

Case Report

Clinical Aspects of Canine Distemper in 1.5 Year Old Labrador retriever

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ABSTRACT

A 1.5 year old Labrador retriever bitch was presented with the history of anorexia, depression, vomiting, diarrhea and involuntary movement of jaw. On clinical examination the bitch, it was found to have a fever 104°F, heart beat (140 bpm) with no cardiac arrhythmia, respiration 54 bpm, CRT < 3 second. Scanty nasal discharge and serous ocular discharge with blepharitis and cloudiness of eyes, distemper myoclonus and hyper keratinization of foot pad was also found. On the basis of clinical signs and lymphopenia shown in blood report, the disease was diagnosed as canine distemper. The treatment of the animal was done symptomatically and prophylactically. Treatment was found to be effective and animal was recovered up to some extent after 15 days; however, nervous signs were still there.

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Canine distemper is an infectious and contagious disease of carnivores caused by morbillivirus. The ssRNA virus belongs to family paramyxoviridae and cause disease even in vaccinated animals (Appel, 1987; Lan et al., 2005). The virus is closely related to PPR, Rinderpest and Measles virus (Beineke et al. 2009) and results in severe systemic and neurologic signs (Appel et al., 1995).

Canine distemper virus (CDV) spreads through oro–nasal secretion (Krakowka et al., 1980) and enters respiratory tract by inhalation. The monocytes and macrophages reach there and engulf this virus and cause further propagation (Appel, 1970) followed by the spread of virus in lymphatics and blood. The resultant viremia is responsible for biphasic fever and the signs of lethargy, anorexia, and dehydration were observed (Wright et al., 1974). Diagnostic techniques use for the detection of the disease includes ELISA, fluorescent antibody techniques, neutralization (SN) assays and real time–polymerase (RT–PCR) chain reaction (Elia et al., 2006). Vaccination at regular intervals is a basic tool to avoid the disease (Moritz et al., 2000).

A study in Madras, India showed that males were more affected by this disease than females in (60:40) (Alex et al., 1994). In Pakistan, 11% prevalence of CDV has been seen on clinical basis (Shabbir et al., 2010). 63 % mortality is seen in less than 1.5 year of age in distemper encephalitis cases (Swango, 1989).

A 1.5 year old Labrador bitch of approx. 19 kg weight was presented at Clinical Medicine and Surgery department (CMS), Faculty of Veterinary Sciences (FVS), University of Agriculture, Faisalabad (UAF) with history of diarrhea from last night, vomiting anorexia, depressed behavior, ocular and scanty nasal discharge (Figure 1b and c) and involuntary movement of jaw (Figure 2) were observed since last 2 days. Bitch was parturated 1 month before and gave birth to 5 pups. By conversation with owner, it was known that animal faced an accident with

motor–bike a week ago without any serious injury. No vaccination or deworming was done.

On distant examination, animal behavior was found quiet and depressed. No visible change was seen, except occasionally chewing gum fits (Figure 2). Clinical examination of the animal showed high temperature (104°F), increased heart beat (140 bpm) without any arrhythmia, increased respiration (54 bpm) and slight increase in capillary refill time (CRT< 3 second). Along with ocular discharge, blephritis and cloudiness of eyes (Figure 1b) was observed. The foot pad of the animal was found to be hard because of hyper keratinization (Figure 2d). While blood chemistry analysis, complete blood count showed lymphopenia.

On the basis of clinical signs and symptoms especially distemper myoclonus and hardening of foot pad due to hyper keratinization the disease was diagnosed as Canine distemper. The complete blood count revealed lymphopenia, decrease in mean value of Packed cell volume (PCV) 30%, total erythrocytic count (TEC) $4.4 \times 10^6/\mu\text{L}$ and Hemoglobin (Hb) 10.2 g/dL may be due to effect on hematopoietic system by CDV and increase in mean values of Mean corpuscular volume (MCV) 80.7f L, Mean corpuscular hemoglobin (MCH) 30.2 pg and mean corpuscular hemoglobin concentration (MCHC) 39.3 g/dL due to leukemia which further support the diagnosis (Zafar et al., 1999). Other method of diagnosis include ELISA, fluorescent antibody test (FAT) and sero–neutralization (SN) assays but got little importance due to development of antibody titer to CDV by previous use of vaccines (Elia et al., 2006). Confirmation of the disease could be done through isolation of the virus from the clinical samples is highly fastidious and time consuming while detection of the specific gene sequences through polymerase chain reactions more rapid, sensitive and specific method for diagnosis of the disease (Elia et al., 2006).



