Review Article

Update on Canine Parvovirus Infection: A Review from the Literature

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Abstract | Canine parvovirus (CPV) enteritis is a highly contagious, virulent, acute, and fatal gastrointestinal viral disease that fundamentally transforms the pups and collapses the body's posthaste branching organs, such as bone marrow, lymph node, and the flimsy cell of the intestine, causing bloody diarrhea amidst a high mortality and morbidity rate. CPV is distributed in the family Parvoviridae beneath the subfamily Parvovirinae that pertains to the genus Protoparvoviorus with a high genomic replacement unlike other DNA virus; the organism is primarily segmented into three forms acknowledged as (CPV-2a, CPV-2b, and CPV-2c), which resembles to be liable for the infection's statewide spread. For transmission, the fecal-oral pathway is deemed the most obvious route than other permissible routes. Moreover, spreading through contact interactions, environmental pollutants, and the host reservoir, like a stray dog, could be feasible. Distressingly, such a pathogen is currently endemic in Asian nations, including India, Bangladesh, Sri Lanka and China. Thus, the literature review encompassed comprehensive knowledge concerning contemporary disease occurrence with causes and transmission imperative for management practice. Besides, the study converged on afresh advanced treatment procedures, vaccine progressions, and public awareness efforts, which can be a baseline for the policymaker, veterinarians and pet owner to limit further outbreaks.

Editor | Muhammad Abubakar, National Veterinary Laboratories, Park Road, Islamabad, Pakistan.
Received | December 07, 2020; **Accepted** | June 24, 2021; **Published** | July 08, 2021
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Citation | Mia, M.M. and M. Hasan. 2021. Update on canine parvovirus infection: A review from the literature. *Veterinary Sciences: Research and Reviews*, 7(2): 92-100.
DOI | https://dx.doi.org/10.17582/journal.vsrr/2021.7.2.92.100
Keywords | Canine parvovirus, Epidemiology, Transmission, Vaccine, Prevention

Introduction

Companion animals, such as dogs and cats, serve an important meaning in our communities. Therefore, people want to keep them for physiological, social, and emotional explanations (Robertson *et al.*, 2000). Dissimilarly, new emerging disease like the Canine parvoviral (CPV) infection rapidly transmits among the dog populations of developing countries globally, although few studies have conducted regarding the prevalence of the listed virus from those regions. CPV is a DNA virus (small, non-enveloped with single-stranded genome) that belonged to the family Parvoviridae under the subfamily Parvovirinae and the genus Protoparvovirus, conferring to the recent cataloging. The genomic structure of the DNA molecule includes approximately 5,000 nucleotides with dual interpretation edges (ORFs), including ORF1 and ORF2, which encrypt two non-structural proteins (NS1, NS2). Rest two structural proteins identified as VP1 and VP2 encode and from another merging of the similar mRNAs (Decaro and Buonavoglia, 2012).

Throughout the first half of the 1980s, CPV-2 strain rapidly and entirely replaced into the novel CPV-2a;

moreover, between 1991 and 2001, CPV-2b and CPV-2c verities were recognized in the dog population (Zhou *et al.*, 2017). However, though the three alternatives are disseminated worldwide, the ancient CPV-2 variety no longer transmits nowadays (Decaro *et al.*, 2020; Decaro and Buonavoglia, 2017). Considering the significant symptom, the common viral cause of bloody diarrhea spreads rapidly with high morbidity (near about 100%) and mortality rate (approximately 10%) (Schoeman *et al.*, 2013; Shackelton *et al.*, 2005). Analyzing the risk factors, including the age, resilient grade of the dogs, season, the quantity of virus, virulence, and pre-existing parasitic (bacterial or viral) infection, are the foremost clinical manifestation of CPV enteritis (McAdaragh *et al.*, 1982). To be somewhat more specific, clinically, there are two forms of this disease, the enteric form characterized by acute fever, lethargy, anorexia, vomiting, and bloody diarrhea; whereas another type identified as a cardiac form that scarcely finds in neonates causing the failure of the respiratory and cardiovascular manifestations, with the infestation in utero (Schatzberg *et al.*, 2003; Shima *et al.*, 2015). In case of transmission, though the dogs can be infected by pathogens found on fomites, such as shoes, clothes, human fingers, food dishes, and other cookware, CPV can be spread predominantly by the fecal-oral route and from the contaminated of prone dogs (Bajehson, 2010).

Therefore, investigation to detect modes of transmission and make the dog owners more aware of preventive strategies is paramount to reduce its accelerated occurrence. This paper aims to focus on epidemiology, mode of transmission, pathogenesis, treatment, and vaccination to take any action before implementing somewhat control programs for curbing further outbreaks.

