RESEARCH ARTICLE

Molecular Characterization of Circulating Canine Parvovirus Type 2c (CPV-2c) and Baseline Haemato-Biochemical Profiling of Parvoviral Enteritis in Unvaccinated Dogs from Guwahati, Assam

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Abstract

The present study was aimed to characterize the circulating Canine Parvovirus (CPV) strain in Guwahati, Assam, and evaluate the efficacy of rapid diagnostic screening against the polymerase chain reaction (PCR), and documented the clinical manifestation of the infection. 57 faecal samples were screened between July 2024 and December 2025, from unvaccinated dogs exhibiting severe haemorrhagic enteritis were screened. PCR analysis confirmed 32 positive cases. The rapid antigen test (RAT) demonstrated a sensitivity of 78.12% and specificity of 96.0%, failing to detect 7 active infections. Sequencing of the partial *VP2* gene amplicons and subsequent phylogenetic analysis revealed that the isolates from Guwahati strictly clustered within the CPV-2c clade, displaying close genetic proximity to regional and transboundary strains from Arunachal Pradesh, India and Sri Lanka. Epidemiological profiling of the 32 PCR-positive cases highlighted a profound susceptibility in young puppies (average age 52.32 days) and indigenous non-descript (56.25 %) dog population. Clinically, 100% of the positive cases presented had severe haemorrhagic diarrhoea, anorexia, and lethargy, and all were unvaccinated dogs. Baseline haemato-biochmeical evaluation (n=32) at the time of admission revealed profound, leukopenia (3.15± 0.13 $\times 10^3$/µL), neutropenia (2.11± 0.08$\times 10^3$/µL) and lymphopenia (0.652± 0.04$\times 10^3$/µL), accompanied by marked elevations in serum AST (38.7± 0.55 U/L) and depletion of serum albumin (3.28± 0.04 g/dL). This study provides definitive molecular evidence for the active regional circulation of the highly contagious CPV-2c variant in Assam and underscores the critical necessity of PCR-based molecular surveillance, comprehensive baseline haemato-biochmeical profiling, and rigorous early vaccination adherence to curb the high morbidity associated with this evolving pathogen.

Keywords: Biochemistry, Canine parvovirus, CPV-2c, Epidemiology, Guwahati, Haematology

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Introduction

Canine parvovirus (CPV) is a highly contagious viral enteritis particularly affecting young puppies globally (Bahera *et al.*, 2015). Canine Parvo Virus was first identified in the late 1970s, and currently, three distinct genetic variants *viz.*, CPV-2a, CPV-2b, and CPV-2c are circulating worldwide (Nandi and Kumar, 2010). CPV has strong tropism for rapidly dividing cells, most commonly the crypt epithelial cells of the small intestine, leading to severe haemorrhagic diarrhoea, vomiting, and immunosuppression (Sykes, 2014). Rapid and accurate diagnosis is critical for managing outbreaks and tracking epidemiological shifts. While rapid antigen detection kits provide quick field level results, PCR remains highly sensitive, and a gold-standard technique for specific variant identification (Tanwar *et al.*, 2020). Despite the recent reports of CPV-2c in other parts of Northeastern India (Chethan *et al.*, 2025), targeted molecular and epidemiological evaluation in the Guwahati city District of Assam remains scarce. Furthermore, while molecular characterization identifies the circulating variant, establishing the immediate underlying haemato-biochemical status caused by the infection at the time

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of presentation is clinically vital for predicting disease severity. Therefore, this study was aimed to characterize the circulating CPV strain in Guwahati, Assam, to evaluate the comparative efficacy of rapid diagnostic screening, and systematically determine the baseline haemato-biochemical status in naturally exposed, unvaccinated puppies.

Materials And Methods

The study was conducted at the College of Veterinary Science, Assam Agricultural University (AAU), Khanapara, Guwahati, Assam (India) from July 2024 to December 2025. Rectal swabs were collected from 57 non-vaccinated dogs presented with clinical signs of systemic illness, including foul-smelling haemorrhagic diarrhoea, emesis, anorexia, and severe dehydration. Samples were immediately screened using a commercial rapid antigen test kit (Bio one Pvt. Ltd., India) following the manufacturer's instructions. Concurrently, self-defecated faecal samples were processed in sterile phosphate-buffered saline (PBS) to extract the viral DNA using a NucleoSpin DNA stool Kit (MN, Germany). The extracted DNA was subjected to PCR amplification targeting the *VP2* gene utilizing specific primers (Forward: 5'-AGCAGATGGTGATCCAAGAT-3'; Reverse: 3'-TGGATTCCAAGTATGAGAGG-5') (De la Torre *et al*., 2018). Amplicons were visualized by agarose gel electrophoresis. Positive amplicons were gel-purified and outsourced (Eurofins Genomics India, Pvt Ltd) for sequencing. Phylogenetic analysis was subsequently performed based on the partial *VP2* gene sequence using the Neighbour-Joining method with 1000 bootstrap replicates by Mega 11 software (Tamura *et al*., 2021).

