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Vital, hematological, and biochemical alterations in dogs naturally infected with canine parvovirus enteritis at the Veterinary Teaching Hospital, Usmanu Danfodiyo University, Sokoto

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Abstract

Canine Parvovirus Enteritis (CPVE) is a highly contagious and often fatal disease of dogs, characterized by severe gastroenteritis and systemic complications. This study aimed to determine the mean and standard deviation of selected vital, hematological, and biochemical parameters in dogs naturally infected with CPVE presented to the Veterinary Teaching Hospital, Usmanu Danfodiyo University, Sokoto, Nigeria. Eighty (80) dogs confirmed positive for CPVE using the Sensepert CPV Ag rapid test kit were evaluated upon admission. Vital parameters (pulse rate, respiratory rate, temperature), hematological indices (packed cell volume (PCV), red blood cell count [RBC], hemoglobin [Hb], mean corpuscular volume (MCV), mean corpuscular hemoglobin [MCH], mean corpuscular hemoglobin concentration (MCHC), platelet count, total and differential white blood cell counts), and biochemical parameters (electrolytes, urea, creatinine, total protein, albumin, and glucose) were analyzed. Data were expressed as mean±standard deviation. Mean pulse rate, respiratory rate, and temperature were 118±19.1 beats/min, 42±11.0 breaths/min, and 39.3±0.8°C, respectively. The dogs showed anemia (PCV 26.8±4.9%, Hb 7.8±1.6 g/dL, RBC 4.3±0.8 ×10⁶/μL). Biochemical results indicated hyponatremia (Na⁺ 129.3±11.2 mmol/L), hypochloremia (Cl⁻ 91.5±10.2 mmol/L), metabolic acidosis (HCO₃⁻ 20.9±6.1 mmol/L), azotemia (urea 45.9±13.9 mg/dL, creatinine 2.1±0.5 mg/dL), hypoproteinemia (TP 4.7±0.9 g/dL; albumin 1.9±0.5 g/dL), and hypoglycemia (47.4±19.3 mg/dL). Dogs infected with CPVE exhibited marked hematological and biochemical derangements indicative of dehydration, electrolyte imbalance, and renal and metabolic dysfunction. Routine monitoring of these parameters upon admission may serve as a valuable prognostic tool for improving survival outcomes in affected dogs.

Keywords: Canine parvovirus enteritis, hematology, biochemistry, electrolytes, Sokoto, dogs

Introduction

Canine parvovirus-2 (CPV-2) is an important enteropathogen and a significant cause of acute haemorrhagic enteritis and myocarditis in dogs (Green and Decaro, 2012) [6]. Canine parvovirus (CPV) and other viral organisms such as canine coronavirus (CCoV) and canine distemper virus (CDV) have been identified as primary causes of enteritis in dogs. However, canine parvovirus was reported as one of the most common causes of infectious disorders of dogs and the most prevalent virus in dogs with infectious diarrhoea (Waldvogel *et al.* 1991) [17]. The virus is very sturdy, highly contagious causing extremely fatal disease. The virus was first reported in 1977 and over time, it has contracted a large number of dogs around the world with extremely high morbidity (100%) in puppies, moderate mortality in adults and a reported high mortality of 91% in puppies (Appel *et al.* 1979) [2]. It is believed that CPV originated as a host range variant from feline panleukopenia virus (FPV), which includes a direct mutation from FPV, a mutation from a FPV vaccine virus and the adaptation to the new dog host via non-domestic carnivores, like mink and foxes. The disease has two prominent clinical forms depending on the age of dogs: severe enteritis associated with vomiting and diarrhoea reported

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in all age groups and subsequent heart failure reported in very young puppies of less than 3 months of age (Appel *et al.* 1979) [2].

Total white blood cell counts and the presence of leukopenia have previously been considered to be a hallmark in patients with CPV because the virus attacks actively replicating cells of the bone marrow, thymus, and other lymphoid tissues. Lymphopenia can occur in puppies infected with CPV as well as puppies affected with canine coronavirus (Castro *et al.*, 2013) [4] so is used along with other testing to further support a diagnosis of CPV in affected patients. The presence of cytopenia during the course of illness can be useful for predicting outcome in CPV. One study documented no significant difference in neutropenia between survivors and non-survivors with CPV. The same study found that maintenance of total leukocyte count greater than 4500/ μ L and a lymphocyte count greater than 1000/ μ L at the time of admission and through 48 hours of hospitalization were strongly predictive of survival (Goddard *et al.*, 2008) [7]. The precise diagnosis of canine parvoviral enteritis not only aids in proper treatment and recovery of affected dogs but also helps in isolation of affected dogs from other susceptible healthy dogs. Clinical diagnosis in CPV-affected dogs can be inconclusive because of the similarities in clinical signs with several other diseases such as canine distemper, canine coronavirus enteritis and haemorrhagic gastroenteritis. Therefore, suspected cases with typical clinical signs should always be verified with reliable laboratory tests (Greene and Decaro, 2012) [6].