Epidemiology and risk factor

In 1978, CPV-2 became considered the cause of significant canine enteropathy and cardiomyopathy (Goddard and Leisewitz, 2010). The original CPV-2 strain has now been exposed worldwide and categorized into CPV-2a and CPV-2b antigenic forms. In Italy and different parts of the world, CPV-2c, a supplementary antigenic variant, was identified for the first time in 2000. Besides, CPV-2c has previously been isolated sporadically in the United Kingdom, Greece and Bulgaria, where there was a more conspicuous incidence of CPV-2a/ 2b appearance (Filipov *et al.*, 2011). A preceding study informed that the oldest CPV-2c strain was discovered in 1996 (Decaro *et al.*, 2007a), describing that perhaps the variant had been circulating in Germany for four years before its discovery in Italy in 2000 (Decaro and Buonavoglia, 2012). Besides, Tunisia has a tremendous occurrence for all three kinds outside Europe (Touihri *et al.*, 2009). Additionally, in North America (Hong *et al.*, 2007), type 2b and 2c isolates are more prevalent, whereas CPV-2c is more familiar in South America, excluding Brazil, where almost all propagating strains were classified as CPV-2a (Castro *et al.*, 2011). In contrast, within Asia (Nandi *et al.*, 2010) and Australia (Meers *et al.*, 2007), CPV-2a is the most typical variety, with hardly a few CPV-2c strains detected in India (Nandi *et al.*, 2010).

Throughout the last decade, CPV was reported from Pakistan, China, Nigeria, Tunisia, Egypt, Bangladesh and India, whereas the notable prevalence rate was inscribed from Egypt and Tunisia, respectively 59.75% and 32.2% (Table 1). Analyzing the risk factors, varied reasons like age, sex and breed are mainly susceptible to CPV-2 infection (Castro etal., 2007; Gombač *et al.*, 2008). In Table 1, most of the evidence designates

Table 1: *Prevalence with risk factors of CPV since 2010 to present.*

Year	Country	Total prevalence (%)	Prevalence (%)									Reference
			Sex		Age		Breed		Season			
			Male	Female	<1 year	>1 year	Indigenous	Pure	Summer	Winter	Rain	
2010-2011	Pakistan	22.7	41.5	58.5	95.5	4.5	5.5	94.5	N/A	38	62	(Umar *et al.*, 2015)
2010-2014	Chin	19.6	18.9	20.2	14.6	5	2.7	30.2	12.9	14.4	13.3	(Luo *et al.*, 2017)
2010-2016	Nigeria	5.7	60.7	39.3	93.3	6.7	N/A	N/A	N/A	48.3	51.7	(Tion *et al.*, 2018)
2012-2013	Tunisia	32.32	50.26	40.7	32.3	N/A	N/A	N/A	31.4	11.1	57.4	(Tagorti, 2018)
2012-2013	Egypt	59.7	N/A	N/A	92.5	20	N/A	18.1	77.1	16.6	46.1	(Sayed-Ahmed *et al.*, 2020)
2016-2017	Bangladesh	13.94	18.45	11	11.74	8.27	4.4	14.8	17.5	12.1	11.6	(Roy *et al.*, 2018)
2017-2018	Jabalpur (India)	7.24	7.91	6.36	N/A	N/A	12.57	4.43	N/A	N/A	N/A	(Khare, 2019)

that the proportion of CPV contamination is very eminent in male individuals rather than female; meanwhile, the puppies below one year aged and pure breed dogs are more receptive to the viral infection. Therefore, the study is in line with the previous study reported that young puppies are vulnerable to infection due to the biological half-life of the parvovirus maternal antibody (only around ten days), and this natural resistance emanates from the mother, rendering puppies more prone to infections (Roy *et al*., 2018). However, (Nandi and Kumar, 2010) proclaimed that CPV has also a devastating effect on pure breeds like Rottweilers, German Shepherd, English Springer Spaniels, and Doberman Pinchers.