Haemato-biochemical evaluation was performed on 32 PCR-confirmed animals immediately upon admission (Day 0), prior to the administration of any supportive fluid or therapeutic regimens. EDTA whole blood aliquots were automatically processed via an automated haematology analyzer (Erba Mannheim, Germany) to determine absolute leukocyte values, erythrocyte count, and thrombocyte parameters. Concurrently, separated serum profiles were evaluated using an automated chemistry analyzer (Mispa ChemDx, India) to establish baseline values for alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood urea nitrogen (BUN), creatinine, total protein, and albumin.

For the objective execution of the one-sample statistical hypotheses across all parameters, the exact mid-value of the standard physiological reference intervals established by the Merck Veterinary Manual (2023) was utilized as the population test value (μ). Normality of the dataset columns was checked via the Shapiro-Wilk test. The haemato-biochemical variables demonstrating normal or symmetric distributions ($p>0.05$) were analyzed using the parametric Student's one-sample t-test. Conversely, variables displaying skewed or non-parametric distributions ($p<0.05$) namely PCV, RBC, and thrombocytes were evaluated using the non-parametric Wilcoxon signed-rank test in Jamovi (Version 2.3), with statistical significance set at $p < 0.05$.

Results and Discussion

Initial field screening using the rapid antigen test identified 26 of the clinically suspected cases as positive. However, subsequent PCR analysis of all the 57 samples confirmed 32 positive and 25 negatives for CPV (Table 1). The rapid test detected 25 true positives but failed to detect 7 active cases (false negative) and yielded 1 false positive. Comparatively, the rapid antigen kit demonstrated an apparent sensitivity of 78.12% and a specificity of 96.00% against the PCR gold standard. This discrepancy highlights the inherent limitations of relying solely on antigen-based assays for epidemiological screening, as they require sufficient viral shedding that may occur only during specific windows of the infection (Mehta *et al*., 2020).

Table 1: Comparative diagnostic efficacy of the commercial Rapid antigen test kit versus PCR for the detection of Canine Parvovirus from faecal samples (n=57)

Diagnostic assay	PCR positive	PCR negative	Total
Rapid antigen kit positive	25 (True positive)	1 (False positive)	**26**
Rapid antigen kit negative	7 (False negative)	24 (True negative)	**31**
Total	**32**	**25**	**57**

Clinically, the cohort of 32 CPV-2c positive dogs presented with classical, severe manifestations of canine parvoviral enteritis (Table 2). The most consistent clinical signs observed at the time of admission were severe haemorrhagic diarrhoea, anorexia, and lethargy, occurring universally in 100% of the positive cases. This was closely followed by profound emesis, which was present in 93.75% of the dogs. Furthermore, over half of the cohort (53.13%) presented with severe dehydration (>8%). The high frequency of these severe gastrointestinal signs correlated directly with the rapid fluid loss and subsequent hypovolaemic shock that characterize acute CPV-2c infections in unvaccinated puppies. The aggressive clinical presentation documented in this study underscores the high pathogenicity of the circulating CPV-2c strain in the region. This perfect alignment between severe clinical manifestations characterized by universal haemorrhagic enteritis and rapid dehydration and definitive molecular identification highlights that the circulating CPV-2c variant remains highly virulent and presents a profound clinical threat to the regional canine population (Decaro and Buonavoglia, 2011).

Table 2: Frequency of primary clinical signs recorded at the time of admission in dogs confirmed positive for CPV by PCR (n=32).

Clinical presentation	No. of dogs	%
Anorexia / Inappetence	32	100.00
Lethargy / Depression	32	100.00
Haemorrhagic diarrhoea (Foul-smelling)	32	100.00
Emesis (Vomiting)	30	93.75
Severe dehydration (>8%)	17	53.13
Mild to moderate dehydration (<5%)	15	46.88

Molecular confirmation by PCR yielded a distinct 529 bp amplicon of the partial *VP2* gene (Fig. 1). Sequencing of three representative PCR amplicons and subsequent BLAST analysis confirmed the highest sequence similarity to the CPV-2c strain. While restricting sequencing to a representative subset (n=3) out of the 32 positive cases represents a limitation of this study. This was primarily necessitated by the low viral DNA concentration and an inadequate amplicon yield recovered from the majority of the clinical faecal samples, which did not meet the stringent quality thresholds required for reliable Sanger sequencing. Although we sequenced only three samples in the present study, this is sufficient to confirm the circulation of the CPV-2c strain in the study area. The retrieved sequences were deposited in NCBI GenBank (Table 3).

