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Electrolyte, blood gas and acid-base imbalance in canine chronic renal disease in dogs

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Abstract

Chronic kidney disease (CKD) in dogs is a progressive and often irreversible condition where the kidneys gradually lose their ability to filter waste, regulate fluid balance and maintain essential electrolyte levels. Acid-base disturbances are commonly identified in critically ill veterinary patients. So, the present investigation was aimed to study blood gas, acid base and electrolyte alteration in canine chronic kidney disease. Study was conducted on 20 dogs suffering from stage IV chronic kidney disease out of which 10 cases were found to be positive for haemoprotzoan infection through microscopic examination or rapid diagnostic kits. Haematologically mean values of TEC, PCV and Platelets were significantly reduced. Biochemically the mean values of Creatinine, SGPT and SGOT were significantly increased. Mean values of HCO₃, pCO₂, BE and BB were significantly decreased as compared to healthy dogs on venous blood gas and acid-base analysis. Electrolyte and mineral estimation revealed significant decrease in the values of sodium, chloride, calcium and magnesium whereas phosphorus were increased. The assessment of blood gas, acid-base balance, electrolyte levels and timely presentation of clinical cases played a crucial role in managing canine chronic kidney disease.

Keywords: Chronic kidney disease, Acid-base, Electrolytes

Introduction

Electrolyte and acid-base balance are essential components of physiological homeostasis in dogs, influencing vital processes such as neuromuscular function, fluid regulation, and cellular metabolism. Acid-base disturbances in dogs can occur due to a variety of conditions, including renal insufficiency, diabetic ketoacidosis (DKA), prolonged vomiting or diarrhoea, shock, and respiratory diseases that impair gas exchange (Tvedten *et al.*, 2016). The present study describes on electrolyte, blood gas and acid-base imbalances in dogs with chronic kidney disease.

Materials and Methods

The present study was carried out on dogs presented during the period of February, 2024 to August, 2025 in the Department of Veterinary Medicine, Veterinary Clinical Complex, College of Veterinary

and Animal Sciences, CSKHPKV Palampur (H.P). Preliminary screening was done on the basis of patient's history and presenting clinical signs as depression, inappetence, dehydration, pyrexia, enlarged lymph nodes, vomiting, diarrhoea, polydipsia, polyuria, halitosis, dental tartar and seizures. In addition to haemato-biochemical estimation, electrolyte, mineral analysis, blood gas and acid-base analysis were performed. Blood smear examination (Fig. 1) and serological detection kits (Fig. 2) were used for the detection of haemoprotzoan infection. Dogs in stage IV of chronic kidney disease as per IRIS staging were selected. On this basis, a total of 20 dogs suffering from stage IV CKD were included in the present study. 10 apparently healthy dogs independent of sex and breed presented for routine health check-ups and regular vaccination were considered as healthy control group.

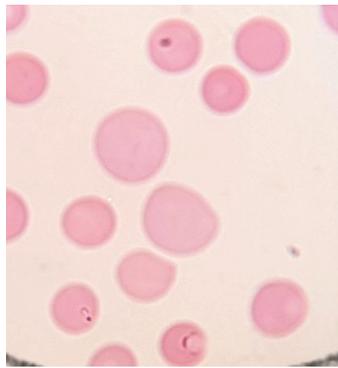


Fig. 1: Babesia gibsoni observed in a Giemsa-stained peripheral blood smear

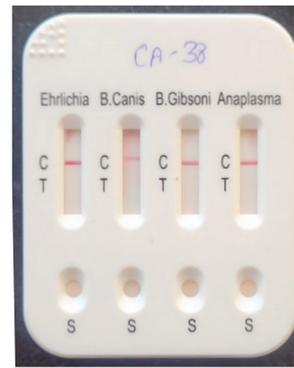


Fig. 2: Qualitative immunochromatographic assay kit

Results and Discussion

Out of 20 dogs in stage IV of chronic kidney disease, 10 were found to be positive for haemoprotozoan infection representing 50% of the cases. Most common clinical signs observed were depression, dehydration, inappetence, vomiting and pale mucous membrane. These findings were in accordance with Nakang *et al.* (2019) and Kumar *et al.* (2020), The mean values of rectal temperature, heart rate and respiration rate varied non significantly as compared to healthy animals. The mean values of haematological parameters of dog with stage IV CKD are presented above in Table 1.

Table 1: Mean values of haematological parameters in dogs with stage IV chronic kidney disease

Parameters	Healthy Control (n=10)	CKD (n=20)
Hb (g/dl)	13.47±0.34	10.93±0.92
PCV (%)	38.87±1.04	28.5±2.3**
TEC (×10 ¹² /L)	6.49±0.21	4.45±0.38 **
TLC (10 ⁹ /L)	11.13±0.6	19.71±4.13
N (%)	79.68±0.85	80.747±2.82
L (%)	15.3±0.62	10.221±2.127
M (%)	4.04±0.27	7.463±0.818**
E (%)	1.35±0.18	1.205±0.2686
MCV (fl)	61.57±0.47	64.68±1.42
MCH (pg)	21.88±0.4	24.69±0.35***
MCHC (g/dl)	35.18±0.58	252.41±41.5**
Platelets (10 ⁹ /L)	299.2±23.72	121.88±15.58***

* Significant at 5% (P<0.05); ** Significant at 1% (P<0.01); ***Significant at 0.1% (P<0.001)

The mean values of PCV, TEC and platelets were significantly decreased whereas mean values of MCH and MCHC were significantly increased. These findings are in accordance with Sharma *et al.* (2015), Devpriya *et al.* (2018) and Eashwar *et al.* (2024). Anaemia in canine chronic kidney disease is multifactorial and associated with decreased erythroid precursor cells,

gastrointestinal bleeding, and systemic inflammation as noted by Crivellenti *et al.* (2023). Decreased platelet count was also reported by Sharma *et al.* (2015) which can be attributed to the reduced thrombopoietic activity in uremic dogs. The mean values of biochemical parameters in dogs with stage IV CKD are presented above in Table 2

Table 2: Mean values of biochemical parameters in dogs with Stage IV Chronic Kidney Disease

Parameters	Healthy (n=10)	CKD (n=20)
Glucose (mg/dl)	102.67±2.66	126.152±10.498
Bilirubin (mg/dl)	0.22±0.04	1.62±1.215
AST (U/L)	38.05±3.98	86.06±15.04*
ALT (U/L)	33.92±2.4	88.35±11.402**
ALP (U/L)	79.25±8.72	179.43±51.606
TP (g/dl)	6.6±0.35	6.632±1.167
Creatinine (mg/dl)	1.01±0.08	15.08±1.48***
BUN (mg/dl)	25.14±4.54	515.31±174.18

* Significant at 5% (P<0.05); ** Significant at 1% (P<0.01); *** Significant at 0.1% (P<0.001)

The mean values of AST, ALT and creatinine were significantly increased as compared to healthy animals. These findings were in accordance with Bradea *et al* (2013), Puri *et al.* (2015), and Devpriya *et al.* (2018). Reason for increased creatinine was its diminished renal excretion in CRF and at least 75% loss of functional nephrons as mentioned by Lefebvre

(2011). Elevations in liver enzymes like ALT and AST are not exclusive to primary liver diseases. They can also occur in extrahepatic conditions including circulatory disturbances and systemic illnesses, which are common in advanced CKD as stated by Alvarez *et al.* (2009). The mean values of electrolyte estimation in dogs with stage IV CKD are presented above in Table 3.

Table 3: Mean values of electrolyte and mineral estimation in dogs with stage IV chronic kidney disease

Parameters	Healthy (n=10)	CKD (n=20)
Sodium (mmol/L)	151.8±1.71	141.3±2.8*
Potassium (mmol/L)	4.7±0.16	5.09±0.4
Chloride (mmol/L)	110.05±1.85	102.96±1.89*
Calcium (mg/dl)	10.56±0.27	9.737±0.219*
Phosphorus (mg/dl)	5.07±0.12	6.774±0.478*
Magnesium (mg/dl)	1.78±0.07	1.39±0.084**

* Significant at 5% (P<0.05); Significant at 1% (P<0.01); ***Significant at 0.1% (P<0.001)

The mean values of electrolytes in dogs with stage IV CKD showed a significant decrease in the values of sodium, chloride, calcium and magnesium whereas the mean values of phosphorus was increased as compared to healthy dogs. These findings are in accordance with Kandula and Karalpudi (2015), Puri *et al.* (2015) and Sumit *et al.* (2018). Suggestive reason for hyperphosphatemia was declining kidney function because the kidneys are the primary route of phosphorus excretion and reduced kidney function results in phosphorus retention (Sonu *et al.*, 2019). Sakaguchi (2022) stated that tubular dysfunction or interstitial fibrosis may play a pivotal role in the development

of hypomagnesemia in CKD, which impairs tubular magnesium reabsorption. Hyponatremia observed could be due to hypotension, pain, and renal injury which activates sympathetic nervous system, renin-angiotensin-aldosterone system and antidiuretic hormone release (Prowle *et al.*, 2010) leading to increased absorption of sodium and water. Hypocalcaemia may be due to increase in GFR leading to abrupt increase in serum phosphorus concentration, causing a decrease calcium concentration due to chelation of phosphorus. This was in agreement with Schenck *et al.* (2005). The mean values of blood gas and acid-base analysis in dogs with stage IV CKD are presented above in Table 4.

Table 4: Mean values of blood gas and acid-base analysis in dogs with stage IV chronic kidney disease

Parameters	Healthy (n=6)	CKD (n=20)
pH (mmHg)	7.35± 0.02	7.2705±0.025
pCO ₂ (mmol/L)	38.15± 2.02	27±0.202***
HCO ₃ (mmol/L)	20.16± 0.52	11.484±0.771***
AnGap (mmol/L)	30.26± 1.25	30.266±2.278
tCO ₂ (mmol/L)	20.8± 0.65	12.273±0.785***
BE (mmol/L)	-6.08± 0.84	-12.773±1.189**
BEact (mmol/L)	-6.26± 1.12	-14.173±1.201**
BEecf (mmol/L)	-5.98± 1.34	-13.805±1.037***
BB (mmol/L)	42.92± 0.46	33.184±1.309***
stHCO ₃ (mmol/L)	20.16± 0.42	13.926±0.8522***
st pH	7.34± 0.01	7.1507±0.027**
cH ⁺ (nmol/L)	49.63± 0.81	55.015±3.364

* Significant at 5% (P<0.05); ** Significant at 1% (P<0.01); ***Significant at 0.1% (P<0.001)

The mean values of HCO₃, pCO₂, BE and BB were significantly decreased as compared to healthy dogs. These findings were in accordance with Koenhemi and Gonul (2019) and Lippi *et al.* (2023). The decreased HCO₃ concentration in CKD patients possibly was due to reduced reabsorption of filtered bicarbonate as number of functioning renal tubules become limited as CKD progresses (Buduk *et al.*, 2020). In CKD a common finding is metabolic acidosis which stimulates compensatory hyperventilation leading to a decrease in arterial pCO₂. BE reflects the amount of excess or insufficient level of bicarbonate in the system and BB refers to the total concentration of buffering anions in the blood, mainly HCO₃ along with proteins, phosphates, and haemoglobin. Altered blood gas and electrolyte changes were also associated with the severity of the disease and loss of kidney function. The assessment of blood gas, acid-base balance, electrolyte levels and timely presentation of clinical cases played a crucial role in managing canine chronic kidney disease.

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References

Alvarez, L. and Whittemore, J. 2009. Liver enzyme elevations in dogs: physiology and pathophysiology. *Compend. Contin. Educ. Vet.*, **31(9)**: 408–410, 412–413.

Crivellenti, S., Crivellenti, L.Z., Gilor, C. 2023. Anaemia in canine chronic kidney disease is multifactorial and associated with decreased erythroid precursor cells, gastrointestinal bleeding, and systemic inflammation. *Am. J. Vet. Res.*, **84(10)**: 1–6.

Bradea, A., Codreanu, M., Vlagioiu, C. and Simion, V. 2013. Hematologic aspects in chronic kidney disease (CKD) in dogs. *Bull. Univ. Agric. Sci. Vet. Med. Cluj-Napoca Vet. Med.*, **70(2)**: 191–194.

Buduk, S.Y., Sinha, R., Bera, A.K. and Suresh, R.V. 2019. Electrolyte and acid-base alterations in dogs with renal dysfunction. *Int. J. Sci. Environ. Technol.* **8(1)**: 10–15.

Carrero, J.J., Johansen, K.L., Lindholm, B., Stenvinkel, P., Cuppari, L. and Avesani, C.M. 2016. Screening for muscle wasting and dysfunction in patients with chronic kidney disease. *Kidney Int.*, **90(1)**: 53–66.

Devipriya, K., Lavanya, C., Selvaraj, P. and Napoleon, R.E. 2018. Early diagnosis of renal insufficiency in dogs with haemato-biochemical findings. *J. Entomol. Zool. Stud.*, **6(5)**: 703–705.

Eashwar, A., Ashwinii, R., Sagare, R., Krishna, C. J., Senthil, N. R. and Tejashree, M. 2024. Haemato-biochemical alterations in dogs suffering from chronic renal failure. *Int. J. Vet. Sci. Anim. Husbandry*, **SP-9(4)**: 01–03.

Kandula, S. and Karlapudi, S. K. 2015. Prevalence of renal disorders in dogs – a clinical study. *Int. J. Agric. Sci. Vet. Med.*, **2(3)**: 146–148.

- Koenhemi, L. and Gonul, R. 2019. Determination of renal blood flow with Doppler ultrasound and the hypertension prevalence and acid-base level in dogs with chronic renal failure. *J. Istanbul Vet. Sci.*, **3**: 6–12.
- Kumar, C., Kamran, A. and Isloor, S. 2020. Clinical signs observed in different stages of chronic kidney disease in dogs. *Int. J. Livest. Res.*, **10**: 249–252.
- Lefebvre, H. 2011. Renal function testing. In: *Nephrology and Urology of Small Animals*, Bartges, J. and Polzin, D. (eds.) Wiley-Blackwell, Ames, IA, USA, pp. 91–96.
- Lippi, I., *et al.* 2023. Serum bicarbonate deficiency in dogs with acute and chronic kidney disease. *Vet. Sci.*, **10(5)**: 363.
- Nakang, H., Changkija, B., Baishya, B.C., Mahato, G., Devi, P. and Kalita, M. 2019. Clinical and haemato-biochemical alterations in canine renal dysfunction. *International Journal of Livestock Research*, **9(8)**: 164–171.
- Prowle, J.R., Echeverri, J.E., Ligabo, E.V., Ronco, C. and Bellomo, R. 2010. Fluid balance and acute kidney injury. *Nat. Rev. Nephrol.*, **6(2)**: 107–115.
- Puri, D., Dua, K., Sood, N.K., Randhawa, S. and Dhaliwal, P.S. 2015. Study of renal dysfunctions in geriatric dogs. *Vet. Pract.*, **16(1)**: 44–46.
- Sakaguchi, Y. (2022). The emerging role of magnesium in CKD. *Clinical and Experimental Nephrology*, **26(5)**: 379–384.
- Schenck, P.A. and Chew, D.J. 2005. Calcium, phosphorus, parathyroid hormone, and renal secondary hyperparathyroidism. *Am. J. Vet. Res.*, **66(8)**: 1330–1336.
- Sharma, A., Ahuja, A., Srivastava, M. and Kachhawa, J.P. 2015. Haemato-biochemical changes in dogs suffering from chronic renal failure. *Indian J. Canine Pract.*, **7(2)**: 102–107.
- Sonu, A.K., Charaya, G., Bangar, Y., Agnihotri, D. and Kumar, T. (2019). Haemato-biochemical alterations in dogs suffering from chronic renal failure. *Indian J. Vet. Med.*, **39(1)**: 31–35.
- Sumit, Goel, P., Kumar, P., Gulia, D., Jhambh, R., Sindhu, N. and Chaudhary, R. N. 2018. Haematobiochemical and serum electrolytes alteration in dogs with chronic kidney disease. *Pharma Innov. J.*, **7(11)**: 302–306.
- Tvedten, H. and Willard, M.D. 2016. Laboratory diagnosis of renal disorders. In: *Small Animal Clinical Diagnosis by Laboratory Methods*. V edn., Elsevier. pp.126-130.

Comparative evaluation of aqueous leaf extract and dried latex powder of *Carica papaya* Linn. for their antidiabetic and antihyperlipidemic activities in Wistar rats

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Abstract

The present study was undertaken to evaluate and compare the effect of dried latex powder (DLPCP) and aqueous leaf extract (ALECP) of *Carica papaya* Linn. in streptozotocin induced diabetic rat model for a period of 21 days for their antidiabetic and antihyperlipidemic properties. A total of 32 male Wistar rats, divided into four groups with eight animals in each group. The groups include normal control (Group-I), diabetic control (Group-II), diabetic rats treated with DLPCP @ 400mg/Kg b.wt (Group-III) and diabetic rats treated with ALECP @ 400 mg/Kg b.wt (Group-IV). Serum was collected on 0th, 4th, 14th and 21st days from all the experimental rats for serum biochemical analysis. On 21st day, the animals were sacrificed for histopathological examination of pancreas and liver. ALECP treatment group has shown antihyperglycemic activity as early as the 4th day (327.7 + 18.65) and hepatoprotective activity on the 4th day (252.9 + 4.6, 155.3 + 4.8), whereas, DLPCP treatment group has shown the similar affect on 21st day (311.7 + 28.83) and on 14th day (228.8 + 10.6, 179.8 + 0.9) of experiment respectively. Antihyperlipidemic affect was exhibited by both the treatment groups with reduced triglycerides, total cholesterol, LDL, VLDL and increased HDL levels, when compared to diabetic control groups. Histopathological examination has endorsed the biochemical findings. ALECP treatment when compared to DLPCP treatment has shown a better antidiabetic activity on the early days of experiment by ameliorating its associated affects. The present study indicate that ALECP acts as an alternative remedy for diabetes milletus.

Keywords: *Carica papaya*- Antidiabetic property- Antihyperlipidemic effect- Aqueous leaf extract- Dried latex powder- Histopathology.

Introduction

Diabetes mellitus is a multifaceted metabolic disorder involving chronic hyperglycemia with associated disturbances in carbohydrate, protein and lipid metabolism due to inadequate insulin secretion or poor sensitivity of target tissues to the metabolic effect of insulin (Guyton and Hall 2006). The incidence of diabetes mellitus (DM) in animals particularly in non-ruminants is on par with humans. In particular, female dogs and male cats are among the most affected with less observed in bovines, equines, pigs, sheep and birds species (Kaoud 2017). Genetics, age, obesity and environmental factors influence the incidence of DM in feline and canine species.

DM is broadly categorised as two types, type I diabetes is also called as insulin dependent diabetes

mellitus (IDDM) is caused by insufficiency of insulin secretion. Dogs, commonly get affected with type I diabetes, breeds like Samoyed, Australian terrier, Keeshond, and Cairn terriers are genetically predisposed (Nelson and Reusch 2014). In developing countries like India, rearing dogs as companion animals is rising gradually, accompanying rapid increase in dogs succumbing to type I diabetes, which makes type I DM a major concern to animal health practice. Type II diabetes is also called as non-insulin dependent diabetes mellitus (NIDDM), which is characterized by the presence of insulin resistance with an inadequate compensatory increase in insulin secretion. High carbohydrate diets in cats will increase the blood glucose and insulin levels, which may predispose them to obesity associated type II diabetes, the commonly observed type in cats (Rand et al. 2004). The risk for development of diabetes increases about 2-fold in overweight cats and about

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4-fold in obese cats (Laflamme 2012). The therapeutic management of DM with minimal side effects either in humans or in animals remains a clinical challenge.. There is an obligatory need in finding alternative drugs of plant origin for DM as they are cheaper, least toxic and with fewer side effects (Nissen and Wolski 2007). A herbal remedy which is having antidiabetic and antihyperlipidemic properties will address the this disease comprehensively.

Carica papaya Linn, commonly known as 'papaya plant', the products of this plant are commonly available and vastly consumed across the country. Evaluating and establishing the antidiabetic properties of this plant shall make it an alternate choice for existing synthetic drugs. In this perspective, numerous scientific investigations have been conducted to evaluate the antidiabetic activities of various parts of *C. papaya*. *Carica papaya* leaves (Rojop et al. 2012; Airaodion et al. 2019), seeds (Venkateshwarlu et al. 2013; Ebenezer et al. 2019), the unripe pulp of fruit (Ezekwe et al. 2014) and root extract (Nimenibo-Uadia and Nwachukwu., 2020) has been reported to be effective in ameliorating the damage caused by diabetes in experimental rats. These studies has revealed that different parts and extracts of *Carica papaya* is having moderate to good antidiabetic and antihyperlipidemic activity, of which aqueous leaf extract is one of the best to have such activity (Maniyar and Bhixavatimath 2011). However, certain components of the plant like latex derived from unripe mature fruit of *Carica papaya*, which is hypothesised to exhibit antidiabetic properties (Ajani and Ogunbiyi 2015) has not been tested comprehensively. With this background the present study was taken up to study and compare the antidiabetic and antihyperlipidemic effects of dried latex powder of *Carica papaya* Linn (DLPCP) and aqueous leaf extract of *Carica papaya* Linn (ALECP).

Materials and methods

The leaves of *C. papaya* plant were collected from the local premises (Tirupati). The leaves of the plant were subjected to surface sterilization using 30% alcohol, and then dried in shade. The dried leaves were subjected to size reduction to a coarse powder by using dry grinder and passed through sieve. The powdered sample (400 g) was boiled in hot water for 30 min after which it was filtered using a piece of white cotton gauze. The filtrate was evaporated to dry at 40°C producing brown color solid residue (yield: 15% w/w). The residue was weighed and stored in an air and water proof containers at 4°C. From this stock, fresh preparation was

made whenever required for the experimental study.

Papaya plant contains specialised cells (laticifers) dispersed throughout the plant tissues that secrete latex. Latex is a thixotropic fluid, with a milky appearance that contains about 85% water. Latex was collected in the early hours of a day as the flow of latex is low as day progresses. Collection was done by making 1-2 mm deep incisions on the skin of unripe mature fruit. The collected latex is passed through 50 mesh sieve to remove dirt. The latex was then dried at room temperature till it became crumbly and non-sticky. The dried latex was triturated using a mortar and pestle, and the same is stored in air tight containers at 4°C.

A pilot study using dried latex of three doses viz 100 mg/Kg b.wt, 200 mg/Kg b.wt and 400mg/Kg b.wt was conducted initially on diabetic rats for antidiabetic dose selection. Nine streptozotocin induced diabetic rats were randomly divided into 3 groups. DLPCP was administered to group D1, D2 and D3, daily for 7 days @ 100mg/Kg b.wt., 200mg/Kg b.wt and 400mg/Kg b.wt. respectively. The selected three doses are lesser than or equal to 1/10th value of the proposed LD50 (4000mg/Kg) of dried latex of *Carica papaya* (Kumar et al. 2018). From the results obtained, the proven best dose was selected for the present study.

The present study was carried out in the Department of Veterinary Biochemistry, College of Veterinary Science, Sri Venkateswara Veterinary University (SVVU), Tirupati, Andhra Pradesh. The experimental procedures were approved by the Institutional Animal Ethics Committee (281/go/ReBi/S/2000/CPCSEA/CVSc/TPTY/011) of College of Veterinary Science, SVVU, Tirupati.

The experimental rats selected for induction of diabetes were fasted overnight before the administration of streptozotocin (STZ). The experimental induction of diabetes in rats was carried out by a single intraperitoneal injection of STZ at the rate of 50mg/Kg b.wt. The induction of diabetes was done for the animals in all the experimental groups except for the animals in normal group to which normal saline was injected..

From the total 32 male Wistar rats weighing 150-200 g, 24 rats were injected with STZ (50 mg/Kg b.wt) through intraperitoneal route, while 8 rats were injected with normal saline. The group of animals which received normal saline served as group I (Normal Control). After 72 hours of STZ administration, the blood glucose levels of the rats were estimated and the

rats with blood glucose levels >200 mg/dl were treated as diabetic animals. Those animals with diabetes (blood glucose levels >200 mg/dl) were distributed into three groups Group II, Group III (DLPCP treatment) and Group IV (ALECP treatment). All the groups were fed on basal diet *ad libitum*.

Blood samples were collected from all the experimental rats on 0th day, 4th day, 14th day and 21st day of the experiment. The collected serum was stored at -20° C for further analysis. Serum biochemical parameters such as, glucose, SGOT, SGPT, triglycerides, total cholesterol and HDL were estimated in automatic analyser (BioSystems A15 analyser), VLDL and LDL levels are calculated based on levels of the triglycerides, total cholesterol and HDL cholesterol.

The rats were sacrificed by cervical dislocation after the routine blood collection on the 21st day of the experiment under deep anaesthesia. The tissues liver and pancreas were collected and preserved till further processing. The liver and pancreas tissues collected were processed and stained with Haematoxylin and Eosin (H & E) stains, slides were observed under light microscope

The group wise and day wise comparison of the biochemical parameters was done statistically by analysing the data with two-way-analysis of variance (ANOVA) followed by bonferroni post-hoc test in graph pad prism version 5.0.

Results and Discussion

The mean blood glucose values in Group II (DC) were consistently increased from 0th day to 21st day with a high of 380.3 ± 23.05 mg/dL on day 21 (Table I).

Significant increase in blood glucose values in diabetic control group compared to normal control group was in accordance with previous findings using streptozotocin to induce diabetes mellitus (Deepak *et al.* 2020 and Azad and Sulaiman., 2020). Among the groups, ALECP treated group (group IV) has shown decreased serum glucose levels from the 4th day itself but the decrease is more significant on 14th and 21st day. Similar results were reported by Maniyar and Bhixavatimath (2011), with significant decrease in serum glucose levels by ALECP treatment as early as day 7 of the experiment. Whereas, DLPCP treated group (group III) has not shown effective reduction of glucose levels in the earlier days of the experiment, but moderate reduction was observed on 21st day. In a similar pattern, the day wise comparison within the group has indicated the superiority of the ALECP which has shown significant decrease of serum glucose levels from 0th day to 21st day of the experiment, which is not observed with DLPCP in any of the days of the experiment. Although, DLPCP has not shown significant decrease in the serum glucose levels from 0th day ($360.0 + 54.63$) to 21st day ($311.7 + 28.83$), it has been found to be effective in ameliorating the increasing trend of serum glucose observed in diabetic group from 0th day ($356.1 + 30.82$) to 21st day ($380.3 + 23.05$). The results with DLPCP were similar with findings of antidiabetic effect showed by ethyl acetate extract of *Atylosia albicans* of 100mg/Kg b.wt in streptozotocin induced diabetic rats (Bhava *et al.* 2020). This antihyperglycemic effect of ALECP & DLPCP may be due to the presence of flavonoids, alkaloids and tanins as reported earlier (Buch *et al.* 2000 and Maniyar and Bhixavatimath., 2011).

Table I: Serum glucose levels in different experimental groups of rats

GLUCOSE (mg/dL)	Day of treatment			
	0	4	14	21
Group-I Normal Control (NC)	$91.0 \pm 6.24^{a,p}$	$92.6 \pm 6.41^{a,p}$	$93.6 \pm 1.47^{a,p}$	$92.3 \pm 4.56^{a,p}$
Group-II Diabetic Control (DC)	$356.1 \pm 30.82^{b,p}$	$360.7 \pm 16.86^{b,p}$	$374.3 \pm 22.07^{c,p}$	$380.3 \pm 23.05^{c,p}$
Group-III (DM+DLPCP @ 400mg/Kg b.wt)	$360.0 \pm 54.63^{b,p}$	$351.0 \pm 32.11^{b,p}$	$320 \pm 30.32^{bc,p}$	$311.7 \pm 28.83^{b,p}$
Group-IV (DM+ALECP @ 400mg/Kg b. wt)	$363.2 \pm 30.20^{b,q}$	$327.7 \pm 18.65^{b,pq}$	$293.7 \pm 13.01^{b,p}$	$267.0 \pm 5.62^{b,p}$

Values are mean \pm SE (n=8); Two way ANOVA (Graph pad prism, version 5.0)

a, b, c Means sharing different superscripts in a column differ significantly ($P \leq 0.05$)

p, q Means sharing different superscripts in a row differ significantly ($P \leq 0.05$).

