B-5010
[1-5]

RESEARCH ARTICLE Indian Journal of Animal Research

Haemato-biochemical Alterations in Canine Rotaviral Enteritis of Dogs and its Management

Chayanika Mazumder[1], Gauranga Mahato[1], Durlav Prasad Bora[2], Deepa Lahkar[1], Syed Abdul Arif[3], Bhaben Chandra Baishya[3]

10.18805/IJAR.B-5010

ABSTRACT

Background: Rotaviruses cause acute diarrhoea, mostly in young puppies. Rotavirus associated gastroenteritis in dog is of great concern not only because of their high morbidity and mortality, but became of the reason that, they act as potential source of zoonotic infection and inter-species transmission. There is no report on rotavirus infection in dog from Assam. Therefore, there is a need to study the haemato-biochemical alterations in rotavirus infected dogs while treating such type of clinical cases.

Methods: A total of 157 faecal and blood samples were collected from the dogs affected with gastroenteritis, registered at Veterinary Clinical Complex, Khanapara, Assam during November, 2020 to April, 2021. Diagnosis of rotavirus was done by RNA-PAGE and RT-PCR. Haematological study of the canine rotavirus (CRV) infected dogs were carried out with automated haematological cell counter and biochemical analysis was done with semi-automated biochemistry analyser using commercial kit.

Result: Out of 157 samples, 8 samples were found positive for canine rotavirus by RNA-PAGE and 17 samples by RT-PCR. Haemato-biochemical alterations in canine rotavirus infected dogs revealed significant increase in Hb, PCV, TEC, monocyte, lymphocyte, AST, ALT, BUN and creatinine while significant decrease in TLC, neutrophil, total protein, serum sodium, potassium and chloride.

Key words: Canine rotavirus, Dogs, Haemato-biochemical alterations, Management.

INTRODUCTION

Gastroenteritis is a common clinical problem in puppies and etiological agents of gastroenteritis are often multi-factorial (Nadia *et al*., 2019). Among the various infectious agents, rotavirus (RV) is considered as an important enteric pathogen of various animal species and human (Martella *et al*., 2010). Rotaviruses (RVs) are classified under the family Reoviridae and genus Rotavirus (Broor *et al*., 2003). It is a non-enveloped virus and consists of 11 segments of double-stranded RNA (dsRNA) genome. Most commonly RV is transmitted by fecal-oral route but airborne or droplet transmission has also been recorded (Ansari *et al*., 1991). Rotaviruses usually multiplies in the intestinal epithelium where it damages the epithelial cells on the tips of the small intestinal villi resulting from mild to moderate villus atrophy (Mosallanejad *et al*., 2015). These viruses induce diarrhoea through destruction of enterocyte by stimulating intestinal secretion and activation of enteric nervous system (Crawford *et al*., 2017). Canine rotaviral enteritis (CRV) usually occurs in sub-clinical to mild forms. Anorexia, abdominal pain, lethargy, hyperthermia, diarrhoea (mucoid, haemorrhagic or non-haemorrhagic) with vomition are the common clinical signs. Rotaviral diarrhoea usually self limiting in nature and infected dog may recover spontaneously within 8-14 days. However, severe enteritis along with secondary bacterial infection may cause death in pups (Fulton *et al*., 1981). The clinical sign of rotavirus infection is not specific; therefore laboratory diagnosis is essential for immediate treatment and control of the disease. There is no specific treatment for the disease, symptomatic and supportive therapy helps in early recovery from gastroenteritis and dehydration. It is necessary to use antibiotics to controls secondary bacterial infection. Rotavirus is species specific but several reports of zoonotic and interspecies transmission of RV under natural conditions have been recorded (Martella *et al*., 2010). It is of utmost importance to isolate the canine rotaviral strains circulating in Assam to adopt preventive and control measures. As detail studies on the haemato-biochemical alterations in rotavirus enteritis in dogs have not been

[1]Department of Veterinary Epidemiology and Preventive Medicine, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati-781 022, Assam, India.
[2]Department of Veterinary Microbiology, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati-781 022, Assam, India.
[3]Department of Veterinary Clinical Medicine Ethics and Jurisprudence, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati-781 022, Assam, India.

