FELINE PANLEUKOPENIA

Synonyms: Also known as "**Feline Distemper", "Feline Enteritis", and "agranulocytosis**",

Introduction: Feline panleukopaenia (FPL) is a highly contagious, and usually fatal febrile disease of cats and other felidae, such as wild cat, cheetah, leopard and tiger, raccoons and mink, caused by feline parvovirus, but it does not harm canids. Kittens are affected most severely.

Etiology: Disease is caused by **FELINE PANLEUKOPENIA VIRUS (Feline Parvovirus)** and the Parvoviruses are the **SMALLEST VIRUSES** of vertebrates. Feline panleukopenia virus (FPV) is closely related to mink enteritis virus and the type 2 canine parvoviruses (CPV) that cause canine parvoviral enteritis.

Single-stranded (DNA) Parvoviruses replicate inside the Nucleus and are Cytocidal. A unique feature of parvoviruses is that they **depend on Cell Proliferation** for viral DNA synthesis to occur. This restricts lesions to those tissues in which cells are undergoing mitosis, and explains the difference in cell tropism seen in foetal, neonatal, and adult animals. Inclusion bodies (if present) are Intranuclear.

Spread: Virus is present in all secretions and excretions during the acute phase of illness and can be shed in the feces of survivors for as long as 6 wk after recovery. Infection spreads by direct contact or through fomites (eg, shoes, clothing). Cats are infected oronasally by exposure to infected animals, their feces, secretions, or contaminated fomites. Most free-roaming cats are thought to be exposed to the virus during their first year of life. In pregnant queens, the virus may spread transplacentally to cause embryonic resorption, fetal mummification, abortion, or stillbirth.

Pathogenesis:

The pathogenesis of parvovirus infections is influenced primarily by the requirement of DNA replication of these autonomous parvoviruses for mitotic cells, which determines many of the differences in the outcome of infections in fetal, neonatal or older animals.

FPV infects and destroys actively dividing cells in bone marrow, lymphoid tissues, intestinal epithelium, and - in very young animals, it targets Cerebellum and Retina. FPV infect the rapidly dividing epithelial cells in the crypts of the intestinal villi of the ileum and jejunum between 3-5 days after inoculation. The virus infection and loss of epithelial cells results in Flattened and attenuated epithelium with shortened intestinal villi leading to

loss of osmotic regulation, with a resulting **diarrhea containing blood and mucus**. Animals may become dehydrated and pyretic, possibly because of endotoxin uptake from the gut.

In pregnant queens, the virus may spread transplacentally to cause embryonic resorption, fetal mummification, abortion, or stillbirth. Alternatively, infection of kittens in the perinatal period may destroy the germinal epithelium of the cerebellum, leading to cerebellar hypoplasia, incoordination, and tremor.

Panleukopenia is a striking feature of many FPV infections of cats, neutrophil counts decrease appreciably, Lymphocyte numbers also decline, although to a lesser degree, but there is very little effect on eosinophil, basophil, monocyte, or red cell numbers.

Signs

Most cats that become ill are usually <1 yr old. Mortality is highest in young kittens < 5 month old. The disease is characterized by **Severe Panleukopaenia** (all types of leukocytes), **Fever**, and **Enteritis**, resulting in extreme **Dehydration**. Disease runs a rapid course, its onset is marked by lassitude (tiredness, lack of energy), and sudden rise of temperature to between 104 - 105˚F.

The **fever is diphasic (biphasic) (so called Feline Distemper)**. It falls after about 24 hours and rises about 48 hours later. Vomiting usually develops 1–2 days after the onset of fever; it is typically Bilious and unrelated to eating. Diarrhea, profound Depression, Extreme Dehydration and Abdominal Pain. Terminal cases are hypothermic and may develop septic shock and disseminated intravascular coagulation. Death usually occurs soon after the second peak of temperature.

In cases of cerebellar hypoplasia, ataxia and tremors with normal mentation are seen. Retinal lesions, if present, appear as discrete gray foci.

Lesions *(Intestine, Lymph node, bone marrow, CNS)*

There are typically few gross lesions, although dehydration is usually marked. The gross lesions consist of extreme dehydration and emaciation, with mucopurulent exudate on the nasal and lachrymal mucosa. The mucosa of the ileum is covered with haemorrhagic exudate. Mesenteric lymph nodes are oedematous and enlarged. The bone marrow in the long bones is often yellowish or white, and semi-fluid.

The main lesions are found in the **gastrointestinal tract**. Bowel loops are usually dilated and may have thickened, hyperemic walls. There may be petechiae or ecchymoses on the intestinal serosal surfaces. Virus replicates in the diving cells in the

Crypts of Lieberkuhn. Histologically, the intestinal crypts are usually dilated and contain debris consisting of sloughed necrotic epithelial cells. Blunting and fusion of villi may be present. Eosinophilic Intranuclear inclusion bodies are occasionally seen in lining epithelium at the sites of erosion.

Lesions always occur in **Lymphoid Organs**, which are site of initial viral replication before dissemination to gastrointestinal epithelium, or other tissues. Initially Lymph nodes are oedematous and hyperaemic, followed by **Necrosis of Lymphocytes in** follicular and paracortical regions in **lymph nodes**, the Malpighian corpuscles of the **Spleen**, the cortex of the **Thymus**, and **Peyer's patches**. In animals surviving infection, marked regenerative hyperplasia of lymphocytes occurs. The bone marrow is markedly hypocellular. This results from necrosis of all stem-cell populations.

Feline Ataxia / Cerebellar Hypoplasia: Infection of kittens either ***in utero*** or shortly after birth can result in viral replication in the cells of the external germinal epithelium of the cerebellum, resulting in cerebella hypoplasia in Kittens. In “Feline ataxia syndrome”, the virus invades cells of the **External Germinal Layer of Foetal CEREBELLUM**, produces intranuclear inclusion bodies and necrosis, and causes gross or microscopic "hypoplasia" of Cerebellum due to lytic infection of the Purkinje cells in the newborn kitten.results from an impaired development of the cerebellum due to lytic infection of the Purkinje cells in the kitten.

Diagnosis

- A presumptive diagnosis can usually be made from **symptoms** and **agranulocytosis.** (Neutropenia is more consistent finding the Lymphopenia). Total WBC counts <2,000 cells/μL are associated with a poorer prognosis. During recovery from infection, there is typically a rebound neutrophilia with a marked left shift.
- Demonstration of intranuclear inclusion bodies in the epithelial cells of the small intestine is helpful in diagnosis.
- In practice, LATEX AGGLUTINATION or Immunochromatographic Tests for FPV antigen detection in faeces.
- PCR-based test on whole blood or faeces.
- Differential diagnoses include other causes of profound depression, Leucopenia, and GI signs, like Salmonellosis, Feline Leukemia Virus and Feline Immunodeficiency Virus (FIV). Concurrent infection with FeLV and FPV can cause a panleukopenia-like syndrome in adult cats.