The serum SGOT and SGPT activities in the group III (DM+DLPCP) and group IV (DM+ALECP) has decreased significantly when compared to diabetic control group from 14th day and 4th day respectively (Table II). Further, day wise comparison within the group has showed that DLPCP shown significant decrease of SGOT and SGPT activities from the 14th day whereas, ALECP shown this effect from 4th day itself, which indicates superiority of ALECP than DLPCP in terms of hepatoprotective action. Hepatoprotective affect shown by treatment groups may be the manifestation of their hypoglycemic activity, which would have effectively

reduced the fatty acid infiltration into the liver and thereby protecting from hepatocyte damage (Juurinen *et al.*2007). Our results with ALECP were similar with findings of hepatoprotective effect showed by aqueous leaf extract of *Carica papaya* of 1.5 g/100ml in streptozotocin induced diabetic rats (Rojop *et al.*2012) and results with DLPCP were similar with findings of hepatoprotective effect showed by hydroethanolic extract of *Artemisia amygdalina* of 250 mg/Kg b.wt in streptozotocin induced diabetic rats (Ghazanfar *et al.*2014).

Table II: SGOT and SGPT (IU/L) activity in different experimental groups of rats

Groups	SGOT(IU/L)				SGPT (IU/L)			
	Day of treatment				Day of treatment			
	0	4	14	21	0	4	14	21
Group-I Normal Control (NC)	151.5 ± 5.3 ^{a,p}	152.3 ± 1.5 ^{a,p}	155.4 ± 3.6 ^{a,p}	162.0 ± 2.0 ^{a,p}	54.3 ± 5.9 ^{a,p}	57.2 ± 6.6 ^{a,p}	55.5 ± 5.4 ^{a,p}	56.6 ± 3.9 ^{a,p}
Group-II Diabetic Control (DC)	274.7 ± 6.8 ^{b,p}	297.7 ± 9.1 ^{c,p}	336.3 ± 2.1 ^{c,q}	356.0 ± 8.2 ^{d,r}	213.9 ± 3.6 ^{b,p}	240.1 ± 3.2 ^{d,q}	264.3 ± 8.9 ^{d,r}	293.0 ± 8.9 ^{c,s}
Group-III (DM+DLPCP @ 400mg/Kg b. wt)	292.8 ± 4.4 ^{b,q}	279.9 ± 10.6 ^{c,q}	228.8 ± 8.5 ^{b,p}	213.0 ± 3.6 ^{c,p}	223.6 ± 6.9 ^{b,r}	200.0 ± 5.2 ^{c,r}	179.8 ± 0.9 ^{c,q}	170.9 ± 2.9 ^{b,p}
Group-IV (DM+ALECP @ 400mg/Kg b. wt)	282.7 ± 8.9 ^{b,s}	252.9 ± 4.6 ^{b,r}	212.8 ± 13.3 ^{b,q}	190.3 ± 7.1 ^{b,p}	224.9 ± 10.5 ^{b,s}	155.3 ± 4.8 ^{b,r}	103.9 ± 4.0 ^{b,q}	63.9 ± 6.9 ^{a,p}

Values are mean ± SE (n=8); Two way ANOVA (Graph pad prism, version 5.0) a,b,c,d Means sharing different superscripts in a column differ significantly ($P \leq 0.05$) p,q,r,s Means sharing different superscripts in a row differ significantly ($P \leq 0.05$)

Among the groups, both ALECP and DLPCP treated groups has shown the significant decreased levels of serum triglycerides, total cholesterol, LDL, VLDL and increased HDL levels (Table III & Table IV) from the 4th day of the experiment, but the ALECP is more effective compared to DLPCP. Within the group comparison, group IV (ALECP + DM) has shown significant decrease in serum levels of triglycerides, total cholesterol, LDL, VLDL and significant increase in HDL levels from 0th day to 21st day of the experiment, which is not observed in the DLPCP treated groups in any of the days of the experiment. Although, DLPCP has not shown significant decrease in the serum levels of serum triglycerides, total cholesterol, LDL, VLDL and significantly increased HDL levels from 0th day to 21st day, it has been effective in ameliorating the increasing trend of serum triglycerides, total cholesterol, LDL, VLDL and decreasing HDL levels observed in diabetic group from 0th day. Our results with DLPCP and ALECP were similar with findings of antihyperlipidemic effect shown by hydroethanolic extract of *Kaemferia galanga*

rhizome of 250 mg/Kg.b.wt and 500 mg/Kg.b.wt respectively in streptozotocin induced diabetic rats (Subbaian and Ragavan., 2020). Antihyperlipidemic effects of treatment groups may be due to increased insulin secretion, which could inhibit lipolysis, thereby decrease the levels of lipids in the serum (Jothivel *et al.*2007). These antihyperlipidemic effects could be attributed to saponins present in DLPCP and ALECP, which inhibits the absorption of dietary lipids from the intestine (Marrelli *et al.*2016).

The above biochemical finding are also endorsed by histopathological studies, where in the section of pancreas from Group III (DM+DLPCP) showed mild regeneration β cells, slight reconstructive appearance of ILH and exocrine acini (Fig 1). The section of liver of Group III (DM+DLPCP) has shown mild reconstructive appearance of hepatocytes along with diminution in inflammatory changes (Fig 2). The histopathological studies with ALECP supplemented Group IV were suggestive of marked regeneration of β cells, moderate reconstructive appearance of ILH and almost normal

appearance of acini of pancreas (Fig 1). The sections from liver of Group IV (DM+ALECP) showed normal number of hepatocytes and minute inflammatory changes resembling almost normal liver (Fig 2). The above results showed that the ALECP has been superior in terms of antidiabetic activity, hepatoprotective activity and antihyperlipidemic activity, when compared to DLPCP.

Hence, the aqueous leaf extract of *Carica papaya* may be considered as better alternative remedy than dried latex of *Carica papaya* in regulating diabetes and its complications. However, further research is needed to gain a better understanding of their potential therapeutic action, the implicated phytochemical constituents and the exact mechanism of action.

Table II: SGOT and SGPT (IU/L) activity in different experimental groups of rats

Groups	SGOT(IU/L)				SGPT (IU/L)			
	Day of treatment				Day of treatment			
	0	4	14	21	0	4	14	21
Group-I Normal Control (NC)	151.5 ± 5.3 ^{a,p}	152.3 ± 1.5 ^{a,p}	155.4 ± 3.6 ^{a,p}	162.0 ± 2.0 ^{a,p}	54.3 ± 5.9 ^{a,p}	57.2 ± 6.6 ^{a,p}	55.5 ± 5.4 ^{a,p}	56.6 ± 3.9 ^{a,p}
Group-II Diabetic Control (DC)	274.7 ± 6.8 ^{b,p}	297.7 ± 9.1 ^{c,p}	336.3 ± 2.1 ^{c,q}	356.0 ± 8.2 ^{d,r}	213.9 ± 3.6 ^{b,p}	240.1 ± 3.2 ^{d,q}	264.3 ± 8.9 ^{d,r}	293.0 ± 8.9 ^{c,s}
Group-III (DM+DLPCP @ 400mg/Kg b. wt)	292.8 ± 4.4 ^{b,q}	279.9 ± 10.6 ^{c,q}	228.8 ± 8.5 ^{b,p}	213.0 ± 3.6 ^{c,p}	223.6 ± 6.9 ^{b,r}	200.0 ± 5.2 ^{c,r}	179.8 ± 0.9 ^{c,q}	170.9 ± 2.9 ^{b,p}
Group-IV (DM+ALECP @ 400mg/Kg b. wt)	282.7 ± 8.9 ^{b,s}	252.9 ± 4.6 ^{b,r}	212.8 ± 13.3 ^{b,q}	190.3 ± 7.1 ^{b,p}	224.9 ± 10.5 ^{b,s}	155.3 ± 4.8 ^{b,r}	103.9 ± 4.0 ^{b,q}	63.9 ± 6.9 ^{a,p}

Values are mean ± SE (n=8); Two way ANOVA (Graph pad prism, version 5.0) a,b,c,d Means sharing different superscripts in a column differ significantly ($P \leq 0.05$) p,q,r,s Means sharing different superscripts in a row differ significantly ($P \leq 0.05$)

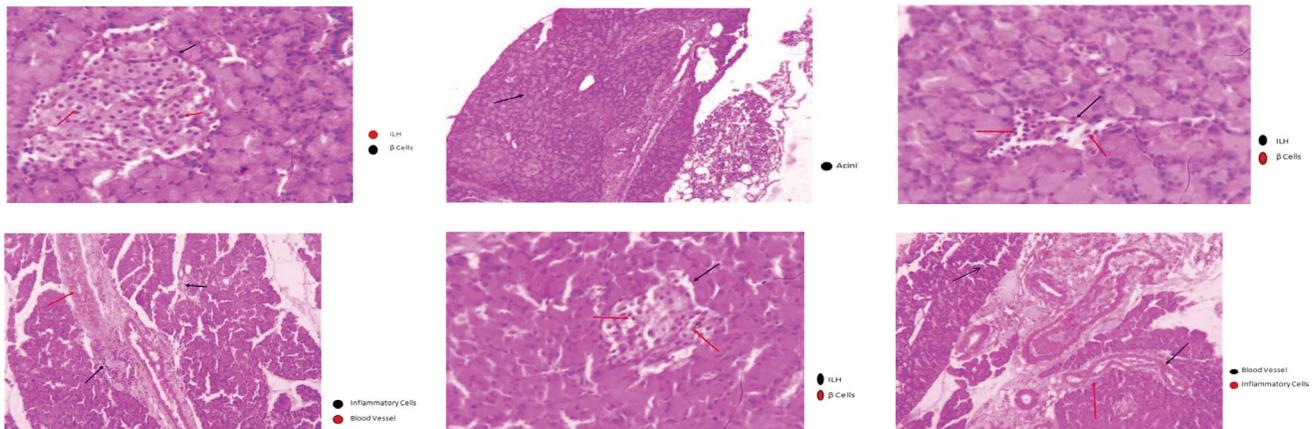


Fig. 1a) Histopathological section of pancreas of group I (NC) rats exhibiting normal sized ILH and normal number of β cells. H&EX400. b) Histopathological section of pancreas of group I (NC) rats showing normal appearance of acinar cells. H&EX100. c) Histopathological section of pancreas of group II (DC) rats showing shrunken ILH with less number of β cells. H&EX400. d) Histopathological section of pancreas of group II (DC) rats showing thickened & dilated blood vessels with severe infiltration of inflammatory cells in acini and around blood vessels. H&EX100. E) Histopathological section of pancreas of group III (DM+DLPCP) rats showing reconstructive appearance of ILH with mild regeneration of β cells. H&EX400. F) Histopathological section of pancreas of group IV (DM+ALECP) rats showing prominent ILH with marked regeneration of β cells. H&EX400.

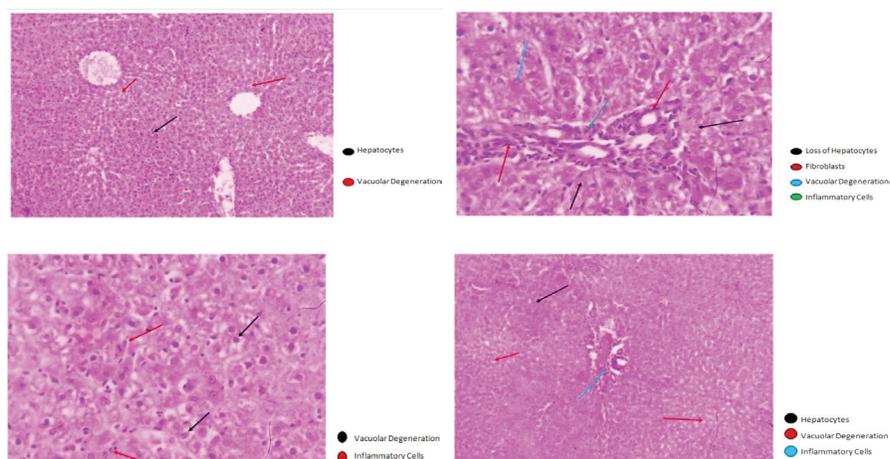


Fig. 2a) Histopathological section of liver of group I (NC) showing normal number and arrangement of hepatocytes with slight vacuolar degeneration. H&EX100. b) Histopathological section of liver of group II (DC) showing loss of hepatocytes, vacuolar degeneration and severe infiltration of fibroblasts and other inflammatory cells. H&EX400. c) Histopathological section of liver of group III (DM+DLPCP) showing vacuolar degeneration and infiltration of inflammatory cells. H&EX400 d) Histopathological section of liver of group IV (DM+ALECP) showing normal number of hepatocytes with mild infiltration of inflammatory cells and less vacuolar degeneration. H&EX100.

Conclusion

The antidiabetic activity of ALECP was much better compared to DLPCP in streptozotocin induced diabetic rats. The effects of ALECP were observed during early days of the experiment when compared to DLPCP. In addition, ALECP showed better hepatoprotective activity and effectively reduced hyperlipidemia associated with diabetes mellitus than DLPCP. In conclusion, dried latex of *Carica papaya* had mild ameliorative effect on streptozotocin induced diabetic changes, whereas the aqueous leaf extract of *Carica papaya* can serve as a promising alternative remedy for diabetes mellitus and its complications.

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Conflict of Interest

The authors declare that there are no any conflict of research and financial interests.

Author Contributions

RN, TVCK & KS performed experiments, TVCK, EP, and KA conceptualized and supervised the study. RN & TVCK wrote the original manuscript. TVCK, KP & EP edited the manuscript.

References

- Airaodion, A.I., Ogbuagu, E.O., Ekenjoku, J., Ogbuagu, U. and Okoroukwu, V.N. 2019. Antidiabetic effect of ethanolic extract of *Carica papaya* leaves in alloxan-induced diabetic rats. *Am. J. Biomed. Sci. Res.*, **5(3)**: 1–6. <http://dx.doi.org/10.34297/AJBSR.2019.05.000917>
- Ajani, R. and Ogunbiyi, K. 2015. *Carica papaya* latex accelerates wound healing in diabetic Wistar rats. *Eur. J. Med. Plants*, **9(3)**: 1–12. <https://doi.org/10.9734/EJMP/2015/17758>
- Bhava, S.B.S., Umasankar, K. and Muthu, K.A. 2020. Studies on antidiabetic activity of *Atylosia albicans* in streptozotocin-induced diabetic rats. *Int. J. Res. Pharm. Sci.*, **11(1)**: 416–424. <https://ijrps.com/home/article/view/507>
- Buch, K.Y., Lewis, H. and Lamba, J. 2000. Phytochemicals as potential hypoglycemic agents. *Stud. Nat. Prod. Chem.*, **21**: 1–20. [https://doi.org/10.1016/S1572-5995\(00\)80012-5](https://doi.org/10.1016/S1572-5995(00)80012-5)
- Ebenezer, A.M., Folasade, O.A. and Senga, K.P. 2019. Antidiabetic effect of aqueous extract of ripe *Carica papaya* seed in alloxan-induced diabetic albino rats. *J. Diabetes Endocrinol.*, **10(2)**: 13–17. <https://doi.org/10.5897/JDE2018.0127>
- Ezekwe, S.A., Elekwa, I., Osuocha, U. and Uzoma, K. (2014). Hypoglycemic, hypolipidemic and body weight effects of unripe pulp of *Carica papaya* using diabetic albino rat model. *J. Pharmacogn. Phytochem.*, **2(6)**: 109–114.

- Ghazanfar, K., Ganai, B.A., Akbar, S., Mubashir, K., Dar, S.A., Dar, M.A. and Tantry, M.A. 2014. Antidiabetic activity of *Artemisia amygdalina* Decne in streptozotocin-induced diabetic rats. *J. Biomed. Biotechnol.*, **2014**: 185676. <https://doi.org/10.1155/2014/185676>
- Guyton, A.C. and Hall, J.E. 2006. Insulin, glucagon, and diabetes mellitus. In: *Textbook of Medical Physiology*, **11**: 961–977.
- Jothivel, N., Ponnusamy, S.P., Appachi, M., Singaravel, S., Rasilingam, D., Deivasigamani, K. and Thangavel, S. 2007. Anti-diabetic activity of methanol leaf extract of *Costus pictus* D. DON in alloxan-induced diabetic rats. *J. Health Sci.*, **53**: 655–663. <http://dx.doi.org/10.1248/jhs.53.655>
- Juurinen, L., Tiikkainen, M., Häkkinen, A., Hakkarainen, A. and Yki-Järvinen, H. 2007. Effects of insulin therapy on liver fat content and hepatic insulin sensitivity in patients with type 2 diabetes. *Am. J. Physiol. Endocrinol. Metab.*, **292(3)**: 829–835. <https://doi.org/10.1152/ajpendo.00133.2006>
- Kaoud, H. 2017. Diabetes in animals: Facts and explanations. In: *Textbook of Diabetes in Animals*, pp. 1–19.
- Kumar, Y., Gautam, G.K. and Mishra, P. 2018. Evaluation of hepatoprotective activity of *Carica papaya* and *Ficus bengalensis* latex on thioacetamide-induced hepatotoxicity in rats. *Int. J. Adv. Res.*, **6(9)**: 294–299. <http://dx.doi.org/10.21474/IJAR01/7674>
- Laflamme, D.P. 2012. Companion animal symposium: Obesity in dogs and cats: What is wrong with being fat? *J. Anim. Sci.*, **90(5)**: 1653–1662. <https://doi.org/10.2527/jas.2011-4571>
- Maniyar, Y. and Bhixavatimath, P. 2011. Antihyperglycemic and hypolipidemic activities of aqueous extract of *Carica papaya* Linn. leaves in alloxan-induced diabetic rats. *J. Ayurveda Integr. Med.*, **3(2)**: 70–74. <https://doi.org/10.4103%2F0975-9476.96519>
- Marrelli, M., Conforti, F., Araniti, F. and Statti, G.A. 2016. Effects of saponins on lipid metabolism: A review of potential health benefits in the treatment of obesity. *Molecules* **21(10)**: 1404. <https://doi.org/10.3390/molecules21101404>
- Nelson, R.W. and Reusch, C.E. 2014. Classification and etiology of diabetes in dogs and cats. *J. Endocrinol.*, **222**: 1–9. <https://doi.org/10.1530/joe-14-0202>
- Nimenibo-Uadia, R. and Nwachukwu, K. 2020. Antidiabetic effect of aqueous root extract of *Carica papaya* L. in alloxan-induced diabetic rats. *J. Nat. Sci. Res.*, **10(6)**: 1–6.
- Nissen, S.E. and Wolski, K. 2007. Effect of rosiglitazone on the risk of myocardial infarction and death from cardiovascular causes. *N. Engl. J. Med.*, **356**: 2457–2471. <http://dx.doi.org/10.1056/NEJMoa072761>
- Rojop, J.I.E., Díaz-Zagoya, J.C., Ble-Castillo, J.L., Miranda-Osorio, P.H., Castell-Rodríguez, A.E., Tovilla-Zárate, C.A., Rodríguez-Hernández, A., Aguilar-Mariscal, H., Ramón-Frías, T. and Bermúdez-Ocaña, D.Y. 2012. Hypoglycemic effect of *Carica papaya* leaves in streptozotocin-induced diabetic rats. *BMC Complement Altern. Med.*, **12**: 236. <https://doi.org/10.1186/1472-6882-12-236>
- Subbaian, K. and Ragavan, B. 2020. Effect of *Kaempferia galanga* rhizome extract on hematological parameters in streptozotocin-induced diabetic Wistar rats. *Int. J. Pharm. Sci. Drug Res.*, **12(3)**: 255–259.
- Venkateshwarlu, E., Dileep, P., Reddy, P.R. and Sandhya, P. 2013. Evaluation of antidiabetic activity of *Carica papaya* seeds on streptozotocin-induced type-II diabetic rats. *J. Adv. Sci. Res.*, **4(3)**: 38–41.

Electrocardiographic Changes in Geriatric Dog with Congestive Heart Failure

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Abstract

Congestive heart failure (CHF) is a leading yet often unnoticed cause of canine deaths in India. The primary culprits behind CHF include dilated cardiomyopathy (DCM), mitral valve disease (MVD), hypertrophic cardiomyopathy (HCM), and pericardial effusion (PE). Sadly, the lack of advanced diagnostic facilities, such as radiography, echocardiography, and cardiac biomarker testing, often results in delayed diagnoses, robbing many pet owners of their cherished companions before timely intervention can be made. Electrocardiographic analysis uncovered a range of supraventricular, ventricular, and morphological abnormalities. In aged dogs affected by congestive heart failure (CHF), atrial fibrillation was the most common finding, observed in 7 dogs (29.17%). Among the other screened dogs (n=251), sinus arrhythmia (6.77%) was the most frequently detected arrhythmia, while deep Q-waves and wide QRS complexes were diagnosed in 3.10%, and 1.59% of cases, respectively. Early diagnosis and regular screening are crucial for identifying and managing cardiac disorders in dogs, enabling timely interventions to improve outcomes and quality of life.

Keywords: Geriatric dogs, Atrial Fibrillation, Dilated Cardiomyopathy, ECG

Introduction

Dogs are becoming more and more common as companion animals among urban populations these days. It had been estimated that around 10-15% of dogs taken to vets in North America are believed to have heart problems (Atkins *et al.*, 2009). In India, a study by Haritha *et al.* (2018) found that the overall prevalence of cardiac disorders among dogs was 1.77%, and it could vary as these diseases are not often properly diagnosed and treated, due to the lack of diagnostic facilities and expertise. Furthermore, Parker *et al.* (2006) emphasized that cardiac diseases in dogs often operate quietly, without showing obvious symptoms, and their sudden impact can leave owners and breeders grappling with feelings of loss and uncertainty. Boswood *et al.* (2016) reported that Congestive heart failure (CHF) CHF was a common outcome of various heart diseases and is particularly prevalent in geriatric dogs, with major causes including myxomatous mitral valve disease (MVD) and dilated cardiomyopathy (DCM). This study describes the electrocardiographic patterns associated with congestive heart failure in geriatric dogs.

Materials and Methods

The study was conducted from June 2023 to August 2024, at the Department of Veterinary Medicine, Dr. G.C. Negi College of Veterinary &

Animal Sciences, CSKHPKV Palampur, Himachal Pradesh, in which 275 geriatric dogs were screened out of total 2,574 registered cases. Among them, 24 geriatric dogs were diagnosed with congestive heart failure (CHF) through electrocardiography (ECG), radiography, and echocardiography. ECG assessments were performed using the RMS Vesta 301i, a 12-lead system, following standard protocols (Tilley and Smith, 1997). Thermosensitive recording paper (80 mm width, 20 metres recording length) from Arrow Medical Recording, Chennai, was used for ECG recordings.

In dogs with respiratory distress, ECG was recorded in standing posture. The collected data was statistically analysed using InStat software from Graphpad (2008) and SPSS 12. Mean values of various parameters between control and diseased groups, as well as pre- and post-treatment data, were compared. Statistical significance at different levels was determined using the “t” test and ANOVA providing a thorough evaluation of the treatment effects.

Results and Discussion

The electrocardiographic findings in dogs with CHF due to DCM, MVD, HCM, and PE are summarized in Table I. In cases of DCM, our study revealed a significant reduction ($p < 0.05$) in P wave amplitude. For dogs with MVD and HCM, no significant changes in ECG parameters were observed. In cases of DCM,

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our study revealed a notably low P wave amplitude. The reduced P wave amplitude may be attributed to the high incidence of atrial fibrillation (AF) (Fig. 7) in dogs with DCM, where the P wave is absent, resulting in a P wave value of zero during AF episodes.

In cases of PE, a significantly reduction in QRS amplitude was noted, which is consistent with the findings of Lakshmi *et al.*, (2017). In our study a

significant reduction in P wave amplitude was also found, in cases of pericardial effusions, which is similar to the findings of Habashy *et al.*, (2004), they observed that low P-wave voltage occurred more often than low QRS voltage (Fig. 3). The low P wave amplitude indicate that some fluid from severe PE can collect around the right atrium, hence P-wave on an ECG gets smaller Madias (2008).

Table I: ECG parameters in CHF-affected dogs (n=24)

Parameter	Control (n=17)	DCM (n=9)	MVD (n=7)	HCM (n=3)	PE (n=5)
P-wave amplitude (mV)	0.26 ± 0.02 ^b	0.035 ± 0.02 ^a	0.13 ± 0.04 ^{ab}	0.11 ± 0.09 ^{ab}	0.06 ± 0.04 ^b
P-wave duration (sec)	0.03 ± 0.01	0.02 ± 0.01	0.03 ± 0.00	0.06 ± 0.02	0.03 ± 0.02
Q-wave amplitude (mV)	1.25 ± 0.06 ^b	1.26 ± 0.13 ^{ab}	0.86 ± 0.27 ^{ab}	1.03 ± 0.31 ^{ab}	0.48 ± 0.13 ^a
QRS (sec)	0.04 ± 0.01	0.06 ± 0.00	0.03 ± 0.00	0.05 ± 0.01	0.04 ± 0.00
T-wave amplitude (mV)	0.20 ± 0.02	0.18 ± 0.02	0.22 ± 0.06	0.3 ± 0.05	0.22 ± 0.04
T-wave duration (sec)	0.05 ± 0.01	0.04 ± 0.00	0.10 ± 0.00	0.06 ± 0.01	0.04 ± 0.06
PR interval (sec)	0.08 ± 0.01	0.05 ± 0.01	0.10 ± 0.01	0.09 ± 0.02	0.07 ± 0.02
QT interval (sec)	0.20 ± 0.01	0.17 ± 0.01	0.21 ± 0.02	0.19 ± 0.02	0.15 ± 0.01

Values with different upper-case alphabets in the same row are significant ($p \leq 0.05$)

In geriatric dogs with congestive heart failure (CHF), various ECG abnormalities were observed (Table II), with atrial fibrillation being the most prevalent, affecting 7 dogs (29.17%), consistent with the findings of Tejaswi (2022) and Noszczyk-Nowak *et al.* (2017). According to Lubitz *et al.*, (2010) higher prevalence of atrial fibrillation (AF) in congestive heart failure (CHF) can be attributed to various underlying mechanisms. Atrial fibrosis which disrupts cell-to-cell electrical coupling and impairs signal conduction thus creating an environment conducive to arrhythmias. Additionally, genetic predisposition may also contribute to the development of AF. Similarly, low QRS complexes were also seen in 7 dogs (29.17%). Other common arrhythmias included ST slurring or coving, R wave notching, wide QRS complexes, and electrical alternans,

each present in 4 dogs (16.67%). Less frequent, but still notable, arrhythmias such as deep Q waves, prolonged PQ intervals (Fig. 13), first degree AV block, and sinus arrest/block were identified in 3 dogs (12.50%). The occurrence of sinus arrest (Fig. 14) is similar to that reported by Tejaswi (2022), while our findings of first-degree AV block align with Noszczyk-Nowak *et al.*, (2017), who noted a 12.63% prevalence of first-degree heart block in dogs with pathological arrhythmias and reported that high-grade atrioventricular blocks are usually claimed to be idiopathic and associated with fibrosis of the conduction system or myocarditis. Sinus pauses are typically observed in brachycephalic breeds. They may occur secondary to fluctuations in vagal tone associated with the respiratory cycle. Such fluctuations are particularly evident in dogs with brachycephalic syndrome due to their increased respiratory effort,

irritation of laryngeal region, and parasympathetic stimulation

Additional arrhythmias, including T(a) wave abnormalities (greater than $\frac{1}{4}$ of the R(a) wave), ST depression, prolonged QT intervals, supraventricular tachycardia, and ventricular premature complexes (VPCs) (Fig. 6), were each detected in 2 dogs (8.34%). Uncommon arrhythmias, such as deep S waves, right bundle branch block (RBBB) (Fig. 2), wandering pacemaker (WPM), and short PQ intervals, were observed in 1 dog each (4.17%). Satish (2009) recorded a lower incidence of RBBB (2.34%) in CHF dogs compared to our study. We also noted increased R wave amplitude in 2 cases (8.34%) (Fig. 12), closely matching

the findings of Satish (2009), who reported an 8.60% occurrence in dogs with cardiac issues. Our study found higher incidences of deep Q waves and low QRS complexes compared to Tejaswi (2022), Satish (2009), and Haritha (2018). Additionally, the findings of ST coving aligned with Tejaswi (2022), and P mitrale was observed in only one case, similar to Satish (2009).