Corresponding Author: Chayanika Mazumder, Department of Veterinary Epidemiology and Preventive Medicine, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati-781 022, Assam, India. Email: chayamazumder12345@gmail.com

How to cite this article: Mazumder, C., Mahato, G., Bora, D.P., Lahkar, D., Arif, S.A. and Baishya, B.C. (2023). Haemato-biochemical Alterations in Canine Rotaviral Enteritis of Dogs and its Management. Indian Journal of Animal Research. doi: 10.18805/IJAR.B-5010.

Submitted: 03-09-2022 **Accepted:** 12-03-2023 **Online:** 28-04-2023

reported, hence it was felt necessary to study the changes for the therapeutic management. Supportive and symptomatic treatment is rendered to the infected pups.

MATERIALS AND METHODS

A total of 157 faecal and blood samples were collected from dogs with complaints of gastroenteritis or related symptoms, registered at Veterinary Clinical Complex (VCC), College of Veterinary Science, Assam Agricultural University, Khanapara, during the period November, 2020 to April, 2021. Approximately 2 g of faecal samples were collected aseptically in sterile vials or rectal swabs and stored at -20°C till diagnosis. Canine rotavirus (CRV)suspected faecal samples were screened by RNA-polyacrylamide gel electrophoresis (RNA-PAGE) as per the method of Herring *et al*. (1982) and by reverse transcription-polymerase chain reaction (RT-PCR) targeting VP6 gene of CRV as per the method mentioned by Ortega *et al*. (2017). Haematological and biochemical study of the rotavirus infected dogs were carried out on 0 day and 7th days of treatment. About 5 ml of whole blood was collected, out of which, 2 ml was transferred into properly labelled EDTA vacutainer for haematological study and 3 ml in serum separator tube with clot activator for biochemical analysis. Haematological parameters *viz*. Hb, PCV, TEC, TLC and DLC were analysed by an automated haematological cell counter (MS 4e, France). For biochemical studies, serum was separated from blood by centrifugation at 3000 rpm for 15 min and was used for estimation of AST, ALT, BUN, creatinine, total protein, serum sodium, potassium and chloride using commercial kit as per manufacturer protocol with the help of semi-automated biochemistry analyzer by spectrophotometric method. Six apparently healthy dogs registered for vaccination were selected as healthy control group to generate normal data for comparison of parameters under study.

Two groups of pups *viz*. Group A (unaffected healthy control with six animals) and Group B (Nine infected pups were selected). In group B, infected dogs were treated with conventional therapy comprising fluid therapy (NS/RL/DNS) @ 10-20 ml/kg body weight), antiemetic (Ondansetron @ 0.1-0.2 mg/kg body weight), antibiotic (Ceftriaxone @ 25 mg/kg body weight daily for 5 days), proton pump inhibitor (Pantoprazole @ 1 mg/kg body weight), multivitamin/vitamin B complex (Tribivet @ 1.0 to 1.5 ml) and haemostatic (Botropase @ 10 mg/kg body weight) through IV route.

Statistical analysis

Statistical analysis was performed using SPSS software. Haematological and biochemical parameters were analysed by one-way analysis of variance (ANOVA).

RESULTS AND DISCUSSION

Out of 157 samples tested, 8 samples were found positive for canine rotavirus (CRV) by RNA-PAGE revealed migration pattern of 4:2:3:2 (Fig 1) indicating group A rotavirus and a total of 17 samples were found positive for CRV infection by RT-PCR. In RT-PCR amplified product targeting VP6 gene produced band of 379 bp (Fig 2).

