Clinical Gastroenterologist International

ISSN: 2691-5642

Perspective

A Global View to Parvoviral Enteritis

Bulent Elitok and Duygu Caliskan*

Department of Internal Medicine, Afyon Kocatepe University, Turkey

Abstract

Parvoviral enteritis caused by *parvovirus* is an important disease of the puppies, especially in the early stages of their lives. Most adult dogs are immune to the disease, either *via* natural infection or immunization. Subclinical infections characterized by seroconversion without evidence of clinical disease, are common in the unvaccinated adult. Infection is a disease characterized by hemorrhagic gastroenteritis and/or myocarditis, with high morbidity and mortality. The aim of this review is to draw attention to this disease, which often causes death in small puppies.

Keywords: Canine; *Parvovirus*; Enteritis; Symptoms; Treatment

Introduction

The most important cause of canine viral enteritis is CPV, which emerged as a clinical problem in 1978. Parvoviruses (*Parvoviridae*) are small, non-enveloped, single-stranded DNA viruses that replicate in actively dividing cells. These viruses are hardy, persisting for long periods of time in the environment (5-7 months), and are ubiquitous [1].

Canine *Parvovirus* type-1 (CPV1) has not been identified as a pathogen for a long time and has been shown to cause diarrhea [2]. Canine *Parvovirus* 2 (CPV-2) believed to have emerged from *Feline Panleukopenia Virus* (FPV) or from a *parvovirus* of another wildlife species was the first variant of this virus associated with the development of hemorrhagic diarrhea. Between 1979 and 1985, CPV-2 was largely replaced by 2 more virulent strains of *parvovirus*, CPV-2a and CPV-2b. More than 80% of the isolated cases of CPV in the United States today are CPV-2b [1,3].

It has been reported that virus excretion, which begins 3-4 days after entering the virus organism, lasts for 1-2 weeks and in experimental studies it is reported that virus fecal spread begins at 3 days after inoculation [4]. Parvoviral enteritis, anaerobic bacteria normally found in the intestinal flora, can lead to sepsis by passing through the damaged intestinal mucosa into the bloodstream. In particular, *Salmonella*, *Clostridium*, *E. coli* and *Campylobacter* have been reported to cause endotoxemia septicemia and death [2]. It has been determined that disturbances in the end-stage renal function of the sepsis result in intestinal nephritis and acute tubular necrosis as a result of these deterioration [5]. Other common symptoms are myocarditis in the heart and pneumonia in the lungs [2]. Subclinical infections are frequently detected in offspring and adult dogs with moderate maternal antigens [6].

In the case of parvoviral enteritis, the evaluation of the hemogram as much as the anamnesis is of great importance. [3,7]. Leukopenia has been implicated in the destruction of hematopoietic stem cells in the bone marrow of leukocytes as well as the inability of lymphoproliferative organs such as the thymus, lymph nodes and spleen to respond to the inflammatory need for neutrophils in the digestive tract [1]. It was reported that dogs with CPV enteritis have a high prevalence of clinical thrombosis or phlebitis and laboratory evidence of hypercoagulability without disseminated intravascular coagulopathy. Thromboelastography may help identify hypercoagulable states in dogs [5]. Clinically, antibody-based methods and tests that detect viral antigens are available methods for diagnosing the disease [6,8].

The most important part of the treatment should be aimed at eliminating the dehydration and eliminating the missing fluid and electrolytes, thereby restoring acid-base balance [1,9]. The use of antiemetics in the treatment of parvoviral enteritis has been suggested to be an indication of the use of antiemetics in treatment of the parvoviral enteritis dogs [10]. Antibiotic use in the treatment of the disease may result in bloody diarrhea or high fever in the dog, intestinal barrier deterioration and intravenous, broad spectrum, bactericidal the use of antibiotics is recommended [1,11]. Albumin supplementation studies on animals are inadequate and the use of plasma transfusions for this purpose is not recommended. More recently, immunotherapy methods have become more important in the treatment of parvoviral enteritis puppies [3,8]. A recent veterinary review article recommended plasma infusion to raise the plasma albumin to 2.0 g/dL to 2.5 g/dL, and the preferential administration of non-protein, synthetic colloids to maintain the plasma COP between 13 mm Hg and 20 mm Hg. However, a large volume of plasma is required to achieve a small increase in plasma albumin (22.5 mL/kg plasma will raise plasma albumin by 0.5 g/dL) [1].

The aim of this review was to draw attention to the parvoviral enteritis table, which has significant morbidity and mortality rates especially in young offspring or immunocompromised dogs. We hope that this compilation will contribute to all scientists, especially clinical or general practitioner veterinarians.

Citation: Elitok B, Caliskan D. A Global View to Parvoviral Enteritis. Clin Gastroenterol Int. 2018; 1(1): 1001.

Publisher Name: MedText Publications LLC

Manuscript compiled: September 24th, 2018

***Corresponding author:** Bulent Elitok, Afyon Kocatepe University, Faculty of Veterinary Medicine, Department of Internal Medicine, Afyonkarahisar, 03200, Turkey, E-mail: elitok1969@hotmail.com

References

1. Prittie J. Canine parvoviral enteritis: A review of diagnosis, management, and prevention. J Vet Emerg Crit Care. 2004;14(3):167-76.
2. Goddard A, Leisewitz AL, Christopher NM, Duncan NM, Becker PJ. Prognostic Usefulness of Blood Leukocyte Changes in Canine Parvoviral Enteritis. J Vet Intern Med. 2008;22(2):309-16.
3. Martella V, Cavall A, Decaro N, Elia G, Desario C, Campolo M, et al. Immunogenicity of an intranasally administered modified live canine parvovirus type 2b vaccine in pups with maternally derived antibodies. Clin Diagn Lab Immunol. 2005;12(10):1243-5.
4. Macartney L, McCandlish IAP, Thompson H, Cornwell HJ. Canine parvovirus enteritis 2: Pathogenesis. Vet Rec. 1984;115(18):453-60.
5. Otto CM, Rieser TM, Brooks MB, Russell MW. Evidence of hypercoagulability in dogs with parvoviral enteritis. J Am Vet Med Assoc. 2000;217(10):1500-4.
6. Decaro N, Desario C, Campolo M, Cavalli A, Cirone F, Elia G, et al. Canine parvovirus infection: which diagnostic test for virüs? J Virol Methods. 2005;126(1-2):179-85.
7. Apel MJG, Cooper BJ, Greisen H, Carmichael LE. Status report: Canine viral enteritis. J Am Vet Med Assoc. 1978;173(11):1516-8.
8. Greene CE, Decaro N. Canine Viral Enteritis. In: Greene CE, editor. Infectious Diseases of the Dog and Cat. 4th ed. Philadelphia, PA: WB Saunders; 2010.
9. Truyen U. Emergence and recent evolution of canine parvovirus. Vet Microbiol. 1999;69(1-2):47-50.
10. Mantione NL, Otto CM. Characterization of the use of antiemetic agents in dogs with parvoviral enteritis treated at a veterinary teaching hospital: 77 cases (1997-2000). J Am Vet Med Assoc. 2005;227(11):1787-93.
11. Macintire DK, Smith-Carr S. Canine parvovirus. Part II. Clinical signs, diagnosis, and treatment. Compendium on Continuing Education for the Practicing Veterinarian. 1997;19(3):291-302.