The Pharma Innovation Journal 2021; SP-10(11): 2432-2434

www.ThePharmaJournal.com

The Pharma Innovation

ISSN (E): 2277- 7695
ISSN (P): 2349-8242
NAAS Rating: 5.23
TPI 2021; SP-10(11): 2432-2434

www.thepharmajournal.com
Received: 21-09-2021
Accepted: 25-10-2021

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Prognostic indicator of canine parvoviral infection in dogs

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Abstract
Canine parvovirus (CPV) is the leading cause of gastroenteritis associated mortality in puppies particularly of less than six months age. Canine parvovirus -2 and its subtypes CPV-2a, 2b and 2c are the etiological factors responsible for haemorrhagic gastroenteritis and myocarditis in puppies. Haemorrhagic gastroenteritis and myocarditis are the usual clinical entities reported in dogs affected with CPV but rarely erythema multiformae, nervous manifestations, retrograde urinary tract infection and phlebitis are reported. Three puppies of less than seven months age with clinical manifestations of pyrexia, vomition, foul smelling haemorrhagic gastroenteritis, significant dehydration were diagnosed for CPV infection by commercially available lateral flow assay kit. Haemogram revealed all three puppies had significant leucopenia of less than 1100 cells/µl on the second day of presentation. Absence of significant increase in leucocyte level was noticed in one puppy with severe leucopaenia (780 cells/ µl) even after administration of Fligrastim (human granulocyte colony stimulating factor (hG-CSF) at the dose rate of 10ug/kg body. All three puppies were succumbed due to CPV infection and it is concluded that severe leucopenia could be used as a prognostic indicator to determine the outcome of CPV infection. Hence it is recommended that periodical immunization of bitches together with weaning of puppies at 45 days age to enhance the passive immunity, immunization as early as 6 weeks with recommended boosters are the best strategies for prevention of CPV infection in dogs.

Keywords: canine parvovirus, dogs, leucopaenia, prognostic indicator

Introduction
Infectious gastroenteritis of viral etiology is most commonly reported clinical entity associated with high mortality and morbidity in young puppies less than six months age. Canine parvoviral infection is considered as the most pathogenic viral infection of dogs from weaning to six months age (Kapil, 1995) [10]. The disease is caused by Canine parvovirus (CPV) type 2 and its variants including CPV-2a, 2b and 2c (Greene and Decaro, 2012) [8]. Even though, dogs of any breed or sex, between 6 weeks and 6 months of age are more susceptible to CPV-2 and its variants. Window of susceptibility of puppies to CPV infection coincides in the above age group due to exponential multiplication of virus in actively dividing intestinal cells in response to change in diet associated with weaning, waning maternal antibodies with advancement of age and failure of complete protection by early vaccination against wild CPV infection (Geetha and Selvaraju, 2021) [7]. Faeco-oral route (direct transmission) or oro-nasal exposure to faeces contaminated fomites (indirect transmission) are usual transmission of CPV in between dogs (Prittie, 2004) [14]. Haemorrhagic gastroenteritis, myocarditis are the usual presentation of CPV infection in dogs characterised by initial leucopaenia followed by leucocytosis (Greene and Decaro, 2012) [8]. The present study describes the significance of leucopaenia as a prognostic indicator in predicting clinical recovery of dogs affected with CPV.

Materials and Methods
Three puppies (two months old Labrador male, Germen Shepherd male of six month age and Greatdane male of two months age) were presented to the Infectious disease unit of Veterinary Clinical Complex, Veterinary College and Research Institute, Namakkal with the history of absence of immunization, vomiting, anorexia, haemorrhagic enteritis. Pyrexia (40 °C), congested conjunctival mucous membranes, semi dry buccal mucosa, sunken eyeballs, nausea, haemorrhagic foul smelling enteritis and absence of detectable abnormality on abdominal palpation were the clinical findings observed in all puppies. Faecal samples were collected aseptically in phosphate buffer saline (PBS) and subjected to commercially available lateral

flow assay kit (Parvofind, Cisgen, India), and all of them were positive for CPV infection. Blood sample was also collected aseptically from the cephalic vein of all dogs in EDTA vials and clot activator vials (2ml) for haematobiochemical analysis in automated serum biochemical analyser (Vetscan2, Abaxis, United Kingdom and Biosystems Diagnostics Pvt. LTD, India). All puppies were treated with Amoxacillin and cloxacillin (Intamox, Intas Pharmaceuticals Ltd., Ahemadabad) @ dose rate of 20 mg/kg body weight, ondansetron @ 0.2 mg/kg body weight, pantaprazole @ 1 mg/kg body weight, crystalloid fluids (Ringers lactate and 5 % dextrose normal saline) after assessing the dehydration status for 7 days as per the recommendations of Greene and Decaro (2012) [8]. Absence of clinical improvement was noticed in all puppies even after intensive treatment for the first two days. Filgrastim, recombinant human granulocyte colony stimulating factor (Neukine, rHu G-CSF, Intas Pharmaceuticals Ltd., Ahmedabad) was administered one puppy (Male Great dane, 2 months age) having severe leucopaenia (780 cells/µl of blood) but there was no significant increase in the leucocytes afters 24 hours of injection and that puppy succumbed after 4 days. Absence of clinical improvement with deterioration was observed in the other two puppies and they were died during the third day of presentation.