Alterations (mean±SE) in haematological parameters of canine rotaviral enteritis (n=9) dogs in pre-treatment and post-treatment group are represented in (Table 1). The mean value of Hb ($P<0.05$), TEC ($P<0.05$) and PCV ($P<0.01$) were significantly higher in pre-treatment group in comparison to healthy control group. This corroborates the findings of Malik *et al*. (2014) in piglets with rotavirus diarrhoea; Bhat *et al*., (2015) in dog with gastroenteritis; Chethan *et al*. (2017) in rotavirus infected piglets. Increased level of Hb, TEC and PCV values might be due to haemoconcentration or fluid loss associated with severe diarrhoea and vomition episodes (Biswas *et al*., 2005). Different mechanisms that attributed to the fluid loss in rotaviral diarrhoea are malabsorption secondary to enterocyte destruction, direct inhibition of glucose-mediated sodium absorption by virus-encoded toxin (NSP4), stimulation of the enteric nervous system (ENS) and villus ischaemia (Raming, 2004; Yin *et al*., 2017). In CRV positive dogs, mean value of TLC and neutrophil were

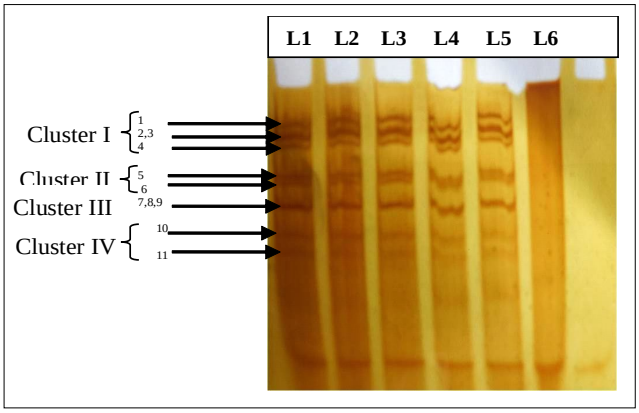

Fig 1: RNA electrophoresis of canine rotavirus RNA (Group A). L1, L2, L3 and L4: Positive faecal samples of dog, L5: Positive control (Human RV), L6: Negative control.

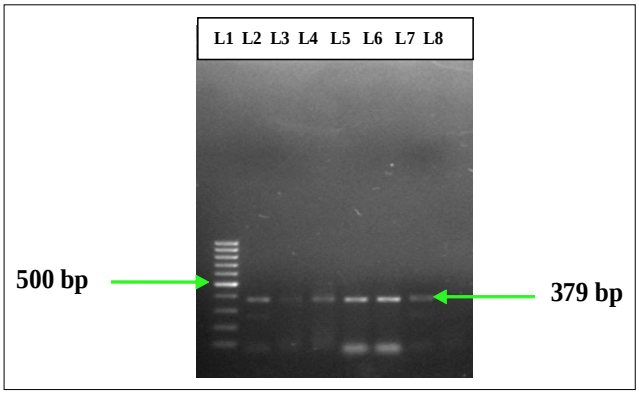

Fig 2: Agarose-gel electrophoresis of RT-PCR product of VP6 region of canine rotavirus. L1: 100 bp ladder, L2: Positive control (Human RV), L3, L4, L5, L6 and L7: Positive faecal samples of dog, L8: Negative control.