There is no specific anti-viral drug for this disease so symptomatic treatment was done (<http://dogtime.com/distemper-in-dogs-vin.html>). The treatment included anti-pyretic (inj. ketoprofen @ 2mg/kg) to reduce the fever, 3rd generation broad spectrum anti-biotic (inj. Ceftriaxone sodium @ 25mg/kg b.w) to avoid secondary bacterial infection. To check diarrhea and dehydration inj. Metronidazole @ 10mg/kg b.w and inf. ringer lactate @ 15ml/kg b.w was given respectively. From 2nd day to onward, with fluid therapy Ketoprofen syrup @ 2mg/kg b.w once a day, Cephalixin (Keflex syrup) @15mg/kg 2 times a day and gravinat syrup @ 3mg/kg b.w after feeding was recommended. A multi-vitamin powder (Bendoz powder) was added in feed @ 10g/day to support the animal. Same treatment was repeated for 3rd, 4th and 5th days. Vita A also play an important role for the treatment of canine distemper but mechanism is still unknown (Rodeheffer et al., 2007).

Morbillivirus of family Paramyxoviridae is the causative agent of an infectious disease of carnivore known as Canine distemper can also effect the vaccinated animals. At age of 6-8 month the disease most likely to occur depends on breed of animal (Chappuis, 1995). Not only canine the members of family Mustelidae and Procyonidae also susceptible (Appel, 1987). Most of times the recently weaned pups get this infection because at this stage the maternal immunity that is coming from milk are at its lowest level (Shabbir et al., 2010). Therefore it is recommended to vaccinate the dogs at 3 month of age. Depending on the age and immune status of host incubation period varies between 1-4 weeks of different strains. About

50% mortality is seen in this disease without clinical signs to severe clinical signs (Appel, 1970, 1987; Krakowka et al., 1980; Moritz et al., 2000). As cold environment favors virus so the disease mostly occur in winter season. High mortality is seen is 63 % in under 1.5 of age in distemper encephalitis cases (Swango, 1989). All body secretions or excretion will contain virus in acute cases. In this case fever, cutaneous rashes, anorexia, diarrhea blisters on the abdominal region ocular and nasal discharge which is serous in nature along with conjunctivitis, blephritis and cloudiness of eye most visible. Gastrointestinal signs become complicated when secondary infection occur. Progressive neurological signs are seen (Greene and Appel, 1998). Myoclonus, ataxia, plegia, and nystagmus are includes in nervous signs (Amude et al., 2007).

CDV main targets are mucous membranes and lymphoid tissue (Appel, 1987). This virus enter in body by air by URT and there in lymph nodes it primarily replicate and causes immunosuppression then spread to epithelium and CNS at about 10 days after transmission (Krakowka et al., 1980). Mainly cause lymphopenia in initial stages. When it reaches to lower respiratory tract (LRT), gastrointestinal tract and CNS cause lesions to be formed on these organs and responsible for appearance of systemic, cutaneous and nervous signs (Greene and Appel, 1998). Out of all CNS inflammation 15% of deaths of dogs caused by encephalomyelitis produce in Canine distemper (Appel and Summers, 1995). CDV produce encephalitic lesions and multifocal demyelination in CNS instead of inflammatory changes cause death of dogs after passing of systemic phase (Beineke et al. 2009).



Figure 2: Involuntary movement of jaw (Distemper myoclonus)

Presence of distemper myoclonus (Figure 2) and hyperkeratinization (Figure 1d) and lymphopenia in blood report confirm the disease as *Canine Distemper*. Because myoclonus and keratinization of foot pad are considered as the pathognomonic signs of this disease (<http://www.2ndchance.info/dogdistemper.htm>).

