Damanhour Journal of Veterinary Sciences 7 (1), (2022) 23-27

Damanhour Journal of Veterinary Sciences

Journal homepage: https://djvs.journals.ekb.eg/

E-ISSN 2636-3003 | ISSN 2636-3011

Clinical and haemato-biochemical alterations with acute phase response in canine parvoviral enteritis

Ibrahim Abdullaziz[1], Mahmoud Aly[2,*], Ibrahim ElShahawy[1]

[1]Department of Animal Medicine, Faculty of Veterinary Medicine, Alexandria University, Egypt.

[2]Departement of Medicine and infectious diseases, faculty of veterinary medicine, university of sadat city

A B S T R A C T

Canine parvoviral enteritis (CPV) is one of the most contagious fatal viral diseases in young puppies with subsequent alterations in homeostasis; This study was conducted on a total number of 35 puppies of different breeds, with age range of 2-6 months old with signs compatible with canine parvovirus enteritis. Another apparently healthy five puppies within similar age range were enrolled as healthy control group. Up on admission, clinical signs were recorded and rapid in-clinic IC test kit for detection of CPV Ag in feces. Blood samples were used to determine haemato-biochemical alterations along with Acute phase response values. Vomiting and foul-smelling bloody diarrhea with marked dehydration were the main recorded clinical signs. Hemogram of CPV infected dogs, revealed the presence of microcytic hypochromic anemia, significant leukopenia with marked lymphopenia and neutropenia. The total serum proteins, albumin, total globulins, sodium, potassium, chloride, levels were significantly decreased in CPV infected group than its level in healthy control dogs. On contrary the mean values of, aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), blood urea nitrogen (BUN) and creatinine were significantly increased in diseased dogs. Results of acute phase biomarkers revealed Significant increase in serum amyloid A (SAA), haptoglobin (Hp) and C-reactive protein (CRP) with significant reduction in mean values of serum albumin in diseased puppies. Based on obtained results, CPV enteritis has negative impact on haemato-biochemical biomarkers and strong expression of acute phase reaction in diseased dogs.

Keywords: Clinical, haemato-biochemical, Parvo, Dogs .

1. Introduction

Canine parvovirus enteritis is a highly contagious fatal viral disease of dogs caused by parvovirus type -2, which has a great affinity to invade lymphoid and intestinal tissues, and it is transmitted via fecal-oral routes through direct or indirect contact between infected and susceptible dogs (Prittie, 2004).Acute parvoviral enteritis has been seen in dogs of all breeds, age, and sex, but puppies between 6 weeks and 6 months appear to be more susceptible (Gombak et al., 2017). Factors that predispose to parvoviral infection in puppies are lack of protective immunity, intestinal parasites and overcrowded, unsanitary and stressful environmental conditions (Khare et al., 2020).Fever, lethargy, vomiting, dehydration, and diarrhea which alternated from mucoid to hemorrhagic are the most common recorded clinical signs associated with CPV (Lamm and Rezabek, 2008; Goddard and Leisewitz, 2010).

Acute phase response (APR) is a state of early, defense systemic reaction of the body modulated by trauma, neoplastic growth, bacterial, parasitic, and viral infection, burns, surgery and immunological disorders (Petersen et al., 2004). The main function of APR is mainly to restore the homeostasis by isolating and destroying the harmful agent and to activate the repair process (Janeway et al., 2001). Acute phase proteins (APPs) is a large group of proteins synthetized mainly in liver and consider one of the most important metabolic alterations accompanying early acute stages of diseases; named acute phase response (APR) (Ceciliani et al., 2012). Nowadays, APPs are considered as a sensitive markers of inflammation and can be classified according to the magnitude of their increase into [positive APPs such as haptoglobin (Hp), C-reactive protein (CRP), α1-acid-glycoprotein and serum amyloid A] or decrease into (negative APPs such as albumin and transferrin) in serum concentrations within a few hours following infection (Murata et al., 2004; Gruys et al., 2005).