The study highlights distinct ECG patterns associated with mitral valve disease (MVD), hypertrophic cardiomyopathy (HCM), pericardial effusion, and dilated cardiomyopathy (DCM) that are associated with various forms of CHF. P wave, QRS duration, and T wave changes provided useful hints for the diagnosis of cardiac disorders.

Table II: Various ECG findings in CHF affected dogs (n=24)

Abnormal rhythm	Number of Dogs	Abnormal rhythm (%)
T wave (a) > $\frac{1}{4}$ R wave (a)	2	8.34%
ST slurring/coving	4	16.67%
R wave notching	4	16.67%
Deep S wave	1	4.17%
Right bundle branch block (RBBB)	1	4.17%
Low QRS Complexes	7	29.17%
Sinus arrest/block	3	12.50%
Wandering pacemaker (WPM)	1	4.17%
ST depression	2	8.34%
Wide QRS complex	4	16.67%
Atrial fibrillation	7	29.17%
Prolong QT interval (>0.25 sec)	2	8.34%
Electrical alterans	4	16.67%
Supraventricular tachycardia	2	8.34%
First degree AV block	3	12.50%
Ventricular premature complexes (VPCs)	2	8.34%
Prolong PQ/PR interval	3	12.50%
Short PQ interval	1	4.17%
Deep Q wave	3	12.50%
Increased R amplitude	2	8.34%
P-mitrale	1	4.17%
Total	58	100.00%

Table III: Various ECG abnormalities found during screening of geriatric dogs (n=251) excluding those diagnosed with CHF

Abnormal rhythm	Number of Dogs	Abnormal rhythm (%)
Sinus arrhythmia	17	6.77%
Deep Q-wave	8	3.10%
Wide-QRS complexes	4	1.59%
Notched R wave	7	2.78%
ST elevation	2	0.79%
Tall T-wave	6	2.39%
ST-coving	4	1.59%
Right bundle branch block	4	1.59%
VPCs	2	0.79%
Electrical alternans	7	2.78%
Atrial flutter	2	0.79%
Tall R wave	3	1.19%
Sinus arrest	2	0.79%
Sinus tachycardia	4	1.59%
Atrial fibrillation	4	1.59%
ST depression	2	0.79%
Sinus bradycardia	2	0.79%
Atrial standstill	3	1.19%
Asystole	3	1.19%
Low QRS complexes	5	1.99%
P- pulmonale	2	0.79%
P- mitrale	1	0.39%
First degree AV block	4	1.59%
Second degree AV block	2	0.79%
WPM	4	1.59%
Total	104	41.43%

Table III presents the types and frequency of arrhythmias identified during a screening of 275 geriatric dogs. The data includes the specific types of abnormal heart rhythms, the number of dogs affected by each, and the percentage of the total population (275) that each arrhythmia impacts. Out of these, 24 dogs were diagnosed with CHF and 104 dogs (41.43%) were identified with abnormal heart rhythms, indicating that over one-quarter of the elderly dogs exhibited some form of cardiac irregularity. The most common type of arrhythmia found was sinus arrhythmia, affecting 17 dogs (6.77%) (Fig. 1), followed by deep Q-wave abnormalities in 8 dogs (3.10%) and wide-QRS complexes, in 4 dogs (1.59%). Several other arrhythmias, including notched R-wave, ST elevation, tall T-wave, and ventricular premature complexes (VPCs), affected

smaller proportion (1% to 3%) of the population. Severe conditions like atrial fibrillation, asystole (Fig. 10), and atrial flutter were rare, affecting nearly 1.59%, 1.19% and 0.79% of the dogs. Additionally, conditions such as AV blocks (first and second degree), electrical alternans and WPM syndrome were also found, though they were less frequent. Sinus arrhythmias are considered physiological in dogs due to their strong parasympathetic (vagal) influence on the heart. Physiological sinus tachycardia can occur as a transient response to catecholamine release, often triggered by the stress of an electrocardiographic examination. Conversely, sinus bradycardia is typically seen in athletic dogs of giant breeds. In smaller breeds, sinus bradycardia may result from elevated vagal tone, the presence of toxemia, or as a secondary manifestation of sick sinus syndrome

(Noszczyk-Nowak et al. 2017). The findings underscore the variety of arrhythmias present in geriatric dogs, with some being more prevalent than others. So, the routine screening for heart conditions in older dogs is essential for early detection and management of both common and rare arrhythmias.

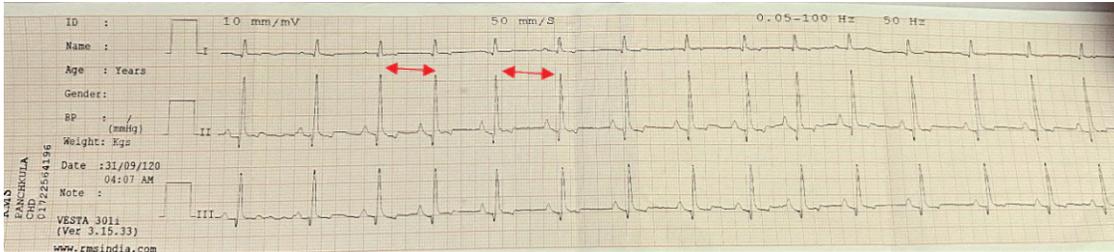


Fig. 1: Sinus arrhythmia (50 mm/s paper speed; 10 mm/mV gain)

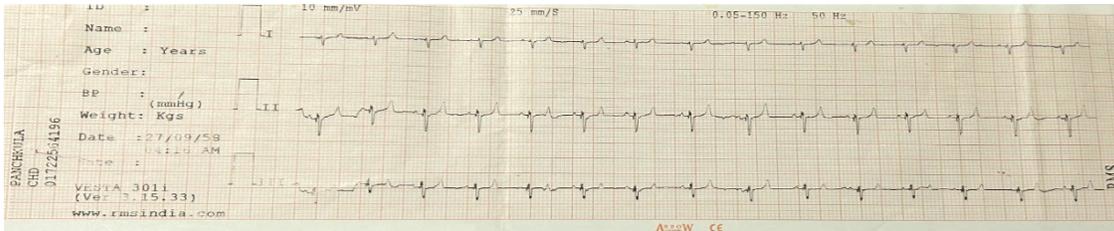


Fig. 2: Right bundle branch block (25 mm/s paper speed, gain 10 mV)

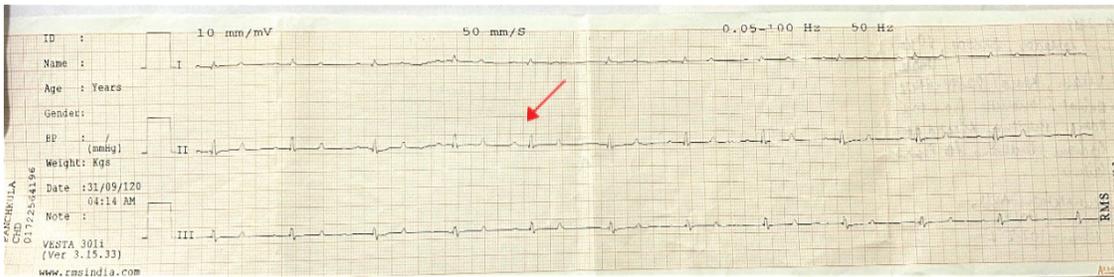


Fig. 3: Low QRS complex in a dog with pericardial effusion (50 mm/s paper speed; 10 mm/mV gain).

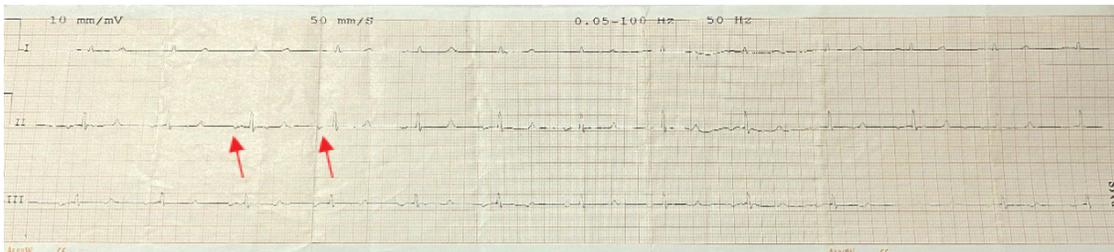


Fig. 4: Negative p wave and normal QRS complexes, suggestive of AV junctional premature complexes (50 mm/s paper speed; 10 mm/mV gain).

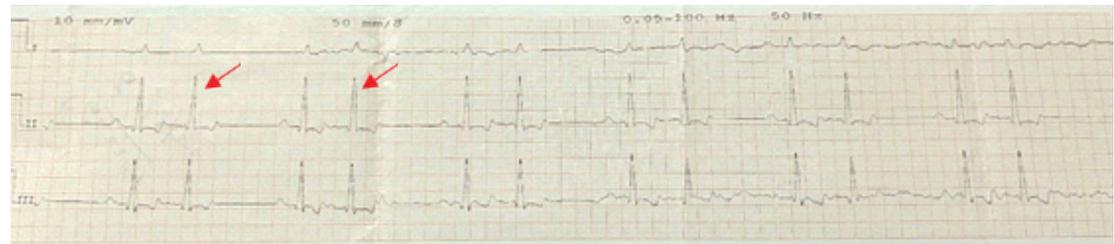


Fig. 5: Atrial premature complexes (APCs) in a dog with severe DCM and severe mitral valve regurgitation (50 mm/s paper speed; 10 mm/mV gain).

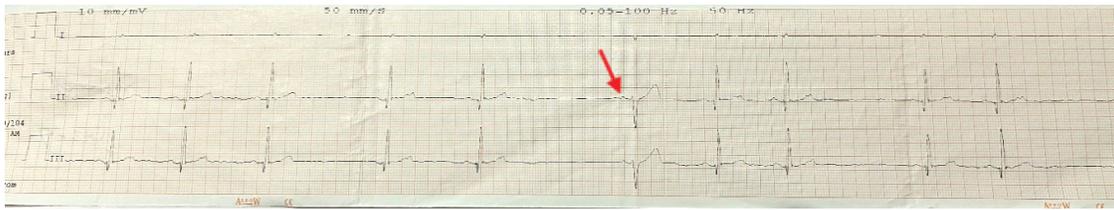


Fig. 6: Ventricular premature complex (VPC) in a dog affected with DCM (50 mm/s paper speed; 10 mm/mV gain).

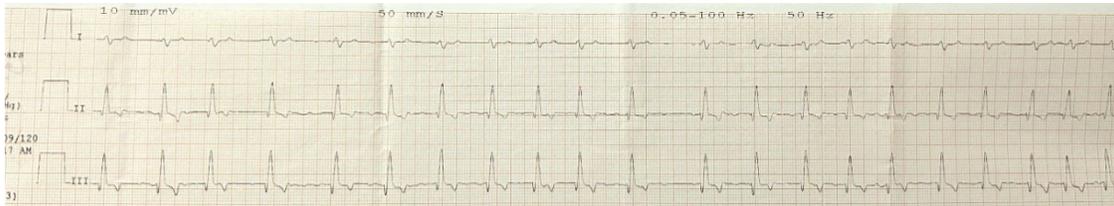


Fig. 7: Atrial fibrillation (AF) in a dog affected with DCM (50 mm/s paper speed; 10 mm/mV gain).

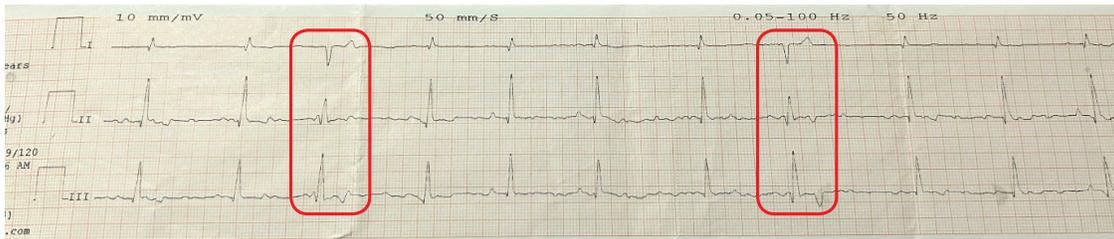


Fig. 8: Ventricular ectopic complex (square box) (50 mm/s paper speed; 10 mm/mV gain).

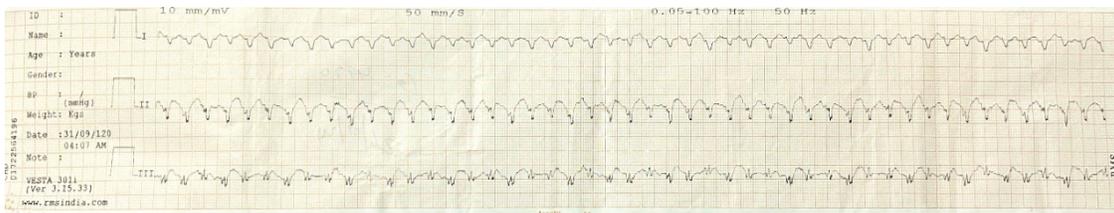


Fig. 9: Ventricular tachycardia (50 mm/s paper speed; 10 mm/mV gain).

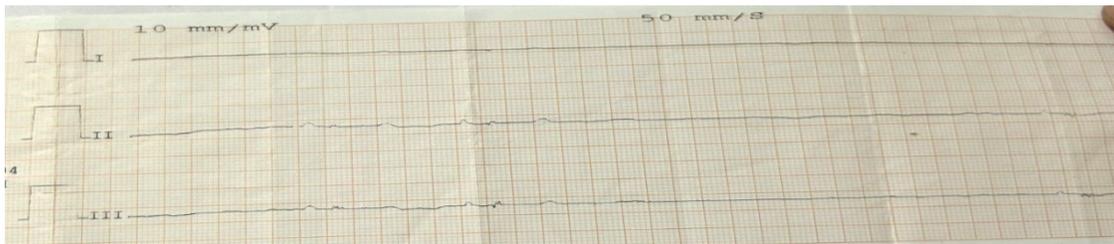


Fig. 10: Asystole (50 mm/s paper speed; 10 mm/mV gain).

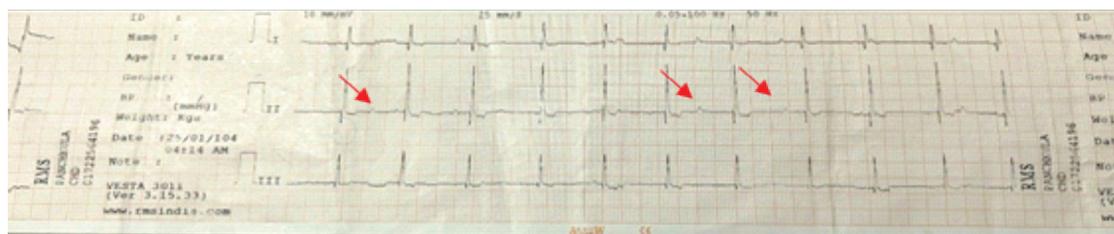


Fig. 11: Second Degree AV Block–Mobitz Type II (red arrows: represents P wave followed by no QRS complex) (50 mm/s paper speed; 10 mm/mV gain).

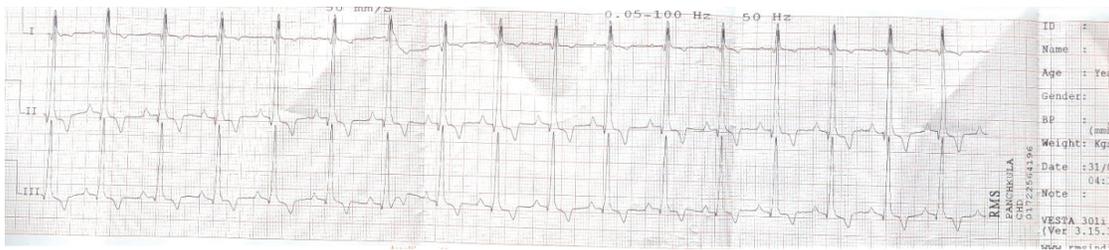


Fig. 12: Spiked R wave with amplitude >3mV, suggestive of left ventricular enlargement (50 mm/s paper speed; 10 mm/mV gain).

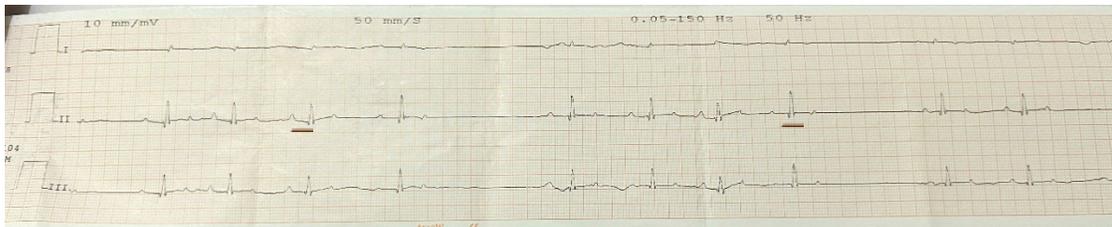


Fig. 13: Second-degree AV block–Mobitz Type I, with prolong PQ interval and with escape of a beat (50 mm/s paper speed and 10 mm/mV gain).

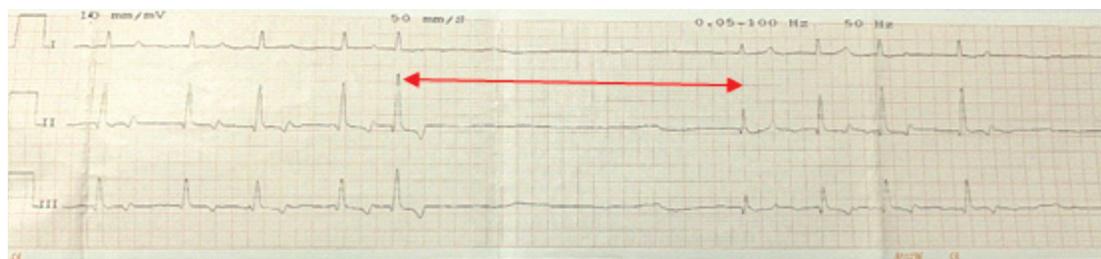


Fig. 14: Atrial standstill and sinus block (50 mm/s paper speed; 10 mm/mV gain).

Conclusion

This study highlights the timely detection and treatment of congestive heart failure (CHF) in geriatric dogs can be facilitated by routine electrocardiogram (ECG) examinations. The most frequent arrhythmia, occurring in 29.17% of cases of CHF, was atrial fibrillation and low QRS amplitude. In DCM, atrial fibrillation was prevalent, along with low P wave amplitude, whereas in PE patients, low QRS amplitude and low P wave amplitudes were typical.

Conflict of interest: Authors have no conflict of interest in this study.

References

- Atkins, C., Bonagura, J., Ettinger, S., Fox, P., Gordon, S., Haggstrom, J., Hamlin, R., Keene, B., Luis, F.V., & Stepien, R. 2009. Guidelines for the diagnosis and treatment of canine chronic valvular heart disease. *J. Vet. Intern. Med.*, **23(6)**: 1142-1150.
- Boswood, A., Haggstrom, J., Gordon, S.G., Wess, G., Stepien, R.L., & Oyama, M.A. 2016. Effect of pimobendan in dogs with preclinical myxomatous mitral valve disease and cardiomegaly: the EPIC study—a randomized clinical trial. *J. Vet. Intern. Med.*, **30**: 1765–1779.
- Ettinger, S.J. 1983. Criteria for the normal canine electrocardiograms. *Textbook of Veterinary Internal Medicine*. 2nd edn., W.B. Saunders, Philadelphia, USA, pp. 984.
- Habashy, A.G., Mittal, A., Ravichandran, N., & Cherian, G. 2004. The electrocardiogram in large pericardial effusion: The forgotten “P” wave and the influence of tamponade, size, etiology, and pericardial thickness on QRS voltage. *Angiology*, **55(3)**: 303-307.
- Haritha, G.S., Satish, K.K., Ayodhya, S., & Amruth, K.V.V.V. 2018. Prevalence of cardiac disorders in canines—a clinical study. *Intas Polivet*, **39(2)**: 8-91.
- Lakshmi, K., Padmaja, K., & Nagaraj, P. 2017. Clinico-diagnostic aspects of right sided heart failure in dogs. *J. Pharm. Innov.*, **6(6)**: 49-53.

- Lubitz, S.A., Benjamin, E.J., & Ellinor, P.T. 2010. Atrial fibrillation in congestive heart failure. *Heart Fail. Clin.*, **6(2)**: 187-200.
- Madias, J.E. 2008. The importance of the P-waves in the differentiation of attenuation of the QRS voltage due to pericardial effusion versus peripheral edema. *J. Card. Fail.*, **14(1)**: 55-60.
- Noszczyk-Nowak, A., Michałek, M., Kałuża, E., Cepiel, A., & Paślawska, U. 2017. Prevalence of arrhythmias in dogs examined between 2008 and 2014. *J. Vet. Res.*, **61(1)**: 103-110.
- Parker, H.G., Meurs, K.M., & Ostrander, E.A. 2006. Finding cardiovascular disease genes in the dog. *J. Vet. Cardiol.*, **8**: 115-127.
- Tejaswi, G. (022. Studies on electrocardiographic changes in dogs. M.V.Sc. Thesis Dept. of Veterinary Medicine, College of Veterinary Science, PVNRTVU, Rajendranagar, Hyderabad.
- Tilley, L.P. and Smith, F. 1997. The 5-minute veterinary consult: Canine and Feline. pp. 1056.

Haematobiochemical examination and urinalysis in dogs with renal disease

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Abstract

Early detection of renal diseases in canines is crucial for improving prognosis and extending quality of life. Urinalysis plays major role in identifying renal dysfunction. This study was aimed to evaluate different haemato-biochemical parameters along with urinalysis in dogs with Stage I and II of renal disease. Study was conducted on 13 dogs with Stage I and II renal disease. Mean values of TEC, PCV, Sodium, Potassium and Calcium were decreased while mean values of TLC, Monocytes, MCH, MCHC, Creatinine and ALT were increased. The urine pH of 10 dogs ranged from 5.0-6.5 whereas 3 dogs had pH 8. Mean value of UP:C was found to be significantly increased. Crystaluria (5), struvites (4) and bilirubin crystals (1) were found.

Keywords: Urinalysis- renal dysfunction – dogs

Introduction

Renal diseases are among the most prevalent and potentially life-threatening conditions affecting canines, often progressing silently until significant kidney function is lost. Early detection is critical for improving prognosis, guiding therapeutic interventions, and enhancing the quality of life in affected dogs. Among various diagnostic modalities, urinalysis remains a cornerstone in veterinary nephrology due to its non-invasive nature, affordability, and diagnostic value. Diagnosing kidney issues in dogs primarily relies on their medical history, clinical assessment, and elevated levels of serum creatinine.

Urinalysis provides essential information about the physical, chemical, and microscopic characteristics of urine, reflecting both renal and systemic health. Parameters such as urine specific gravity, proteinuria, haematuria, and the presence of casts or crystals indicate renal pathology before clinical signs or serum biochemical changes become evident. The present study includes detailed clinical examination, haemato-biochemical examination and urinalysis to assess renal diseases in canines.

Materials and Methods

The present study was carried out on dogs presented during the period of November 2024 to May 2025 in the Department of Veterinary Medicine, Veterinary Clinical Complex, College of Veterinary & Animal Sciences, CSKHPKV Palampur (H.P). Initial screening was done on the basis of patient's history and presenting clinical signs as inappetence, dehydration, fever, occasional vomiting, diarrhoea, urine dribbling, oliguria and halitosis. Haemato-biochemical estimation, electrolytes analysis and urinalysis were performed. Physical, chemical and microscopic examination of urine samples were done. Thirteen dogs in Stage I and II based on IRIS CKD classification were included for the study. Ten apparently healthy dogs presented for general checkup and vaccination were included as control group.

Results and Discussion

Out of 13 dogs, 53% (7/13) were in Stage I while 46.15% (6/13) were in Stage II of renal disease. Different aetiologies in these dogs are presented in Table 1. Most common clinical signs observed were urine dribbling, occasional vomiting, halitosis, inappetence, fever, dehydration and weight loss. Clinical, haematobiochemical parameters of dogs in Stage I and II of renal disease are presented in Table 2

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Table 1: Etiologies in dogs with Stage I and II of renal disease

S.No.	Etiology	No. of cases (n=13)
1.	Haemoprotozoan infection	4/13 (30%)
2.	Cystitis	3/13 (23%)
3.	Urethral Calculi	2/13 (15%)
4.	Haematuria	2/13 (15%)
5.	Megaesophagus	1/13 (7.6%)
6.	Endometrial hyperplasia	1/13 (7.6%)

Table 2: Clinical, Haematobiochemical parameters of dogs in Stage I and II of renal disease

Sl.No.	Parameter	Healthy control (n=10)	Stage I and II renal disease (n=13)
Clinical parameters			
1.	Rectal temperature (⁰ F)	101.88±0.17	101.01±0.54
2.	Heart rate (per minute)	132.40±2.78	120±5.46
3.	Respiratory rate (per minute)	32.40±1.24	35.27±2.43
Haematology			
4.	Hb (g/dl)	13.47±0.34	12.28±1.021
5.	PCV (%)	38.87±1.04	33.85±2.63
6.	TEC (10 ¹² /L)	6.49±0.21	5.21±0.42*
7.	TLC (10 ⁹ /L)	11.13±0.6	21.14±5.24
8.	N (%)	79.68±0.85	79.5±2.75
9.	L (%)	15.3±0.62	12.53±2.60
10.	M (%)	4.04±0.27	6.16±0.70*
11.	E (%)	1.35±0.18	2±0.73
12.	PLT (10 ⁹ /L)	299.2±23.72	279.23±48.29
13.	MCV (fl)	61.57±0.47	64.43±1.97
14.	MCH (pg)	21.88±0.4	24.16±0.45**
15.	MCHC (g/dl)	35.18±0.58	375.84±8.27***
Biochemical Parameters			
16.	Creatinine (mg/dL)	1.01±0.08	1.8±0.306*
17.	BUN (mg/dL)	25.14±4.54	82.88±29.14
18.	Total Protein (g/dL)	6.6±0.35	6.723±0.265
19.	Glucose (mg/dL)	102.67±2.66	102.60±5.732
20.	Bilirubin (mg/dL)	0.22±0.04	0.76±0.366
21.	AST (U/L)	38.05±3.98	175.56±112.36
22.	ALT (U/L)	33.92±2.4	110.78±23.738*
23.	ALP (U/L)	79.25±8.72	307.29±97.23
Serum electrolytes			
24.	Sodium (mmol/L)	151.8±1.71	146.20±1.64*
25.	Potassium (mmol/L)	4.7±0.16	3.97±0.24*
26.	Chloride (mmol/L)	110.05±1.85	109.53±1.121
27.	Calcium (mg/dl)	10.56±0.27	9.825±0.218*

*Significant at 5% (P<0.05); ** Significant at 1% (P<0.01); ***Significant at 0.1% (P<0.001)

The mean rectal temperature (101.01 ± 0.54), heart rate (120 ± 5.46) and respiratory rate (35.27 ± 2.43) of the dogs with renal dysfunction were non-significant compared to respective values of apparently healthy animals. Mean values of TEC (5.21 ± 0.42) were found to be significantly lower in dogs with renal dysfunction and are similar to the findings of Senthil *et al.* (2024) and Devipriya *et al.* (2018). Low values of TEC, PCV and Hb suggested dogs suffered from mild anaemia due to decreased erythropoietin production by diseased kidneys. These findings are in accordance with Silverberg *et al.* (2002). Mean values of ALT (110.78 ± 23.738) and creatinine (1.8 ± 0.306) were found to be significantly higher in the dogs with renal dysfunction and was similar to the observations of Devipriya *et al.* (2018). Sonu *et al.* (2019) reported that the increase in creatinine could be due to diminished renal excretion and enhanced tubular absorption of urea. The mean values of sodium (146.20 ± 1.64), potassium (3.97 ± 0.24) and calcium (9.825 ± 0.218) showed significant decrease as compared to healthy group.