Transmission

CPV is a highly contagious disease that can transmit from the fecal waste of diseased individuals, whose severity can alter from slight to over 90% if the individuals do not get treatment or receive any faulty treatment (Kumrul *et al*., 2017). Going dipper, CPV is resistant to disinfectant with extremely stable power to stay in the environment for a long period (Khatri *et al*., 2017). The incubation period of this virus is 4-5 days (Bajehson, 2010). Excluding the fecal-oral route, the feces of dogs, pet shops, kennels, breeding equipment, and veterinary clinic can act as a secondary source of infection among the canine population (Bajehson, 2010; Behdenna *et al*., 2019; Nandi and Kumar, 2010). Likewise, dogs can also infect by fomites, including shoes, human hands, clothing, food bowls, and other utensils when they usually lick those items (Decaro *et al*., 2005). In addition, direct interaction or pollution of the atmosphere can also aid CPV transmission (Bagshaw *et al*., 2014; Behdenna *et al*., 2019).

Moreover, street dogs can act as a reservoir and perform a crucial role for operating the life cycle of the pathogen (Islam *et al*., 2014). A recent study held in Australia also reported that feral dogs in a particular area could be responsible for the growing infection rate of the pet dogs in those areas, but further investigation is important for evaluating the transmission (Kelman *et al*., 2020). In the case of cross-transmission, (Behdenna *et al*., 2019) stated that dogs serve as a vector of infection for lions in unidirectional cross-species transmission, and lions do not maintain CPV infection independently of dogs. Despite the unusual direct contact of domestic dogs and lions, several other carnivorous animals could serve as intermediate hosts (Craft *et al*., 2017). However, recent studies indicated that thermal persistence and enhanced potential exposure of CPV pathogen could be responsible for the extended drought periods. Moreover, a strong correlation between CPV frequency and rainfall distribution periods has been indicated (Clark *et al*., 2018; Decaro *et al*., 2020; Rika-Heke *et al*., 2015). Furthermore, the parasitic and other particular causes can also predispose puppies to parvoviral infection, including premature weaning, overcapacity, starvation, and additional anxiety factors (Gamage *et al*., 2020; Mylonakis *et al*., 2016). Thus, comprehending the exact cause of a disease is essential to its prevention.

Pathogenesis

The virus comes in contact when the dog cleans its own body or eats food off the ground or floor through the mouth. Following entered the virus into a dog, it takes (3-7) days development to show signs and symptoms. Then, it replicates an immense number within the lymph node (Khatri *et al*., 2017; Nandi and Kumar, 2010). A few days later, significant masses of viruses in the bloodstream are discharged, and the pathogen invades 3-4 days later in bone marrow (fast dividing cell). Then the virus delicates the intestinal cells and formed broad inclusion bodies (eosinophilic intranuclear) by multiplying viruses. After that, privileged the bone marrow, the pathogen abolishes the new cells of the defense system. Alongside, it commences to knock out the best defensive mechanism of the body. Afterward, the pathogen causes the vastest shocking consequences in the gastrointestinal tract, and due to inflammation of the bone marrow, CPV infection is signalized by a reduction in white blood cells. The typical intestine has small finger-like protuberances called villi. These tiny fingers are immensely strengthened in the surface areas that make it difficult to access liquid and nutrient absorption in the gastrointestinal tract. The villi are compact transitory cells that are readily replaced by the new cells. The rapidly dividing portion at the base of the villi known as the Crypts of Lieberkühn acts as a source of the new cells that provide the actual number of cells in the villi (Nandi and Kumar, 2010). The parvovirus raided the crypt and remodeled the villi into blunted shape, and makes it unable to engross nutrients; thus, diarrhea results due to a reduction of new cells from the crypt and addresses the easy entry of bacteria causing widespread infection. The etiology of recurrent enteropathies in dogs is associated with Enterobacteriaceae (Proteobacteria phylum)

(Cassmann *et al*., 2016; Park *et al*., 2019); belatedly, extreme fluid loss leads to bloody diarrhea and vomiting until shock and death (Nandi and Kumar, 2010).

Treatment

For fluid therapy, after accessing the vascular balance, a crystalloid isotonic solution should be administrated, and the initial fluid application volume delimits the degree of interstitial dehydration, whether the hypovolemia present. For the consequence of hypothermia, tachycardia, bradycardia, hypotension, and overdue capillary refill time, it is more gratifying to give intravenous (IV) solution as promptly as practicable in boluses (20 ml/kg) (Decaro *et al*., 2007b). Besides, serum glucose concentration needs to be monitored regularly and supplemented as necessary for hypoglycemia. Furthermore, for decreasing the serum glucose level (below 60 mg/dL tailed by the adding of 2.5% to 5% dextrose solution in crystalloid fluids), it is mandatory to the administration of IV dextrose solution (Vet One Dextrose 50%) besides bolus (1-2 ml/kg 25% dextrose) (Mazzaferro, 2020).