This study aimed to highlights the haemato-biochemical alterations associated with cases of canine parvoviral enteritis presented to small animal clinic, teaching hospital, Faculty of Veterinary Medicine, Alexandria University, Egypt with special focus on acute phase proteins [CRP, Hp, SAA and albumin] as a diagnostic indicator in CPV infected dogs..

*Corresponding author:
Email address: mahmoudaly@vet.usc.edu.eg
Departement of Medicine and infectious diseases, faculty of veterinary medicine, university of sadat city

2. Materials and Methods

2.1. Study design:

A total number of 35 puppies of different breeds, with age range of 2-6 months old were admitted to small animal clinic, teaching hospital, Faculty of Veterinary Medicine, Alexandria University in the period from January 2019 to June 2021, with signs compatible with canine parvovirus enteritis. Another apparently healthy five puppies within similar age range were enrolled as healthy control group.

All dogs were clinically examined, and clinical signs were recorded at time of admission (Table 1, 2). Fecal samples from diseased dogs were collected for microscopical examination to ensure that dogs were free from parasitic infestation (Bellwood and Andrasik-Catton 2014). In- clinic rapid CPV Ag rapid kits were used on stool of suspected dogs (Quacking Biotech Co, Ltd, Shanghai, China) (Fig. 1).

Only dogs give positive results upon IC- test were enrolled in this study as diseased group (CPV infected dogs). Blood samples from all dogs were withdrawn and divided into 2 tubes; EDTA-containing tubes that was used for hematological analysis and plain tube was used for serum separation for further biochemical analysis.

2.2. Clinical examination:

Case history and clinical examination of all dogs under investigation was performed according to the method described by (Ettinger, 2010). Including temperature, pulse, respiration, mucous membrane evaluation and capillary refill time (CRT) (Table 1).

2.3. Sampling and Measurements:

Blood samples that were collected from cephalic vein of each dog are conducted for determination of: Red blood Cell count (RBCs X106/µl), hemoglobin concentration (Hb g/l), packed cell volume (PCV%), mean corpuscular volume (MCV fl), mean corpuscular hemoglobin (MCH pg), mean corpuscular hemoglobin concentration (MCHC g/dl), WBCs count (X103/µl) and differential leucocytic count all were determined by using of fully automated veterinary hematology analyzer (Exigo, Boule medical AB., Sweden) in the central laboratory, Faculty of veterinary medicine, Alexandria University.

Determination of serum concentrations of Sodium, Potassium, Chloride, AST, ALT, Alkaline phosphatase (ALP), Total protiens and Albumin all were carried out by using commercial test kits supplied by (Bio-labo, France) while analysis of BUN (blood urea nitrogen) and creatinine were carried out by using commercial test kits supplied by (Ben-Biochemical Enterprise, Italy) all were analyzed follwing standard methods mentioned in the leaflet of the manufacturer. Serum globulins was calculated by

substraction of the amount of serum albumin from the amount of total serum proteins. Concentrations of [CRP, Hp, SAA] in the serum were determined with ELISA kits according to the method described by (Sahinduran et al., 2016).

2.4. Statistical analysis:

Data collected were subjected to analysis by T- independent student test to assess significant differences between groups with the aid of (SAS, 2004). All values were expressed as mean ± standard error (SE). Significance level was set at P≤ 0.05.

3.Results:

3.1. Clinical examination:

In CPV infected dogs, very offensive bloody diarrhea, frequent vomiting, fever, depression, anorexia, weight loss with marked dehydration were the most recorded clinical signs. Confirmation of infection was done via in-clinic rapid CPV Ag test which gave positive results with fecal swabs (Fig. 1). Compatible thorough clinical examination reveals significant elevated body temperature, tachycardia, significant tachypnea with congested mucous membrane in CPV infected dogs compared to healthy control dogs as shown in (Table 1& 2).