Treatment for PVE is largely supportive and symptomatic. The principal components of treatment include; fluid therapy, antibiotic treatment, antiemetic treatment, and nutritional support. An array of other treatment measures including, though not limited to, antiviral treatments, immunotherapy and pain management have been assessed in the past or are currently under investigation regarding their potential utility in PVE (Mylonakis *et al.*, 2016) [13].

Materials and Methods

This study was hospital based where dogs with clinical signs of gastroenteritis presenting to clinics from July 2024 to July 2025 with signs of vomiting, lethargy, dehydration, foul-smelling diarrhea were recruited for the study with client consent after education about benefits and implications of study. A total of 80 dogs were diagnosed with canine parvovirus enteritis with the aid of Sensepert Ag rapid test kit. Faecal sample was collected by inserting a swab into the rectum as described by the manufacturer. The swab containing fecal sample was inserted into a sample collection tube and emulsified in 0.1M Phosphate Buffered Saline (PBS) with pH of 7.4 and stirred 10 times. The swab was squeezed against the wall of tube to remove as much sample as possible and discarded. The supernatant part of the sample was aspirated using the Pasteur dropper provided and 5 drops of the sample was dispensed into the hole of the test device. Result was read in 5 minutes and interpreted based on appearance as positive if double lines appear and negative if single line appears.

Vital signs, hematological (PCV, total white blood cell count (TWBC), platelet count), and biochemical parameters (Na⁺, K⁺, Cl⁻, Urea, Creatinine, Glucose, Total Protein) were obtained from CPV-positive dogs upon admission. A total of 5ml of blood was collected from the cephalic vein after swabbing the area with cotton wool and alcohol, 2ml aliquot was dispensed into EDTA sample bottle for haematology, while remaining 3ml was dispensed into plain sample bottle

to harvest serum for determination of glucose, BUN, creatinine, total protein, albumin, Na⁺, K⁺, Cl⁻ and HCO₃⁻. Samples were immediately taken to Veterinary Clinical Pathology unit of Usmanu Danfodiyo University, Sokoto for analysis. Auto hemoanalyzer model HB7021 and biochemical analyzer SK3002B were used for CBC and biochemical analysis respectively.

Descriptive statistic was used to analyze data and results were presented as mean and standard deviation

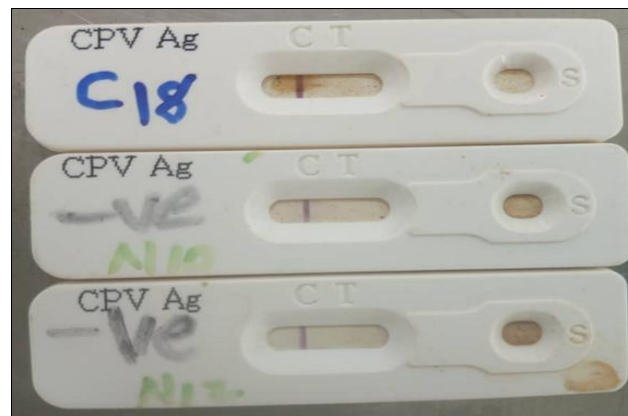


Plate 1: Negative result with single line

Table 1: Mean vital, hematological and biochemical parameters of CPVE positive dogs

Parameters (N=80)	Values (mean \pm SD)
Pulse rate (beats/min)	118 \pm 19.1
Respiratory rate (Cycles/min)	42 \pm 11.0
Temperature ($^{\circ}$ C)	39.3 \pm 0.8
PCV (%)	26.8 \pm 4.9
RBC ($\times 10^6/\mu$ L)	4.3 \pm 0.8
HB (g/dl)	7.8 \pm 1.6
MCV (fl)	61.9 \pm 8.2
MCH (pg)	18.0 \pm 2.8
MCHC (g/dl)	29.2 \pm 2.5
PLATELETS ($\times 10^3/\mu$ L)	249.7 \pm 105.1
TWBC ($\times 10^3/\mu$ L)	10.6 \pm 4.4
Lymphocytes ($\times 10^3/\mu$ L)	1.2 \pm 0.9
Monocytes ($\times 10^3/\mu$ L)	0.7 \pm 0.4
Eosinophils ($\times 10^3/\mu$ L)	0.5 \pm 0.3
Neutrophils ($\times 10^3/\mu$ L)	8.0 \pm 3.5
Basophils ($\times 10^3/\mu$ L)	0.01 \pm 0.04
Na ⁺ (mmol/L)	129.3 \pm 11.2
K ⁺ (mmol/L)	6.4 \pm 1.7
Cl ⁻ (mmol/L)	91.5 \pm 10.2
CO ₃ (mmol/L)	20.9 \pm 6.1
UREA (mg/dl)	45.9 \pm 13.9
Creatinine (mg/dl)	2.1 \pm 0.5
Total Protein (g/dl)	4.7 \pm 0.9
Albumin (g/dl)	1.9 \pm 0.5
Glucose (mg/dl)	47.4 \pm 19.3

