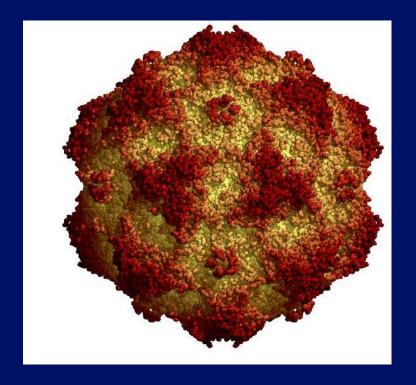


PARVOVIRIDAE





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MORPHOLOGY OF PARVOVIRUSES

- The members of this family are small sized as the name itself means (parvo= small).
- Icosahedral symmetry (spherical).
- Size 20-25 nm (smallest DNA virus).
- Non enveloped virus
- Hardy in nature & resistant to environmental stress.

Continue...

- DNA of virus is linear
- Size of the genome is 5.3 kb
- Two types of proteins present in virus i.e structural proteins :VP1,VP2,VP3 nonstructural proteins:NSP1,NSP2

Out of these proteins VP2 is most immunogenic

Physiochemical Properties

- Virus is stable at pH 3-9
- Virus is heat stable, resistant at temp of 70° C for 60 min.

Main host

Bovine, canine, porcine, feline

Division of parvoviridae

- Parvoviridae family is further divided into 2 sub family namely:
- 1. Parvovirinae: infects birds & mammals.
- 2. Densovirinae: infects insects only.

Continue...

Parvovirinae contains three genera:

1)Parvovirus: it includes members of which infects vertebrates & replicate autonomously.

2) Erythrovirus:

Includes human parvovirus B19 and a related virus of monkeys which also replicate autonomously.

3)Dependovirus:

includes members of which are called adenoassociated viruses because they are defective and unable to replicate except in the presence of a helper virus, usually an adenovirus.

Genus: Parvovirus

- Viral replication takes place in the nucleus so intranuclear inclusion bodies are produced in the nucleus.
- In infections of the fetus (pig or cat) or newborn (dog or cat) where there is considerable cell division in many organs, the infection may be widespread; in older animals a narrower range of tissues is affected.

 At all ages, the continuous division of cells in lymphoid tissues and the intestinal epithelium leads to common occurrence of leukopenia and enteritis.

Manifestations of Parvovirus Diseases in Animals

- Feline panleukopenia virus: Generalized disease in kittens, with panleukopenia, enteritis; cerebellar hypoplasia.
- Canine parvovirus 2 (subtypes 2a, 2b, 2c): Generalized disease in puppies; enteritis, myocarditis (rarely), lymphopenia.
- Porcine parvovirus: Stillbirth, abortion, fetal death, mummification, infertility

Continue...

- Mink enteritis virus: Leukopenia, enteritis
- Goose parvovirus: Hepatitis, myocarditis, myositis
- Duck parvovirus: Hepatitis, myocarditis, myositis

CANINE PARVOVIRUS

 Earlier this virus was known as canineparvo virus 1 (CPV1)/ minute virus of canines.

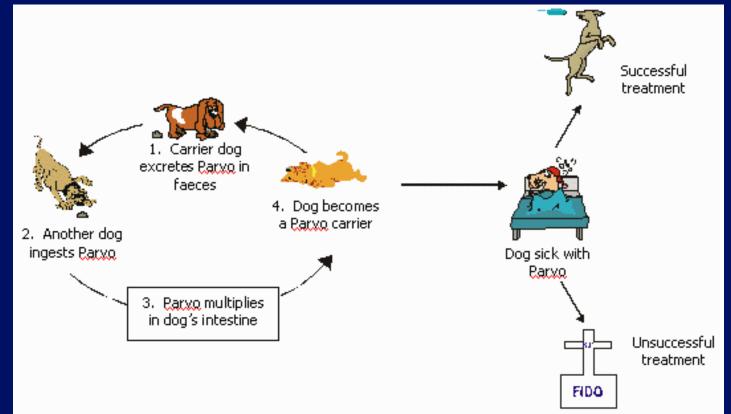
 Later virus structure was evovled & than it was named as canine parvo virus (CPV)2 which is further divided into CPV2a, CPV2b & CPV2c.

Epidemiology

- Canine parvovirus 1 was recognized in 1978 as a cause of hemorrhagic gastroenteritis simultaneously from USA, Australia, UK & others parts of the world
- In India virus was first reported in 1981 near Chennai.
- Mostly affects young pups of 4-8 weeks of age.
 The relative availability of mitotically active cells in specific tissues during differentiation in early life confers an age-dependent susceptibility to several parvovirus-induced diseases.