Hematological analysis:

Erythrogram of CPV infected dogs, shown in (Table 3) revealed the presence of microcytic hypochromic anemia, which is markered by significant reduction in mean values of RBCs count, Hb concentration, PCV%, MCV, MCH and MCHC when compared to healthy control dogs. Where leukogram of CPV infected dogs showed significant leukopenia with marked lymphopenia and neutropenia, while total number of MID "eosinophils, basophils and monocytes" showed non-significant changes between diseased and healthy ones. Moreover, CPV infected dogs revealed significant thrombocytopenia in comparison to the mean values of healthy dogs.

3.2. Biochemical analysis:

The mean values of total serum proteins, albumin, total globulins, sodium, potassium, and chloride were significantly decreased in CPV infected group than its level in healthy control dogs. On contrary the mean values of, AST, ALT, ALP, blood urea and creatinine were significantly increased in diseased dogs (Table 4).

Results of acute phase biomarkers are shown in (Table 5) Significant increase in SAA, Hp and CRP with significant reduction in mean values of serum albumin was recorded in the diseased group compared with healthy control group.

4. Discussion:

Canine Parvo viral enteritis is one of the most common destructive diseases in dogs, especially unvaccinated puppies below six months old. It is responsible for serious morbidity and mortality (Decaro et al., 2006).

All examined dogs shown the characteristic clinical signs of Parvo virus infection including lethargy, anorexia, persistent vomiting, and foul-smelling diarrhea varying in color from bloody to yellow with traces of blood (Table 1, Fig. 1). This vomiting and foul-smelling bloody diarrhea is closely related to the erosive inflammatory damage of the stomach and intestinal mucosal barrier (Mccaw & Hoskins, 2006 and Streck et al., 2009).

Most of CPV infected dogs showed marked dehydration with varying degree which is markered by prolonged (CRT ≥2 seconds) values, this significant dehydration is mainly attributed to vomiting and diarrhea which is associated with large fluid and protein losses through damaged GIT (Biswas et al, 2005). Additionally, Compatible thorough clinical examination reveals significant elevated body temperature, tachycardia, significant tachypnea with congested mucous membrane in CPV infected dogs compared to healthy control dogs which comes in agreement with (AL-Hosary, 2016 and Kubesy et al., 2017). Damage to intestinal tract secondary to parvoviral infection increases markedly the risk of bacterial translocation and subsequent coliform septicemia, which may lead to development of a systemic inflammatory response syndrome (SIRS) that can progress to septic shock and ultimate death. Moreover, mortality rate was higher in puppies that met the criteria for SIRS (heart rate > 140 beats/min, respiratory rate > 30 breaths/min, temperature > 39.20 ℃ or < 37.80 ℃) (Kalli et al., 2010).

Erythrogram of CPV infected dogs revealed the presence of microcytic hypochromic anemia; this results were compatible with that observed by (Arslan et al., 2017). This anemia might be resulted from suppression of erythropoiesis through the direct inhibitory effect of CPV on bone marrow (Elsayed et al., 2020) and, due to intestinal bleeding associated with loss of large volume of blood through diarrhea (Abd El-baky et al., 2017 and Arora et al., 2018).

Where leukogram of CPV infected dogs showed significant leukopenia with marked lymphopenia and neutropenia, while total number of MID "eosinophils, basophils and monocytes" showed non-significant changes between diseased and healthy ones, as stated by (Mylonakis et al., 2016) who attributed the resultant leukocytic changes to huge request of inflamed mucosa of intestine along with exhaustion of lymphoid tissue in addition to destruction of bone marrow precursors However, leukocytosis and neutrophilia maybe also recorded (Kubesy et al., 2019) due to secondary bacterial infection .

In accordance with (Decaro et al., 2005) and (Shah et al., 2013) CPV infected dogs revealed significant thrombocytopenia in comparison to the mean values of healthy dogs which may attributed to loss of blood through bloody diarrhea, increased platelets utilization through injured gastrointestinal mucosa or from decreased platelets production as a direct inhibitory effect of CPV on bone marrow precursor cell.