significantly (P<0.01) lower in pre-treatment group than healthy control group. A marked leukopenia along with neutropenia in the study is in agreement with the finding of Green *et al*. (2003), who has reported neutropenia in rotavirus positive children; Chethan *et al*. (2017) reported decrease in TLC values along with marked neutropenia in rotavirus infected piglets; Arora *et al*. (2018) in dog with gastroenteritis; Barua *et al*. (2018) in rotavirus infected calves. This might be due to result of non bacterial systemic infection caused by rotavirus (Dalgic *et al*., 2010). Generally, the demand for WBC (particularly neutrophil) in inflamed gastrointestinal tract is more but during infection due to destruction of haemopoietic progenitor cells of various leucocytes types in bone marrow and other lymph proliferative organs and also due to loss of neutrophils through the damaged gastrointestinal tract, causes inadequate supply of leukocytes (specifically neutrophils) in the inflamed gastrointestinal tract (Goddard *et al*., 2008). The mean value of lymphocyte (P<0.01) and monocyte (P<0.05) were significantly higher in CRV positive dogs in pre-treatment group in comparison to healthy control group. In the study, lymphocytosis was recorded in rotavirus infected dogs and the finding reframes with that of Chethan *et al*. (2017); Arora *et al*. (2018); Barua *et al*. (2018). Lymphocytosis might be due to viral etiology of the disease. Increased level of monocyte in the study was in accordance with the finding of Chethan *et al*. (2017). The increased level of monocytes might be due to general immune reaction to infection.

Alterations (mean±SE) in biochemical parameters of CRV infected dog in pre-treatment and post-treatment group are presented in (Table 2). The mean values of AST, ALT, BUN and creatinine were significantly (P<0.01) higher in pre-treatment group when compared to healthy control group. The increased level of AST, ALT, BUN and creatinine in the study might be due to extra-intestinal spread of the rotaviruses. In previous study, RV has been demonstrated in other organs like lamina propria, Peyer's patches, mesenteric lymph nodes, liver, lung, kidney and bile duct of children, animals and laboratory animals, which indicated extra-intestinal spread of the virus (Mossel and Ramig, 2003; Alfajaro and Cho, 2014). Increase level of AST was noted in the study which corroborates the findings of Khatib and Khan, (2012) in children with rotavirus diarrhoea; Chethan *et al*. (2017) in piglets with rotavirus diarrhoea; Arora *et al*. (2018) in dogs with gastroenteritis. Increase in AST level might be due to involvement of liver (Grigonis *et al*., 2002a). Hepatic damage caused by viral infections lead to increased levels of enzyme activity in serum. Increase level of ALT is consistent with the previous reports of Chethan *et al*. (2017), which might be due to hepatic hypoxia secondary to severe hypovolemia or the absorption of toxic substances due to loss of the gut barrier (Shah *et al*., 2013). Increased level of BUN and creatinine in the study were similar with the findings of Chethan *et al*. (2017). The increased level of BUN and creatinine are suggestive of pre-renal uremia which might be due to reduced glomerular filtration rate (GFR) because

Table 1: Haematological alterations (mean±SE) of rotavirus infected dog in pre-treatment and post-treatment group.

Parameters	Healthy control group (n=6)	Pre-treatment group (n=9)	Post-treatment group (n=9)
Hb (gm/dL)	12.52±0.03^{a}	12.95±0.15^{b}	12.86±0.04^{ab}
PCV (%)	38.19±0.45^{a}	41.91±0.84^{b}	39.96±0.63^{ab}
TEC (10^{6}/cumm)	6.48±0.04^{a}	6.99±0.14^{b}	6.88±0.12^{ab}
TLC (10^{3}/cumm)	10.38±0.31^{a}	6.81±0.25^{b}	8.73±0.77^{a}
Neutrophil (%)	72.01±0.61^{a}	59.29±0.94^{b}	69.83±0.41^{a}
Lymphocyte (%)	26.40±0.90^{a}	36.53±1.30^{b}	29.42±0.77^{a}
Monocyte (%)	2.42±0.03^{a}	2.56±0.03^{b}	2.49±0.04^{ab}
Eosinophil (%)	1.18±0.16	1.02±0.11	1.09±0.12

Hb: Haemoglobin; PCV: Packed cell volume; TEC: Total erythrocyte count; TLC: Total leukocyte count; Mean values in a row bearing different superscript differ significantly (P<0.05).

Table 2: Biochemical alterations (mean±SE) of rotavirus infected dog in pre-treatment and post-treatment group.