Urine pH of 76.9% (10/13) dogs had acidic pH with (pH 5-6.5) whereas 23% (3/13) dogs had alkaline (pH=8). The mean values of urinary pH and specific

gravity were 6.42 ± 0.29 and 1.017 ± 0.002 respectively. These values are in accordance with Oburai *et al.* (2015). The decrease in urine specific gravity is a result of decreased concentrating ability of kidneys. The color of urine was colorless (30.76%), yellow (38.46%), red (15.38%) and brown (15.38%). Increased levels of urinary proteins in (23.07%) +++, (15.38%) ++, (7.6%) +, (23.07%) trace were observed which are similar to the findings of Sonu *et al.* (2024). UP:C ratio (1.38 ± 0.36) was found to be significantly increased which could be due to glomerular damage as observed by Oburai *et al.* (2015). Sonu *et al.* (2024) in his study reported that the most common cause for renal tubular injury in protein losing disease could be increased tubular uptake of filtered proteins or protein bound substances. Microscopic examination of urine of dogs with renal disease (Table 5) revealed presence of pus cells in 53.84% (7/13), increased number of RBCs in 38.46% (5/13) dogs (Plate 1). Crystaluria was found in 38.5% (5/13) dogs with struvite (triple phosphate) crystals in 4 dogs and bilirubin crystals in 1 dog (Plates 2 and 3). Epithelial cells were found in 69.23% (9/13) dogs (Plate 4). These findings are in accordance with Punia *et al.* (2018).

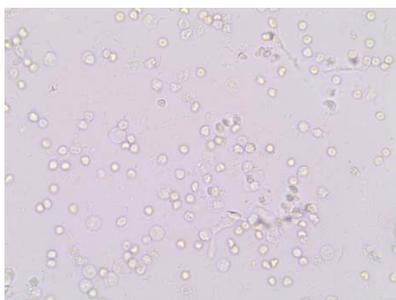


Plate 1. Abundant RBCs, WBCs along with few bacteria, 20X



Plate 2. Struvite crystal with typical coffin-lid appearance, 20X

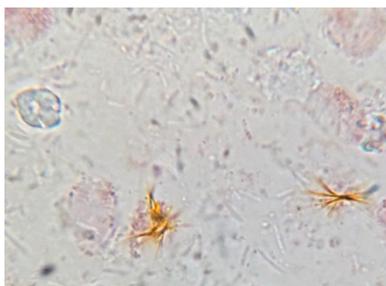


Plate 3. Bilirubin crystals characterised by yellow-brown granular appearance, 20X

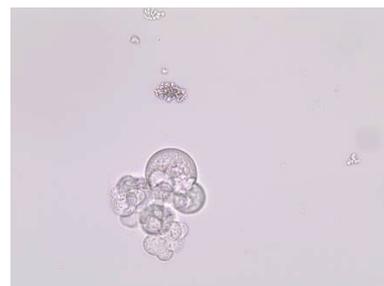


Plate 4. Cellular cast, containing variety of cells (Epithelial cells, WBCs), 20X

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References

- Athaley, A., Bhojne, G.R., Khanolkar, V.M., Dhoot, V.M., Upadhye, S.V. and Panchbhai, C.K., 2018. Urine analysis and ultrasonographic findings of dogs suffering from renal failure. *Journal homepage*, **7**(10).
- Devipriya, K., Lavanya, C., Selvaraj, P. and Napoleon, R.E. 2018. Early diagnosis of renal insufficiency in dogs with haemato: Biochemical findings. *Journal of Entomology and Zoology Studies*, **6**(5):703-5.
- Kumar, G.C., Kamran, C.A. and Ramesh P.T. 2020. Hematological Changes in Different Stages of Canine Chronic Kidney Disease. *International Journal of Livestock Research*, **10**(2):1.
- Sharma, A., Ahuja, A., Srivastava, M. and Kachhawa, J.P. 2015. Haemato-biochemical changes in Dogs suffering from chronic renal failure. *Indian Journal of Canine Practice*. **7**(2):102-7.
- Silverberg, D.S., Wexler, D., Blum, M., Tchebiner, J., Sheps, D., Keren, G., Schwartz, D., Baruch, R., Yachnin, T., Shaked, M. and Zubkov, A. 2002. The correction of anemia in severe resistant heart failure with erythropoietin and intravenous iron prevents the progression of both the heart and the renal failure and markedly reduces hospitalization. *Clinical Nephrology*, **58**:37-45.
- Sonu, A.K., Charaya, G., Bangar, Y., Agnihotri, D. and Kumar, T. 2019. Haemato-biochemical alterations in dogs suffering from chronic renal failure. *Indian Journal Veterinary Medicine*, **39**(1):31-5.
- Subapriya, S., Vairamuthu, S., Chandrasekar, M., Balagangatharathilagar, M., Ramesh, S., Areshkumar, M. and Thangaraj, M.J. 2020. Clinicopathological profile of canine renal disorders. *Journal of Entomology and Zoology Studies*, **8**(2):770-774.
- Oburai, L.N., Vaikunta Rao, V. and Naik, B.R., 2015. Clinical and nephrosonographic findings in canine chronic renal failure: A Prospective Study. *IOSR Journal of Agriculture and Veterinary Science*, **8**(6):11-16.
- Yogeshpriya, S., Pillai, U.N., Ajithkumar, S. and Unny, M. 2018. Clinico-haemato-biochemical profile of dogs with urinary tract infection: A retrospective study of 32 cases (2010-2012). *International Journal of Current Microbiology and Applied Sciences*, **7**(9):2797-2802.

Occurrence of Canine Periodontal Disease

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Abstract

Canine periodontal disease was one of the most common oral cavity diseases affecting both health and quality of life. The present study was conducted to record the occurrence of canine periodontal disease in relation to age, breed, sex and type of diet. The overall occurrence recorded in the present study was 83.44 per cent. The occurrence of canine periodontal disease in relation to age, breed and sex revealed highest in more than 8 years, smaller breeds of dogs and in male dogs. The occurrence in relation to diet revealed highest in dogs fed with soft diet when compared with to those fed with mixed and hard diets.

Keywords: periodontal disease, occurrence, age, breed, diet

Periodontal disease (PD) was one among the most widespread disease of oral cavity affecting 44 to 80 per cent of dogs (Jeusette *et al.*, 2016). Periodontal disease was a plaque-inducing disease affecting any part of the periodontium i.e., gingiva, cementum, periodontal ligament and alveolar bone (Nabi *et al.*, 2014) and bacterial biofilm was mainly responsible for the development of disease (Hasan and Palmer 2014).

The present study on occurrence of periodontal disease was conducted in Veterinary Clinical Complex, NTR College of Veterinary Science, Gannavaram from May 2024 to December 2024. A total of 302 dogs with an age of more than one year were screened for recording the occurrence of periodontal disease based on clinical signs like gingivitis, halitosis, drooling of saliva, deposition of calculus, discomfort on eating, gingival bleeding and pawing the mouth. The occurrence of canine periodontal disease (CPD) was further analysed in relation to age, breed, gender and diet.

In the present study, the occurrence of canine periodontal disease recorded was 83.44 per cent (252/302) in relation with the number affected to the number screened in per cent from May 2024 to December 2024 (Table 1). Age wise occurrence of canine periodontal disease revealed that dogs of more than 8 years age group (43/46, 93.47%) were most commonly affected followed by 4 to 8 years age group

(116/138, 84.05%) and least in dogs aged between 1 to 4 years (93/118, 78.81 %).

Occurrence of canine periodontal disease recorded in the present study was in concurrence with that of Shearer (2009), Kouki *et al.* (2013) and Eyarefe *et al.* (2014) who reported the occurrence as 85.00, 80.00 and 82.46 per cent respectively. The present findings are in partial agreement with Ranjan *et al.* (2010), Oba *et al.* (2018) and Kumar and Xaxa (2021) who recorded the occurrence as 68.90, 60.00 and 59.67 percent respectively. Sarangamath *et al.* (2022) reported that the variation in the occurrence of canine periodontal disease documented by different workers might be due to the differences in sampling size, age, food habits, concurrent disease, owner awareness and adaptability of preventive measures which had a direct influence the accumulation of plaque and calculus. The present findings are almost in agreement with Berryhill (2005), Ray and Eubanks (2009) and Garanayak *et al.* (2019) who reported that approximately 75-85 per cent of dogs exhibit some form of periodontal disease by two years of age.

In the present study the highest occurrence of CPD was seen in Shih tzu (25/25, 100%) followed by Pomeranian (24/25, 96.00%), Spitz (21/22, 95.45%), Pug(18/19, 94.74%), Labrador Retriever (34/36, 94.44%), Mongrel (42/46, 91.30%), Golden Retriever (25/29, 86.21%), German Shepherd (18/21, 85.71%), Beagle (14/18, 77.78%), Rottweiler (11/20, 55.00%), Belgian Malinois (12/24, 50.00%) and least in Great Dane (8/17, 47.06%). Breed wise occurrence

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of periodontal disease in the present study was in agreement with Debowes (2010), Ranjan *et al.* (2010) and Khatariya *et al.* (2020) who reported that small, toy and brachycephalic breeds were more susceptible for the development of periodontal disease due to the overcrowding and rotation of teeth. Hendy *et al.* (2022)

reported that malocclusion and overcrowding of teeth in smaller breeds of dogs increased the occurrence of PD when compared with larger breeds of dogs. Shewale *et al.* (2021) reported that overcrowding resulted in the development of more crevices leading to the accumulation of dental plaque.

Table I: Occurrence of canine periodontal disease ase

S.No	No. of dogs screened	No. affected with CPD	Percentage affected with CPD out of No. Screened
1.	302	252	83.44

Table II: Age wise occurrence of canine periodontal dise

S.No.	Age group	No. of dogs examined (n=302)	No. of dogs affected with CPD (n=252)	Percentage affected with CPD out of the dogs examined
1.	1 to 4 years	118	93	78.81
2.	4 to 8 years	138	116	84.05
3.	8 years and above	46	43	93.47
Total		302	252	

The present study revealed highest percentage of occurrence in male dogs (137/159, 86.16%) when compared with female dogs (115/143, 80.42%). The occurrence of periodontal disease in the present study and was in accordance with Vani *et al.* (2007) and Kumar *et al.* (2008) who recorded higher occurrence in male dogs when compared to female dogs. Bhardwaj *et al.* (2021) reported that the higher occurrence of periodontal disease in the present study might be due to the preference of rearing male dogs over female dogs in the area under study.

CPD in relation to diet in the present study revealed highest occurrence in dogs fed with soft diet (148/156, 94.87 %) followed by mixed diet (67/93, 72.04%) and least in dogs fed with dry diet (37/53, 69.81 %). The present study findings are in accordance with Kyllar and Witter (2005), Gawor *et al.* (2006) and Oba *et al.* (2018) who found that feeding on soft diet increased the frequency and severity of periodontal disease. Cunha *et al.* (2022) reported that when dogs were fed with soft diet mechanical abrasion on the dental surface decreases resulting in the accumulation of plaque and calculus. Ranjan *et al.* (2010) opined that when dogs were fed with dry food, chewing results in the mechanical disruption of the accumulated plaque and promotes the self cleaning action while Bjone *et al.*

(2005) reported that chewing stimulated the production of saliva which contains antibacterial agents that helps in keeping the mouth clean.

References

- Berryhill, S. 2005 The complete dental” prophylaxis”. *The North American Veterinary Conference – 2005 Proceedings*:7-8.
- Bhardwaj, V., Chaudhary, R. N., Kumar, A. and Tiwari, D. K. 2021 Clinical effects of enrofloxacin and oflox-ornidazole on periodontal diseases in dogs. *Haryana Veterinarian* **60 (SI)**:16-20.
- Cunha, .E, Tavares, L. and Oliveira, M. 2022 Revisiting periodontal disease in dogs: how to manage this new old problem?. *Antibiotics*, **11(12)**: 1729.
- Debowes, L. J. 2010 Problems with the gingiva. *Small animal dental, oral and maxillofacial disease, A color handbook* pp. 159-181.
- Garanayak, N., Das, M., Patra, R. C., Biswal, S. and Panda, S. K. 2019. Effect of age on dental plaque deposition and its control by ultrasonic scaling, dental hygiene chew, and chlorhexidine (0.2% w/v) in dogs. *Vet. World*, **12(11)**: 1872.
- Gawor, J. P., Reiter, A. M., Jodkowska, K., Kurski, G., Wojtacki, M. P. and Kurek, A. 2006. Influence of diet

- on oral health in cats and dogs. *J. Nutrition* **136(7)**: 2021S-2023S.
- Hasan, A. and Palmer, R. M. .2014. A clinical guide to periodontology: pathology of periodontal disease. *British Dental Journal*, **216(8)**: 457-461.
- Hendy, E. A., Elseddawy, F., Behery, A. E. and Ezzeldein, S.A. 2022. A retrospective study on periodontal diseases in companion animals. *Zagazig Veterinary Journal*, **50(3)**: 255-263.
- Jeusette, I .C., Román, A. M., Torre, C., Crusafont ,J., Sánchez, N., Sánchez, M. C. and Herrera, D. 2016. 24-hour evaluation of dental plaque bacteria and halitosis after consumption of a single placebo or dental treat by dogs. *Am. J. Vet. Res.*, **77(6)**: 613-619.
- Khatariya, M. D., Talekar, S. H., Dodia, V. D., Ahlawat, A. R. and Kalaria, V. A. 2020 Periodontal diseases and their surgical management in dogs. *Indian J. Vet.Sci. Biotechnol.*, **15(4)**: 19-23.
- Kouki, M. I., Papadimitriou, S. A., Kazakos, G. M., Savas, I. and Bitchava, D. 2013. Periodontal disease as a potential factor for systemic inflammatory response in the dog. *Journal of Veterinary Dentistry*, **30(1)**: 26-29.
- Kumar, V., Kelawala, N., Patil, D. B., Parikh, P. V. and Barvalia, D. R. 2008. Epidemiological studies on periodontal disease in dogs. *Indian J. Vet. Sur.*, **29(2)**:112-113
- Kyllar, M. and Witter, K. 2005. Prevalence of dental disorders in pet dogs. *Veterinarni Medicina-Praha-* **50(11)**: 496.
- Nabi, S.. U, Wani, A. R., Sha,h O. S. and Dey, S. 2014. Association of periodontitis and chronic kidney disease in dogs. *Vet. World*, **7(6)**:403-407.
- Oba, P. M., Rentas, M. F., Vendramini, T. H. and Brunetto, M. A. 2018. Nutrition as a tool to control periodontal diseases in dogs and cats. *Nutrition Food Science*, **24(4)**: 236-239.
- Ranjan, R., Zahid, U. N., Gupta, .D K., Bansal, B. K. and Dua, K. 2010. An epidemiological study on periodontal diseases in dogs-a clinical study of 103 canine patients. *Intas Polivet*, **11(2)**: 274-277.
- Ray, Jr. J. D. and Eubanks, D. L. 2009. Dental homecare: teaching your clients to care for their pet's teeth. *Journal of Veterinary Dentistry*, **26(1)**: 57-60.
- Shearer, P. 2010. Periodontal literature review. *Banfield Applied Research and Knowledge*, Oregon pp.1-8.
- Vani, G., Haragopal, V., Kumar, R .V. S. and Rao, T. S. C. 2007. Incidence of dental affections in dogs. *Indian Vet. J.*, **84(9)**: 974-975.

Outbreak of Newcastle Disease in a Turkey Farm in Andhra Pradesh

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Abstract

The present study reports incidence of Newcastle disease in turkeys housed in an integrated avian farm. Mortality recorded was more than 75% and noticed in both young and adult birds. Infected birds exhibited clinical signs including anorexia, depression, greenish diarrhea along with nervous signs few hours before death. Newcastle disease was confirmed by haemagglutination inhibition test and RT-PCR targeting partial fusion gene. All the infected birds were immediately segregated from the other birds and treated appropriately. This incidence highlights the need to vaccinate all avian species including chicken against Newcastle disease.

Keywords: Newcastle disease; Turkeys; HI; RT-PCR;

Newcastle disease (ND) is a highly infectious viral disease of avian species caused by virulent strains of Avian Paramyxovirus-1 (APMV-1). Wide variety of avian species have been reported to be susceptible to ND (Kaleta and Baldauf, 1988). Severity of the disease and the associated consequences, led to recognition of ND as OIE list A disease (Office Internationale des Epizooties, 2004). Turkeys are considered to be highly resistant to experimental infection with NDV (Tsai *et al.*, 1992; Alexander, 2000). Here we report an incidence of Newcastle disease in turkeys reared in an integrated avian farm in Tanuku, West Godavari district, Andhra Pradesh.

A total of 65 turkeys of different age groups were housed along with other avian species such as chicken, pigeons and quails during November, 2018. The clinical signs were initially noticed in five-month-old poults which included inappetance, depression, greenish diarrhea and nervous signs like neck and leg paralysis (Fig 1). Nervous signs became severe few hours before death. More than 90 percent of the birds exhibited similar clinical signs.

However, average mortality recorded in the poults and adult turkeys was 75 percent (49/65). Postmortem examination revealed focal hemorrhages and necrotic foci in the caecal tonsils and intestines, hemorrhages at the tips of proventricular glands, diffuse congestion in the viscera including kidneys, heart, lung and trachea (Fig 2). During necropsy, samples

viz., proventriculus, lung, kidneys, intestine (including contents), caecal tonsils, spleen, brain, liver and heart tissues as a pool were collected from each bird. The tissues were processed and inoculated into 9-day old embryonated chicken eggs through allantoic route. Embryos that died after 24 h of inoculation were harvested and the allantoic fluid was tested for haemagglutination. Presence of NDV was confirmed by haemagglutination inhibition (HI) test employing NDV- LaSota hyper immune serum (OIE, 2012). Reverse transcription PCR employing universal primer set targeting hyper variable region of the fusion gene *i.e.*, fusion protein cleavage site (FPCS) (Nantha Kumar *et al.*, 2000) was also used for confirmation of NDV.

NDV was isolated from tissues collected during necropsy. Embryos dead after 24 h were harvested and observed for typical lesions which include peri occipital hemorrhages and diffuse congestion of the embryos. As a preliminary test, amnio allantoic fluid (AAF) collected from each embryo was tested by spot haemagglutination test. AAF positive by spot haemagglutination test was quantified by microtitre haemagglutination assay and the titre was found to be 1 in 2⁸. Haemagglutination activity of the sample was specifically inhibited by LaSota specific hyper immune serum in HI test. For RT-PCR, total RNA was extracted from the HA positive amnio allantoic fluid by TRIzol method. In parallel, RNA was also extracted from LaSota vaccine and allantoic fluid of uninoculated embryo are used as positive and negative controls respectively. The cDNA concentration was measured and adjusted to a minimum concentration of 100 ng and then used as template in PCR. Partial fusion

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gene with an amplicon size of 356 bp was amplified as visualized in 2 per cent agarose gel containing ethidium bromide along with 100 bp DNA ladder (Fig 3).

Besides chicken, Newcastle disease was reported in different avian species such as pigeons, cormorants, psittacines, pheasants, peafowl, wild waterfowl and shorebirds (Pearson and McCann, 1975; Garcia *et al.*, 2013). Turkeys are generally considered resistant to ND infection (Alexander, 2001). In the present investigation, seventy five per cent of the affected turkey flock succumbed to infection. Several authors previously reported that mortality can easily

reach 100 per cent in viscerotropic velogenic Newcastle disease and neurotropic velogenic Newcastle disease outbreaks (Aldous and Alexander, 2001, Cattoli *et al.*, 2011 and Ganar *et al.*, 2014). Balachandran *et al.* (2014) reported 3-7 per cent drop in egg production in ND affected flocks. Integrated or mixed poultry farming is a new trend resulting in good returns to the farmers. Nevertheless, there is every chance for spill over of pathogens among different avian species. Furthermore, vaccination against ND is focused and limited only to the commercial poultry sector which makes other avian species vulnerable.



Fig. 1: Turkey poult exhibiting leg and neck paralysis along with greenish diarrhea



Fig. 2: Postmortem lesions- Necrotic foci in the caecal tonsils, haemorrhages on the proventricular glands and visceral congestion.

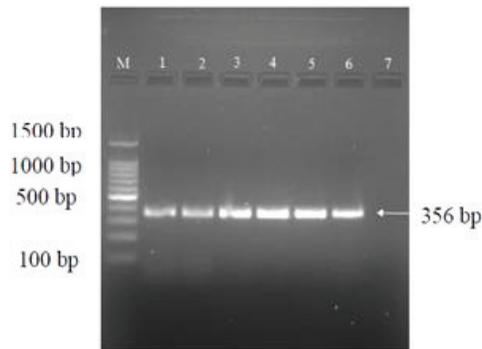


Fig. 3: RT-PCR for partial fusion gene. M- 100 bp marker; Lanes 1-5: Positive samples; Lane 6: Positive control; Lane 7: Negative control

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References

- Alders, R.G. 2014. Making Newcastle disease vaccines available at village level. *Vet. Rec.* **174** (20), 502-503.
- Aldous, E.W. and Alexander, D.J. 2001. Detection and differentiation of Newcastle disease virus (avian paramyxovirus type 1). *Avian Pathol.* **30**(2): 117-128.
- Alexander, D.J. 2000. Newcastle disease and other avian paramyxoviruses. *Revue Scientifique et Technique-Office International des Epizooties*, **19**(2): 443-462.
- Balachandran, P., Srinivasan, P., Sivaseelan, S., Balasubramaniam, G.A. and Murthy, T.G.K. 2014. Isolation and characterization of Newcastle disease virus from vaccinated commercial layer chicken. *Vet. World.* **7**(7):457-462.
- Cattoli, G., Fusaro, A., Monne, I., Molia, S., Le Menach, A., Maregeya, B., Nchare, A., Bangana, I., Maina, A.G., Koffi, J.N., Thiam, H., Bezeid, O.E., Salviato, A., Nisi, R., Terregino, C. and Capua, I. 2010. Emergence of a new genetic lineage of Newcastle disease virus in West and Central Africa – Implications for diagnosis and control. *Vet. Microbiol.* **142** (3-4): 168–176.
- Dimitrov, K.M., Abolnik, C., Afonso, CL., Albina, E., Bahl, J., Berg, M., Briand, F.X., Brown, I.H., Choi, K.S., Chvala, I. and Diel, D.G. 2019. Updated unified phylogenetic classification system and revised nomenclature for Newcastle disease virus. *Infect. Genet. Evol.* **74**:103917.
- Ganar, K., Das, M., Sinha, S. and Kumar, S. 2014. Newcastle disease virus: current status and our understanding. *Virus res.* **184**: 71-81.
- Garcia, C.S., Lopez, R.N., Morales, R., Olvera, M.A., Marquez, M.A., Merino, R., Miller, P.J. and Afonso, C.L. 2013. Molecular epidemiology of Newcastle disease in Mexico and the potential spillover of viruses from poultry into wild bird species. *Appl. Environ. Microbiol.* **79** (16): 4985–4992.
- Kaleta, E.F and Baldauf, C. 1988. Newcastle disease in free-living and pet birds. In: Newcastle disease , Springer, Boston, MA. pp 197-246.
- Miller, P. J. and Koch, G. 2013. Newcastle disease. *Dis. Poult.* **13**:89–138.
- Nantha kumar, T., Kataria, R.S., Tiwari, A.K., Butchaiah, G. and Kataria J.M. 2000. Pathotyping of Newcastle disease viruses by RT-PCR and restriction enzyme analysis. *Vet. Res. Commun.* **24**(4): 275-286.
- OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. 2004. Newcastle disease. Chapter 2.1.15. <http://www.oie.int>.
- OIE Manual of diagnostic tests and vaccines for terrestrial animals: mammals, birds and bees, 2012. Newcastle disease. Biological Standards Commission. World Organisation for Animal Health, Paris, France. 555–574.
- Pearson, G. L. and Mccann, M. K. 1975. The role of indigenous wild, semi domestic, and exotic birds in the epizootiology of velogenic viscerotropic Newcastle disease in southern California, 1972-1973. *J. Am. Vet. Med.* **167**(7): 610-614.
- Taxonomy IV. Release. 2019. International committee on taxonomy of viruses. v/ msw054.
- Tsai, H.J., Saif, Y.M., Nestor, K.E., Emmerson, D.A. and Patterson, R.A. 1992. Genetic variation in resistance of turkeys to experimental infection with Newcastle disease virus. *Avian diseases*, pp.561-565.

Prevalence of Mast Cell Tumour in Dogs in Chennai

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Abstract

The present study was carried out to assess the prevalence of mast cell tumours in dogs presented at Madras Veterinary College Teaching Hospital, Chennai for five months (July 2024- November 2024). A total of 802 cytological smears prepared each from the nodule of different breeds of dogs were referred to the Centralised Clinical Laboratory, Madras Veterinary College, Chennai for cytological examination. The overall prevalence of mast cell tumour was found to be 2.86 %, of which, females showed a higher prevalence (56.52 %) compared to males (43.47%). Among various age groups affected, an increased prevalence was recorded in dogs belonging to more than six years of age (56.52%), followed by 0-3 years (26.08%) and 4-6 years (17.39%). Non-descript breeds showed the highest prevalence (52.17 %), followed by Labrador Retriever (34.78%), and Golden Retrievers, Boxer and Pug (4.34 % each).

Keywords: Chennai, Dog, Mast cell tumour, Prevalence,

Mast cell tumour (MCT) also known as mastocytoma, the most common haematopoietic neoplasm accounts for 16 to 21% of all cutaneous tumours encountered in dogs (Pierini *et al.*, 2019). The tumour composed of mast cells appears as small, demarcated, single or multiple growths that may infiltrate the adjacent tissues and metastases to the distant organs, namely lymph nodes, skin, spleen, liver, and lungs. MCTs can develop in any part of the body but mostly affect the torso, limbs, head, and neck. Research data about the epizootological studies on MCT in dogs are scanty. Hence the present work was undertaken to study the prevalence associated with MCT in dogs in Chennai.

A total of 802 cytological smears were prepared each from various breeds of dogs presented at Madras Veterinary College Teaching Hospital, Chennai were referred to the Centralised Clinical Laboratory, Madras Veterinary College, Chennai for cytological examination. The present study was conducted for five

months (July 2024- November 2024). The cytological smears were air dried and stained with Leishman and Giemsa cocktail stain for microscopic examination as described by Garbyal *et al.* (2006). The risk factors such as age, sex, and breed associated with MCT in dogs in Chennai were analyzed.

On microscopic examination of 802 smears, 23 samples showed cytological features suggestive of mast cell tumour (Fig. 1&2) with an overall prevalence of 2.86%.

MCT was observed in 13 females and 10 males, and thus females revealed a higher prevalence (56.52%) than males (43.47%). The highest prevalence was recorded in dogs aged more than 6 years (n=13;56.52%), followed by 0-3 years (n=6 ; 26.08%) and 4-6 years (n=4; 17.39%). MCT was observed in 12 non-descript breeds (52.17%), eight Labrador Retrievers and Golden Retrievers, Boxer and Pug (4.34 %). each.

The present study recorded an overall prevalence of 2.86 %. In contrast to the present findings, Smiech *et al.* (2018) reported a high prevalence of 17.8% of all

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skin tumours examined in dogs and 16 to 21% of all cutaneous tumours encountered in dogs (Pierini *et al.*, 2019). There is no association observed in the occurrence of MCT among the sexes in the present findings agreed with that of Shoop *et al.* (2015). The highest prevalence observed in dogs aged more than six years during the present study agrees with the observations of Śmiech *et al.* (2018) who also noticed an increased incidence in older dogs compared to the younger ones. Similar findings were also recorded by Pierini *et al.* (2019) who also observed the median age of MCT in dogs was 6.22 years. An increased prevalence of MCT in non-descript

breeds in this study was in accordance with that of Pierini *et al.* (2019). Śmiech *et al.* (2018) reported the highest prevalence in Labrador Retriever, Boxer, French Bulldog, American Staffordshire Terrier, and Shar-pei while the lowest incidence in Cocker Spaniel, German Shepherd, and Yorkshire Terrier. The Boxer and Bulldog related breeds including Bullmastiffs, Boston Terriers and Staffordshire Bull Terriers were found to be more susceptible to MCT (Mochizuki *et al.*, 2017) and the breed predisposition to develop MCT might be because these breeds of dogs belong to the same phylogenetic tree (Peters, 1969).