The significant reduction in serum total protein, albumin and globulins concentration in diseased dogs was also reported by (Tefft, 2014) which resulted from anorexia with decreased food consumption and reduced protein synthesis in the liver as amino acids are shunted into synthesis of positive acute phase proteins as a body reaction to inflammation and tissue impairment which comes on the expanse of albumin and other proteins synthesis (Mazzaferro et al., 2002). Also, these results are in concurrence with those obtained by (Li and humm, 2015). This observation may be due to inadequate dietary intake, reduced absorption associated with intestinal hemorrhage and altered gastrointestinal mucosal barrier with protein losing-enteropathy.Serum values of sodium, potassium and chloride showed significant decrease in CPV infected group where these findings come in contact with (Ukwueze et al., 2020) this reduction could be attributed to poor appetite, reduced absorption from the disrupted gut with marked fluid and electrolytes loss through vomiting and diarrhea which mainly contribute to depression and general muscular weakness (Burchell et al., 2014).

The elevated ALT, AST and ALP values in diseased dogs were agreeable with those of (Kubesy et al., 2019) and (Salem, 2014). Nutritional imbalances with dehydration and hypovolemia have a strong impact on liver functions and vice versa. In this respect, increased Levels of AST, ALT and ALP activities could be attributed to hepatic hypoxia secondary to severe hypovolemia or absorption of toxic substances through disrupted gut barrier as well (Shah et al., 2013). Furthermore, highly significant increase in BUN and serum creatinine values may be due to dehydration and hypovolemia as a consequence to vomiting and diarrhea with subsequent decreased renal blood flow of affected dogs causing BUN and creatinine significantly increased (Barsanti et al., 2004). Regarding acute phase response, the obtained data showed Significant increase in positive APPS (SAA, Hp and CRP) with significant reduction in mean values of Negative APPs (serum albumin) in CPV infected dogs which is similar to those previously described by (kogika et al., 2003) and (McClure et al., 2013) who mentioned that acute phase response expression occurs at the expense of albumin synthesis in canine parvoviral enteritis as a response to severely inflamed gastrointestinal tract. In this respect These results can be explained as, during infection, hepatocytes responded by producing a large number of APPs, which are part of the innate immune system. Nowadays it is widely speculated that APPs are important components of the antimicrobial response, frequently involved directly or indirectly in the inhibition of viral replication and spread within the host body, in accordance to (Mazzaferro, 2020).

Conclusion:

This study concluded that, Canine parvoviral enteritis causing gastro-enteritis, fever and dehydration with microcytic hypochromic anemia and significant leukopenia with marked lymphopenia and neutropenia which are the main causes of death in infected animals, especially with neglected treatment and healthcare. Moreover, A typical APP response characterized by significant increase in the major (CRP and SAA) and moderate (Hp) with marked decrease in negative (albumin) APPs occurs in CPV infected dogs.

Authors contribution: IA ,MA and IE conceived and designed the study. IA performed the study. IA ,MA and IE analyzed the data. IA ,MA and IE and IA interpreted the data, IA ,MA and IE wrote the paper. IA ,MA and IE revised the final manuscript. IA ,MA and IE reviewed the manuscript.

Competing interests

There is no competing interest and we don't have any financial support from any institutions.

Acknowledgments

We would like from our heart to appreciate the owners of the cases.

5. References

Abd El-Baky, A. A., Mousa, S. A. and. Kelany, W. M. 2017. Diagnosis of hemorrhagic gastroenteritis in dogs. Bioscience Research, 14(4): 1223-1229.

AL-Hosary, A., A. 2016. Prevalence of Parvovirus Infection in Household Dogs with Special Reference to its Effects on Some Blood Parameters. Alexandria Journal of Veterinary Sciences, 51 (2), 174-177. doi:10.5455/ajvs.236999

Arora, R., Tyagi, A., Shekhar, S., Rajora, V., and Arora, N. 2018. Haemato-biochemical alterations in gastroenteritis affected dogs. Journal of Entomology and Zoology Studies; 6(5): 972-974.

