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Canine Parvovirus – Pathogenesis, Pathology and Diagnosis

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Abstract

Canine parvovirus is one of the most important intestinal pathogens in puppies and dogs. It is a single stranded non-enveloped DNA virus belonging to family of *Parvoviridae*. CPV-2 was first recognized in 1978 in USA and soon after that in 1980s two antigenic variants termed as CPV-2a and CPV-2b was reported. Later in 2000s CPV-2c was identified in Italy. On postmortem examination, intestine showed hyperaemia and haemorrhages along with accumulation of blood. In young pups myocarditis was a consistent finding. Histopathlogical examination revealed intranuclear basophilic inclusion bodies in the intestinal epithelial cells and cardiac muscle fibres. Necrosis and depletion of the peyer's patches, mesenteric lymph nodes, thymus and spleen was also observed.

Introduction

Canine parvovirus is one of the causative agents of acute hemorrhagic gastroenteritis and myocarditis in dogs. The disease cause severe illness among young and unvaccinated dogs. The disease occurs into two clinical manifestation gastroenteritis with vomition and diarrhea in dogs of all age groups (Appel *et al.,* 1978), myocarditis in young pups of less than 3 months of age (Hayes *et al.,* 1979). CPV-2 was first recognized in 1978 and soon after that in 1980s, two antigenic variants termed as CPV-2a and CPV-2b were reported. CPV-2c was first identified in 2000s in Italy (Decara and Buonavoglia, 2012) and later reported in Europe, Asia and South America (Grecco *et al.,* 2018). In India, the disease was first reported in 1982 and after that a large number of outbreaks have been reported from different parts of the country (Ramadass and Khadher, 1982).

Etiology

It is a non-envelope single stranded DNA virus under the family *Parvoviridae,* genus *Protoparvovirus,* subfamily *Parvovirinae* (Dogonyara, 2010).

Pathogenesis

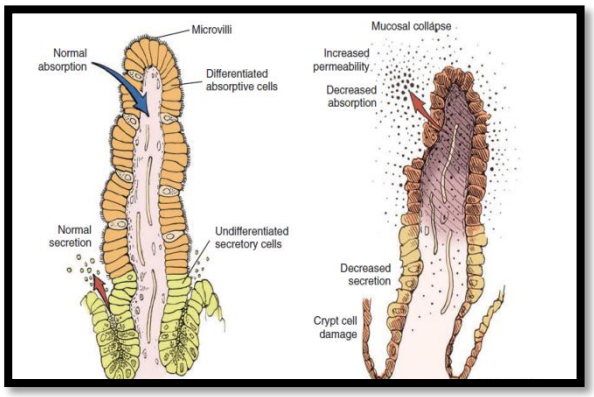


Fig. : A- Normal intestinal villus showing cellular differentiation along the villus
B-Parvovirus-infected villus showing collapse and necrosis of intestinal villus

Virus start replication in lymphoid tissues of oropharynx, mesenteric lymph nodes, bone marrow and thymus. Virus enters into the blood stream (Viraemia). It goes onto the gastrointestinal tract of the tongue, oral cavity, oesophagus and intestinal tract especially the germinal epithelial cells of the intestinal crypts causing malabsorption and increased intestinal permeability. Infection of leukocytes mainly circulating and tissue-associated lymphocytes induces lymphopenia and neutropenia (Pollock, 1982).

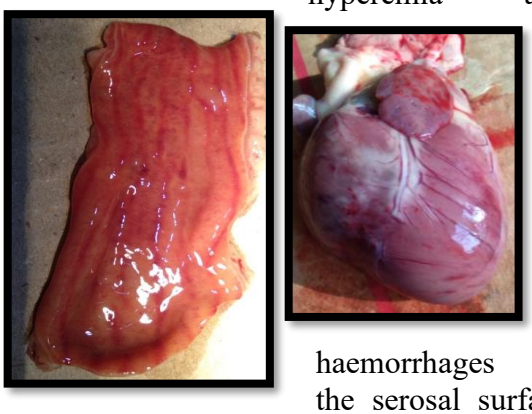
Clinical signs

Anorexia or lethargy, weakness, depression, foul-smelling diarrhea which could be mucoid or completely hemorrhagic, vomition, dehydration and fever are common clinical signs of CPV-2 infection that are non-specific or referable to enteritis (Houston *et al.,* 1996; Kalli *et al.,* 2010).

Pathology

Gross lesion

Carcass showed emaciation and dehydration. The most gross lesions were hyperemia and



haemorrhages on the serosal surface of small intestine. In some dogs the color of the intestinal content ranged from yellowish or reddish or brownish (de Oliveira *et al.,* 2018). Heart showed hemorrhages in the epicardium and pale streaks may be visible in the myocardium of pups with sub-acute heart failure.

Histopathology

Microscopically, necrosis of crypts of epithelium in small intestine and depletion of lymphoid tissues were observed in affected dogs. Pulmonary congestion, alveolar edema and moderate mixed inflammatory infiltrate in lungs were frequently observed (de Oliveira *et al.,* 2018). Intranuclear basophilic inclusion bodies in the intestinal epithelial cells and cardiac muscle fibers were consistent findings (Behera *et al.,* 2014).

Diagnosis

A presumptive diagnosis of CPV can be made based on the clinical signs such as vomition, bloody diarrhea with foul smell, anorexia and fever. The diagnostic tests which were employed include Haemagglutination, electron microscopy, virus isolation using Madin-Darby Canine Kidney Cells (MDCK), ELISA, Fluorescent antibody test (FAT) and PCR (Appel *et al.,* 1979; Mohan *et al.,* 1993; Nandi *et al.,* 2007).

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