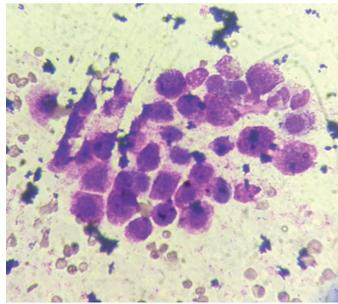


Fig. 1: Cytology- Dog –MCT L&G stain x100

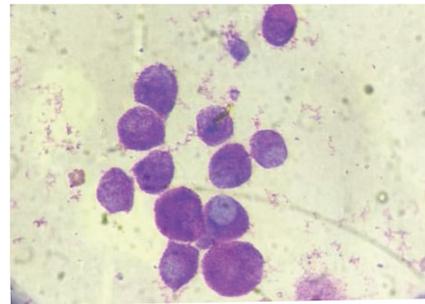


Fig. 2: Cytology- Dog –MCT L&G stain x40

References

- de Nardi, A.B., Dos Santos Horta, R., Fonseca-Alves, C.E., de Paiva, F.N., Linhares, L.C.M., Firmo, B.F., Ruiz Sueiro, F.A., de Oliveira, K.D., Lourenço, S.V., De Francisco Strefezzi, R., Brunner, C.H.M., Rangel, M.M.M., Jark, P.C., Castro, J.L.C., Ubukata, R., Batschinski, K., Sobral, R.A., da Cruz, N.O., Nishiya, A.T., Fernandes, S.C., Dos Santos Cunha, S.C., Gerardi, D.G., Challoub, G.S.G., Biondi, L.R., Laufer-Amorim, R., de Oliveira Paes, P.R., Lavalle, G.E., Huppel, R.R., Grandi, F., de Carvalho Vasconcellos, C.H., Dos Anjos, D.S., Luzo, Â.C.M., Matera, J.M., Vozdova, M. and Dagli, M.L.Z. 2022. Diagnosis, Prognosis and Treatment of Canine Cutaneous and Subcutaneous Mast Cell Tumors. *Cells*, **11**:618.
- Garbayl, R.S., Agarwal, N. and Kumar, P. 2006. Leishman-Giemsa cocktail, an effective Romanowsky stain for air dried cytologic smears. *Acta Cytol.*, **50**:403-406.
- Mochizuki, H., Motsinger-Reif, A., Bettini, C., Moroff, S. and Breen, M. 2017. Association of breed and histopathological grade in canine mast cell tumours. *Vet Comp Oncol.*, **15**(3):829-839.
- Peters, J.A. 1969. Canine mastocytoma: excess risk as related to ancestry. *J Natl Cancer Inst.*, **42**(3):435-43.
- Pierini, A., Lubas, G., Gori, E., Binanti, D., Millanta, F. and Marchetti, V. 2019. Epidemiology of Breed-Related Mast Cell Tumour Occurrence and Prognostic Significance of Clinical Features in a Defined Population of Dogs in West-Central Italy. *Vet Sci.*, **6**(2):53.
- Shoop, S.J., Marlow, S., Church, D.B., English, K., McGreevy, P.D., Stell, A.J., Thomson, P.C., O'Neill, D.G. and Brodbelt, D.C. (2015). Prevalence and risk factors for mast cell tumours in dogs in England. *Canine Genet Epidemiol.*, **2**:1.
- Śmiech, A., Ślaska, B., Łopuszyński, W., Jasik, A., Bochyńska, D. and Dąbrowski, R. 2018. Epidemiological assessment of the risk of canine mast cell tumours based on the Kiupel two-grade malignancy classification. *Acta Vet Scand.*, **60**(1):70.

Use of Continuous Glucose Monitoring system in Canine Diabetes

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Abstract

Glycemic control is crucial for managing diabetes mellitus (DM) and preventing complications. Continuous glucose monitoring systems (CGMS) are commonly used in humans with DM, providing real-time glucose levels and detecting hyperglycemic and hypoglycemic episodes. CGMS are increasingly prescribed to diabetic dogs and cats due to their well-tolerated nature. This article describes the use of CGMS in a dog with diabetes.

Keywords: Dog- diabetes- CGMS continuous glucose monitoring system

Glycemic control is crucial for managing diabetes mellitus (DM) and preventing complications in both human and veterinary medicine. Continuous glucose monitoring systems (CGMS) are commonly used in humans with DM, and clinical studies have shown their effectiveness in optimizing and titrating the insulin dosing and management of DM. CGMS measures interstitial concentration using a flexible sensor inserted into the skin. They provide real-time sensor

glucose levels and allow detection of hyperglycemic and hypoglycemic episodes that might otherwise be undetected. The article describes use of CGMS in canine DM.

The present study was initiated after permission from the Institutional Ethics Committee for Veterinary Clinical Research (IEC-VCR) of Mumbai Veterinary College, Maharashtra Animal and Fishery Sciences University (MAFSU), Mumbai-India.

Sl No.	Patient details	Chief complaints	Laboratory investigations
1	Age: 05 years, 9 months; Sex: Male; Breed: Labrador; Body weight: 38 kg.	Polydipsia and polyuria.	Fasting blood glucose: 501 mg/dl; fructosamine: 449.02 µmol/L Basal cortisol: 92.96 nmol/L TT ₄ =43.76 nmol/L. CBC, LFT, and KFT: unremarkable. Urinalysis: glycosuria but nil urine ketones

The CGMS does not require calibration, is accurate, and well tolerated by dogs, its placement was undertaken as per the methods outlined by Tardo *et al.* (2024). A small patch of hair (approximately 3cm x 3cm) is shaved on the lateral thoracic region. The skin was cleaned of excess hair and wiped with an alcohol swab. The sensor and attached stylet were inserted through the skin. After placement of the sensor, the reading with the mobile software was undertaken after one hour. In the present clinical case, installation of the sensors proceeded without any pain reaction, and after removal of the sensor, no skin erythema was observed. The installation of CGMS and its reading

are presented in Fig 1 to 3. Interstitial glucose data was automatically calculated by CGMS on the mobile application software. In 14 days, the insulin dose was titrated. Patient was stabilized (range of blood glucose 160 mg/dl to 250 mg/dl) with a soluble insulin dose of 10 units twice a day and 14 units of insulin glargine (100 IU/ml) at bedtime. CGMS wireless system provides a continuous display of interstitial glucose concentration, minimizing patient handling needs. It enabled clinicians to detect trends and quickly identify glucose fluctuations without repeated phlebotomies. The other advantages of this device are, it has an 8-hour window, connects three devices (patient, caretaker, and physician), and alarm alerts for hypoglycaemia. CGMS is a well-tolerated and pet-friendly tool for monitoring glucose levels at

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home. However, the CGMS for veterinary patients has limitations, including an inability to record glucose concentrations and complete data when detached, necessitating further research for accurate diabetes management.

Conflict of Interest declaration

The authors declare no conflict of interest.



Fig. 1: Placement of CGMS in a dog at the lateral thoracic region



Fig. 2: CGMS sensor

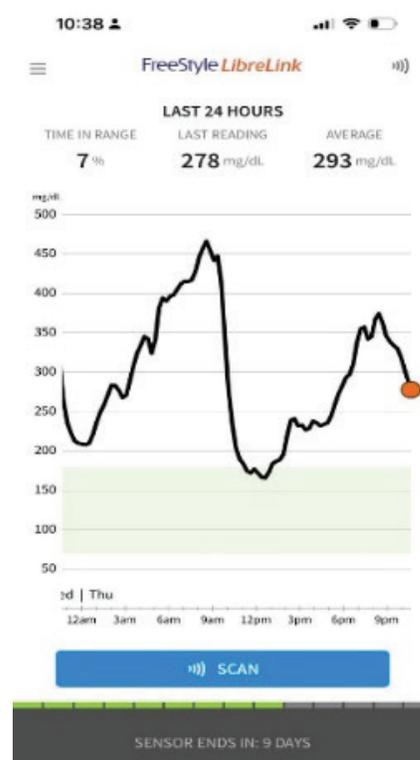
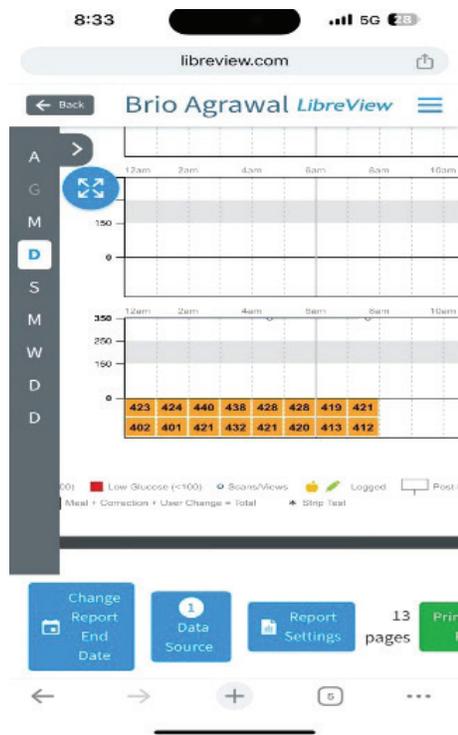


Fig. 3: CGMS reading on mobile app during insulin dose titration

References

- Corradini, S., Pilosio, B., Dondi, F., Linari, G., Testa, S., Brugnoli, F., Gianella, P., Pietra, M. and Fracassi, F. 2016. Accuracy of a flash glucose monitoring system in diabetic dogs. *J. Vet. Intern. Med.*, **30**: 983–988.
- Deiting, V. and Mischke, R. 2021. Use of the “FreeStyle Libre” glucose monitoring system in diabetic cats. *Res. Vet. Sci.*, **135**: 253–259.
- Del Baldo, F., Fracassi, F., Pires, J., Tardo, A.M., Malerba, E., Manassero, E. and Gilor, C. 2021. Accuracy of a flash glucose monitoring system in cats and determination of

- the time lag between blood glucose and interstitial glucose concentrations. *J. Vet. Intern. Med.*, **35**: 1279–1287.
- Malerba, E., Cattani, C., Del Baldo, F., Carotenuto, G., Corradini, S., Golinelli, S., Drudi, I. and Fracassi, F. 2020. Accuracy of a flash glucose monitoring system in dogs with diabetic ketoacidosis. *J. Vet. Intern. Med.*, **34**: 83–91.
- Surman, S. and Fleeman, L. 2013. Continuous glucose monitoring in small animals. *Vet. Clin. N. Am. Small Anim. Pract.*, **43**: 381–406.
- Tardo, A.M., Fleeman, L.M., Fracassi, F., Berg, A.S., Guarino, A.L. and Gilor, C. 2024. A dose titration protocol for once-daily insulin glargine 300 U/mL for the treatment of diabetes mellitus in dogs. *J. Vet. Intern. Med.*, **38**(4):2120-2128.
- Wiedmeyer, C.E. and Declue, A.E. 2008. Continuous glucose monitoring in dogs and cats. *J. Vet. Intern. Med.*, **22**: 2–8.

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Allergic dermatitis in a German Shephard dog in association with *Toxocara canis* infection

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Abstract

A six-month-old German Shephard was brought to the clinic with recurrent skin lesions, pruritus for a period of two months. Upon clinical examination, erythematous patches and crusty lesions were noticed on the ventral abdomen. Microscopic examination of stained tape impression smears showed the presence of neutrophils, eosinophils and cocci. Unembryonated eggs of *Toxocara canis* were found on faecal sedimentation technique. Elevated levels of serum alkaline phosphatase and alanine aminotransferase were noticed. Dog was treated with oral cyclosporine, omega-3 and omega-6 fatty acids, pyrantel pamoate, hepato-protective syrup and external application of fluticasone propionate with mupirocin ointment. Following 21 days of treatment, noticeable clinical improvement was noticed until nine months of post therapy. Allergic dermatitis due to toxocariasis in a dog is reported in this article.

Keywords: *Toxocara canis*, Dog, Dermatitis, Pyrantel pamoate, Cyclosporine

Toxocariasis caused by the *Toxocara canis* is a zoonotic intestinal parasite of dogs. Dogs can become infected through transmammary, transplacental, by eating food contaminated with *Toxocara canis* ova, ingestion of paratenic hosts and less commonly by cutaneous route (Nijse *et al.*, 2016). In dogs, it may result in subclinical or clinical infections that worsen up to mortality. The adult worm load and the host's immune condition determine the clinical manifestation of toxocariasis. Allergic dermatitis due to *Toxocara canis* infection has been rarely documented.

A six-month-old German Shephard was brought to the clinic with a history of recurrent skin lesions, pruritus for the period of two months which had responded well to the therapy and recurrence noticed after completion of therapy. Clinical examination revealed erythematous patches and crusty lesions in the inner thigh and ventral abdomen. Microscopic examination of methylene blue stained tape impression smears showed the presence of neutrophils, eosinophils and cocci. Unembryonated eggs of *Toxocara* spp. were discovered upon sedimentation faecal smear analysis. Haemato-biochemical evaluation showed haemoglobin 11.5 g/dL, packed cell volume 34.5%, total erythrocyte count 5.64 million/cumm, total leucocyte count 14660 cells/cumm, percentage of neutrophils 70%, percentage of lymphocytes 18%, percentage of monocytes 2%, percentage of eosinophils

10%, serum albumin 1.77 g/dL, alkaline phosphatase 128 IU/L, alanine aminotransferase 212 IU/L. The clinical condition was determined to be toxocariasis in association with the allergic dermatitis. The dog was treated with oral cyclosporine (with initial dose of 5 mg/kg body weight twice daily followed by gradual tapering for a period of 14 days), syrup containing omega 3 and 6 fatty acids (@ 5 ml twice daily orally for one month), three doses of suspension pyrantel pamoate (@ 10 mg/kg body weight), syrup containing the hepato-protective supplement (@ 5 ml twice daily orally for a month) and external application of ointment containing the fluticasone propionate (0.005%) and mupirocin (2.0%) until resolution of skin lesions. The dog showed marked recovery after 10 days of therapy and complete clinical recovery was noticed by 21 days of therapy. There was no recurrence was noticed until nine months of post therapy (Fig. 1A, 1B, 1C and 1D).

Present case report about the possible role of *Toxocara canis* in inducing chronic allergic dermatitis. The causative role of *Toxocara canis* in development of dermatitis is being supported by eosinophilia and elevated hepatic enzymes in the present study. Pinelli and Aranzamendi (2012) reported that dermatological lesions consist of sensitization phase with allergic reaction activated by the innate immunity and further induces the inflammation, recruitment of leukocytes and further activation of resident and dendritic cells. Gavignet *et al.* (2008) reported that CD8+

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cells produced cytokines and caused the keratinocyte apoptosis, CD4⁺ T cells down regulated the allergic dermatitis by controlling the expansion of CD8⁺ T cells in the lymphoid organs and their activation in the skin. *Toxocara canis* infected dogs exhibited higher levels

of IgG and IgE levels and noticed the development of skin lesion scores and pruritus (Fischer *et al.*, 2018). Further research studies are warranted to record the role of *Toxocara canis* infection in associate with the dermatological lesions in dogs.

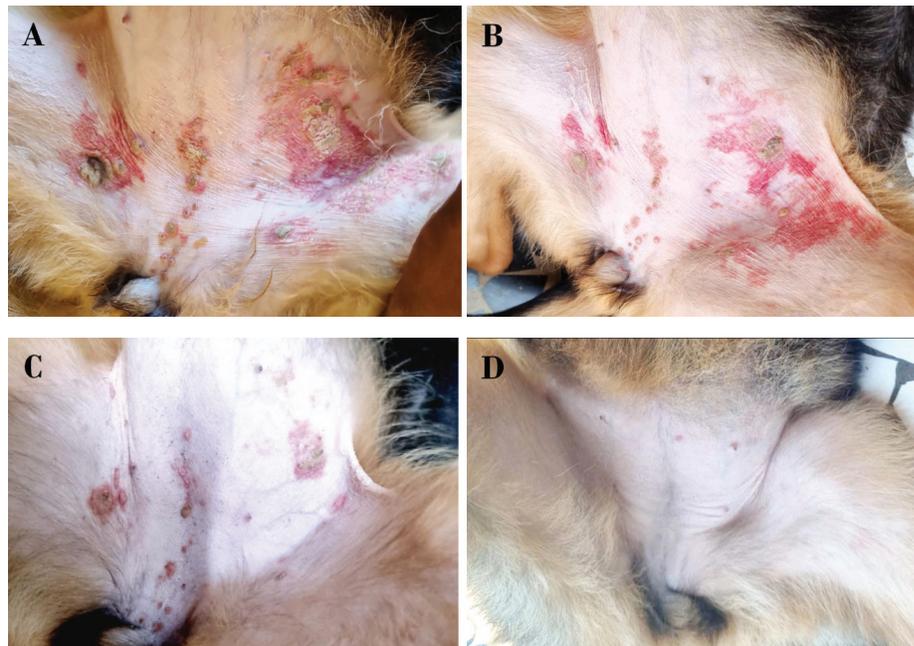


Fig. 1: Clinical response to the therapy (0th; 3rd; 10th; 21st day of post therapy)

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References

- Fischer, N., Rostaer, A., Zwickl, L., Deplazes, P., Olivry, T. and Favrot, C. 2018. A *Toxocara canis* infection influences the immune response to house dust mite allergens in dogs. *Vet. Immunol. Immunopathol.*, **202**:11-17.
- Gavignet, B., Piarroux, R., Aubin, F., Millon, L. and Humbert, P. 2008. Cutaneous manifestations of human toxocariasis. *J. Am. Aca. Dermatol.*, **59** (6): 031–1042.
- Nijse, R., Mughini-Gras, L., Wagenaar, J.A. and Ploeger, H.W. 2016. Recurrent patent infections with *Toxocara canis* in household dogs older than six months: a prospective study. *Parasites and Vectors*, **9**: 531.
- Pinelli, E. and Aranzamendi, C. 2012. *Toxocara* infection and its association with allergic manifestations. *Endocrine, Metabolic & Immune Disorders: Drug Targets*, **12** (1): 33–44.
- Reddy, B.S. and Sivajothi, S. 2016. Recurrent *Malassezia* dermatitis due to hypothyroidism in a dog and its management. *Comp. Clinical Pathol.*, **25**: 531–533.

Occurrence of Mitral Value disease in Large Breed Dogs

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Abstract

The present study aimed to investigate occurrence of Mitral valve disease (MVD) a common acquired heart disease, in large breed dogs in Gannavaram, Andhra Pradesh. An occurrence of 0.32 % was noticed among the total dogs presented with higher prevalence in dogs aged above eight years. Male dogs revealed a higher incidence of MVD than female dogs.

Keywords: Mitral valve disease, large breed dog, occurrence

Mitral valve disease (MVD) is the common acquired heart disease, that affects 10-15% of dogs. Mitral valvular dysfunction leads to valvular regurgitation, volume overload, and eventual heart failure (Ettinger *et al.*, 2017). Perusal of literature revealed the data available about MVD in large breeds is scanty.

The present study was conducted at Veterinary Clinical Complex (VCC), NTR College of Veterinary Science, Gannavaram during the period, from March 2024 to November 2024. Large breed dogs presented to the VCC with clinical signs like exercise intolerance, cough, lethargy, dyspnoea and body weight greater than 20 kg, were selected and screened for the presence of Mitral valve disease. Diagnosis was based on history, clinical examination, radiographic, electrocardiographic and echocardiographic findings. Diagnostic criteria for MVD included presence of heart murmur over mitral area, evident cardiomegaly along with one or more of following echocardiographic criteria like mitral valve thickening, mitral valve prolapse, mitral regurgitation on colour doppler and fractional shortening > 20 % were considered for study.

During the study period, a total of 7791 dogs were registered at VCC, Gannavaram. The overall occurrence of Mitral valve disease among the total dogs registered was 1.10 % (25/7791) of which large breeds accounted for 0.32 % (25/7791) (Table 1). This finding was in close agreement with the observations of Mattin *et al.* (2015) who reported the occurrence as

0.36 %. Age wise occurrence of Mitral valve disease (MVD) in large breed dogs revealed that majority of the affected dogs were in between 8-11 year age followed by 5-8 year accounting to an occurrence of 40.00 % (10/25) and 36.00 % (9/25) while above 11 years and 3-5 years age groups represented 12.00 % (3/25) each (Figure 1). These findings commensurate with the observations of Keene *et al.* (2019) who opined that MVD was more common in aged dogs which were above eight years. Large breed dogs were affected with MVD at an earlier age compared to small breed dogs (Borgarelli *et al.*, 2004). Higher occurrence of MVD in geriatric dogs was in agreement with the reports of Kumar and Kumar (2024) who stated that aging results in altered homeostasis by affecting cells, tissues and organs there by making geriatric population more prone to the condition. Labrador Retriever were most common among the affected large breed dogs with an occurrence of 68.00 % (17 cases). Mongrels accounted for 16.00 % (4 cases) while lowest occurrence was observed in Bull Mastiff, Dalmatian, German Shepherd and Golden Retriever, each representing 4.00 % of the affected cases (one each) (Figure 2). Labrador Retriever were most prone to MVD as per the current study which were in congruence with the findings of Jong *et al.* (2022) while Svensson *et al.* (2024) recorded a lower occurrence of 14.3 % in Labrador Retrievers. However genetic make up and the breed predisposition to MVD in case of Labrador Retrievers need to be investigated as in case of Cavalier King Charles Spaniel. Occurrence of MVD in male dogs (76.00 %, 19/25) was higher than females (24.00 %, 6/25) in the present study. These findings were similar to the reports of Borgarelli *et al.* (2004)

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and Thirunavukkarasu *et al.* (2019) while Svensson *et al.* (2024) reported comparatively lower incidence (67.3%) in males than current study. These variations could be attributed to over representation of male dogs which might be due to preference of pet owners to male dogs.

Table 1 : Occurrence of Mitral valve disease in Large breed dogs.

Dogs under study	Number	Number of dogs affected with MVD	Percentage	Percentage among total dogs
Large breed dogs	2337	25	1.07	0.32
Small and medium breed dogs	5454	61	1.12	0.78
Total	7791	86	-	1.10

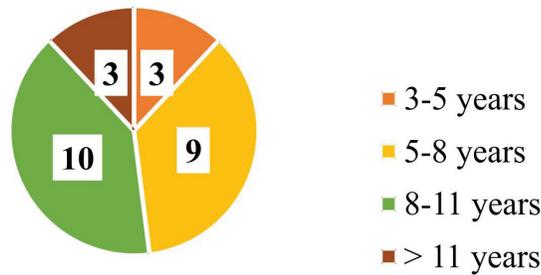


Figure 1: Age wise distribution of Mitral valve disease in large breed dogs (n=25)

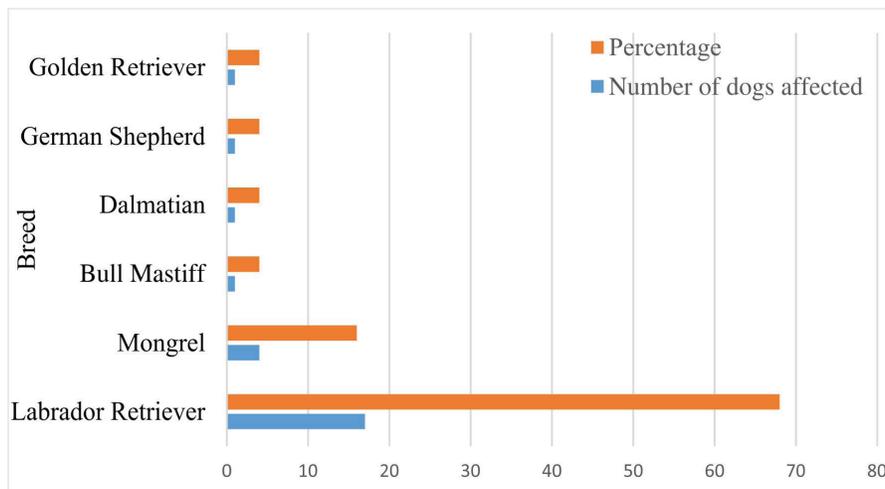


Figure 2: Breed wise distribution of Mitral valve disease in large breed dogs (n=25)

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References

Borgarelli, M., Zini, E., D’Agnolo, G., Tarducci, A., Santilli, R. A., Chiavegato, D. and Haggstrom, J. 2004. Comparison of primary mitral valve disease in German Shepherd dogs and in small breeds. *J. of Vet. Card.*, **6(2)** :27-34.

- Ettinger, S. J., Feldman, E. C. and Cote, E. 2017. Textbook of veterinary internal medicine : diseases of the dog and the cat. 8th ed., Elsevier.
- Jong, M. V. D., Leegwater, P. A., Fieten, H. and Szatmari, V. 2022. Prevalence of echocardiographic evidence of trace mitral and aortic valve regurgitation in 50 clinically healthy young adult Labrador retrievers without heart murmur. *Animals*, **12(18)** :2442.
- Keene, B. W., Atkins, C. E., Bonagura, J. D., Fox, P. R., Haggstrom, J., Fuentes, V.L. and Uechi, M. 2019. ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs. *J. of Vet. Int. Med.*, **33(3)** :1127-1140.
- Kumar, K. S. and Kumar, V. V. V. 2024. Diagnosis of myxomatous mitral valve disease (MMVD) in aged dogs. *Indian J. of Anim. Res.*, **58(1)** :129-134.
- Mattin, M. J., Boswood, A., Church, D. B., Lopez-Alvarez, J., McGreevy, P. D., O'neill, D. G. and Brodbelt, D. C. 2015. Prevalence of and risk factors for degenerative mitral valve disease in dogs attending primary-care veterinary practices in England. *J. of Vet. Int. Med.*, **29(3)** :847-854.
- Svensson, M., Selling, J. and Dirven, M. 2024. Myxomatous mitral valve disease in large breed dogs: survival characteristics and prognostic variables. *Vet. Sci.* , **11** :136.
- Thirunavukkarasu, P., Nagarajan, B., Vijayarani, K. and Kumanan, K. 2019. Canine N-terminal-pro brain natriuretic peptides (NT Probnp): A promising marker of heart failure. *IJCS* **7(1)** :718-719.

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Clinico pathological changes in cattle with theileriosis

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Abstract

The paper reports the clinical and pathological findings in cattle suffering with abomasal ulcers in association with theileriosis. Six cattle with abomasal ulcers in association with theileriosis was identified by presence of intra-erythrocytic piroplasms of *Theileria annulata* in stained blood smears. Abomasal ulcers were identified based on the positive benzidine test. The clinical findings noticed were absence of rumination, anorexia, loss of milk yield, melena, depressed demeanour, emaciation, abdominal guarding, pale mucous membranes, hyperthermia, lymphadenopathy, kyphosis, tachypnea and dyspnea. Reduced serum levels of albumin, calcium, phosphorus, chloride, potassium and increased serum levels of aspartate transaminase, blood urea nitrogen, creatinine were noticed.

Keywords: cattle- theileria- abomasal ulcer

Tropical theileriosis is one of the economically important haemoprotozoan disease affecting bovines and it is caused by *Theileria annulata*. Reports in the cattle with theileriosis associated with abomasal ulcers were very limited. This article reports on the clinical signs and serum-biochemical changes in dairy cattle with abomasal ulcers due to theileriosis.

During the one-year period of study, from January 2022 to December 2022 at Department of Veterinary Medicine, College of Veterinary Science, Proddatur, Andhra Pradesh cattle attending Out-patient unit were screened for theileriosis. Cattle with history of emaciation, frequent fever, chronic illness were selected for collection of peripheral blood and lymph node aspirates for smears preparation. The observed clinical signs were absence of rumination, anorexia, loss of milk yield, melena, depressed demeanour, emaciation, abdominal guarding, pale mucous membranes, hyperthermia, lymphadenopathy, kyphosis, tachypnea, dyspnea, hypothermia and dependent oedema. The serum biochemical parameters were mentioned in the Table 1. A significant decrease ($P < 0.01$) in serum albumin, A/G ratio, calcium and significant decrease ($P < 0.05$) in phosphorous, sodium, potassium, chloride and significant increase ($P < 0.01$) in globulin, AST, bilirubin, BUN, creatinine were noticed in when compared to the apparently healthy cattle. Giemsa's staining was carried out and examined the smears under oil immersion lens at 100x magnification of microscope. Benzidine test was selected to assess the presence of occult blood in dung.