Arslan, H. H., Guzel, M., Meral, Y., Dalgin, D., Gokalp, G. and Ozcan, U. 2017. A new approach to blood parameters in dogs with hemorrhagic enteritis. Acta Scientiae Veterinariae 45: 1458.

Barsanti JA, GE Lees, MD Willard and RA Green, 2004. Urinary disorders. In: Willard MD and Tvedten H (editor), Small Animal Clinical Diagnosis by Laboratory Methods. 4th ed., Saunders.

Bellwood, B., Andrasik-Catton, M. 2014. Parasitology: veterinary technician's handbook of laboratory procedures, 1st edn. Wiley, pp 77–83.

Biswas, S., Chakravorty, D. and Pradhan, N.R. 2005. Clinical and haemato-biochemical changes in parvovirus infection in dogs. Indian J Vet Med 25:16-18.

Burchell, R. K., Schoeman, J. P., and Leisewitz, A. L. 2014. The central role of chloride in the metabolic acid-base changes in canine parvoviral enteritis. The Veterinary Journal 200(1):152-6.

Castro TX, Miranda SC, Labarthe NV, Silva LE, Cubel Garcia RCN. 2007. Clinical and epidemiological aspects of canine parvovirus (CPV) enteritis in the State of Rio de Janeiro: 1995-2004. Arq Brasileiro de Med Vet Zoo.;59(2):333-339.

Ceciliani, F., J. J. Ceron P. D. Eckersall & H. Sauerwein, 2012. Acute phase proteins in ruminants. Journal of Proteomics, 75, 4207–4231.

Decaro, N., Desario, C., Campolo, M. et al, 2005. Clinical and virological findings in pups naturally infected by canine parvovirus type 2 Glu-426 mutant. J Vet Diagn Invest 17(2):133-138.

Decaro, N., Desario, C., Elia, G., Campolo, M., Lorusso, E., Mari, V., Martella, V. and Buonavoglia, C. 2006. Occurrence of severe gastroenteritis in pups after canine parvovirus vaccine administration: Clinical laboratory diagnostic dilemma. Vaccine 25(7):1161-1166.

Elsayed, N. M., Kubesy, A. A., and Salem, N. Y. 2019. Altered blood oxidative stress biomarkers in association with canine parvovirus enteritis. Comparative Clinical Pathology (2020) 29:355–359.

Ettinger, S. J. (2010): The physical examination of the dog and cat. In: Ettinger SJ, Feldman EC, eds. Textbook of Veterinary Internal Medicine. 7th ed. St. Louis, MO: Saunders Elsevier. 1-9.

Goddard, A. and Leisewitz, A. L. 2010. Canine parvovirus. Vet Clin North Am Small Anim Pract, 40, 1041-1053.

Gombac, M., Tadic, M., Svara, T. and Pogaenik, M. 2017. Canine Parvovirus: A Review.Inter J Sci Appl Res.;2(2):74-95. Doi: 10.1036/1097-8542.757249.

Gruys, E., Toussaint, M. J., Niewold, T. A. and Koopmans, S. J. 2005. Acute phase reaction and acute phase proteins. Journal of Zhejiang University Science B 6, 1045-1056

Janeway, C. A., P. Travers, M. Walport and M. J. Schlomschik, 2001. Immunology, 5th edn, Taylor and Francis, London, pp. 732.

Judge, P. 2015. Management of the Patient with Canine Parvovirus Enteritis. Vet Education, proceedings of the New Zealand veterinary nursing association annual conference, 5-11.

Kalli I, Leontides LS, Mylonakis ME, Adamama-Moraitou K, Rallis T and Koutinas AF, 2010. Factors affecting the occurrence, duration of hospitalization and final outcome in canine parvovirus infection. Res Vet Sci 89: 174.

