, and urogenital epithelium, as well as the CNS and optic nerves. Disease follows virus replication in these tissues. The degree of viremia and extent of viral spread to various tissues is moderated by the level of specific humoral immunity in the host during the viremic period.

Clinical and Pathological Findings

A transient fever usually occurs 3–6 days after infection, and there may be a leukopenia (especially lymphopenia) at this time; these signs may go unnoticed or be accompanied by anorexia. The fever subsides for several days before a second fever occurs, which may be accompanied by serous nasal discharge, mucopurulent ocular discharge, lethargy, and anorexia. GI and respiratory signs, typically complicated by secondary bacterial infections, may follow; rarely, pustular dermatitis may be seen. Encephalomyelitis may occur in association with these signs, follow the systemic disease, or occur in the absence of systemic manifestations. Dogs surviving the acute phase may have hyperkeratosis of the footpads and epithelium of the nasal planum, as well as enamel hypoplasia in incompletely erupted teeth.

Overall, a longer course of illness is associated with the presence of neurologic signs; however, there is no way to anticipate whether an infected dog will develop neurologic manifestations. CNS signs include circling, head tilt, nystagmus, paresis to paralysis, and focal to generalized seizures. Localized involuntary twitching of a muscle or group of muscles (myoclonus, chorea, flexor spasm, hyperkinesia) and

convulsions characterized by salivation and, often, chewing movements of the jaw ("chewing-gum fits") are considered classic neurologic signs.

A dog may exhibit any or all of these multisystemic signs during the course of the disease. Infection may be mild and inapparent or lead to severe disease with most of the described signs. The course of the systemic disease may be as short as 10 days, but the onset of neurologic signs may be delayed for several weeks or months as a result of chronic progressive demyelination within the CNS.

Clinicopathologic findings are nonspecific and include lymphopenia, with the possible finding of viral inclusion bodies in circulating leukocytes very early in the course of the disease. Thoracic radiographs may reveal an interstitial pattern typical of viral pneumonia.

Chronic distemper encephalitis (old dog encephalitis, [ODE]), a condition often marked by ataxia, compulsive movements such as head pressing or continual pacing, and incoordinated hypermetria, may be seen in fully vaccinated adult dogs without a history suggestive of systemic canine distemper infection. Although canine distemper antigen has been detected in the brains of some dogs with ODE by fluorescent antibody staining or genetic methods, dogs with ODE are not infectious, and replication-competent virus has not been isolated. The disease is caused by an inflammatory reaction associated with persistent canine distemper virus infection in the CNS, but mechanisms that trigger this syndrome are unknown.

Thymic atrophy is a consistent postmortem finding in infected young puppies. Hyperkeratosis of the nose and footpads is often found in dogs with neurologic manifestations. Depending on the degree of secondary bacterial infection, bronchopneumonia, enteritis, and skin pustules also may be present. In cases of acute to peracute death, exclusively respiratory abnormalities may be found. Histologically, canine distemper virus produces necrosis of lymphatic tissues, interstitial pneumonia, and cytoplasmic and intranuclear inclusion bodies in respiratory, urinary, and GI epithelium. Lesions found in the brains of dogs with neurologic complications include neuronal degeneration, gliosis, noninflammatory demyelination, perivascular cuffing, nonsuppurative leptomeningitis, and intranuclear inclusion bodies predominately within glial cells.

Diagnosis

Distemper should be considered in the diagnosis of any febrile condition in dogs with multisystemic manifestations. Characteristic signs sometimes do not appear until late in the disease, and the clinical picture may be modified by concurrent parasitism and numerous viral or bacterial infections. A febrile catarrhal illness with neurologic sequelae justifies a clinical diagnosis of distemper.

In dogs with multisystemic signs, the following can be examined by immunofluorescent assay or reverse transcriptase (RT) PCR: smears of conjunctival, tracheal, vaginal, or other epithelium; the buffy coat of the blood; urine sediment; or bone marrow aspirates. Commercially available quantitative RT-PCR can usually distinguish natural infection from vaccinal virus. A combined two-step RT-PCR to distinguish vaccinal strains from emerging wild-type strains has also been described; this assay would be of particular value in epidemiologic investigations or in outbreaks in non-canine species. Antibody titers or ELISA can be performed on CSF and compared with peripheral blood; a relatively higher level in the CSF is typical of natural infection versus vaccination. Viral antigen immunofluorescent assay (IFA) or fluorescent in situ hybridization for viral DNA can be performed on biopsies from the footpads or from the haired skin of the dorsal neck.

At necropsy, diagnosis is usually confirmed by histologic lesions, IFA, or both. These samples are often negative when the dog is showing only neurologic manifestations or when circulating antibody is present (or both), requiring that the diagnosis be made by CSF evaluation or RT-PCR as described above.