Results and Discussion

Canine parvoviruses (CPV) are the leading cause of most dangerous life threatening highly contagious, fatal haemorrhagic gastroenteritis of puppies of less than six months age. Parvoviruses are small, single stranded, non - enveloped DNA viruses (Carr *et al*., 1997) [6]. Canine parvovirus -2 (CPV-2) was first described as a cause of deadliest haemorrhagic gastroenteritis of puppies in 1978 and was named as CPV type 2 differentiated from Canine parvovirus -1 (CPV-1), the minute virus of canines (Binn *et al*., 1970) [3]. After its first emergence, the virus spread worldwide and now endemic in domestic and wild canids (Parrish *et al*., 1988) [13]. Periodical antigenic mutations of CPV-2 ended in the emergence of newer sub types including CPV-2a, CPV-2b and recently CPV- 2c (Buonavoglia *et al*., 2001) which affects felines apart from canines (Greene and Decaro, 2012) [8]. The above antigenic variants have completely replaced their original type 2, which is still used in most commercial vaccines (Behera *et al*., 2015) [4]. The disease is endemic in India and reported in various states including Tamil Nadu (Surendhar *et al*., 2019) [15].

Classical clinical presentation of CPV-2 and its subtypes infection in canines is haemorrhagic gastroenteritis with pyrexia, septicaemia associated with gram negative sepsis secondary to pathological damage of gastrointestinal tract of CPV - 2 affected puppies (Appel *et al*., 1978; Macartney *et al*., 1984) [1, 11]. Typical clinical manifestations described by the above authors were reported in all three dogs brought for therapy. All dogs were positive for CPV infection by detection of antigen in the commercially available lateral flow assay kit. Inspite of intensive antibiotic treatment, with recommended antiemetic, antiulcer drugs with appropriate crystalloid fluid therapy as recommended by Greene and Decaro (2012) [8], clinical improvement was not significant in all puppies and two of them died on third day. Significant leucopaenia (<1700 cells/µl), mild anaemia (10.5 + 1.1 gm/dl), hypoalbunameia (2.2 g/dl) were the major haemato-biochemical abnormalities noticed in all puppies. Remarkable leucopaenia of <1050 cells/ µl of blood could be a bad prognostic marker on the survivability of dogs affected with CPV (Greene and Decaro (2012) [8]. The above authors findings clearly reflected in this study also where all the puppies had leucocytes of less than 2000 cells/ µl, and they were succumbed to the deadliest viral infection. Hence it is recommended that along with confirmation of CPV infection, haemato-biochemical analysis can also be carried out to find out the prognosis.

Leucopaenia, although not found in all dogs is usually proportional to the severity of illness and its monitoring may yield prognostic information about the likely course of infection (Goddard *et al*., 2008) [9]. Median white blood cells (WBC) of survivors of CPV infection was never decreased below the lower reference limit (4050/µl) and started increasing 24 hours after admission, where as in non survivors it was markedly below the lower reference limit at 24 and 48 hours post admission and significant differences between survivors and non survivors with respect to the proportion of cases below the lower reference limit for WBC was also observed by Goddard *et al*. (2008) [9]. He recommended that accurate prognosis could be obtained at 24 hours after admission by evaluating the change in total leukocyte, band neutrophil, lymphocyte, monocyte, and eosinophil counts.

One puppy with severe leucopaenia (780 cells/µl of blood) was treated with Neukine (Filgrastim), a commercially available recombinant human granulocyte colony stimulating factor as per the recommendation of Areshkumar *et al.* (2017) [2]. The above author reported that there was a notable elevation of leucocytes from 1000 cells/µl to 8000 cells/µl after two days of administration of filgrastim but it was not noticed in this study which could be explained as prominent hematological abnormality in canine parvoviral enteritis is leucopaenia due to the destruction of bone marrow precursors, the depletion of lymphoid tissues, and the increased demands of the massively inflamed intestinal tract of affected dogs (Mathios *et al*. 2016) [12].

Periodical vaccination of bitches against CPV infection for enhancement of passive immunity of puppies through maternally derived antibodies (MDA), augmenting the duration of protection by MDA by following appropriate weaning strategies, ie. Weaning at 45 days of age are secondary prevention strategies to be addressed at the field level to reduce mortality puppies in CPV infection. Completion of series of recommended vaccination in the early life of puppies will be definitely help to reduce the immunocompromisy of CPV infection in puppies.

Acknowledgement

The authors acknowledge Tamil Nadu Veterinary and Animal Sciences University for necessary facilities for the study.

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