Khare, DS Gupta, DK Shukla, PC Das, G Meena NS and Ravi Khare, 2020. Clinical and haemato-biochemical changes in canine parvovirus infection. Journal of Pharmacognosy and Phytochemistry 2020; 9(4): 1601-1604.

Kogika, M. M., Pereira, D. A., Elias, F., Notomi, M. K., Delayte, D. E., Kawahara. R. and Hagiwara, M. K. 2003.Determination of serum haptoglobin, ceruloplasmin and acid-alpha-glycoprotein in dogs with haemorrhagic gastroenteritis. Ciência Rural Santa Maria 33, 513-517.

Kubesy AA, Salem NY and Jaheen AH. 2017. Altered blood oxidative stress biomarkers in association with canine pyoderma and allergic contact dermatitis. Comp Clin Pathology 26(3):643–646.

Kubesy AA, Rakha, G. M., Salem, S. I. and Jaheen, A. H. 2019. Altered blood procalcitonin, C-reactive protein, and leucocytes count in association with canine parvovirus (CPV) enteritis. Comp Clin Pathology 28, 1095–1099. https://doi.org/10.1007/s00580-019-02941-y

Lamm, C.G. and Rezabek, G.B., 2008. Parvovirus infection in domestic companion animals. Vet Clin North Am Small Anim Pract, 38, 837-850, viii-ix.

Li, R. and Humm KR, 2015. Canine parvovirus infection. In: Silverstein DC, Hoper K, editors. Small Animal Critical Care Medicine. 2nd Ed, St Louis, MO: Elsevier: 509-513.

Mazzaferro EM, E Rudleff and R Kirby, 2002. The role of albumin replacement in the critically ill veterinary patient. J Vet Emerg. Crit Care, 12: 113-124.

Mazzaferro, E. M. 2020. Update on canine parvoviral enteritis. Vet Clin North AM Amall Anim Pract; 50 (6): 1307-1325.

Mccaw, D.L. and Hoskins, J.D. 2006. Canine Viral Enteritis. In: Greene C. Infectious Diseases of the dog and cat. 3rd edn. Rio de Janeiro: Guanabara Koogan. 63-71.

McClure V, van Schoor M, Thompson PN, Kjelgaard-Hansen M and Goddard A, 2013. Evaluation of the use of serum C-reactive protein concentration to predict outcome in puppies infected with canine parvovirus. J Am Vet Med 243: 361-266.

Murata, H., Shimada, N. and Yoshioka, M. 2004. Current research on acute phase proteins in veterinary diagnosis: an overview. Veterinary Journal 168, 28-40.

Mylonakis ME, Iris K and Timoleon SR, 2016. Canine parvoviral enteritis: an update on the clinical diagnosis, treatment, and prevention. Veterinary Medicine: Research and Reports 7: 91-100.

Petersen, H. H., J. P., Nielsen and P. M. H. Heegaard, 2004. Application of acute phase protein measurements in veterinary clinical chemistry. Veterinary Research, 35, 163–187.

Sahinduran S, Albay MK, Karakurum MC, Ozmen O and Kale M, 2016. Investigation of Some Cytokines, Acute Phase Proteins and Hepcidin Concentrations Before and After Treatment in Dogs with Parvoviral Gastroenteritis.Pakistan Veterinary Journal,36(4), 487-492.

Salem N.Y. 2014. Canine viral diarrhea: clinical, hematologic and biochemical alterations with particular reference to in-clinic rapid diagnosis. Glob Vet 13(3):302–307

SAS. 2004. Users guide statistics. As. Institute Cary, North Carolina. USA.

Shah SA, Sood NK, Wani N, Gupta K. and Singh A. 2013. Haemato-biochemical changes in canine parvoviral infection. Indian Journal of Veterinary Pathology; 37(2):131-133.

Streck, A.F., de Souza, C.K., Goncalves, K.R., Zang, L., Pinto, L.D. and Canal, C.W. 2009. First detection of canine parvovirus type 2c in Brazil. Braz. J. Microbiol. 40(3): 465-469.