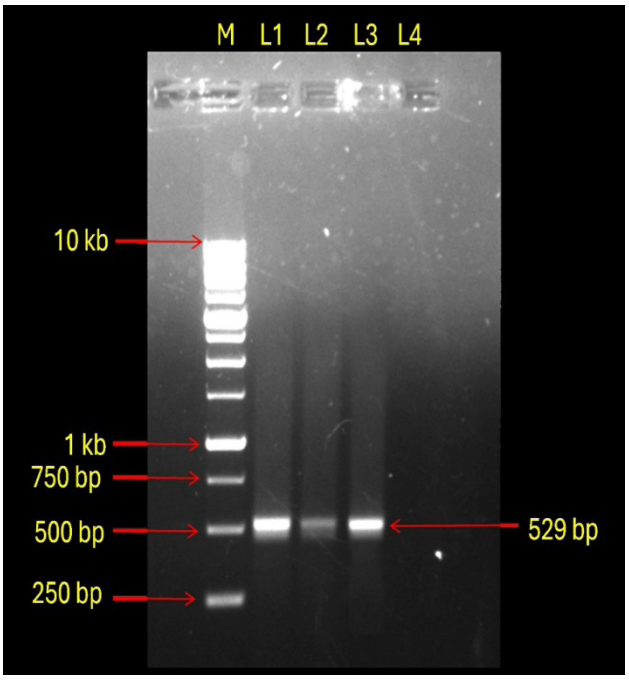


Fig. 1: PCR amplification of the *VP2* gene for the confirmatory diagnosis of Canine Parvovirus. Lane M: 1 kb + DNA ladder; Lanes L1-L3: Positive clinical samples showing the specific 529 bp amplicon; L4: Non Template Control (NTC).

Table 3: Details of the Canine Parvovirus (CPV) *VP2* gene sequences isolated from dogs in Guwahati, Assam, and submitted to GenBank GenBank

Isolate Designation	GenBank Accession No.	Confirmed Strain	Target Gene	Origin/ Location
CPV1_VP2_ CVSc_2025	PV424420	CPV-2c	VP2 (Partial)	Guwahati, Assam
CPV2_VP2_ CVSc_2025	PX682472	CPV-2c	VP2 (Partial)	Guwahati, Assam
CPV3_VP2_ CVSc_2025	PX833276	CPV-2c	VP2 (Partial)	Guwahati, Assam

Phylogenetic analysis of the *VP2* gene sequences revealed that the Guwahati isolates clustered strictly within the CPV-2c clade (Fig. 2). The isolates of the present study grouped very closely with the sequences from Sri Lanka and Arunachal Pradesh, India on the phylogenetic tree. The genetic similarity suggests that this rapidly evolving variant is actively spreading across regional and national borders (Decaro and Buonavoglia, 2011)

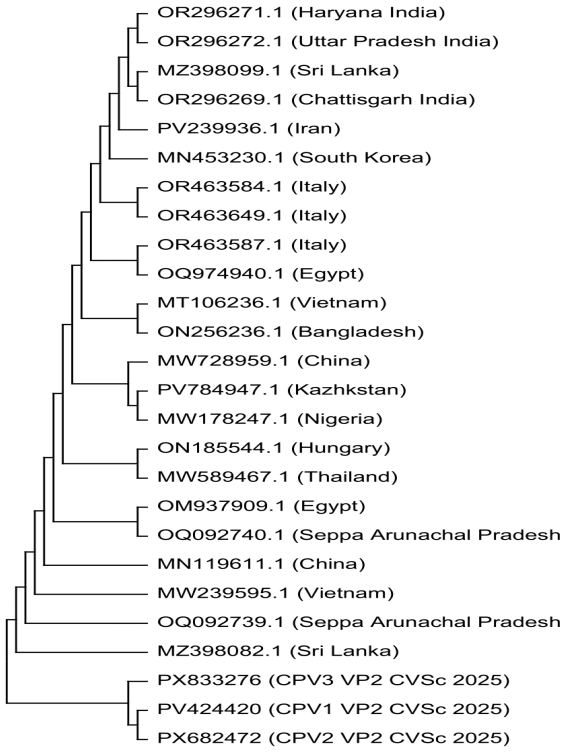


Fig. 2: Phylogenetic tree based on the partial nucleotide sequence of the *VP2* gene of Canine Parvovirus (CPV) The isolates from the present study in Guwahati, Assam were CPV1_VP2_CVSc_2025, CPV2_VP2_CVSc_2025 & CPV2_VP3_CVSc_2025 respectively.