In patients with CPV, use of antiemetics is vital to use antiemetic drugs for lessening vomiting. Administration of anthelmintic in pups with CPV enteritis showed lessened clinic stay time in patients who have endured antiemetics (Mantione and Otto, 2005). Coequally active in minimizing the number of vomiting incidents in another study was also antiemetics, mainly metoclopramide (0.5 mg/kg IV every 8 hours). Moreover, administration of ondansetron (0.5 mg/kg) through IV route after every 8 hours and maropitant (1 mg/kg) subcutaneously after every 24 hours are also recommended (Mazzaferro, 2020; Yalcin and Keser, 2017).

For antibiotic treatment, administration of Ampicillin (20-40) mg/kg, Ampicillin-sulbactam (30–50) mg/kg, Cefovecin 8 mg/kg, Cefoxitin from cephalosporin group (20–30) mg/kg body weight, Enrofloxacin 10 mg/kg body weight, and Metronidazole 10 mg/kg via IV route except for Cefovecin (subcutaneous) is highly effective (Mazzaferro, 2020). Moreover, in analgesia, vomiting and potential with ileus intussusception may cause abdominal pain in many patients with CPV infection (Mylonakis *et al*., 2016). Partial inhibitors like as buprenorphine (0.01-0.02 mg/kg IV every 8 hours) or else butorphanol (0.1-0.2 mg/kg/hour) can be suggested to genuine agonists, including methadone (0.1-0.2 mg/kg IV every 6 hours), hydromorphone (0.1 mg/kg intravenous, intramuscular every 8 hours) morphine (0.1-0.2 mg/kg, intravenous, intramuscular, or subcutaneous every 8 hours), or fentanyl (1-5 mg/kg/hour IV) (Marquez *et al*., 2015; Mazzaferro, 2020). Furthermore, the current findings examining the rectal application to puppies with CPV enteritis of 10 g of fecal content from the recovered dog, diluted in 10 ml of sterile 0.9% saline, demonstrated the earlier onset of diarrheal resolution increased survival in the fecal transplant community (Pereira *et al*., 2018). Finally, to lessen the fever, diarrhea, vomiting, and mortality and increases appetite, transgenic feline omega-interferon (1–5 106 IU/kg/d IV for three days) can be provided (De Mari *et al*., 2003; Mazzaferro, 2020).

Prevention

Canine parvovirus enteritis disease is extremely virulent and contagious; prevention is imperative to assure the good health of the puppy and dog. The virus is remarkably hardy and can live in feces and soil (organic material) for more than one year (Bajehson, 2010). Besides, CPV is versatile, environmentally stable, and can survive in favorable conditions for many months (Bajehson, 2010; Decaro *et al*., 2020). Thus, for indoor decontamination, about one month, the pathogen loses its virulence; it should perhaps be safe to insert a new puppy indoors, one month after the conclusion of the active infection. On the contemporary, if the exterior is poisoned and cold, one should perpetually wait to heat it before a regulated manner adopting a newborn pup. Shaded areas for seven months and zones with adequate daylight should be known as polluted (Houston *et al*., 1996; Nandi and Kumar, 2010). Though CPV can resist broadly used disinfectants, likely quaternary ammonium compounds), a sodium hypochlorite solution (0.75%) verified respectable effectiveness against the pathogen (Cavalli *et al*., 2018; Decaro *et al*., 2020).

Additionally, mechanical refinement through irrigation after drying the permissible area may also be supportive. Spreading peroxymonosulfate (organic matter) has relatively good action on filthy areas (Nandi and Kumar, 2010). Segregating an at-risk puppy is the best way to avoid infection from CPV exposure. Vaccinated adults' dog (with normal feces) can spread the CPV pathogen via the possible path of contamination. So, to minimize contamination, the risk puppy should not expose till the puppy has

acquired its complete spectrum of vaccines. Barrier methods, including protective gloves, hat, gown, and booties, must be worn in the diarrheic patient, including the negative response of fecal ELISA test, once treating the patient to prevent cross-contamination as well as the advancement of the disease (Mazzaferro, 2020).