TRANSMISSION

- By direct contact
- Indirectly through excretions of the animal & the infected animal excretes the virus in high concentration in faeces



PATHOGENESIS

Virus enters through oro-feacal route



Viraemia after 2-7 days



Multiplication of virus in lymphoid tissue



Virus replicates in epithetial cells of intestinal villi

Effects osmoregulatory function of intestine

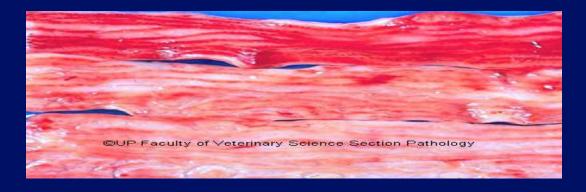


Leads to diarrhoea



Clinical symptoms

- Enteritic form: Rise in temp, Anorexia, vomition, haemorrhagic diarrhoea (due to destruction of epithelial cells of intestinal villi) Vomition & diarrhoea leads to dehydration & death.
- Myocarditic form: Affects pups of 3-8wks of age.
 There is cardiac arythemia, dysponea, laboured breathing leading to acute heart failure.



DIAGNOSIS

- Clinical symptoms & history of infection.
- Detection of antibody by ELISA.
- Detection of antigen by hemagglutination, hemagglutination inhibition.
- Detection of virus by PCR.
- Cultivation of virus in different cell lines:

MDCK-Maden Darby Canine Kidney cell line CRFK-Crandle Feline Kidney cell line

PREVENTION & CONTROL

- Vaccination of pups at 6-8 weeks of age followed by booster at 11th weeks of age.
- Both inactivated and liveattenuated virus vaccines available
- Control can done be by strict hygiene, disinfection of contaminated premises & segregation of infected animals.



FELINE PANLEUKOPENIA

- Also known as FELINE INFECTIOUS ENTERITIS or FELINE DISTEMPER
- Highly contagious generalized disease of domestic and wild cats caused by FELINE PANLEUKOPENIA VIRUS. Only one serotype of this virus has been identified.

Host

- Domestic and wild cats, although cats of all ages are susceptible to infection
- Desease occurs predominantly in young recently-weaned kittens, as maternally-derived antibody levels wane.

Transmission

- Transplacental infection occurs in fully susceptible queens.
- High rates of virus excretion occur during the acute stage of the disease, mainly in faeces but also in saliva, urine, vomitus and blood.
- Faecal shedding usually continues for some weeks following clinical recovery.
- Fleas and **humans** may act as mechanical vectors. Prognosis is grave if the white blood cell count falls **below 1000 cells** per cubic millimeter of blood.

PATHOGENESIS AND PATHOLOGY

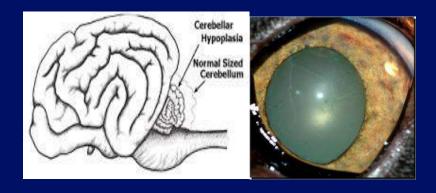
- Following ingestion or inhalation, replication occurs in the mitotically active lymphoid tissues of the oropharynx and associated lymph nodes.
- Viraemia develops within 24 hours, producing infection of the cells of the intestinal crypts and the lymphopoietic cells of the bone marrow, thymus, lymph nodes and spleen.
- Destruction of these target tissues results in panleukopenia and villous atrophy.
- The crypts of Lieberkiihn are dilated and contain necrotic epithelial cells. Intestinal villi become blunted and may fuse.
- The effects of transplacental infection range from cerebellar hypoplasia and retinal dysplasia to foetal death.

CLINICAL SIGNS

- The incubation period of ranges from two to ten days but is typically four to five days.
- Subacute disease presents as depression, fever and diarrhoea lasting one to three days, followed by rapid recovery.
- The disease is most severe in young unvaccinated kittens between 6 and 24 weeks of age.
- Vomiting, sometimes accompanied by diarrhoea or dysentery, follows within two days and can result in severe dehydration and electrolyte imbalance.

Continue...

- Intrauterine infection of the developing foetus often occurs.
- Foetal infections early in gestation may result in resorption or abortion.
- Stillbirths, early neonatal death and teratological changes such as cerebellar hypoplasia and retinal dysplasia may occur in the litters of queens infected during late pregnancy.
- Kittens with cerebellar hypoplasia exhibit cerebellar ataxia manifested as hypermetria, incoordination and frequently, intention tremors.



DIAGNOSIS

- Specimens for virus isolation in primary feline cell lines include oropharyngeal swabs, faeces, spleen, mesenteric lymph nodes and ileum.
- A white cell count of less than 7 x 10⁹/L is often encountered in acutely affected animals. Neutropenia is more common than lymphopenia.
- Intranuclear inclusion bodies may be detected in crypt cells.
- Viral antigen can be detected in faeces using ELISA or haemagglutination employing pig or Rhesus monkey red cells.
- A rising antibody titre may be detected in serum samples by Haemagglutination- inhibition (HAI) or Virus neutralization (VN) tests.
- PCR

CONTROL

- Inactivated vaccines are less effective than modified live vaccines and require booster inoculations. They are safe for pregnant queens and might be considered for vaccination of Siamese and Burmese kittens.
- Modified live vaccines can be used to immunize kittens at 8 to 10 weeks of age, with a booster dose at 12 to 14 weeks of age. These vaccines should not be used in pregnant queens because replicating virus may cause cerebellar hypoplasia in developing foetuses.
- Premises should be thoroughly disinfected with 1% sodium hypochlorite or 2% formalin.