Tefft KM, 2014. Successful Management Strategies for Canine Parvovirus. Indiana Veterinary Medical Association Annual Meeting. C.S.

Ukwueze, C.S., Akpan, E.S., Ezeokonkwo, R.C., Nwosuh, C.I. and Anene, B.M.2020. Haematological, oxidative stress and electrolyte alterations in puppies with canine parvoviral enteritis. Iraqi Journal of Veterinary Sciences, 34(1): 65-69.

Fig. 1: Bloody diarrhea with positive IC rapid test kits for CPV infection

Table (1): Clinical picture of healthy control dogs compared to CPV infected dogs:

Parameters \ Groups	Control	Diseased
Rectal temperature °C	38.6±0.21	40.1±0.15*
Respiratory rate / min.	21.63±1.25	34.22±1.12*
Pulse rate / min.	98.2±2.29	129.25±2.8*
Lymph nodes	Normal	Normal
Mucous membranes	Rosy red	congested*
CRT in seconds.	< 2 seconds	≥2 seconds*

Table (2): Clinical manifestation scale upon presentation in CPV infected dogs:

No.	Clinical findings	No. of Dogs	Percent (%)
1	**Anorexia**	31	88.57
2	**Vomiting**	35	100
3	**General Body condition:**		
	a. Good	24	68.57
	b. Poor	11	31.43
4	**Color of conjunctival mucus membrane:**		
	a. Congested	27	77.14
	b. Pale	8	22.86
5	**Dehydration (%)**		
	a. Mild (4-6%)	8	22.86
	b. Moderate (6-8%)	22	62.85
	c. Severe (8-10%)	5	14. 29
6	**Body temperature:**		
	a. Decreased	6	17. 14
	b. Normal	11	31.43
	C. Increased	18	51.43

Table (3): Mean values (±SE) of some hematological changes in both healthy control dogs compared to CPV infected dogs:

Group	Control	Diseased
RBCS ($X10^6$/µl)	6.57±0.98	4.48±0.34*
Hb (g/dl)	13.69±0.86	10.31±0.52*
PCV (%)	43.35±1.55	32.27±1.12*
MCH (*Pg.*)	22.13±0.34	20.48±0.38*
MCHC (g/dl)	33.80±0.53	27.81±0.32*
MCV (*fl*)	66.76±1.96	60.85±0.98*
WBCS ($X10^3$/µl)	12.16±1.34	7.16±0.91*
Lymphocytes ($X10^3$/µl)	3.64±0.87	2.01±0.66*
MID($X10^3$/µl)	1.10±0.10	0.98±0.09
Neutrophils ($X10^3$/µl)	7.42±0.63	4.17±0.58*
Platelets ($X10^3$/µl)	427.32±6.4	300.61±5.15*

Table (4): Mean values (±SE) of some serum biochemical changes in healthy control dogs compared to CPV infected dogs:

Group	Control	Diseased
Total Proteins (g/dl)	6.99±0.55	5.07±0.48*
Albumin (g/dl)	3.98±0.16	2.92±0.11*
Globulins (g/dl)	3.01±0.12	2.15±0.14*
ALT (u/l)	27.39±0.84	53.82±0.97*
AST (u/l)	48.37±1.98	87.63±1.23*
ALP (u/l)	95.72±1.6	176.42±5.63 *
Na (mmol/l)	142.05±3.2	132.45±2.63*
Cl (mmol/l)	113.43±1.52	102.4±1.12*
K (mmol/l)	4.46±0.62	3.48±0.14*
Urea (mg/dl)	16.39±0.74	39.42±0.45*
Creatinine (mg/dl)	0.87±0.26	3.82±0.47*

Table (5): Mean values (±SE) of APPs profile in both healthy Control dogs compared to CPV infected dogs.

Group	SAA (µg/mL)	Hp (g/l)	CRP (mg/l)
Control	2.16±0.3	0.56±0.09	4.7±0.08
CPV	5.91±1.16 *	2.81±0.39 *	18.45±3.5 *