Animal can recover if promote the production of antibodies against the virus. But in footpads and lymphatic cells, this virus can persist and cause hyperkeratinization (Appel, 1970, 1987; Greene and Appel, 1998) Due to this reason this disease is also called as *hard pad disease*.

At this time the only thing that we can do to prevent this disease is vaccination. By vaccination at regular intervals will help us a lot to control this disease until the specific treatment is not discover.

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REFERENCE

Alex PC and Dhanapalan P (1994). Distemper encephalitis in dogs: incidence, symptomology and electroencephalographic findings. *J. Vet. Anim. Sci.* 25: 127– 31.

Amude AM, Alfieri AA and Alfieri AF (2007). Clinicopathological findings in dogs with distemper encephalomyelitis presented without characteristic signs of the disease. *Res. Vet. Sci.* 82: 416– 422.

Appel MJ (1970). Distemper pathogenesis in dogs. *J. Am. Vet. Med. Assoc.* 156: 1681– 1684.

Appel MJ (1987). Canine distemper virus. In: Horzinek, M.C.M. (Ed.), *Virus Infection of Vertebrates*, vol. 1. Elsevier Science Publisher B.V., Amsterdam, Oxford, New York, Tokyo, 133– 159.

Appel MJG and Summers BA (1995). Pathogenicity of morbilliviruses for terrestrial carnivores. *Vet. Microbiol.* 44: 187– 191.

Beineke A, Puff C, Seehusen F and Baumgartner (2009). Pathogenesis and immunopathology of systemic and nervous canine distemper. *Vet. Immunol. Immunopathol.* 127: 1– 18.

Chappuis G (1995). Control of canine distemper. *Vet. Microbiol.* 44(2/4): 351– 358.

Elia G, Decaro N, Martella V, Cirone F, Lucente MS, Lorusso E, Trani LD and Buonavoglia C (2006) Detection of canine distemper virus in dogs by real-time RT-PCR. *J. Virol. Meth.* 136: 171– 176.

Greene CE and Appel MJ (1998). Canine distemper. In: *Infectious diseases of dogs and cats*. WB Saunders Company, Philadelphia, London, Toronto, Montreal, Sydney, Tokyo. pp. 9– 22.

Krakowka S, Higgins RJ and Koestner A (1980). Canine distemper virus: review of structural and functional modulations in lymphoid tissue. *Am. J. Vet. Res.* 41: 284– 292.

Lan NT, Yamaguchi R, Unchida K, Sugano S and Tateyama (2005). Growth profiles of recent canine distemper isolates on Vero cells expressing canine signaling lymphocyte activation molecule (SLAM). *J. Comp. Pathol.* 133: 77– 81.

Mortiz A, Frisk A and Baumgartner W (2000). The evaluation of diagnostic procedures for the detection of canine distemper virus infection. *Eur. J. Comp. Anim. Pract.* 10: 37– 47.

Swango LJ (1989). Canine viral diseases. In: *Textbook of veterinary internal medicine*. 3rd ed. Philadelphia: W.B Saunders Company, Philadelphia, London, Toronto, Montreal, Sydney, Tokyo. pp. 301– 303.

Shabbir MZ, Arfanahmad M, Aliahmed, Muhammad K, Anwar I, 2010. Comparative evaluation of clinical samples from naturally infected dogs for early detection of canine distemper virus. *Turk. J. Vet. Anim. Sci.* 34(6): 547– 552.

Wright NG, Cornwell AJ, Thompson H and Lauder IM (1974). Canine distemper: current concepts in laboratory and clinical diagnosis. *Vet. Rec.* 94: 86– 92.

Zafar M. S., Khan S. A. and Rabbani A., 1999. Hematological studies and estimation of electrolytes in dogs. *P. Vet. J.* 19(1): 35– 39.

Rodeheffer C, Messling V, Milot S, Lepine F, Manges AR, Ward BJ (2007). Disease manifestations of canine distemper virus infection in ferrets are modulated by vitamin A status. *J. Nutrition* 137 (8): 1916– 22.