Parameters	Healthy control group (n=6)	Pre-treatment group (n=9)	Post-treatment group (n=9)
AST (IU/L)	33.97±0.61^{a}	39.02±0.51^{b}	36.96±0.97^{ab}
ALT (IU/L)	38.46±0.42^{a}	41.32±0.51^{b}	39.88±0.61^{ab}
BUN (mg/dL)	20.94±0.39^{a}	31.22±0.41^{b}	22.37±0.55^{a}
Creatinine (mg/dL)	0.81±0.01^{a}	0.96±0.02^{b}	0.88±0.03^{a}
TP (g/dL)	6.86±0.02^{a}	6.49±0.06^{b}	6.66±0.07^{ab}
Na (mmol/L)	144.24±0.32^{a}	141.15±0.41^{b}	142.91±0.59^{a}
K (mmol/L)	4.70±0.04^{a}	4.20±0.08^{b}	4.48±0.08^{a}
Cl (mmol/L)	111.00±0.49^{a}	109.91±0.17^{b}	110.42±0.18^{ab}

AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; BUN: Blood urea nitrogen; TP: Total protein; Na: Sodium; K: Potassium; Cl: Chloride; Mean values in a row bearing different superscript differ significantly (P<0.05).

of haemoconcentration, dehydration, decrease tissue perfusion and also due to catabolic breakdown of tissues as a result of fever (Biswas *et al*., 2005; Bhat *et al*., 2015). CRV infected dogs showed, significant (P<0.01) lower values of TP in pre-treatment group in comparison to healthy control group. Decrease level of TP in the study was in agreement with the findings of Biswas *et al*. (2005); Khatib and Khan, (2012); Bhat *et al*. (2015) and Chethan *et al*. (2017). Hypoprotenemia might be due to anorexia, leakage of serum protein through damaged capillaries of the villi of intestines and also due to malabsorption through villi. The mean values of sodium (P<0.01), potassium (P<0.01) and chloride (P<0.05) were significantly lower in pre- treatment group comparison to healthy control group. Hyponatremia, hypochloremia and hypokalaemia in the study corroborates with the findings of Bhat *et al*. (2015) in dog with gastroenteritis; Khatib and Khan, (2012) who has recorded significant decrease of serum sodium and potassium in rotavirus infected children. Whereas, Barua *et al*. (2018) revealed lower value of serum sodium; higher level of potassium in diarrhoeic calves and significantly higher level of serum chloride in rotavirus positive cases. Hyponatremia might be due to loss of sodium ions through vomition and diarrhoea (Joshi *et al*., 2012). Hypochloremia might be due to the loss of chloride ions through vomition and loss in the secretion of intestinal fluid during diarrhoea, resulting in intestinal villous atrophy (Burchell *et al*., 2014). Hypokalaemia might be due to the loss of potassium in the diarrhoeic fluid along with sodium and bicarbonate (Agnihotri *et al*., 2017).

The dogs after treatment were active, alert and there was improvement in appetite. Haemato-biochemical parameters revealed significant increase in TLC (P<0.01), neutrophil (P<0.01), serum sodium (P<0.05) and potassium (P<0.05) with non-significant (P>0.05) increase in TP and serum chloride in post-treatment group in comparison to pre-treatment group. While, significant decrease in lymphocyte (P<0.01), BUN (P<0.01) and creatinine (P<0.05) with non-significant (P>0.05) decrease in Hb, PCV, TEC, monocyte, eosinophil, AST and ALT values were recorded in post-treatment group in comparison to pre-treatment group. Haemato-biochemical profiles also have reached nearer to the blood profile of healthy control group post 7th day of treatment. Fluid therapy helped in improvement of electrolyte and acid-base imbalances. Balanced isotonic crystalloid solution (lactated Ringer's) was the fluid of choice for initial restoration of intravascular fluid and rehydration. Lactated Ringer's solution approximately related to the composition of extracellular fluid which is lost in diarrhoea and vomition episodes (Waterman, 1991). Restoration of Hb, PCV, TEC and TP in post-treatment group was because of hydration improvement with fluid therapy. The values of AST and ALT were restored to normal in post-treatment group. The level of BUN and creatinine decreased to normal after 7th days of treatment indicating increased glomerular filtration rate. In the study, ceftriaxone (third generation cephalosporin) has been used which has broad spectrum activity. Bhat *et al*. (2015) has mentioned in his study that in vitro sensitivity of associated bacteria responsible for secondary infection was 91.66% (highly sensitive) for ceftriaxone.

