



International Journal of Veterinary Sciences and Animal Husbandry



ISSN: 2456-2912

NAAS Rating (2025): 4.61

VET 2025; 10(12): 253-256

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www.veterinarypaper.com

Received: 13-10-2025

Accepted: 14-11-2025

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Diagnostic and therapeutic management of canine distemper

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DOI: <https://www.doi.org/10.22271/veterinary.2025.v10.i12d.2826>

Abstract

Canine Distemper is a highly contagious viral disease. It belongs to the genus *Morbili virus* within the family *Paramyxoviridae*. It is a highly contagious disease of domestic dogs and wild carnivores, associated with high morbidity and mortality. Despite vaccination and health management strategies, no specific antiviral therapy is currently available. This study reports clinical, hematological, and biochemical findings in six dogs naturally infected with CD compared with six healthy control dogs of similar age and breed. Affected animals presented with characteristic clinical signs, including fever, anorexia, nasal and ocular discharge, temporal twitching, frothy salivation during feeding, and prolapse of the third eyelid. Canine Distemper infection was confirmed using a rapid antigen detection test and further verified by reverse transcription polymerase chain reaction. All affected dogs received supportive and symptomatic therapy, including intravenous fluids, antihistamines (Histanil), digestive enzyme supplements (Pregamyl Forte), atropine sulphate, anti-inflammatory medication (Melonex), neurotonic (Neurokind injection), and antibiotics (Intacef). Subsequently, a combination of *Cicuta virosa* 30C, Pregamyl Forte, and Neurokind was administered for one month. Gradual neurological improvement was observed, and follow-up PCR results were negative for Canine Distemper, with full recovery in all treated dogs. Hematological analysis of Canine Distemper-infected dogs revealed significant leukopenia, lymphopenia, and mild anemia compared with healthy controls. Biochemical evaluation demonstrated elevated liver enzymes, increased total bilirubin, and reduced total protein and albumin levels in affected animals. Supportive management combined with *Cicuta virosa* 30C and nutritional supplementation appeared to facilitate recovery and normalization of clinical and laboratory parameters.

Keywords: Canine distemper virus, hematology, biochemistry, RT-PCR, *Cicuta virosa*, supportive therapy

Introduction

Canine Distemper (CD) is a highly contagious and often fatal viral disease that affects domestic dogs and a variety of wild carnivores globally. The causative agent belongs to the genus *Morbillivirus* under the family *Paramyxoviridae* (Geetha & Selvaraju, 2021) [3]. CDV exhibits a broad host range, infecting not only domestic dogs but also wildlife species such as lions, tigers, leopards, and foxes. The disease is characterized by multi systemic disease development with highest mortality rate after rabies (Latha *et al.*, 2007) [6]. Following infection through the upper respiratory tract, the virus primarily targets lymphoid tissues, resulting in marked immunosuppression, (Krakowka *et al.*, 1980; Krakowka, 1985) [1, 5]. As the disease progresses, virus disseminates systemically, affecting multiple organ systems including the respiratory, gastrointestinal, integumentary, and central nervous systems. Neurological complications are particularly severe and may persist as chronic sequelae even after the acute phase of infection (Summers *et al.*, 1978) [9]. Despite the availability of effective vaccines, outbreaks of CDV continue to occur, particularly in inadequately vaccinated or stray dog populations. No specific antiviral therapy exists, and current treatment is largely based on symptomatic therapy.

Materials and Methods

A total of twelve dogs (n = 12) that were presented to Veterinary Clinical Complex,

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Rajendranagar were included in the present study, consisting of six clinically affected dogs (Group I) and six apparently healthy dogs (Group II, control group). All animals were of similar age (4–7 years), different breeds, and sex (male), and were maintained under comparable managerial and nutritional conditions.

Clinical Examination

The dogs of canine distemper that were presented to the Veterinary Clinical Complex, Rajendranagar with clinical signs such as temporal muscle twitching, inappetence, and pyrexia were selected in present study. A detailed clinical and physical examination was performed in all the animals.

Approximately 5 mL of blood was collected aseptically from the cephalic vein of each dog.

- 2 mL was placed into EDTA-coated vials for hematological analysis.
- 3 mL was placed into plain vials, allowed to clot, and centrifuged at 3,000 rpm for 10 minutes to obtain serum for biochemical and CD antigen testing.

Serum samples were subjected to a rapid Canine Distemper antigen test (Snap Test). The parameters evaluated included:

- Hemoglobin (Hb)
- Packed Cell Volume (PCV)
- Total Erythrocyte Count (TEC)
- Total Leukocyte Count (TLC)
- Differential Leukocyte Count (neutrophils, lymphocytes, monocytes, eosinophil)

Serum biochemical parameters were analyzed using a semi-automated biochemical analyzer and commercially available reagent kits. The following parameters were estimated:

- Total protein (TP)
- Albumin
- Globulin
- Aspartate aminotransferase (AST)
- Alanine aminotransferase (ALT)
- Blood urea nitrogen (BUN)
- Creatinine
- Glucose