The epidemiological data from this study reveals key patterns about the local CPV-2c circulation (Table 4). The infection primarily affected young puppies, with an average age of 52.32 days. This corroborated with the findings of Bahera *et al.* (2015), who noted that puppies under three months of age are most susceptible for CPV infection. This age group is highly vulnerable to CPV infection due to a critical immunity gap. During this period,

the protective antibodies they received from their mother drop to very low levels. Because their own immune system is not yet fully mature, they are left highly exposed to environmental infections (Goddard and Leisewitz, 2010). Among the breed, the highest incidence was recorded in indigenous non-descript animals (56.25%). This high incidence makes sense given the large population of local dogs in the area. It also highlights a perilous lack of herd immunity among both street dogs and adopted strays due to non-vaccination. Previous work suggests a higher risk for male dogs or specific purebreds due to underlying genetic susceptibilities (Kalli *et al*., 2010). However, present study showed the opposite, with females accounting higher number of cases (53.12%). Several factors may explain this difference in sex distribution. First, our sample animal consists mostly of indigenous dogs, which inherently possess greater genetic diversity and lack the specific genetic vulnerabilities commonly documented in purebreds (Houston *et al*., 1996; Mylonakis *et al*., 2016). Second, socio-behavioural factors significantly influence hospital-based demographics. Local caregivers actively prioritize female street dogs for rescue and veterinary treatment, which frequently leads to an over-representation of females in clinical settings (Totton *et al*., 2010; Paul *et al*., 2016). Lastly, with a limited sample size, localized litter clustering can easily skew the final sex ratio. All the confirmed CPV-2c cases in the present study occurred in completely unvaccinated dogs.

Table 4: Epidemiological profile of dogs positive for Canine Parvovirus infection by PCR in Guwahati, Assam (n=32)

Parameter	Category	No.	%
Age	<45 days	8	25.00
	45-60 days	14	43.75
	>60 days	10	31.25
Sex	Male	15	46.88
	Female	17	53.12
Breed	Non-Descript	18	56.25
	Labrador Retriever	4	12.50
	German Shepherd	3	9.38
	Golden Retriever	2	6.25
	Others (Husky, Spitz, Pug, Beagle)	5	15.62
Vaccination	Unvaccinated	32	100
	Vaccinated	0	0

While the global emergence of the CPV-2c variant characterized by the specific Asp426Glu mutation on the major capsid protein has raised concerns regarding reduced cross-neutralization and potential breakthrough infections in vaccinated populations (Miranda and Thompson, 2016). The complete lack of vaccination in the study positive samples highlights the failure of basic prophylactic coverage, which remains the primary driver of high viral circulation and morbidity in Guwahati.

Evaluation of the haematological parameters against standard Merck reference values revealed severe, uniform deviations across all parameters (Table 5). The definitive haematological hallmark identified in these 32 dogs was leukopenia (3.15±0.13 $\times 10^3$/µL, p<0.001), with values below the standard physiological range. Leukopenia reflects the typical bone-marrow tropism of canine parvovirus, where active lytic replication shreds the rapidly dividing hematopoietic stem cell, affecting the host's primary immune defense mechanism (Goddard and Leisewitz, 2010). This bone marrow suppression, further exacerbated by the massive, unmitigated pooling and consumption of peripheral immune cells within the necrotic, severely damaged layers of the intestinal wall, heavily reflected in the absolute neutropenia (2.11±0.08 $\times 10^3$/µL, p<0.001) and lymphopenia (0.652± 0.04$\times 10^3$/µL, p<0.001), findings which strongly aligned with variant-induced lymphoid depletion dynamics already reported (Decaro and Buonavoglia, 2011).