Vaccine development

Though the control of canine parvovirus is a global challenge, the most important criterion for minimizing the transmission of infection in dogs is vaccination (Decaro *et al*., 2020). For all dogs, irrespective of situations or geographic region, a core vaccination is important because the vaccine can protect the animals from dangerous and serious diseases (Day *et al*., 2016). Though the vaccination program is increasing, the CPV infection among licensed and confined dogs was 4.12 times among one thousand dogs, according to the latest study of veterinary hospitals in Australia (Kelman *et al*., 2019). In the circumstance of the vaccine category, modified live vaccine (MLV), frequently uses because, by duplicating inside the host, it can induce a durable, lifelong response without causing any tissue harm or clinical signs. Currently, only two CPV forms, the inventive CPV-2 stain, besides its additional variant CPV-2b comprise the CPV MLV vaccine formulations in most countries. However, the MLV can be synthesized in the intestinal mucosa and can be shed (feces of immunized dogs) for at least 3-4 weeks after vaccination (Freisl *et al*., 2017; Zhou *et al*., 2017). This vaccine is also responsible for causing viremia (Decaro *et al*., 2020; Freisl *et al*., 2017). However, for heightening equally antibody and cell-mediated immune responses, Modified Live Virus (MLV) vaccines are used to protect CPV.

Moreover, it offers decent, lifelong coverage against subsequent obstacles of virulent viruses (Ford *et al*., 2017). On the other hand, the "World Small Animal Veterinary Association" Vaccination Community is not strongly suggesting CPV MLV vaccines for wild animals. Also, it is restricted in pups (age less than 4-6 weeks) and at the time of pregnancy because of the potentially harmful impact of vaccines (Day *et al*., 2016; Decaro *et al*., 2020).

To better overcome MDA intervention, it is now suggesting trial and marketable vaccines for oral and intranasal administration, but all commercially viable CPV vaccines offer parenteral route administration (Cavalli *et al*., 2020). Failures in immunization may be vaccinated or host-related, and inappropriate vaccine schedules, vaccine processing or implementation defects, and the defects of vaccine immunogenicity are causes of vaccine-related failures (Altman *et al*., 2017; Decaro *et al*., 2020). On the other hand, age, hereditary factors, and compromised fitness, diet, or immune status are correlated with host-related factors (Wiedermann *et al*., 2016).

Serological testing may be a valuable tool for evaluating the feasibility of vaccination in rising kennels to explore diseased animals and determine the letdown influences to vaccinate (Rota *et al*., 2019). In most studies, CPV seroprevalence already published within the last twenty years, ranging from 86% to 98.5% in typically vaccinated owned dogs (Killey *et al*., 2018; Riedl *et al*., 2015; Rota *et al*., 2019). On the other hand, unvaccinated homeless dogs showed a seroprevalences rate of 67% to 84% (Spindel *et al*., 2018). 70-75% vaccination coverage recommended for the lowest possible in owned dog population groups to avoid disease outbreaks (Decaro *et al*., 2020; Riedl *et al*., 2015).

Vaccination protocol

Protocols for CPV vaccination are a customized action with various variables, including age, breed, dog's way of living, and the occurrence of diseases in a specific geographic area. Therefore, some general guidelines for the canine core vaccinations that include MLV CPV and Canine Distemper are recommended to use, but no standard vaccination policy can cover all possible circumstances (Decaro *et al*., 2020). The minimum age for beginning the primary CPV vaccinated protocol is 6-8 weeks. Then a revaccination period of (2-4) weeks protocol is recommended until the age of 16 weeks or even later after vaccination. Dogs must undergo a booster as a pup within one year of the primary vaccine course (Decaro *et al*., 2020; Ford *et al*., 2017).

Economic impact

People from poor and under developing countries are not highly familiar with pure breed pet dog rearing. The price of a puppy is very high, though a few people are starting to make dog farms for business purposes. As the canine parvovirus is a contagious disease, the contaminated dog can easily spread the virus in the environment. If the outbreak of the disease rises bit by bit, individuals do not express interest in chasing

the puppy. Then, not only for those who engage with the pet dog selling trade but also for the economy created for this business, detrimental factors can arise. Besides, the cost of animal diseases will increase the expenditure on treatment, which typically increases expenses. To sum up, animal disease output depends on the expenses regarding the animal disease, deaths, decreased productivity, and declines in commerce and other receipts (Rushton, 2008).

Acknowledgments

The authors are thankful to the all members of Department of Medicine, Sylhet Agricultural University, for providing guidelines.

Novelty Statement

Previously, many studies reported scattered top to bottom information about CPV. However, our study reviewed the history of the most contagious CPV disease's contemporary prevalence with risk factors, the adversarial impact on dogs, newly ascertained dynamic transmission with treatment procedures, and significant economic repercussions. Furthermore, our study affirmed that the CPV is fast progressing in impoverished and developing countries due to a lack of awareness.

Author's Contribution

MH: Conceptualization, writing original draft preparation and writing, reviewing and editing.
MMM: Supervision, writing, reviewing and editing.

Conflict of interest

The authors have declared no conflict of interest.

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