CONCLUSION

This study was conducted on dogs infected with rota viral enteritis. CRV positive dogs showed significant increase in Hb, PCV, TEC, monocyte, lymphocyte, AST, ALT, BUN and creatinine with significant decrease in TLC, neutrophil, total protein, serum sodium, potassium and chloride. All the infected dogs recovered and the haemato-biochemical values became almost similar to healthy control group post 7th day treatment. Being the first report of RV detection in dog from Assam, this data will serve as a baseline to conduct further detailed study of RV infection in dogs. The evaluation of haemato-biochemical profiles might be helpful to clinicians while treating such type of clinical cases.

ACKNOWLEDGEMENT

The article is a part of MVSc research work of the first author. The authors gratefully acknowledged all the necessary facilities and helps received from the VCC Khanapara, Assam Agricultural University, Department of Veterinary Epidemiology and Preventive Medicine and Department of Microbiology for providing necessary facilities and support required during research period.

Conflict of interest: None.

REFERENCES

Agnihotri, D., Singh, Y., Maan, S., Jain, V.K. and Kumar, A. (2017). Molecular detection and clinico-haematological study of viral gastroenteritis in dogs. Haryana Veterinary. 56(1): 72-76.

Alfajaro, M.M. and Cho, K.O. (2014). Evidences and consequences of extra-intestinal spread of rotaviruses in humans and animals. Virus Disease. 25: 186-194.

Ansari, S.A., Springthorpe, V.S. and Sattar, S.A. (1991). Survival and vehicular spread of human rotaviruses: Possible relation to seasonality of outbreaks. Reviews of Infectious Diseases. 13: 448-461.

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

Barua, S.R., Rakib, T.M., Das, S., Mohammad, M., Hossain, A.M. and Chowdhury, S. (2018). Hematological and serological changes in neonatal diarrheic calves infected with bovine rotavirus. Multidisciplinary Advances in Veterinary Science. 2(3): 356-366.

Bhat, A.A., Wadhwa, D.R., Mandial, R.K., Sharma, A., Katoch, A. and Sharma, P. (2015). Clinico-biochemical alterations and therapeutic management of canine gastroenteritis. Journal of Animal Research. 5(1): 149. DOI: 10.5958/2277-940X.2015.00025.X

Biswas, S., Chakravorty, D. and Pradhan, N.R. (2005). Clinical and haemato-biochemical changes in parvovirus infection in dogs. Indian Journal of Veterinary Medicine. 25(1): 16-18.

Broor, S., Ghosh, D. and Mathur, P. (2003). Molecular epidemiology of rotaviruses in India. Indian Journal of Medical Research. 118: 59-67.

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): 152-156.

Chethan, G.E., Garkhal, J., Sircar, S., Malik, Y.P.S., Mukherjee, R., Gupta, V.K., Sahoo, N.R., Agarwal, R.K. and De, U.K. (2017). Changes of haemogram and serum biochemistry in neonatal piglet diarrhoea associated with porcine rotavirus type A. Tropical Animal Health and Production. 49(7): 1517-1522.

Crawford, S.E., Ramani, S., Tate, J.E., Parashar, U.D., Svensson, L., Hagbom, M., Franco, M.A., Greenberg, H.A., O'Ryan, M., Kang, G., Desselberger, U. and Estes, M.K. (2017). Rotavirus infection. Nature Reviews Disease Primers. 3: 17083.