The baseline red blood cell parameters (Erythrocytes: 7.01±0.06 $\times 10^6$/µL; Packed Cell Volume: 43.40±0.23 %) initially remained within normal reference intervals, yet showed a significant statistical variation against their respective reference test values (p< 0.001). Rather than representing structural blood stability, these elevated values are a classic indicator of acute haemoconcentration driven by dehydration due to diarrhoea and vomiting (Castro *et al*., 2013). The catastrophic fluid loss out of the vascular space into the intestinal lumen thickens the blood, creating a relative elevation in cell percentages that temporarily masks the true severity of the underlying anaemia caused by active gastrointestinal bleeding. Conversely, circulating haemoglobin concentrations dropped significantly below standard parameters (13.60± 0.12g/dL p< 0.001), confirming structural blood breakdown and early mucosal blood loss. This was further accompanied by a significant thrombocytopenia, with thrombocyte counts dropping to 3.52±0.01 $\times 10^5$/µL, p<0.001), suggesting localized platelet consumption and early microvascular clotting within the shredded mucosal layers of the gut, a process typical of viral-induced endothelial injury (Goddard and Leisewitz, 2010).

This severe tissue destruction is further supported by the initial serum biochemical analysis (Table 5). While ALT (33.00± 0.49, U/L) remained within normal boundaries (p<0.001), AST levels exhibited a highly significant, massive spike (38.70± 0.55 U/L, p<0.001) over the baseline reference standard. This profound hepatic enzyme divergence typically arises from acute systemic tissue hypoperfusion, localized cell death, and early ischemic injury resulting from poor hepatic tissue perfusion as a consequence of progressive hypovolemic shock rather than primary hepatocyte necrosis (Schoeman *et al*., 2013). This multi-organ crisis is further reflected in the early stages of protein-losing enteropathy. Total serum protein (6.08 ± 0.04 g/dL) and albumin (3.28 ± 0.04 g/dL) were significantly depleted relative to their normal test standards (p<0.001), serving as a clear functional indicator of

massive structural barrier breakdown, where plasma proteins actively leak across the damaged surface of the intestinal villi into the gut lumen (Kalli *et al*., 2010). Interestingly, BUN (17.80 ± 0.37 mg/dL, p=0.540) showed a non-significant lower value against the standard baseline midpoint, while serum creatinine dropped significantly to 0.98 ± 0.04 mg/dL, p=0.006. This demonstrates that at the early presentation phase, the kidneys are temporarily maintaining functional clearance prior to the full onset of pre-renal azotemia, which commonly develops as hypovolemia advances without intervention (Goddard and Leisewitz, 2010).

Table 5: Haemato-biochemical profile of dogs positive for Canine parvovirus cohort at presentation (Mean ± SE, n=32)

Parameters	CPV positive	Healthy @	p-Value
Haemoglobin (g/dL)	13.60±0.12	15.4	<0.001***
Packed Cell Volume (%)	43.40±0.23	56	<0.001***
RBC (×10^6/µL)	7.01±0.06	6.41	<0.001***
TLC (×10^3/µL)	3.15±0.13	9.55	<0.001***
Neutrophils (×10^3/µL)	2.11±0.08	7.45	<0.001***
Lymphocytes (×10^3/µL)	0.652±0.04	2.9	<0.001***
Thrombocytes (×10^5/µL)	3.52±0.01	4.26	<0.001***
ALT (U/L)	33.00±0.49	59.5	<0.001***
AST (U/L)	38.70±0.55	14	<0.001***
Albumin (g/dL)	3.28±0.04	3.85	<0.001***
Total Protein (g/dL)	6.08±0.04	6.45	<0.001***
BUN (mg/dL)	17.80±0.37	18	0.540
Creatinine (mg/dL)	0.98±0.04	1.1	0.006**

@Merck Veterinary Manual (2023), ***p<0.001 highly significant.

Conclusions

The emergence of CPV-2c in Assam emphasizes the urgent need to update regional vaccination strategies. The new variants often drive outbreak of infection. This study provides definitive molecular evidence for the active circulation of the highly contagious CPV-2c genetic variant in Guwahati, Assam. The integration of absolute baseline haemato-biochemical profiling reveals that this circulating variant triggers an immediate, multi-systemic physiological crisis at the time of clinical presentation characterized by a profound cellular collapse within white blood cell lineages resulting in severe leukopenia, absolute neutropenia, and lymphopenia alongside early-stage protein-losing enteropathy marked by significant serum albumin depletion. Taken together, these severe systemic deviations and the absolute lack of vaccine adherence in infected puppies emphasize the critical necessity of transitioning from narrow field tests to a comprehensive diagnostic approach. Continuous molecular surveillance utilizing PCR and extended sequencing, combined with early clinicopathological baseline profiling, is highly recommended to monitor variant evolution, track transboundary spread, and guide aggressive, early therapeutic fluid and metabolic management. Ultimately, these findings underscore an urgent need to update regional prophylactic strategies and enforce strict early vaccination adherence to curb the high mortality associated with this evolving viral pathogen.

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