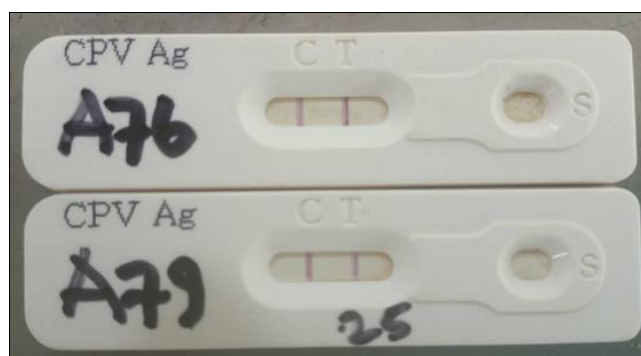


Plate 2: Positive result with double line

Discussion

The findings of this study revealed significant alterations in vital, hematological, and biochemical parameters among dogs naturally infected with canine parvovirus enteritis (CPVE). These changes are consistent with the pathophysiological effects of severe gastroenteritis, dehydration, electrolyte imbalance, and systemic inflammatory response induced by canine parvovirus (CPV).

The elevated mean pulse rate (118 ± 19.1 beats/min) and respiratory rate (42 ± 11.0 breaths/min) observed in this study are typical of dogs suffering from hypovolemia, fever, and systemic infection. The mean body temperature ($39.3 \pm 0.8^\circ\text{C}$) was slightly above the normal reference range for dogs, suggesting that most affected dogs were febrile at presentation. Similar findings were reported by Decaro and Buonavoglia (2012)^[6] and Ogbu *et al.* (2020)^[14], who attributed pyrexia in CPVE to viral replication, intestinal inflammation, and bacterial translocation.

The mean packed cell volume (PCV = $26.8 \pm 4.9\%$) and hemoglobin concentration (7.8 ± 1.6 g/dL) indicate moderate anemia. This may result from intestinal hemorrhage, malabsorption of nutrients, and hemodilution following aggressive intravenous fluid therapy before hospital admission in some cases. The reduction in red blood cell count ($4.3 \pm 0.8 \times 10^6/\mu\text{L}$) supports this observation. Similar hematological profiles have been reported in CPV-infected dogs in Nigeria and other endemic regions (Olaifa *et al.*, 2025; Mekky *et al.*, 2024)^[15, 12]. The anemia is typically non regenerative reflecting bone marrow suppression during the viremic phase, but regenerative anemia can also be observed during recovery stage of the disease.

Biochemical findings indicated notable electrolyte imbalances. Hyponatremia (129.3 ± 11.2 mmol/L) and hypochloremia (91.5 ± 10.2 mmol/L) are consistent with the loss of sodium- and chloride-rich intestinal secretions during vomiting and diarrhea, compounded by inadequate fluid intake. Hyperkalemia (6.4 ± 1.7 mmol/L) was observed, possibly reflecting metabolic acidosis, tissue catabolism, or transient renal compromise. The mean bicarbonate concentration (20.9 ± 6.1 mmol/L) suggests metabolic acidosis, a hallmark of CPVE due to lactic acid accumulation from dehydration and hypoperfusion (Burchell *et al.*, 2020)^[3].

Azotemia, as indicated by elevated mean urea (45.9 ± 13.9 mg/dL) and creatinine (2.1 ± 0.5 mg/dL), likely reflects pre-renal dehydration and reduced glomerular filtration rate. These alterations are commonly associated with severe fluid loss and circulatory collapse (Van den Berg *et al.*, 2018). The observed hypoproteinemia (total protein = 4.7 ± 0.9 g/dL) and hypoalbuminemia (1.9 ± 0.5 g/dL) may result from intestinal protein loss, malabsorption, and systemic inflammatory response, leading to poor oncotic pressure and tissue edema (Mazzaferro, 2020)^[11].

Marked hypoglycemia (47.4 ± 19.3 mg/dL) was a prominent feature and could be due to starvation, intestinal malabsorption, and increased glucose utilization by septic tissues which are findings or complications observed in dogs with CPVE (Idowu and Heading, 2018)^[9]. This finding aligns with Kowalski (1980)^[10] and Chalifoux *et al.* (2021)^[5], who associated hypoglycemia with poor prognosis in CPV-infected dogs, especially in young puppies.

Overall, these hematological and biochemical changes reflect the complex pathophysiological interactions during CPV infection dehydration, inflammation, secondary bacterial infection, and metabolic derangements. Monitoring these indices at presentation provides valuable diagnostic and prognostic information that can guide fluid, electrolyte, and metabolic therapy. Early correction of electrolyte deficits and hypoglycemia has been shown to improve survival in affected dogs (Mylonakis *et al.*, 2016)^[13].

Conclusion

The present study demonstrates that dogs infected with canine parvovirus enteritis exhibit characteristic hematological and biochemical abnormalities, including anemia, neutrophilia, electrolyte imbalances, azotemia, hypoproteinemia and hypoglycemia. These findings are consistent with dehydration, systemic inflammation, and metabolic derangements associated with CPVE. Routine monitoring of vital, hematological, and biochemical parameters at the time of admission is essential for assessing disease severity, guiding therapeutic interventions, and improving survival outcomes. Future studies should focus on correlating these parameters with survival rates and treatment responses to enhance prognostic evaluation in affected dogs.

Conflict of Interest

Not available

Financial Support

Not available

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