Theileriosis was identified based on the presence of intra-erythrocytic piroplasms of *Theileria annulata* in stained blood smears. Confirmation of the abomasal ulcers was done by positive benzidine test results by development of blue or blue-green colour. Necropsy was carried out on two cattle which died during the study period and the serosal surface of abomasum showed varied degree of congestion, hemorrhages, mucosal surface revealed diffuse hyperaemia, round to oval shaped and linear abomasal ulcers and multifocal necrotic lesions on the abomasal rugae (Fig 1a and 1b). Histopathology revealed mucosal epithelial detachment, necrosis, loss of mucosal lining epithelial cells, infiltration of mononuclear cells in glandular portion of mucosa and submucosal edema. Degeneration, desquamation of glands, goblet cell hyperplasia, and congested blood vessels in the mucosa was noticed (Fig. 2a and 2b).

In the present study confirmation of the theileriosis was done by demonstration of piroplasms and it was in association with the Aktas *et al.* (2006) and Ramazan and Ugur (2006). Durrani *et al.* (2008) and Bhosale *et al.* (2020) stated that high fever, swelling of sub mandibular and sub scapular lymph nodes, increased respiration rate and pulse rate, anorexia, loss of condition, pale conjunctiva and anemia observed in cattle with theileriosis. In the present study positive benzidine test was appreciated in the cattle with abomasal ulcers and it was associated with Hund and Wittek (2018). The histopathological findings in the present study are in agreement with Tharwat and Ahmed (2012) and Tajik *et al.* (2012).

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Fig.1a and 1b. Post mortem examination of abomasum

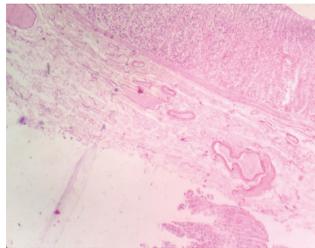


Fig.2a Section of abomasum showing necrosis, loss of mucosal lining epithelial cells, infiltration of mononuclear cells in glandular portion of mucosa and submucosal edema. H&E:40

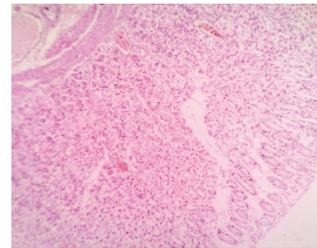


Fig.2b Section of abomasum showing the degeneration, desquamation of glands, goblet cell hyperplasia, and congested blood vessels in the mucosa. H&Ex100

Table 1: Serum biochemical examination in buffaloes with theileriosis associated with abomasal ulcers

S.No.	Parameter	Apparently healthy cattle (6)	Theileriosis associated with abomasal ulcers (6)	P value
1.	Total protein (g/dL)	6.33 ^a ±0.24	6.42 ^a ±0.12	0.065 ^{NS}
2.	Albumin (g/dL)	2.94 ^b ±0.23	1.66 ^a ±0.03	0.002 [*]
3.	Globulin (g/dL)	3.39 ^a ±0.12	4.76 ^b ±0.04	0.003 [*]
4.	AST (IU/L)	58.33 ^a ±4.12	812.5 ^b ±4.08	0.089 [*]
5.	Bilirubin (mg/dL)	1.21 ^a ±0.64	1.66 ^b ±0.09	0.038 [*]
6.	BUN (mg/dL)	26.34 ^a ±1.11	37.31 ^b ±2.09	0.033 [*]
7.	Creatinine (mg/dL)	1.87 ^a ±0.21	2.61 ^b ±0.31	0.021 [*]
8.	Calcium (mg/dL)	9.88 ^b ±0.51	6.68 ^a ±0.33	0.006 [*]
9.	Phosphorous (mg/dL)	5.37 ^b ±0.23	3.47 ^a ±0.07	0.034 [*]
10.	Sodium (mEq/L)	142.39 ^b ±7.06	134.9 ^a ±5.01	0.035 [*]
11.	Potassium (mEq/L)	4.64 ^b ±0.21	4.28 ^a ±0.07	0.038 [*]
12.	Chloride (mEq/L)	102.29 ^b ±3.54	81.09 ^a ±3.08	0.021 [*]

*P<0.05; **P<0.01; ^{NS}P>0.05; Columns bearing different superscripts differ significantly

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References

Aktas, M., Altay, K. and Dumanli, N. 2006. A molecular survey of bovine *Theileria* parasites among apparently healthy cattle and with a note on the distribution of ticks in eastern Turkey. *Vet Parasitol.*, **138**: 179–185.

- Bhosale, A. A. , Bhikane, A .U., Chavhan, S. G., Jadhav, R .K., Mohan, A. and Kushwaha, N. 2020. Prevalence and Clinico-Therapeutic Management of Bubaline Theileriosis in Marathwada Region of Maharashtra. *International Journal of Livestock Research* **10 (9)**: 112-114
- Durrani, A. Z., Ahmad, M., Ashraf, M., Khan, M. S., Khan, J .A., Kamal, N. and Mumtaz, N. 2008. Prevalence of theileriosis in buffaloes and detection through blood smear examination and polymerase chain reaction test in district Lahore. *J. Anim . Pl. Sci.*, **18**:(2-3).
- Hund, A. and Wittek, T. 2018. Abomasal and third compartment ulcers in ruminants and South American camelids. *Vet. Clinics North Am. Food Anim. Pract.*, **34**:35–54.
- Ramazan, C. and Ugur, U. 2006. Haematological and coagulation profiles during severe tropical theileriosis in buffaloes. *Turk. J. Vet. Anim. Sci.*, **30**: 577–582.
- Tajik, .J, Tafti, A.K., Heidari, M. and Babazadeh, M. 2013. Prevalence, histopathological, and some epidemiological aspects of abomasal ulcers in water buffalo (*Bubalus bubalis*) in Iran. *Comp. Clin. Pathol.*, **22**:271–275
- Tharwat, M. and Ahmed, A.F. 2012. Abomasal Ulceration in Buffaloes and cattle: clinic biochemical and pathological findings. *J Anim. Vet. Adv.*, **11**:1327–1331

Prevalence of Rabies Among Hospital-Admitted Rabies Suspected Dogs in and around Chennai

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Abstract

The present study is aimed to identify the prevalence of rabies cases in and around Chennai from January 2024 to December 2024 among the hospital admitted rabies suspected dogs. Data were collected from rabies suspected dog cases which were admitted in under observation for rabies (UOR) ward of Madras Veterinary College, Chennai, Tamilnadu. Cases were confirmed in post-mortem based on FAT and modified William's stain method. The prevalence of rabies for one year was 77.8%. The prevalence in male dog was 75.8% while in female dog was 69.6%. Breed-wise prevalence of non-descript and others (German shepherd, Doberman, Spitz, Kombai, Chippiparai) was 79 and 1 percent, respectively. Positive prevalence of susceptible age group was 68.18, 66.6, 61.5, 50, 45 and 40% in > one year age group, 3-6 months age group, 6-9 months age group, 0-3 months age group and 9-12 months age group respectively. The positive prevalence of unvaccinated dogs and booster-given dogs were 77.2 and 54.5% respectively.

Keywords: Rabies, FAT, prevalence, Chennai.

Rabies is a fatal and zoonotic disease of mammals and is endemic in India (Hampson *et al.*, 2015). Most of the human cases of rabies occur on the globe due to dog bites of more than 95% (Ghosh, 2006). Mortality due to rabies in Asia is mainly due to free-roaming dogs (Shah *et al.*, 2012). In India, rabies is endemic due to its poor dog population management, lack of vaccination awareness, pre-post bite exposure vaccine awareness and poor education knowledge on rabies (Sudarshan *et al.*, 2006). This article describes the prevalence of rabies among hospital-admitted rabies suspected dogs in and around Chennai at Tamilnadu in India.

Rabies-suspected dogs were admitted in under observation of rabies ward of Madras Veterinary College, Chennai Tamilnadu from January 2024 to December 2024 were included for the study. The dogs were kept in UOR for 10 days and dead dogs were sent to post-mortem examination for the confirmation of rabies. The results of the Fluorescent Antibody Test and Williams modified Van Gieson's stain for Negri bodies used as a criterion for the confirmation of rabies in the dogs admitted. The present study was to identify the rabies cases in and around Chennai from January 2024 to December 2024.

Number of dogs admitted in UOR ward from January 2024 to December 2024 was 104 while 81 (77.88%) of these dogs were confirmed to be positive for rabies.

Various breed admitted in the UOR ward that were found to be positive for rabies were non-descript dogs (64/81; 79.01%), spitz (8/8; 100%), German shepherd (6/6; 100%), dalmatian and dobermann (3/3; 100% each), Kombai (2/2; 100%), and Chippiparai (1/1; 100%). In this study, the highest level of positive prevalence of rabies was recorded in non-descript breeds (Table 1) followed by pedigree dogs agreed with the findings of Thiptara *et al.* (2011), Karshima *et al.* (2013) and Yale *et al.* (2013). This may be due to the increased local population with a lack of vaccination knowledge and irregular vaccination was predisposed the nondescript breed to higher positivity than the other breeds.

Male dogs (74.6%) had high prevalence than female dogs (67.6%) (Table 2) which coincides with the findings of Gunaseelan *et al.*, 2004; Thiptara *et al.*, 2011; Kujul *et al.*, 2012; Karshima *et al.*, 2013 and Yale *et al.*, 2013 may be due to dominant territorial activity and fighting among male dogs.

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Table 1: Breed-wise Positive prevalence of rabies-infected dogs

Breeds	Total number of dogs-admitted	Positive	Positive prevalence (Percentage)
ND	81	64	79.01
Dalmatian	3	3	100
GSD	6	6	100
Chippiparai	1	1	100
Kombi	2	2	100
Dobermann	3	3	100
Spitz	8	8	100

Table 2: Sex-wise -Positive prevalence of Rabies

Sex	Total Number of dogs admitted	Positive	Positive prevalence (% age)
Male	67	50	74.6
Female	37	25	67.6

Prevalence of rabies was higher in the above 1 year age group (66.6%) followed by 3-6 months age group (61.5%), 6-9 months age group (50%), 0-3 months age group (45%), 9-12 months age group (40%) (Table 3). Thiptara *et al.* (2011) reported that the age group of above one year was more susceptible than the other age group. This may be due to greater activity during the breeding season and irregular vaccination and unawareness of rabies to the owner may play a role in the transmission of rabies during fighting in the breeding season (Yale *et al.*, 2013). Three - six months of age group showed positivity (Karshima *et al.*, 2013) might be due to a lack of maternal immunity highly susceptible of puppies when exposed.

Table 3: Age-wise Positive prevalence of Rabies

Age	Total number	Positive	Positive prevalence (Percentage)
0-3 mon	20	9	45
3-6mon	13	8	61.5
6-9 mon	6	3	50
9-12mon	5	2	40
Above 1YR	60	40	66.7

May month (92.8%) had highest prevalence of rabies followed by February (90.9%) (Table 4).

Table 4: Month-wise positive prevalence of rabies

Months	No. of cases admitted	No. of positive cases	Positive prevalence
January	12	8	66.6
February	11	10	90.9
March	10	6	60
April	14	13	92.85
May	7	6	85.71
June	9	5	55.5
July	8	7	87.5
August	4	3	75
September	7	6	85.71
October	6	4	66.6
November	11	9	81.8
December	5	1	20

The highest positive prevalence of rabies was noticed in April (92.85%) and February (90.9%) (Table 4). This is similar to the reports of Ezeokoli and Umoh (1987) who reported that higher rabies positive cases in April due to increased breeding activity. It can also be due to increased chances of contact, higher mobility and interaction between dogs as stated by Gunseelan *et al.*

(2004), Ehizibolo *et al.* (2009), Thiptara *et al.* (2011) and Yale *et al.* (2013).

Unvaccinated dogs showed a higher positive prevalence (77.1%) than primary vaccinated dogs (52.4%) (Table 5). This may be due to unawareness and irregular vaccination of dogs by the owner as reported by Yale *et al.* (2013).

Table 5: Vaccination status of the Rabies infected dogs

Vaccination status	Total number of animals	Positive	Positive Prevalence (Percentage)
Unvaccinated	83	64	77.1
Primary vaccination	21	11	52.4

References

- de la Rocque, S., Errecaborde, K.M.M. and Belot, G. 2023 One health systems strengthening in countries: Tripartite tools and approaches at the human-animal-environment interface. *BMJ Global Health*, doi.org/10.1136/bmjgh-2022-011236
- Ehizibolo, D.O., Nwosuh, C.I., Ehizibolo, E.E., Kia, G.S.N. 2009. Comparison of the Fluorescent Antibody test and Direct Microscopic examination for rabies Diagnosis at the National Veterinary Research Institute, Vom, Nigeria. *Afr J Biomed Res.*, **12(1)**:73-6.
- Ezeokoli, C.D. and Umoh, J.U. 1987. Epidemiology of rabies in northern Nigeria. *Trans R Soc Trop Med Hyg*, **81(2)**: 268-72.
- Ghosh, T.K. 2006. Rabies. Proceedings of the IX national conference of pediatric infectious diseases. Chennai, India.
- Gunaseelan, L., Selvi, D., Manohar, B.M., and Thilagar, S. 2004. Epidemiological observations of canine rabies in Chennai city. *Cherion*, **33**:1-6.
- Hampson, K., Coudeville, L., Lembo, T. 2020. Estimating the global burden of endemic canine rabies, <https://dx.plos.org/10.1371/journal.pntd.0003709>.
- Karshima, N.S., Kujul, N.B., Ogbu, K.I., Abdullateef, M.H., Dung, P.A., Salihu, A.A., Obalisa, A. and Paman, N.D. 2013. Incidence and risk factors associated with rabies and dog bites in Plateau State, Nigeria between 2011 and 2012. *J. Anim. Sci. Adv.*, **3**: 114-20.
- Kujul, N.B., Karishma, N.S., Chukwuedu, A.A., Olaleye, S. and Salisu, A.A. 2012. Retrospective study on puppy bites reported to veterinary clinic. Federal College of Animal Health and Production Technology, Vom, Plateau state from 2024 to 2010. *Nigeria Vet. J.*, **33(3)**: 549-57.
- Shah, V., Bala, D.V., Thakker, J., Dalal, A., Shah, U. and Chauhan, S. 2012. Epidemiological determinants of animal bite cases attending the anti-rabies clinic at V S General hospital, Ahmedabad. *Indian J. Prev. Soc. Med.*, **3(1)**: 66-8.
- Sudarshan, M.K., Mahendra, B.J., Madhusudana, S.N., Ashwath-Narayana, D.H., Abdul-Rahman, S. and Meslin, F. and 2006. An epidemiological study of animal bites results of a WHO-sponsored National multi-centric rabies survey. *J. Commun. Dis.*, **38(1)**: 32-9.
- Thiptara, A., Atwill, .ER., Kongknew, W. and Chomel, B.B. 2011. Epidemiologic trends of rabies in domestic animals in Southern Thailand, 1994-2008. *Am. J. Trop. Med. Hyg.*, **85**: 138-45.
- Yale, G., Ganesan, P.I., Bharathi, M.V., Tirumurugaan, K.G., Thangavelu, A. and Srinivasan, S.R. 2013. A retrospective b study of canine rabies incidence in Chennai. *Int. J. Adv. Res.*, **1**: 821-4.

Diagnosis and Management of Demodicosis in Dogs

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Abstract

Demodicosis is the most common skin condition predominantly found in canines. Demodex is a cigar shaped mite which normally grow in hair follicles. Overgrowth of these mites in the follicles will result in a condition called Demodicosis. Fifteen dogs with signs and lesions suggestive for demodicosis were subjected for skin scraping, tape impression and hair pluck from the same area and examined under microscope for confirmation of demodicosis. Positive dogs were treated with oral ivermectin @400mcg daily for three weeks along with other supplements and topical amitraz. Dogs that were treated with both ivermectin and amitraz showed a marked recovery.

Keywords: Canine Demodicosis, Hair follicle, Skin Scrapping, Acetate tape impression.

Demodectic mange is the most prevalent parasitic infection in canine practice caused by, *Demodex canis*. These cigar-shaped mites are known to be commensals inhabiting canine skin, residing in hair follicles and feeding on cells, sebum and epidermal debris (Craig, 2020). Breeds such as Bulldogs, Pugs, Pit bulls and Terriers are particularly found to be highly susceptible (Mueller et al., 2020). Furthermore, animals suffering from immune suppressive disorders are at a higher risk of developing this condition (Dryden, 2024). This article describes diagnosis and management of demodicosis in dogs.

A total of 15 dogs of various breed and gender that were presented to the Veterinary Clinical Complex, College of Veterinary Science, Rajendranagar with the history and signs of erythematous lesions generally distributed over the skin along with alopecia and scabs were considered for the present investigation. After thorough examination hair follicles (trichogram) were plucked out using artery forceps, scotch tape impressions and skin scrapings were collected from the lesions by gently squeezing around the area and were subjected to subsequent microscopic examination at 10x and 40x magnification.

Dogs that presented with pustules were also evaluated for presence of secondary bacterial infection. Swab samples were collected from the animals presented with pustules which are inoculated in a nutrient broth for 24hrs at 37°C.

Detailed clinical examination revealed various degrees of erythematous lesions, severe pruritis, alopecia, folliculitis, scales, comedon formation hyperpigmentation and pododemodicosis (Fig 1a and 1b). On microscopic examination all the skin scraping samples turned out to be positive for the presence of Demodex mites (fig. 2). 12 of 15 samples collected for trichogram (fig. 3), and 8 of 15 scotch tape impression samples evaluated have given a positive result for presence of mites (Fig. 4). Dogs with moderate to severe form of the infection have shown higher sensitivity for trichogram (Reddy & Sivajothi, 2017). Trichogram being non-invasive and less painful can be effective as a primary means of diagnosis specially if the lesions are confined to sensitive areas like eyes, face, inter digital space. A negative trichogram result can be followed by deep skin scraping for confirmatory results. Scotch tape impressions can be used in case of severe generalised form of this disease.

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Fig. 1a. Erythema, alopecia, furunculosis, pustules, crust formation in an 8-month-old dalmatian presented with Generalised form of demodicosis



Fig. 1b. Erythema, focal alopecia in periorbital area, paws in a 2-year-old labrador and a 5-year-old German shepherd. Erythema, alopecia, pustules in a 1-year-old pug

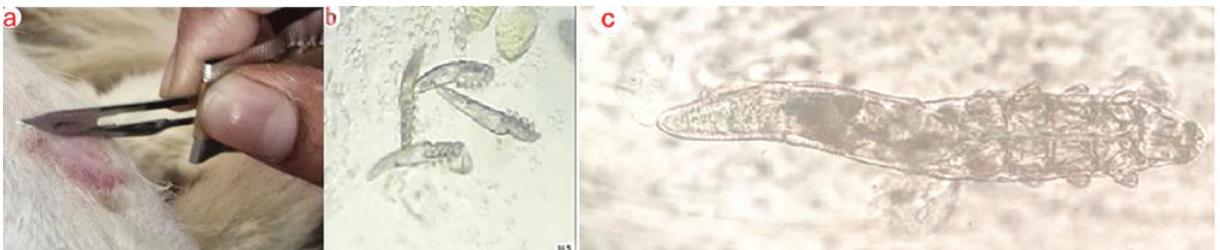


Fig. 2 Detection of Demodex mites from the sample collected by deep skin scraping: (a) Skin scraping sample collection, (b) Demodex mites as observed under 10X magnification, (c) Demodex mites as observed under 40X magnification



Fig. 3 Detection of Demodex mites by trichogram method: (a) hair follicle sample collection for trichogram, (b & c) Demodex mites as observed under 10X magnification



Fig. 4 Sample collection by acetate tape impression method (a and b), Demodex mite from the sample (c)

Animals tested positive for demodicosis were treated with ivermectin (oral @ 400mcg SID, for 3 weeks) and topical application of amitraz solution at a concentration of 0.025% once a week for 4 weeks. Animals suffering from secondary bacterial infection underwent a specific antibiotic therapy for 5-7 days. Following treatment all the dogs showed marked clinical improvement from day 7 and complete clinical recovery with absence of lesions along with reappearance of hair by day 30 (Fig. 7). These findings are in accordance with Mueller (2012) who reported that combination therapy of ivermectin and amitraz is more effective in

the management of generalised demodicosis. However, pyoderma is one of the most common secondary infection associated with demodicosis which can be managed with broad spectrum antibiotics like cephalexin at 30 mg/kg q12h (for superficial pyoderma) or q8h (for deep pyoderma). Other antibiotics which can be used are enrofloxacin 5-10 mg/kg q24 h, amoxicillin-clavulanate 13.75 mg/kg q12h or marbofloxacin 3-5 mg/kg q 24 h. Out of a total 15 dogs that were evaluated for demodicosis, 15/15, 12/15 and 08/15 dogs were found positive for Demodex mites on scraping, trichogram and tape impression.



Fig. 5. Demodicosis dogs shown in fig 1a and 1b showed regression of lesions by day 30.

References

- Craig, M. 2020. Demodicosis. Foster, A.P. and Foil, C.S. (eds.) *BSAVA Manual of Small Animal Dermatology*. 2nd edn. Gloucester: British Small Animal Veterinary Association, pp. 153–157.
- Dryden, M.W. 2025. Mange in dogs and cats. *The Merck Veterinary Manual*, pp. 922–923
- Mueller, R.S. 2012. An update on the therapy of canine demodicosis. *Compendium (Yardley, PA)*, **34(4)**: 1-4.
- Mueller, R.S., Rosenkrantz, W., Bensignor, E., Karaś-Tęcza, J., Paterson, T. and Shipstone, M.A. 2020. Diagnosis and treatment of demodicosis in dogs and cats: Clinical consensus guidelines of the World Association for Veterinary Dermatology. *Vet. Dermatol.*, **31(1)**: 4-e2.
- Reddy, B.S. and Sivajothi, S. 2017. Importance of diagnostic procedures and client education in demodicosis: An evidence based study. *J. Parasit. Dis. Diag.n Ther.*, **2 (2)**: 25-27

Blood transfusion in a dog with Ehrlichiosis

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Abstract

Blood transfusion is a crucial medical intervention used to address anemia, effectively restoring essential red blood cell levels. A nine-year-old male Rottweiler was presented at the Veterinary Clinical Complex, CVSc., Rajendranagar, with a history of severe tick infestation, decreased water intake, and lethargy lasting for six days. A physical examination revealed enlarged lymph nodes, anemic signs and severe dehydration. Haematological analysis showed anaemia and thrombocytopenia. Lateral flow assay revealed positive for *E. canis*. Blood transfusion was performed along with supportive therapy. The dog was also administered oral Doxycycline @10 mg/Kg for two weeks. The combination of blood transfusion and subsequent treatments significantly contributed to the dog's recovery.

Keywords: Anemia, tick infestation, Blood transfusion, Hematology, Doxycycline

Anemia can be defined as a reduced erythrocyte content or diminished oxygen carrying capacity of blood resulting from a decrease in PCV, RBC count or hemoglobin concentration below normal reference range. The etiology often includes infectious diseases (Ehrlichiosis, Babesiosis, Haemotropic Mycoplasmosis etc), autoimmune disorders, nutritional deficiencies, parasitic infestations, chronic diseases, neoplasia and hemorrhage. This case report highlights the successful management of severe anemia in a Rottweiler, demonstrating how blood transfusion effectively served as a critical supportive therapy alongside doxycycline, and contributed significantly to the overall recovery.

Nine year-old male Rottweiler was presented to the Veterinary Clinical Complex, CVSc, Rajendranagar, with a history of anorexia, fever, tick infestation and epistaxis. The animal was dull and lethargic with pyrexia. The conjunctival and buccal mucous membranes were markedly pale. Enlargement of the submandibular, prescapular, and popliteal lymph nodes were noted.

Haematobiochemical examination, SNAP test and PCR were carried out. Peripheral blood smear revealed the presence of morulae within neutrophils. Both the SNAP test and lateral flow assay yielded positive results for ehrlichiosis. The diagnosis was further confirmed by PCR analysis (Fig. 1). Decreased RBC count (2.38

$\times 10^{12}/L$), PCV (11.4%) and thrombocytopenia ($89 \times 10^9/L$) were noticed. Liver function test parameters were within normal limits, whereas kidney function tests revealed an elevated blood urea level of 43.7 mg/dL.

Both direct (tridrop method) and indirect cross matchings (Wardrop and Davidow, 2023) were performed to detect any incompatibility between the donor and the recipient and also to rule out the presence of any previously sensitised antibodies both of which showed negative results for agglutination (Fig.2). Using the formula, Blood transfusion volume = $80 \times \text{body weight} \times (\text{desired PCV} - \text{recipient PCV}) / (\text{PCV transfused blood})$ (Wardrop *et al.*, 2016). Blood was successfully collected from the donor dog and transfused into the recipient dog. The blood transfusion served as an adjunct therapy, effectively raising the PCV to a level that facilitated clinical improvement and enhanced the animal's response to concurrent treatments.

The animal was administered Ferritas and Vitamin B-complex supplement (@1.5 ml, to support haematopoiesis). The dog was administered doxycycline (orally @ 10 mg/kg S.I.D for 3 weeks) Anti-tick shampoo and fipronil spray were used for effective tick control. Following the blood transfusion and treatment, the animal gradually recovered and became active by day 7. The hematinics and multivitamin supplements aided in full recovery. Although often recorded as an independent diagnosis, anemia is actually a clinical

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sign with several underlying causes. Treatment of anemia in veterinary medicine includes addressing the underlying cause, providing supportive care such as blood transfusions, and administering hematinic agents

like iron, vitamin B12, and folic acid as appropriate. Doxycycline addressed the underlying ehrlichial infection, blood transfusion played a crucial supportive role in stabilizing the patient.

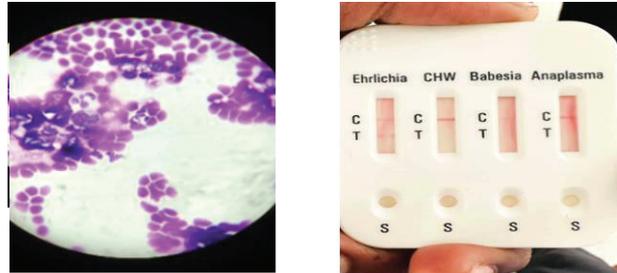


Fig. 1: (a) Presence of morulae in neutrophil (b) snap test showing positive result

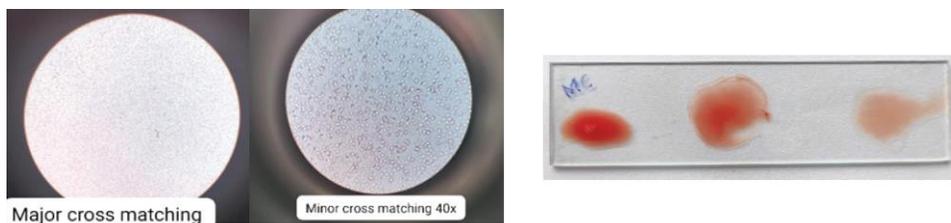


Fig. 2: Indirect and direct crpps matchings showing no signs of agglutination



Fig. 3: Collection and weighing of blood in citrate phosphate dextrose blood bag; b) collection of blood from donor and transfusion to recipient.

References

- Wardrop, K.J., Birkenheuer, A. and Blais, M.C. 2016. Update on canine and feline blood donor screening for blood-borne pathogens. *J. Vet. Intern. Med.*, **6;30(1)**:15-35.
- Wardrop, K.J. and Davidow, E.B. 2023. Laboratory testing in transfusion medicine. *Vet. Clin. North Am. Small Anim. Pract.*, **53(1)**:265-278.

Management of Cystitis in two dogs

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Abstract

Two dogs were presented with complaint of haematuria, inappetance, dyspepsia and stranguria. Leukocytosis, low Hb while urinalysis revealed proteinuria with pH 9, erythrocytes, pus cells and abundant struvite crystals in female. Culture result showed *Staphylococcus* growth in female and mixed growth of *Pseudomonas*, *Proteus* and *Klebsiella* in male. Antibiotic sensitivity was present predominantly for cephalosporins. On abdominal ultrasonography examination, the bladder displayed increased wall thickness, and hyperechoic crystals which concluded the case as cystitis. Culture result showed *Staphylococcus* growth. Antibiotic sensitivity was present predominantly for cephalosporins and enrofloxacin. They were treated with cephalexin with other supplements and was advised for struvite diet. Improvement with resolution of signs and normal appetite was noticed after 15 days.