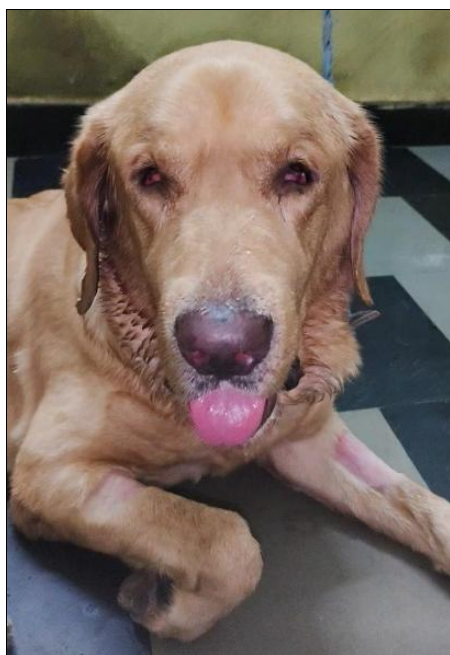


Fig 1: Prolapse of third eyelid



Fig 2: Frothy salivation while taking food

Table 1: Hematological Parameters of CD-Affected and Healthy Dogs (Mean $\pm$ SE)

Parameter	Affected Dogs (n=6)	Healthy Dogs (n=6)
Hemoglobin (g/dL)	10.2 $\pm$ 0.4	13.5 $\pm$ 0.3
PCV (%)	31.4 $\pm$ 1.2	41.7 $\pm$ 1.1
TEC ($\times 10^6/\mu\text{L}$)	4.6 $\pm$ 0.2	5.8 $\pm$ 0.3
TLC ($\times 10^3/\mu\text{L}$)	14.2 $\pm$ 0.8	9.1 $\pm$ 0.7
Neutrophils (%)	78.3 $\pm$ 2.1	65.1 $\pm$ 1.8
Lymphocytes (%)	17.4 $\pm$ 1.2	29.5 $\pm$ 2.0
Monocytes (%)	3.5 $\pm$ 0.3	3.1 $\pm$ 0.2
Eosinophils (%)	0.8 $\pm$ 0.2	2.3 $\pm$ 0.3

Table 2: Serum Biochemical Parameters of CD-Affected and Healthy Dog (Mean $\pm$ SE)

Parameter	Affected Dogs (n=6)	Healthy Dogs (n=6)
Total Protein (g/dL)	5.2 $\pm$ 0.3	6.8 $\pm$ 0.2
Albumin (g/dL)	2.1 $\pm$ 0.2	3.4 $\pm$ 0.2
Globulin (g/dL)	3.1 $\pm$ 0.2	3.4 $\pm$ 0.1
AST (U/L)	85.6 $\pm$ 6.4	45.3 $\pm$ 3.2
ALT (U/L)	76.4 $\pm$ 5.8	41.6 $\pm$ 2.5
BUN (mg/dL)	26.7 $\pm$ 2.3	18.1 $\pm$ 1.5
Creatinine (mg/dL)	1.9 $\pm$ 0.1	1.3 $\pm$ 0.1
Glucose (mg/dL)	68.4 $\pm$ 4.2	92.7 $\pm$ 3.6

Treatment Protocols

Affected animals were treated with following treatment regimen.

1. Intacef (Ceftriaxone + tazobactam) Given intramuscularly @ 20 mg/kg bwt once daily for one week to prevent or treat secondary bacterial infections.
2. Tab Pregamyl Forte 4mg (Pregabalin 75mg and methylcobalamin 1500mcg) – Provided orally once daily to enhance appetite, improve digestion, and provide nutritional support.
3. Melonex (Meloxicam) – Given intramuscularly at 0.2 mg/kg once daily to reduce inflammation and alleviate pain or discomfort.
4. Histanyl (Antihistamine) – Administered intramuscularly at 0.5 mg/kg once daily for three consecutive days to minimize histamine-mediated allergic and inflammatory responses.
5. Atropine Sulphate – Injected intramuscularly for one

week @ 0.04 mg/kg e to control hypersalivation

6. Neurokind Injection – Administered intramuscularly at 1 mL per animal once daily for seven days to support neurological health and nerve regeneration.

After one week of therapy, clinical improvement in appetite was observed, although mild neurological manifestations such as temporal twitching and intermittent salivation persisted. Then *Cicuta virosa* 30C homeopathic tablets were administered (five pills twice daily for one month) alongside continued Pregamyl Forte and Neurokind supplementation. Blood samples were collected from all animals (both affected and control groups) prior to treatment and again at the end of the therapy period. After one month, a follow-up PCR test for Canine Distemper was performed, yielding a negative result. All treated dogs exhibited complete clinical recovery, with resolution of neurological signs and restoration of normal behavior and appetite.

