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Canine and Feline Distemper

Description

Canine and feline distemper are diseases affecting many wild and domestic carnivores. Although both these diseases can cause acute illness and death, canine and feline distemper should not be confused, as they are caused by two distinctly different viral agents.
The following chart indicates the animals which are susceptible to infection by canine and feline distemper.

Family	Canine Distemper	Feline Distemper
Canidae wolf, coyote, fox, domestic dog	Yes	No
Felidae bobcat, lynx, domestic cat	No	Yes
Procyonidae raccoon	Yes	Yes
Mustelidae ferret, mink, weasel, marten, fisher, otter, badger, skunk, wolverine	Yes	Yes (Mink and possibly skunk and otter)

Canine Distemper

Description

Canine distemper is a highly contagious disease of carnivores caused by a paramyxovirus. The virus is widespread and mortality in juveniles is higher than in adults. The canine distemper virus is very resistant to cold and the majority of distemper cases in domestic dogs are seen in the fall and winter. In wild animals, since the juveniles are more

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- Taenia Hydatigena
- Strychnine Poisoning
- Squirrel Pox
- Sarcocystis
- Salmonellosis
- Roundworms
- Raccoon Roundworm (Baylisascaris)
- Rabbit Fibromatosis
- Proventricular or Stomach Worm
- Organophosphate Toxicity
- Oil Intoxication

susceptible to infection, the majority of cases are seen in the spring and summer, but cases are observed year round.

Transmission

Transmission occurs via an aerosol-droplet route, direct contact, or possibly by contact with contaminated objects. It is uncertain whether carrier or unapparent infections exist. The virus is shed in the feces and urine of infected individuals and some evidence exists for transplacental transmission. The usual route of infection is through the upper respiratory tract, following inhalation of infective virus. Occasionally infection occurs from ingestion of infective material. Following entry into the upper respiratory tract, the virus is spread to the tonsils and lymph nodes, where viral replication occurs. The virus then enters the blood stream where it is transported to epithelial cells throughout the body, including the intestinal and respiratory tract.

Clinical Signs and Pathology

Typical signs of canine distemper seen in the domestic dog include respiratory and intestinal problems such as coughing, diarrhea, vomiting, nasal and ocular discharge, anorexia, and hyperkeratosis of the nasal planum and foot pads. Central nervous system signs may follow the above clinical signs. In wild carnivores, signs of abnormal behavior and apparent lack of fear, suggestive of rabies, may be the only signs grossly visible. Often the animals are presented with a purulent conjunctivitis and nasal discharge and the eyelids may be adhered together with crusty exudate. Neurological disturbances are often seen such as aggressiveness, disorientation, lack of alertness, convulsive movements of the head and paws, and aimless wandering. There may be evidence of diarrhea, labored breathing and an unkempt appearance to the fur. Due to the diarrhea and vomiting, the animal may be dehydrated and exhibit excessive thirst. Weakness and emaciation have been associated with canine distemper, but often animals will be acutely affected and be presented in good nutritional condition.

The pathological lesions of canine distemper include pulmonary congestion and consolidation leading to focal pneumonitis. Eosinophilic rounded or ovoid bodies with refractile particles are found in the epithelial cells of skin, bronchi, intestinal tract, urinary tract, bile duct, salivary glands, adrenal glands, central nervous system, lymph nodes and spleen. At the time of necropsy, an enlarged spleen is usually seen.

Diagnosis

Presumptive diagnosis is based on clinical signs, the demonstration of inclusion bodies in neutrophils on blood smear and inclusion bodies in conjunctival smears. Definitive diagnosis is based on laboratory analysis of affected tissues by fluorescent antibody techniques.

In wild carnivores, the presenting signs are often neurological and the disease must be differentiated from rabies and other encephalitides. Other diseases which may mimic distemper include tularemia, listeriosis, Chastek's paralysis (in captive mink and fox), histoplasmosis (raccoons) and poisonings.

Treatment and Control

No treatment other than supportive care exists for canine distemper. Control of canine distemper outbreaks includes the removal of carcasses of animals which have died from the disease, vaccination of susceptible domestic species to decrease the number of susceptible hosts, and a reduction in wildlife populations which also reduces the number of potential hosts. The virus is inactivated by heat, formalin, and Roccal R.

Significance

In Michigan, die-offs of raccoons due to canine distemper occur yearly. The impact of this disease on other wildlife populations is not known at this time. Unvaccinated domestic dogs are fully susceptible to the canine distemper virus, therefore, annual vaccination is recommended. Due to the similarity of some of the clinical signs of canine distemper and rabies, affected animals should be handled with caution until a diagnosis is confirmed. Canine distemper is of no public health significance to humans.

Feline Distemper

Description

Feline distemper, also called feline panleukopenia, cat plague, cat fever, feline agranulocytosis, and feline infectious enteritis, is an acute, highly infectious viral disease affecting members of the *Felidae*, *Procyonidae* and *Mustelidae*.

Transmission and Development

Feline distemper virus is shed in all body secretions and excretions of affected animals. Recovered animals may shed virus for months. Fleas and other insects, especially flies, may play a role in transmission of the disease. The route of infection is either inhalation or ingestion of infective material by a susceptible host. Feline distemper virus affects all rapidly dividing cells including cells of the intestinal mucosa, bone marrow and reticulo-endothelial system.

Clinical Signs and Pathology

Feline distemper usually begins suddenly with a high fever. Some animals may die peracutely at this stage with a minimum of gross lesions. More commonly, the high fever is followed by depression, vomiting, anorexia, diarrhea, and a profound leukopenia. These signs rapidly lead to severe dehydration. In a wild animal, feline distemper may progress as described above or the disease may be characterized by an encephalitis syndrome with central nervous system disturbances, convulsions, or ataxia. The course of the disease is short and rarely lasts over one week, but mortality is high and may reach 100% in susceptible animals.

The pathological lesions of feline distemper are found primarily in the bone marrow and small intestine.

Necropsy findings include an empty intestinal tract, hemorrhagic small intestine, hemorrhagic and edematous mesenteric lymph nodes, and a fluid-like appearance of the bone marrow of the long bones.

Diagnosis

A presumptive diagnosis can be made based on necropsy findings and the demonstration of leukopenia with a marked absence of granulocytes on differential blood cell count. Definitive diagnosis is based on histological examination and laboratory analysis of affected tissues.

Treatment and Control

No treatment other than supportive care and prevention of secondary bacterial infection exists for feline distemper. There is no means of control in wildlife populations other than the vaccination of susceptible domestic species to decrease the number of potential hosts.

Significance

The impact of this disease on wildlife populations in Michigan is thought to be small. Bobcats are very susceptible to feline distemper; however, we are aware of only one being positively diagnosed with the disease. Feline distemper is not transmitted to humans.

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For questions about wildlife diseases, please contact the *Michigan DNR Wildlife Disease Laboratory**.***