Keywords: canine cystitis, hematuria, struvite

Cystitis is inflammation of wall of urinary bladder caused by ascending migration, multiplication, and establishment of microorganisms from the lower parts of the urinary tract. It is caused by various infectious agents, remarkably by bacteria like *Streptococcus*, *Staphylococcus*, *Escherichia coli*, *Proteus*, *Klebsiella* and *Pseudomonas spp.*, and rarely by fungi and viruses (Krane and Levine, 1992 and Ettinger *et al.*, 2005). In the present study, the cystitis was diagnosed and confirmed using haematological, biochemical, microbiological, and imaging investigations, and was successfully managed.

Two dogs (2 and 11 years; one male and one female) were presented to the Telangana Veterinary Clinical Complex, CVSc Rajendranagar, Hyderabad, Telangana, India with the history and signs of reduced urine volume but increased frequency, dysuria, pigmented urine and frequent licking. Haematology revealed marked neutrophilia (97%). Ultrasound imaging displayed, increased bladder wall thickness, an irregular mucosal surface and hyperechoic sediments. Urine samples were blood tinged and the staining of the urine smear demonstrated cocci, pus cells and epithelial cells (Fig.1). Quinn *et al.* (2011) reported that *Staphylococcus spp.* form golden-yellow colonies on Mannitol Salt Agar (MSA) due to mannitol fermentation and pigment production, which was observed in the samples collected from the dogs (Fig. 2). ABST revealed

sensitive to cephalosporin and fluoroquinolones. The diagnostic tests revealed a severe bacterial infection along with crystalluria, indicative of cystitis, based on which appropriate treatment was initiated.

Clinical manifestations of cystitis typically include hematuria, stranguria, pollakiuria, dysuria, urinary incontinence, and in some cases, systemic signs such as lethargy, dehydration, and pyrexia. Early diagnosis is critical and involves a combination of hematology, biochemistry, complete urinalysis, urine sediment examination, and advanced imaging modalities such as ultrasonography to assess bladder wall architecture, intraluminal debris, and crystalluria (Stamm & Norrby, 2001; Nickel, 2005). Definitive diagnosis requires urine culture and sensitivity testing, ideally performed on samples collected via cystocentesis to avoid contamination, which enables pathogen identification and guides appropriate antimicrobial therapy (Flores-Mireles *et al.*, 2015). A comprehensive, individualized, and evidence-based diagnostic and therapeutic approach remains critical for successful management of cystitis in canines, ensuring rapid clinical improvement and minimizing further complications that might lead to nephritis. The dogs had uneventful recovery following treatment with cephalexin (@20mg /kg bid PO) for a period of 15 days.

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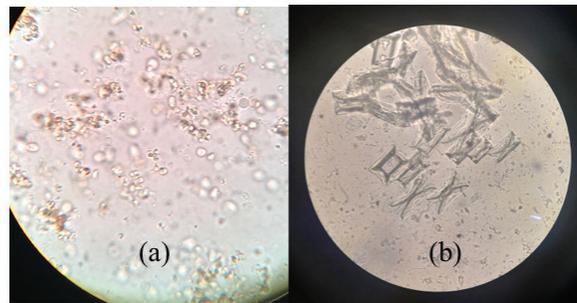


Fig. 1: Microscopic examination of urine sediment revealed RBCs, pus cells and struvite crystals



Fig. 2: Culture of urine sample of MSA agar showing golden yellow colonies of Staphylococcus spp.

References

- Ettinger, S.J. and Feldman, E.C. 2005. Textbook of veterinary internal medicine. 6th ed. Vol. 2. Chapter 262: Urinary tract infections by J.W. Bartges. St. Louis: Saunders Elsevier.
- Flores-Mireles, A.L., Walker, J.N., Caparon, M. and Hultgren, S.J. 2015. Urinary tract infections: Epidemiology, mechanisms of infection and treatment options. *Nature Reviews Microbiology*, 13(5), pp.269–284. <https://doi.org/10.1038/nrmicro3432>
- Krane, D.M. and Levine, L.A. 1992. Haemorrhagic cystitis. AUA Update Series, 11, Lesson 31.
- Nickel, J.C. 2005. The bladder epithelium: A scientific update. *Journal of Urology*, 173(3), pp.848–854. <https://doi.org/10.1097/01.ju.0000154642.60516.d7>.
- Stamm, W.E. and Norrby, S.R. 2001. Urinary tract infections: Disease panorama and challenges. *Journal of Infectious Diseases*, 183(Supplement 1), pp.S1–S4. <https://doi.org/10.1086/318850>

Incidence of canine parvovirus infection in and around Tirupati

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Abstract

Canine parvovirus (CPV) is the most significant viral cause of acute haemorrhagic enteritis. The objective of the present study is to detect the incidence of canine parvovirus infection in and around Tirupati. A total of 154 faecal samples were collected from the dogs showing clinical signs of vomiting, diarrhoea, anorexia, dehydration and pyrexia. Out of 154 samples, 62 samples were positive for CPV 2 by PCR with an overall prevalence of 40.25 %. Occurrence of CPV infection in relation to age was highest in 0 to 3 months (45.16 %) and least was recorded in dogs above 1 year of age (9.67 %). Breed wise analysis of data indicated highest occurrence in Non-descript dogs (38.70 %). The occurrence of CPV in the present study was more in male dogs (66.12 %) than female dogs (33.87 %). Out of 62 dogs positive for canine parvoviral enteritis, 15 dogs (24.19%) were vaccinated whereas, 47 (75.80%) were non- vaccinated. The results indicate the necessity of implementing control and preventive measures to combat canine parvoviral infection..

Keywords: Canine parvovirus, incidence, Age, Breed, Sex

Canine parvovirus is a highly contagious disease caused by canine parvovirus 2. Outbreaks of CPV have been reported from many countries including India. The factors that predispose puppies to parvovirus infection are lack of protective immunity, overcrowding of animals in a small space, unhygienic, stressful environmental conditions (Dash *et al.* 2020). Survival of infected puppies has ranged from 9% in untreated to more than 90% in those treated at veterinary facilities (Kalli *et al.* 2010). This article describes the incidence of parvo viral enteritis in dogs in and around Tirupati.

In present study, faecal samples from dogs brought to the College of Veterinary Science, Tirupati from April 2024 to November 2024 showing symptoms of vomiting, haemorrhagic diarrhoea, pyrexia, inappetence and dehydration suspected for CPV gastroenteritis were collected. The DNA was extracted from faecal samples by boiling and chilling method (Vieira *et al.*, 2008) and subjected to PCR for detection of canine parvovirus-2 by using CPV 2 primers. Information on age, sex, breed was collected from the owners of the animals selected for the study.

Out of 154 faecal samples, 62 were found positive for canine parvovirus infection by PCR with an overall prevalence of 40.25% in and around Tirupati. This finding was in agreement with the studies of Reddy *et al.* (2015) and Mehta *et al.* (2017) who also reported incidence of 40 %, 33.17 % and 43.44 %, respectively for CPV gastroenteritis. In contrast, Sagar *et al.* (2008) reported higher incidence of 66.66 % and 63% respectively and Khare *et al.* (2019) reported low incidence of 7.7 % in their study. These differences in the incidence rate might be due to changes in sample sizes or from the various geographic locations where studies have been conducted.

Out of 62 parvovirus infected dogs, highest occurrence (45.16 %) was recorded in the age group of 0 to 3 months, followed by 3 to 6 months age group (32.25%), 6 to 12 months (12.90 %) and lowest occurrence was in dogs above 1 year of age (9.67 %). These findings were in agreement with Sayed-Ahmed *et al.* (2020) and Wark *et al.* (2024), who also reported a higher prevalence of canine parvoviral enteritis in dogs aged between 1 to 3 months and 0 to 3 months, respectively. In contrast to this, Vivek *et al.* (2013) and Behera *et al.* (2015) reported higher prevalence of CPV infection in dogs aged between 3 to 6 months. The

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higher prevalence of CPV infection in 0-3 months of age dogs might be attributed to the higher susceptibility of enterocytes to the viral tropism (Sayed-Ahmed *et al.*, 2020). The fall in maternal antibody level after 3 months of age might be one of the predisposing factors in endemic areas, that resulted in second highest occurrence of CPV infection in 3 to 6 months age group of dogs. Further, the higher incidence of CPV infection in dogs below 6 months might be due to the affinity of the virus for rapidly multiplying intestinal crypt cells with higher mitotic index due to changes in bacterial flora as well as in the diet due to weaning (Behera *et al.*, 2015).

In the current study, highest occurrence (38.70 %) of canine parvovirus infection was recorded in Non-descript dogs, followed by Labrador (20.96 %), German Shepherds (11.29%), Shih Tzu (9.67%), Doberman (6.45 %), Golden Retriever (4.83 %) and Pomeranian (3.22 %). Lowest occurrence of CPV infection was recorded in Husky, Dachshund and Beagle (1.61 % each). The highest prevalence of canine parvoviral infection in Non descriptive dogs might be due to the higher population density of this breed making their vicinity to spread the infection and poor vaccination schedule being followed by the owners of Non descript breeds due to the lack of awareness among them (Khare *et al.*, 2019 and Sayed-Ahmed *et al.*, 2020). Labrador retriever dogs were at the second position in most affected dogs in the present

study, this might be due to the preference of dog owners for this breed in this region.

The occurrence of canine parvovirus infection in the present study was more in male dogs (66.12%) than female dogs (33.87 %). These findings were corroborated with the reports of Reddy *et al.* (2015), Tion *et al.* (2018), Vivek *et al.* (2013) and Wark *et al.* (2024) who also reported higher prevalence of CPV infections in male dogs. The higher prevalence of CPV in male dogs might be attributed to more danger of exposure to infection due to their behaviour and selective fondness of keeping male dogs as pets by the pet owners (Reddy *et al.*, 2015).

Out of 62 dogs positive for canine parvoviral enteritis, 15 dogs (24.19 %) were vaccinated whereas, 47 (75.80 %) were non- vaccinated. This was in agreement with the findings of Reddy *et al.* (2015), Tion *et al.* (2018), and Tagorti *et al.* (2018). High prevalence of CPV infection in non- vaccinated dogs might be due to lack of protective immunity and in vaccinated dogs the probable reason for occurrence of CPV infection might be due to progressive decline in maternal antibodies after 38 days (Kataria *et al.*, 2020). Moreover, vaccine failure can also occur due to poor vaccine quality, lack of maintenance of cold chain and poor storage of vaccine as a result of erratic power supply (Ukwueze *et al.*, 2018).

Table 1: Age-wise distribution of CPV positive cases

Age group	Number of dogs positive for CPV (n = 62)	Percentage (%)
0 to 3 months	28	
3 to 6 months	20	32.25
6 months to 1 year	8	12.90
Above 1 year	6	9.67

Table 2: Breed-wise distribution of CPV positive cases

Breed	Number of dogs positive for CPV (n = 62)	Percentage (%)
Non-descript	24	38.70
Labrador	13	20.96
German Shepherd	7	11.29
Shih Tzu	6	9.67
Doberman	4	6.45
Golden Retriever	3	4.83
Pomeranian	2	3.22
Husky	1	1.61
Dachshund	1	1.61
Beagle	1	1.61

Table 3: Sex-wise distribution of CPV positive cases

Sex	Number of dogs positive for CPV (n = 62)	Percentage (%)
Males	41	66.12
Females	21	33.87

Table 4: Vaccination status of CPV positive cases

Vaccination status	Number of dogs positive for CPV (n = 62)	Percentage (%)
Vaccinated	15	24.19
Unvaccinated	47	75.80

The overall prevalence of CPV infection was 40.25 % in and around Tirupati. Highest occurrence of CPV infection was noticed in Non-descript dogs between 0 to 3 months of age. Male dogs and unvaccinated were found to be more susceptible for CPV infection suggesting the necessity of implementing control and preventive measures to combat canine parvoviral infection.

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References

- Behera, M., Panda, S. K., Sahoo, P. K., Acharya, A. P., Patra, R. C., Das, S. and Pati, S. 2015. Epidemiological study of canine parvovirus infection in and around Bhubaneswar, Odisha, India. *Veterinary world*, **8(1)**: 33.
- Dash, S., Das, M., Senapati, S., Patra, R., Behera, P. and Sathapathy, S. 2020. Effect of therapeutic regimen on the survivability and mortality rates in canine Parvovirus infection. *J.Entomol. Zoology Studies*, **8**: 392-395.
- Kalli, I., Leontides, L. S., Mylonakis, M.E., Adamama-Moraitou, K., Rallis, T. and Koutinas, A. F. 2010. Factors affecting the occurrence, duration of hospitalization and final outcome in canine parvovirus infection. *Res.Vet. Sci.*, **89(2)**: 174-178.
- Kataria, D., Agnihotri, D., Jain, V., Charaya, G. and Singh, Y. 2020. Molecular occurrence and therapeutic management of canine parvovirus infection in dogs. *International J. Current Microbiol. Applied Sciences*, **9**: 1770-1779.
- Khare, D. S., Gupta, D. K., Shukla, P. C., Das, G., Tiwari, A., Meena, N. S. and Khare, R. 2019. Prevalence of canine parvovirus infection in dogs in Jabalpur (MP). *J. Entomology and Zoology Studies*, **7(3)**: 1495-1498.
- Mehta, S. A., Patel, R. M., Vagh, A. A., Mavadiya, S. V., Patel, M. D., Vala. J. A. and Parmar, S, M. 2017. Prevalence of canine parvo viral infection in dogs in and around Navsari district of Gujarat State, India. *Indian Journal of Veterinary Sciences & Biotechnology*, **13(2)**: 67-72.
- Reddy, K. B., Shobhamani, B., Sreedevi, B., Prameela, D. R. and Reddy, B. R. 2015. Prevalence of canine parvoviral infection in dogs in and around Tirupathi of India. *International J. Livestock Res.*, **5(3)**: 93-99
- Sagar, A., Roy, S. and Roy, M. 2008. Clinico, haemato-biochemical changes and diagnosis of canine parvoviral enteritis. *Intas Polivet*, **9(2)**: 262-265.
- Sayed-Ahmed, M. Z., Elbaz, E., Younis, E. and Khodier, M. 2020. Canine parvovirus infection in dogs: Prevalence and associated risk factors in Egypt. *World's Veterinary Journal* **(4)**: 571-577.
- Tagorti, G. 2018. Prevalence of canine parvovirus infection in Grand Tunis, Tunisia. *J. Advanced Vet. Anim. Res.*, **5(1)**: 93-97.
- Tion, M. T., Fotina, H. A. and Saganuwan, A. S. 2018. A retrospective study of Canine parvovirus in Private veterinary clinic 'Health', Sumy Region, Ukraine (2015–2018). *Journal for Veterinary Medicine, Biotechnology and Biosafety*, **(4, Iss. 3)**: 5-9.
- Ukwueze, C. S., Anene, B. M., Ezeokonkwo, R, C. and Nwosuhm, C. I. 2018. Prevalence of canine parvovirus infection in South Eastern region, Nigeria. *Bangladesh J.Vet. Med.*, **16(2)**: 153-161.

Therapeutic Management of Traumatic Reticuloperitonitis in a cow

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Abstract

Four -year-old Jersey cross-bred cow was brought with history of inappetence, reduced milk production and brisket oedema for the period of 10 days. Detailed clinical examination revealed fever, tachycardia, tachypnoea, positive venous stasis and dehydration. Elevated haematocrit, leucocytosis with neutrophilia, presence of a greater number of immature neutrophils and decreased lymphocyte count were noticed. Hypoproteinemia, hypophosphataemia and increased calcium and AST values were noticed. Radiography revealed the presence of linear foreign body in the reticulum. The ultrasonography revealed presence of anechoic fluid in the peritoneum, mixed echogenic mass on the serosal surface of reticulum suggestive of reticular abscess. The cow was managed medically with antibiotics, diuretics, NSAIDs and fluid therapy for three days. Subsequently rumenotomy was performed to relieve the foreign body and the cow had uneventful recovery.

Keywords: Cattle, Reticular abscess, Rumenotomy

Traumatic reticuloperitonitis, also known as hardware disease, continues to be one of the most significant digestive disorders in cattle (Mousavi *et al.*, 2007). Bovines are more prone to ingesting foreign objects than small ruminants because they do not differentiate between metallic materials in their feed, particularly those raised in urban and peri-urban areas (Aiello *et al.*, 2016). Traumatic reticuloperitonitis (TRP) occurs when a metallic foreign body is accidentally swallowed and penetrates the reticular wall, leading to acute inflammation around the reticulum, along with adhesions and abscess formation (Abdelaal *et al.*, 2009). A case of TRP in a cattle and its successful management is reported in this article.

Four-year-old Jersey cross-bred cow was brought to the Large Animal Medicine Out Patient Unit of Madras Veterinary College Teaching Hospital with history

of inappetence, reduced milk production and brisket oedema (Fig-1) for a period of 10 days. Detailed clinical examination revealed fever, tachycardia, tachypnoea, positive venous stasis and dehydration. Haematological examination showed elevated PCV, leucocytosis with neutrophilia and decreased lymphocyte count. Serum biochemistry revealed decreased protein, phosphorous levels and increased calcium and AST values than the normal range (Table-1). Radiography revealed the presence of linear foreign body in the reticulum (Fig-2). The ultrasonography revealed presence of anechoic fluid with fibrin strands in the peritoneum (Fig-3) and mixed echogenic mass on the serosal side of the reticulum suggestive of reticular abscess (Fig-4). Thoracic ultrasonography was performed to rule out pericarditis. Based on the above findings the case was diagnosed as TRP.



Fig.1: Brisket edema

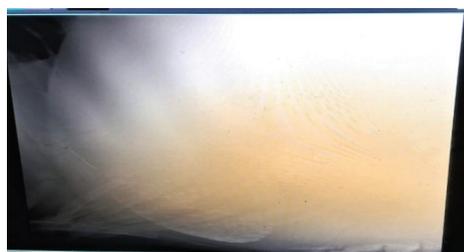


Fig.2: Linear foreign body in the reticulum



Fig.3: Fibrin strands in the peritoneal fluid



Fig.4: Reticular abscess

Table-I Haematobiochemical parameters

Parameters	Cattle with TRP	Normal range
Haemoglobin (g/dl)	9.3	8.0–15.0
Packed cell volume (%)	55	24-46
RBC (m/cmm)	6.9 m	5.0-10.0
WBC/ cmm	21,000	4.0-12.0
PLATELETS /cmm	3,51,000	1.0-8.0
Neutrophils (%)	70	14-45
Lymphocytes (%)	30	48-75
Monocytes (%)	4	2-7
Eosinophils (%)	2	2-15
Basophils (%)	-	0-2
Glucose mg/dl)	74	45-75
Total protein (g/dl)	5.2	6.7-7.4
Albumin (g/dl)	2.7	3.0-3.5
Calcium (mg/dl)	14.6	9.7-12.4
Phosphorous (mg/dl)	3.7	5.5-8.0
ALT (U/L)	35	11-40
AST (U/L)	186	78-132
GGT (U/L)	15	6.0-17.4
BUN (mg/dl)	27	20-30
Creatinine (mg/dl)	1.2	1.0-2.0

The cow was conservatively managed with confinement, broad-spectrum antibiotic streptopenicillin (@20,0000 IU/kg B.wt, IM), meloxica (@ 0.5 mg/kg B.wt, IM), and furosemide (@ 2 mg/ kg B.wt, IM) for three days. Rumenotomy (Fig-5) was performed standing

position under paravertebral nerve block and inverted L block using 2% lignocaine. On exploration of reticulum, the foreign body (Fig-6) was removed successfully. All the incision made was closed as per standard technique and the cow had uneventful recovery.

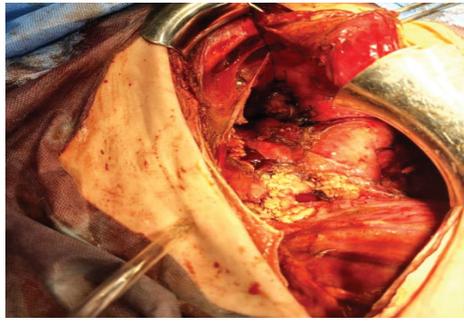


Fig.5: Rumenotomy



Fig.6: Metallic foreign body recovered from cow

Predisposing factors for hardware disease include the indiscriminate feeding behaviour of cattle, as well as conditions like phosphorus deficiency, which can lead to pica (Divers and Peek, 2008). Common symptoms include anorexia, reduced milk production, fever, tachypnea, reluctance to move, and a characteristic stance with an arched back and outwardly spread elbows (Fubini *et al.*, 2008). Other signs may include abdominal pain, fever, toxemia, and a decrease in faecal output (Radostits *et al.*, 2007). Abdominal ultrasonography proved to be an excellent diagnostic tool (Braun *et al.*, 2018). The treatment of TRP can be either surgical or conservative (Orpin and Harwood, 2008). Surgical treatment is considered the gold standard for managing TRP. In this study, the cow was treated with both medical and surgical interventions. A proactive strategy, which includes thorough risk assessments, careful culling decisions, and both clinical and postmortem evaluations, is essential for early identification and preventing further issues within the herd.

References

- Abdelaal, A.M., Floeck, M., El Maghawry, S. and Baumgartner, W. 2009. Clinical and ultrasonographic differences between cattle and buffaloes with various sequelae of traumatic reticuloperitonitis. *Vet. Med.*, **54**(9): 399-406.
- Aiello, S.E., Mosesand, M.A. and Allen, D.G. 2016. The Merck Veterinary Manual 11th ed., Merck and Co Inc., Kenilworth, NJ, pp.3177-3181.
- Braun, U., Warislohner, S., Torgerson, P., Nuss, K. and Gerspach, C. 2018. Clinical and laboratory findings in cattle with traumatic reticuloperitonitis. *Vet. Res.*, **14** : 66.
- Divers, T. J. and Peek, S.P. 2008. Rebhun's disease of dairy cattle. 2th Edition, Elsevier Inc, United States America. pp. 141-145.
- Fubini, S. and Divers, T.J. 2008. Noninfectious diseases of the gastrointestinal tract. In Rebhun's diseases of dairy cattle pp.130-199.
- Mousavi, G., Hassanpour, A., Tabrizi, A. and Rezaie, A. 2007. Electrocardiographic changes in buffaloes with Traumatic reticuloperitonitis. *Ital. J. Anim. Sci.*, **6**(sup2):1029-1031.
- Orpin, P. and Harwood, D. 2008. Clinical management of traumatic reticuloperitonitis in cattle. *In Pract.*, **30**: 544-551.
- Radostits, O.M., Gay, C.C., Hinchcliffand, K.W. and Constable, P.D. 2007. Veterinary medicine. A textbook of the diseases of the cattle, horses, sheep, pigs and goats. 10 edn. Saunders Elsevier, Philadelphia. pp. 189-382

Coagulation Profile and D-dimer in Labrador Puppies with Parvo Viral Enteritis

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Abstract

Management of puppies with parvo viral enteritis is a major challenge. Complicating factors include sepsis and coagulation status. In a study of PCR confirmed parvo affected labrador puppies, a hypercoagulable status was observed. Haematobiochemical examination revealed decreased hemoglobin, Packed Cell Volume and total erythrocyte count, hypoproteinaemia and hypoalbuminemia. The Activated Partial Thromboplastin Time (aPTT) was markedly prolonged. D – Dimer status was negative and was unremarkable.

Keywords: Parvo viral Enteritis - Prothrombin Time - aPTT - D-Dimer

Introduction

Canine Parvovirus (CPV) is considered as the most pathogenic that affects the young dogs and remains an important cause of morbidity and mortality in young dogs. Critical care management of these puppies are essential and it requires advanced monitoring, as there exists laboratory evidences of hypercoagulability. This study documents the D-Dimer and Coagulation Profile of Labrador puppies affected with parvo viral enteritis.

Materials and Methods

Eight Labrador retriever puppies aged between 3 months to 5 months with the signs of canine parvo viral enteritis such as vomiting, bloody diarrhoea, fever, weakness, inappetence, lethargy etc., were randomly selected for this study. These dogs were confirmed to be positive through PCR (polymerase Chain Reaction) for parvo viral enteritis. The selected cases were subjected to thorough physical examination. Haemoglobin (Hb), Packed Cell Volume (PCV), Total Erythrocyte Count (TEC), total leucocyte count, differential count and platelet count were analyzed using automated hematology analyzer (BC-2800 Vet, Mindray, Asia Pacific).

Total proteins, albumin and globulin were estimated with automated biochemistry analyzer (A15 random access analyzer, Biosystems, Barcelona, Spain)

using standard diagnostic kits (Agappe Diagnostics, India). For Coagulation profile, nine parts of blood collected non-traumatically was placed in a tube containing 1 part of 3.2 per cent Tri-sodium citrate solution (0.109M) and mixed gently. Plasma was harvested by centrifugation at 3000rpm for 15min. The harvested plasma was then used for coagulation assays. Activated Partial Thromboplastin Time (aPTT) and Prothrombin Time (PT) with the help of commercial coagulation test kits (Agappe Diagnostics, India) using a Coagulation Analyzer (Mispa Clog, India). ELISA assay was used for D-dimer estimation. DNA was extracted by stool DNA extraction kit. PCR was standardised for the primer set pCPV- 2a and pCPV- 2b, as reported by Pereira *et al.* (2000) with slight modifications.

Results and Discussion

Clinical signs observed included vomiting, bloody diarrhoea, fever, weakness, inappetence and lethargy and was in accordance with previous reports (Salem, 2014). Decrease in values of haemoglobin, packed cell volume, total erythrocyte count (RBC) was observed (Table 1). This might be due to virus induced suppression of bone marrow along with alterations in erythroid, myeloid and megakaryocytes causing these alterations. It is also attributed to intestinal haemorrhage (Salem, 2014). Leucopaenia was observed to be associated with poor prognosis and needed aggressive treatments (Woods *et al.*, 1980; Pogietlerl *et al.*, 1981;

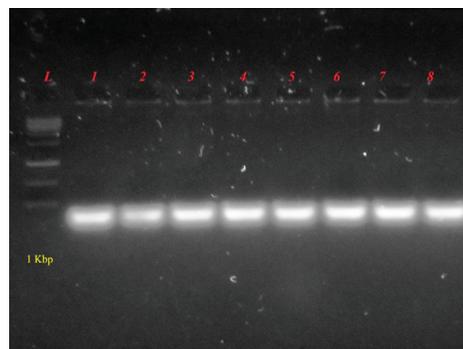
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O'Sullivan *et al.*, 1984). In this study, hypoalbuminemia was observed in all cases and hypoproteinaemia was observed in five cases (Table 2). Similar findings were reported by Dongre (2014) and might be due to direct loss of protein in the form of blood and other proteinous content, the shift of protein poor extra vascular fluid in to the vascular compartment and delay in synthesis of protein. Documented PT in this study ranged from 7.2 to 7.8sec (Table 3), which correlated with findings of Ford and Mazzaferro (2006).