Results

Before treatment, all six affected dogs exhibited classical clinical signs of canine distemper, including inappetence, temporal muscle twitching, prolapse of the third eyelid (Fig. 1), and excessive frothy salivation (Fig. 2). Supportive therapy resulted in gradual improvement of appetite within one week; however, neurological manifestations persisted in all cases. After one month (Fig. 3) of administration of *Cicuta virosa* 30CC, all neurological symptoms resolved completely in the affected dogs. Post-treatment, the dogs regained normal appetite, activity levels, and behaviour. PCR testing for canine distemper performed after therapy yielded negative results, confirming recovery. Hematological analysis of affected dogs prior to treatment revealed leukopenia, lymphopenia, and mild anemia compared to healthy control dogs. Following recovery, hematological values returned to normal ranges, comparable to those of the healthy group. Biochemical evaluation before treatment showed elevated liver enzymes (ALT, AST) and reduced total protein and glucose levels in affected dogs relative to controls. After one month of therapy, biochemical parameters normalized, indicating restoration of hepatic function and metabolic status. Overall, the group treated with *Cicuta virosa* 30C demonstrated complete clinical and laboratory recovery, comparable to the healthy control group.

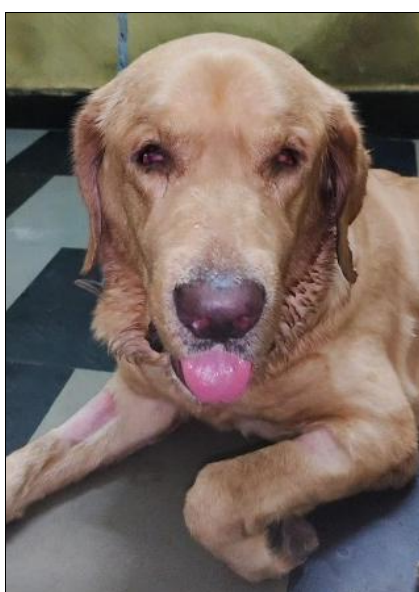


Fig 3: Prolapse of third eyelid



Fig 4: Frothy salivation while taking food

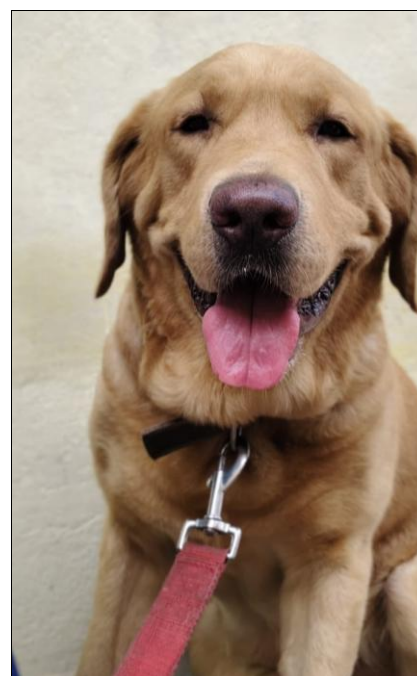


Fig 5: Recovery after 1 month

Discussion

Canine distemper in dogs is characterized by temporal

twitching, high temperature, in appetite, jaw champing, and hind limb paralysis. In our study and other researchers have reported similar clinical progressions of canine distemper in dogs (Martella *et al.*, 2008; Amude *et al.*, 2010) [7, 2]. The initial improvement in appetite and general health following supportive therapy aligns with the symptomatic management in CD, which aims to stabilize the animal while its immune system combats the virus. Persistent neurological signs after the acute phase of CD are well documented. These are believed to result from demyelination and neuronal degeneration caused by viral persistence in the central nervous system (Summers *et al.*, 1979) [8]. Age, immunity, viral strain and environmental impact of secondary pathogens determines the severity of Canine distemper infection.

Oral administration of homeopathic medicine *Cicuta virosa* (Drops. *Cicuta*-30 C) was followed in all affected dogs showed remarkable clinical recovery. In this case, gradual improvement of neurological signs after its administration suggests a potential supportive role in neuronal recovery.

The combination of conventional supportive care with complementary therapies may offer a more comprehensive approach for managing chronic CD cases, particularly those with neurological involvement.

The negative PCR result following treatment indicates viral clearance and supports the clinical recovery observed.

Conclusion

This study demonstrates that *Cicuta virosa* 30C, when administered alongside conventional supportive therapy, may contribute to the improvement of neurological symptoms associated with Canine Distemper infection in dogs. The infected animals treated with *Cicuta virosa* 30C, revealed notable variations in hematological and biochemical parameters. Treated groups showed a normalization of key hematological indices, including hemoglobin concentration, total leukocyte count, and differential leukocyte profiles, compared to untreated infected controls. Similarly, biochemical parameters such as liver and kidney function markers (ALT, AST, ALP, creatinine, and BUN) indicated improved organ function in *Cicuta virosa* treated animals. When compared with healthy animals, the *Cicuta virosa*-treated group demonstrated partial restoration of physiological parameters toward normal reference ranges, suggesting a potential modulatory role of the remedy in mitigating systemic effects of CDV infection.

Conflict of Interest

Not available

Financial Support

Not available

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How to Cite This Article

Kumar VVVA, Sirisha K. Diagnostic and therapeutic management of canine distemper. *International Journal of Veterinary Sciences and Animal Husbandry*. 2025; 10(12): 253-256.

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