Dalgic, N., Hasim, O., Pullu, M., Sancar, M., Kafadar, Y. and Yilmaz, A. (2010). Is rotavirus diarrhoea a systemic viral infection? Journal of the Pediatric Infectious Diseases. 2: 48-56.

Fulton, R.W., Johnson, C.A., Pearson, N.J. and Woode, G.N. (1981). Isolation of a rotavirus from newborn dog with diarrhea. American Journal Veterinary Research. 42: 841-843.

Goddard, A., Leisewitz, A.L., Christopher, M.M., Duncan, N.M. and Becker, P.J. (2008). Prognostic usefulness of blood leukocyte changes in canine parvoviral enteritis. Journal of Veterinary Internal Medicine. 22: 309-316.

Green, D., Wilimas, J.A. and Buckingham, S.C. (2003). Hematologic findings in children with rotavirus-positive and rota-negative diarrhea. Pediatric Hematology and Oncology. 20(6): 453-456.

Grigonis, A., Macijauskas, V. and Zamokas, G. (2002a). Examination of liver functions in dogs with parvovirus enteritis. Veterinarijair Zootechnika. 17: 23-28.

Herring, A.J., Inglis, N.F., Ojelt, C.K., Snodgrass, D.R. and James, D. (1982). Rapid diagnosis of rotavirus infection by direct detection of viral nucleic acid in silver-stained polyacrylamide gels. Journal of Clinical Microbiology. 16: 473-477.

Joshi, G., Singathia, R., Gattani, A., Yadav, R. and Lakhotia, R.L. (2012). Microbiochemical studies of canine parvovirus infection in puppies. Veterinary Practitioner. 13(2): 347-348.

Khatib, H.K. and Khan, H.Z. (2012). Biochemical studies in serum of children suffering from rotavirus diarrhoea. Bioscience Discovery. 3(1): 25-29.

Malik, Y.P.S., Kumar, N., Sharma, K., Sircar, S. and Bora, D.P. (2014). Rotavirus diarrhoea in piglets: A review on epidemiology, genetic diversity and zoonotic risks. Indian Journal of Animal Sciences. 84: 1035-1942.

Martella, V., Banyai, K., Matthijnssens, J., Buonavoglia, C. and Ciarlet, M. (2010). Zoonotic aspects of rotaviruses. Veterinary Microbiology. 140: 246-255.

Mosallanejad, B., Shapouri, M.R.S., Avizeh, R. and Pourmahdi, M. (2015). Antigenic detection of Canine rotavirus group A in diarrheic dogs in Ahvaz district, Southwestern Iran. Comparative Clinical Pathology. 24: 899-902.

Mossel E.C. and Ramig R.F. (2003). A lymphatic mechanism of rotavirus extra intestinal spread in the neonatal mouse. Journal of Virology. 77: 12352-12356.

Nadia, H. (2019). Algerian indigenous dog: haemato-biobiochemical profile in healthy and gastroenteritis diseases case. Agricultural Science Digest. 39(3): 244-249.

Ortega, A.F., Martinez-Castaneda J.S., Bautista-Gomez, L.G., Munoz, R.F. and Hernandez, I.Q. (2017). Identification of co-infection by rotavirus and parvovirus in dogs with gastroenteritis in Mexico. Brazilian Journal of Microbiology. 48: 767-773.

Ramig, R.F. (2004). Pathogenesis of intestinal and systemic rotavirus infection. Journal of Virology. 78: 10213-20.

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

Waterman, A.E. (1991). Fluid therapy and intensive care. In: Canine Medicine and Therapeutics, [Editor (Chandler, E.A.)], Blackwell Scientific Publications, Oxford, pp. 103-140.

Yin, L., Menon, R., Gupta, R., Vaught, L., Okunieff, P. and Vidyasagar, S. (2017). Glucose enhances rotavirus enterotoxin-induced intestinal chloride secretion. Pflugers Archives. doi: 10.1007/s00424-017-1987- x.