Table 1. Haematobiochemical, clotting profile and D- dimer (MEAN±S.E)

Parameters	Units	Observed Range	Mean±S.E.	Reference range*
AGE (Months)		3-5	3.75±0.31	
Sex		n=8 (6-male & 2-female)		
PCV	%	18.9-40.0	29.03±2.58	37-55
Hb	g%	7.1-14.1	10.44±0.92	12.0-18.0
RBC	m/Cmm	3.82-6.41	4.88±0.35	5.5-8.5
WBC	/Cmm	2600-12800	6787.50±1387.76	6000-17000
Neutrophil	×10 ³ /Cmm	70-78	73.00±1.02	3000-11500
Lymphocyte	×10 ³ /Cmm	18-26	22.88±1.09	1000-4800
Monocyte	×10 ³ /Cmm	0-5	2.25±0.53	150-1350
Eosinophils	×10 ³ /Cmm	0-4	1.63±0.53	100-1250
Basophils	×10 ³ /Cmm	0	0.00±0.00	Rare
Platelets	Lakhs/Cmm	228000-595000	377250.00±46037.16	200000-500000
Total Protein	g/dl	3.12-7.73	4.84±0.49	5.3-7.6
Albumin	g/dl	1.20-2.18	1.69±0.12	3.2-4.7
Globulin	g/dl	1.79-5.55	3.14±0.44	1.5-3.5
Prothrombin Time (PT)		7.2-7.8	7.46±0.08	5.1-7.9 sec
International Normalized Ratio (INR) Value		0.44-0.66	0.53±0.03	-
Activated partial Thromboplastin Time (aPTT)		12.6-18.0	15.54±0.77	8.6-12.9 sec
D-Dimer		-	Negative (< 0.5)	-

Plate 1. Diagnosis Of Canine Parvo Virus (Cpv) By Pcr



Lane 1-8: Positive samples L: 1Kbp DNA ladder

Activated Partial Thromboplastin Time (aPTT) in the present study ranged from 12.6 to 18.0sec, which was markedly prolonged when compared to the findings of Ford and Mazzaferro (2006). A prolonged aPTT could be due to deficiency of intrinsic pathway or contact factors. If PT is normal, then a prolonged aPTT is considered to be due to a deficiency of factors VIII (Hemophilia A and von Willebrand Disease), IX and XI. Prolongation of both aPTT and PT suggests a deficiency or inhibition of the common pathway coagulation factors (factor X, V, and II), or a qualitative or quantitative fibrinogen defect (Lopez *et al.*, 2005; Vlasin *et al.*, 2004). As aPTT is prolonged in this study, this underscores the need for tailor made therapeutic planning with inclusion of plasma products administration as part of therapy in severely affected puppies. In the present study, the Mean±S.E. values of D-Dimer was Negative (<0.5) which correlated with the findings of Otto *et al.* (2000).

Conclusion

Prothrombin Time (PT) in parvo enteritis affected puppies ranged from 7.2 to 7.8sec and the Activated Partial Thromboplastin Time (aPTT) ranged from 12.6 to 18.0sec and is indicator of hypercoagulable state. A prolonged aPTT underscores the need for inclusion of plasma products administration as part of critical care management.

Acknowledgement

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References

- Dongre, J., Mehta, H.K. and Maheshwari, P. 2014. Serum Biochemical Observations in Dogs Affected with Canine Parvo Virus Infection. *Indian Vet. J.*, **91**: 96 – 97.
- Ford, R.B. and Mazzaferro, E. 2006. Kirk and Bistner's Handbook of Veterinary Procedures and Emergency Treatment 8th Edn. Saunders, St. Louis, Missouri, pp:578.
- Lopez, S.T.A., Emanuelli, M.P., Schmidt, C., Raiser, A.G., Mazzanti, A. and Alves, A.S. 2005. Reference ranges of prothrombin time (PT) and activated partial thromboplastin time (aPTT) in dogs. *Cienc. Rural.*, **35**: 381-384.
- O'Sullivan, G., Durham, P.J.K., Smith, J.R. and Campbell, R.S.F. 1984. Experimentally induced severe canine parvoviral enteritis. *Aust. Vet. J.*, **61**: 1-4.
- Otto, C.M., Rieser, T.M., Brooks, M.B. and Russell, M.W. 2000. Evidence of hypercoagulability in dogs with parvoviral enteritis. *J. Am. Vet. Med. Assoc.*, **217**: 1500-1504.
- Pereira, C.A, Monezi, T.A., Mehnert, D.U., D'Angelo, M. and Durigon, E.L. 2000. Molecular characterization of canine parvovirus in Brazil by PCR. *Vet. Microbiol.*, **75**: 127–33.
- Pogietler, L.N.D., Jones, J.B., Patton, S.C. and Webb-Martin, T.A. 1981. Experimental parvovirus infection in dogs. *Can. J. Comp. Med. Vet. Sci.*, **45**: 212-216.
- Salem, N.Y. 2014. Canine Viral Diarrhea: Clinical, Hematologic and Biochemical Alterations with Particular Reference to In-Clinic Rapid Diagnosis. *Glob. Vet.*, **13**: 302-307.
- Vlasin, M., Rauser, R., Fichtel, T. and Novotny, J. 2004. Disseminated intravascular coagulopathy of the Dog. *Acta Vet. Brno.*, **73**: 497-505
- Woods, C.B., Pollock, R.V.H. and Carmichael, L.E. 1980. Canine parvoviral enteritis. *J. Am. Anim. Hosp. Assoc.*, **16**: 171-179.

Effect of Selamectin spot-on against the *Lynxacarus radovskyi* infestation in cats

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Abstract

Lynxacarus radovskyi is a fur mite that is found on the hair shafts of cats. Two adult Persian cats from the same household presented to the clinic with mild alopecia, pruritus and “pepper and salt” appearance over the dorsum region. *Lynxacarus radovskyi* mites were found on the hair upon microscopic inspection. Cats were administered with selamectin spot-on once in month for a period of three months. The cat had complete full recovery following therapy.

Keywords: Selamectin, *Lynxacarus radovskyi*, Cats

Introduction

Domestic cats fur mite is *Lynxacarus radovskyi* which is a member of the Listrophoridae family, is about 0.5 mm long, has an elongated and flattened body, and parasitizes the hair stems. Nichols and Heath (2017) reported that *Lynxacarus radovskyi* had been observed in cats less frequently than *Otodectes cynotis*, *Notoedres cati* and *Sarcoptes scabiei*. This article presents the *Lynxacarus radovskyi* mite infestation in cats.

Case History and Observations

Two adult Persian cats with mild alopecia and pruritus were brought to the clinic for treatment. They had a powder-like substance with “pepper and salt” appearance over the dorsum (Fig.1). Both tape impression smears, superficial and deep skin scrapings were taken and examined under microscope for mites and parasitic eggs. *Lynxacarus radovskyi* mites were found on the hair upon microscopic inspection (Fig.2). Cats were given selamectin (6%) spot-on.

Results and Discussion

Lynxacarus radovskyi mites were found crawling on the hair shafts of the infected cats, as confirmed by microscopic analysis of the adhesive tape impression smears. Jayanthi *et al.* (2017) and Rohini *et al.*, (2020) reported similar findings in cats with lynxacarosis in which the current history and clinical findings of alopecia and pepper salt appearance of the hair coat.

Divya *et al.* (2021) reported that the intensity of dermatitis varied from mild to severe and the outcome could be due to the co-infestation with other parasites, such as lice, fleas, or ear mites. Various products were

used for the management of mite infestation and included oral afoxolaner and moxidectin (Han *et al.*, 2016), sub cutaneous ivermectin injection (Jayanthi *et al.*, 2017), oral sarolaner (Campos *et al.*, 2020), selamectin spot-on (Divya *et al.*, 2021), transdermal fluralaner (Guimaraes *et al.*, 2023). In the present study utilised the spot on containing selamectin and noticed the complete recovery following therapy. Selamectin specifically target the nervous system of invertebrate parasites such as mites. It binds to the glutamate-gated chloride channels, which were present in the nerve and muscle cells of these mites. This binding causes the increased permeability to chloride ions, leading to a continuous influx of chloride ions into nerve cells further causing the hyperpolarization of the nerve cells, disrupting the normal neurotransmission leads to neuro muscular paralysis and eventual death of the mites.



Fig.1: Presence of pepper salt like powder over the dorsum region of a cat with lynxacarosis



Fig.2: Microscopic examination of adhesive tape impression smears showing the *Lynxacarus* mites (400x)

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References

- Campos, D.R., Chaves. J.K.O., Assis, R.C.P., Fernandes, J.I. and Scott, F.B. 2020. Efficacy of oral sarolaner against *Lynxacarus radovskyi* in naturally infested cats. *Veterinary Dermatol.*, **31**: 355-e92.
- Divya, V., Gopalakrishnan, M.A., James, A.A., Ram, R.S., Linija, M.L. and Mohan, R.M. 2021. Concurrent infestation of *Lynxacarus radovskyi* and *Otodectes cynotis* in a Persian cat. *Journal of Pharma Innovation*, **10(10)**: 325-328.
- Guimaraes, B.M.C., Tortoriello, R., Christ, L.X., Manier, C.S.M.L., Campos, D.R., Alonso, L.S. 2023 Effectiveness of transdermal fluralaner in the treatment of *Lynxacarus radovskyi* (Acari: Lirophoridae) in naturally infested domestic cats. *Brazilian J. Vet. Parasitol.*, **32(4)**: e011423
- Han, H.S., Noli, C. and Cena, T. 2016. Efficacy and duration of action of oral fluralaner and spot-on moxidectin/imidacloprid in cats infested with *Lynxacarus radovskyi*. *Vet. Dermatol.*, **27**: 474–e127.
- Jayanthy, C., Nagarajan, B. and Latha, B.R. 2017. Cat fur mite *Lynxacarus radovskyi* in India. *J. Parasitic Diseases*, **41(4)**:1102-1104.
- Nichols, A.J. and Heath, A. 2017. Discovery of the feline fur-mite *Lynxacarus radovskyi* in a New Zealand resident cat. *New Zealand Vet. J.*, **66(1)**:1-7.
- Rohini, B.G., Shyma, V.H. and Pillai, U.N. 2020. *Lynxacarus radovskyi* infestation in a Persian cat: A case report. *Indian J. Vet. Med.*, **40(2)**:30-31.

Therapeutic Management of Monocytic Ehrlichiosis in a dog

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Abstract

One year old German Shepherd dog was presented with history of unresponsive fever, weakness, epistaxis tick infestation and dyspepsia. Clinical examination revealed pale mucosa with mild enlargement of peripheral lymph nodes. Reduced haematocrit, haemoglobin, WBC counts, thrombocytopenia, elevated alkaline phosphate were noticed. Splenomegaly was appreciated in abdominal ultrasonography. Peripheral blood smear revealed morulae of *E. canis* and PCR technique confirmed the presence of canine monocytic ehrlichiosis. The dog was treated with doxycycline for three weeks, along with other supportive drugs and fluid therapy. Dog showed uneventful recovery following therapy.

Keywords: canine monocytic ehrlichiosis, doxycycline

Canine monocytic ehrlichiosis is a tick-borne infectious disease of dogs, which has the potential to be fatal. *Ehrlichia canis* predominantly infects dogs and other members of the Canidae family. All dog breeds are susceptible to canine monocytic ehrlichiosis (CME). This article reports successful management of ehrlichiosis in a German Shepherd dog.

Case History and Observations

A German shepherd dog aged one year was presented to TVCC, Rajendranagar, Hyderabad with history of epistaxis, anorexia, dyschezia, inactiveness and infestation of ticks. Serous oculo nasal discharges, congested conjunctival mucosa and enlarged lymph nodes. Haematological analysis revealed marked thrombocytopenia ($92 \times 10^3/\mu\text{l}$), low levels of Haemoglobin (13.1 g/dL), Total Erythrocyte Count ($6.1 \times 10^6/\mu\text{l}$), Leucocytes ($10.7 \times 10^3/\mu\text{l}$), PCV (37.7%) and increased serum alkaline phosphate (304.6 U/L). Ultrasonographic examination revealed splenomegaly and peripheral blood smear examination revealed morulae in monocytes. Blood sample was subjected for PCR and lateral flow assay that tested positive for Ehrlichiosis (Fig 1).

Treatment and Discussion

The dog was treated with doxycycline @10mg per kg bodyweight, orally, BID for 15 days along with anti-inflammatory drugs and fluid therapy. The dog

showed marked improvement following therapy. Canine Ehrlichiosis is a zoonotic disease transmitted by ticks, posing a global challenge to veterinary and public health. Haematologic abnormalities and clinical signs such as thrombocytopenia, anemia, depression, anorexia, weight loss, fever, and bleeding mark the acute phase (Diniz *et al.* 2022). During the subclinical phase, dogs may either clear the infection spontaneously or remain infected while appearing clinically healthy for months to years (Diniz *et al.*, 2022). Eventually, some dogs may progress to a severe chronic infection characterized by hypoplastic bone marrow, bleeding, and death (Diniz *et al.*, 2022). Thrombocytopenia is considered to be the most common and consistent haematological abnormality of dogs naturally or experimentally infected with *E. canis* (Harrus *et al.* 1999). When diagnosed, treatment involves using antibiotics such as doxycycline, tetracycline, or rifampicin. Doxycycline may be administered orally at 10 mg/kg of dog body weight, divided into two daily doses, for 28 days (Alcón-Chino & De-Simone, 2025).

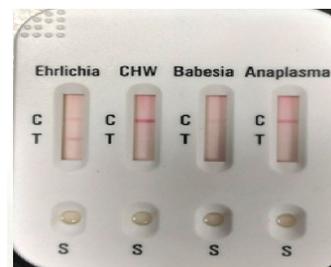


Fig.1: Image showing reports positive for ehrlichiosis by lateral flow assay.

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References

- Alcón-Chino, M.E. and De-Simone, S.G. 2025. Understanding the Diagnosing of Canine Ehrlichiosis: A Comprehensive Review. DOI: 10.5772/intechopen.1010408
- Diniz, P.P.V. and de Aguiar, D.M. 2022. Ehrlichiosis and anaplasmosis: An update. *Veterinary Clinics: Small Animal Practice*, **52(6)**: 1225-1266.
- Harrus, S., Waner, T., Bark, H., Jongejan, F. and Cornelissen, A.W. 1999. Recent advances in determining the pathogenesis of canine monocytic ehrlichiosis. *J. cli. microbiol.*, **37(9)**: 2745-2749.
- Pugliese, M., Biondi, V., Merola, G., Landi, A. and Passantino, A. 2022. Oxidative stress evaluation in dogs affected with Canine Monocytic Ehrlichiosis. *Antioxidants*, **11(2)**: 328.

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Breaking the biofilm: A persistent case of deep pyoderma in a Bullykutta dog

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Abstract

An eleven-month-old male Bullykutta dog was presented with a complaint of persistent skin infection in spite of treatment. The history revealed recurrence over four months, with the latest episode persisting for more than one month. Clinical examination showed hard, adherent crusts with erythema, alopecia, scaling and draining tracts of blood oozing lesions localized at the dorsum. Skin scrapings tested negative for mites, and no fleas or flea dirt's were observed. Direct impression cytology revealed cocci, clusters of bacilli, and neutrophilic infiltration. Trichogram analysis identified trichorrhexis, with all hairs in the telogen phase. Culture confirmed *Staphylococcus* and *Pseudomonas* spp. ABST results showed sensitivity to ceftriaxone and resistance to other drugs. Based on history of draining tracts of blood oozing lesions and laboratory investigations the present was diagnosed as deep pyoderma with a mixed bacterial infection. Treatment began with cephalexin but was later switched to Sulphamethoxazole and trimethoprim due to recurrence. A 10-day regimen of Sulphamethoxazole and trimethoprim combined with omega fatty acid supplements and adjunctive topical therapy resulted in complete resolution and effectively preventing further recurrence of infection.

Keywords: Deep pyoderma, Bullykutta

Introduction

Canine pyoderma is the most frequently occurring bacterial skin infection in dogs. It is primarily associated with staphylococcal species, which naturally inhabit the skin of healthy dogs as common colonizers (Pinchbeck *et al.*, 2006). Causes for recurrent pyoderma infection include ectoparasitic infestations, allergic skin diseases such as atopic dermatitis and food allergic dermatitis where pyoderma is a secondary causative, endocrine conditions, keratinization disorders, genodermatoses (follicular dysplasia, color dilution alopecia, sebaceous adenitis), immunodeficiency (congenital, acquired), bacterial hypersensitivity, resistant strains of *Staphylococcus* sp., non-staphylococcal pyoderma such as *Pseudomonas* leading to prolonged or recurring infections, (Peter, 2005 ; Loeffler and Lloyd, 2018).

Case History and Observations

An eleven-month-old male Bullykutta dog was presented to the Dermatology Unit of Madras

Veterinary College Hospital with major complaint of crusting lesions with erythematous patches and scales, folliculitis, and draining tracts of blood oozing lesions localized all over the dorsum region of the body for the past one month but there is recurrence of infection since four months. Upon clinical examination the lesion is with hard crusts and erythematous patches with loss of hair over the lesion. Deep skin scraping was negative for mites. No traces of flea and flea dirt which ruled out parasitic infestation. Trichogram revealed trichorrhexis, follicular casts and hairs in telogen phase. Direct impression cytology revealed cocci, sheets of bacilli and neutrophilic infiltration. Culture and ABST revealed causatives as *Staphylococcus* and *Pseudomonas* species and sensitive for cephalosporins in ABST. Based on diagnosis it is confirmed as deep pyoderma involving dermis layer.

Treatment and Discussion

Treatment is initiated with cephalexin as there is recurrence observed antibiotic was changed to Trimethoprim and sulpha methoxazole, along with omega fatty acids supplements with biotin and niacinamide and topical adjunctives such as 2% chlorhexidine spray and

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ketoconazole- chlorhexidine shampoo and mupirocin ointment. The patient showed significant improvement with no recurrence observed during follow-up visits. Regular follow-ups were scheduled to monitor recovery and ensure early detection if recurrence occurs. This

case highlights the importance of prompt diagnosis and treatment in managing recurrent deep pyoderma were there are draining tracts indicating damage to dermis layer which is due to biofilm formation and cutaneous barrier dysfunction



Fig.1 Initial presentation: Bullykutta with adherent crusts and scaling along with hyperpigmentation and deep folliculitis



Fig.2 Erythematous crusting all over dorsum



Fig.3 Lesions localized only over dorsum

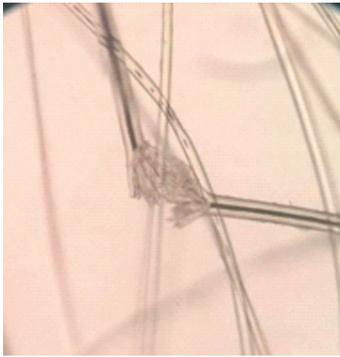


Fig. 4 Trichorrhhexis nodosa

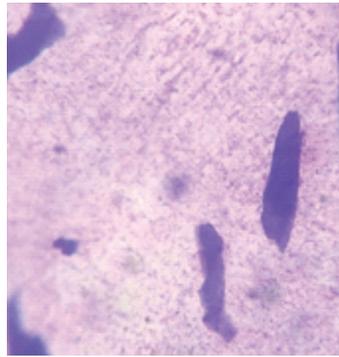


Fig. 5 Cytology impression with rods

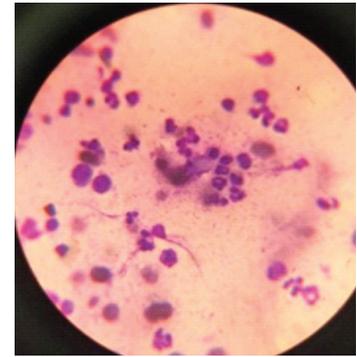


Fig. 6 Cytology impression with neutrophil infiltration and cocci

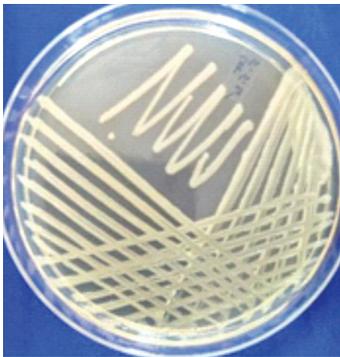


Fig. 7.1 On cetrimide agar the colonies appeared as yellowish mucoid colonies which were suspected for *Pseudomonas sp*

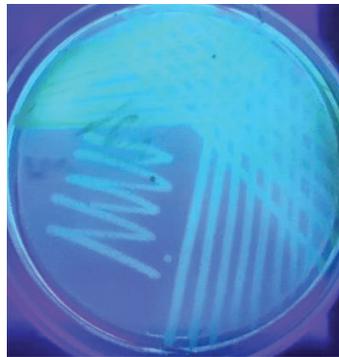


Fig.7.2 Under UV light colonies exhibit fluorescence which were presumptive of *Pseudomonas sp*



Fig. 8 After treatment

Canine pyoderma, a prevalent skin infection, manifests through primary lesions like papules and pustules, with secondary lesions including crusting, epidermal collarettes, alopecia, scaling, erythema, pruritus, lichenification, and hyperpigmentation (Chaudhary *et al.*, 2019). The predominant bacterial agents responsible for pyoderma include *Staphylococcus intermedius* and *Staphylococcus aureus* (Scott *et al.*, 1998), though deep pyoderma often involves opportunistic pathogens such as *Pseudomonas*, *Proteus*, *Escherichia coli*, *Actinomyces*, *Actinobacillus*, *Fusobacterium*, and *Mycobacterium* spp. (Paradis *et al.*, 2001). Deep pyoderma, the most severe variant, extends into the dermis and subcutaneous tissue, manifesting as draining sinuses, nodules, hemorrhagic crusts, and painful swelling, with a risk of systemic spread and bacteremia (Loeffler, 2018). Rather *et al.* (2021) reported that the bacterial persistence in pyoderma was largely attributed to extracellular polymeric substances (EPS), which created a protective barrier

aiding survival and bacterial slime enhances adhesion to host cells, further complicating treatment efforts. Cerasela (2013) and Hill & Moriello (1994) reported that *Staphylococci* produced protein A, a virulence factor that triggered the complement cascade, leading to neutrophil recruitment and heightened inflammation and additionally, protein A contributed to both immediate and delayed hypersensitivity reactions, exacerbating tissue damage and immune dysregulation. Andrade *et al.* (2022) reported that the biofilm production was a key factor in chronic infections, with 51% of *S. pseudointermedius*, 94.6% of *S. aureus*, and 88.9% of *S. coagulans* isolates demonstrating biofilm-forming capability. Most species of *Pseudomonas* are known to readily form biofilms, which play a significant role in causing biofilm-associated infections that often become recurrent and lead to chronic skin infection (Vetrivel *et al.*, 2021). First-line options for Methicillin-susceptible *Staphylococcus pseudointermedius* (MSSP) included clindamycin, cefalexin, and amoxicillin-clavulanate

(Loeffler *et al.*, 2025). Mupirocin, an antibiotic derived from *Pseudomonas fluorescens*, selectively inhibits iso-leucyl transfer RNA synthetase, disrupting bacterial protein synthesis. This case illustrates the intricate interplay between biofilm formation, cutaneous barrier dysfunction, and antimicrobial resistance in recurrent deep pyoderma. Successful resolution through a multifaceted approach incorporating targeted antimicrobial therapy, adjunctive topical treatments, and omega fatty acid supplementation highlights the necessity of comprehensive management strategies for persistent infections.

References

- Andrade, M., Oliveira, K., Morais, C., Abrantes, P., Pomba, C., Rosato, A. E. and Costa, S. S. 2022. Virulence potential of biofilm-producing *Staphylococcus pseudintermedius*, *Staphylococcus aureus* and *Staphylococcus coagulans* causing skin infections in companion animals. *Antibiotics*, **11(10)**: 1339.
- Cerasela, V. (2013). Bacterial pyoderma in dogs and bacterial pathogens isolated from canine pyoderma. *Int. J. Curr. Microbiol. Appl. Sci.*, **8(1)**: 2305-2311.
- Faccin, M., Wiener, D. J., Rech, R. R., Santoro, D. and Rodrigues Hoffmann, A. 2023. Common superficial and deep cutaneous bacterial infections in domestic animals: A review. *Vet. Pathol.*, **60(6)**: 796-811.
- Hill, P. B. and Moriello, K. A. 1994. Canine pyoderma. *J. Am. Vet. Med. Assoc.*, **204(3)**: 334-340.
- Loeffler, A. and Lloyd, D. H. 2018. What has changed in canine pyoderma? A narrative review. *Vet. J.*, **235**: 73-82.
- Loeffler, A., Cain, C. L., Ferrer, L., Nishifuji, K., Varjonen, K., Papich, M. G. and Weese, J. S. 2025. Antimicrobial use guidelines for canine pyoderma by the International Society for Companion Animal Infectious Diseases (ISCAID). *Vet. Dermatol.*, **36(3)**: 234-282.
- Paradis, M., Abbey, L., Baker, B., Coyne, M., Hannigan, M., Joffe, D. and Wellington, J. 2001. Evaluation of the clinical efficacy of marbofloxacin (Zeniquin) tablets for the treatment of canine pyoderma: an open clinical trial. *Vet. Dermatol.*, **12(3)**: 163-169.
- Pinchbeck, L. R., Cole, L. K., Hillier, A., Kowalski, J. J., Rajala-Schultz, P. J., Bannerman, T. L. and York, S. 2006. Genotypic relatedness of staphylococcal strains isolated from pustules and carriage sites in dogs with superficial bacterial folliculitis. *Am. J. Vet. Res.*, **67(8)**: 1337-1346.
- Rather, M. A., Gupta, K. and Mandal, M. 2021. Microbial biofilm: formation, architecture, antibiotic resistance, and control strategies. *Brazilian J. Microbiol.*, **6**:1-18.
- Scott, D.W., Beningo, K.E., Miller, W.H. and Rothstein, E. (1998). Efficacy of clindamycin hydrochloride capsules for the treatment of deep pyoderma due to *Staphylococcus intermedius* infection in dogs. *Can. Vet. J.*, **39**:753-756.
- Vetrivel, A., Ramasamy, M., Vetrivel, P., Natchimuthu, S., Arunachalam, S., Kim, G. S. and Murugesan, R. 2021. *Pseudomonas aeruginosa* biofilm formation and its control. *Biologics*, **1(3)**: 312-336.

Juvenile Cellulitis in a Labrador retriever Puppy

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Abstract

Juvenile cellulitis, commonly known as puppy strangles is characterised by acute swelling, pustular dermatitis and lymphadenopathy. An eight-week-old Labrador retriever puppy was presented with the history of nodular skin lesions and facial oedema that was not resolving with antibiotic therapy. Clinical examination revealed lymphadenopathy and secondary otitis. From skin scrapings, lymph node cytology, bacterial culture and response to therapy, the condition was diagnosed as puppy strangles. The pup made a full recovery upon glucocorticoid therapy.

Keywords: Juvenile pyoderma, sterile granulomatous dermatitis and lymphadenitis, young dogs, immune-mediated

Introduction

Puppy strangles is a sterile vesiculopustular to granulomatous lympho-cutaneous disease primarily affecting dogs under 16 weeks of age; involving the face, ears (pinnae) and submandibular lymph nodes (Cohn and Cote, 2019). One or several puppies in a litter can be affected with breed predilection observed in Golden retrievers, Dachshunds, Labrador Retriever, Siberian Husky and Lhasa Apso (Bassett *et al.*, 2005). A case of juvenile cellulitis in a Labrador puppy is presented in this article.

Case history and Observations

An 8-week-old male unvaccinated Labrador retriever pup was presented to Teaching Veterinary Clinical Complex, Pookode with nodular oedematous lesions in and around face, symmetrical lumps on either side of the lateral neck and severe head shaking. Pet was bright and alert and was reported to have normal food and water intake. All the vital parameters were within the normal range. Severe painful bilateral enlargement of prescapular, mandibular and popliteal lymph nodes could be appreciated (Fig. 2).

Oedema and folliculitis could be noticed primarily in the periocular region, muzzle, rostral chin and aural canals (Fig. 3). Purulent discharge and ulceration could be observed inside both the ear canals (Fig. 4). Fleas were observed on the body. Laboratory investigations included wet film being negative for

filariasis, blood smear negative for haemo-parasites, impression cytology from the ear pinna positive for cocci, rods and Malassezia species. Interpretation of clinicopathologic data revealed leucocytosis ($27.28 \times 10^3/\mu\text{L}$) and mild anaemia (RBC – $4.32 \times 10^6/\mu\text{L}$, Hb – 9.1 g/dL, HCT – 28 %). Serum biochemistry values were normal for the age of the pup. The discharge from ears were given for culture and antibiotic sensitivity testing, which showed sensitivity for amoxicillin-clavulanate, gentamicin, neomycin and ceftriaxone-tazobactam. Prescapular lymph node aspiration cytology revealed the presence of a mixed pattern with greater percentage of degenerative neutrophils and reactive macrophages suggestive of a pyogranulomatous lymphadenitis (Fig. 5). No growth was observed upon culture of the prescapular lymph node aspirate.

Treatment and Discussion

The pet was treated with oral prednisolone sodium phosphate (2 mg/kg PO q24h), amoxicillin-clavulanate (12.5 mg/kg PO q12h), ranitidine (2 mg/kg PO ac q12h) and an ear drop containing ofloxacin, clotrimazole, betamethasone and lignocaine initially given for a span of 14 days. Profound improvement could be noticed on day-5 of the therapy. The enlarged lymph nodes were reduced in size by half and inflammation of the ears were almost resolved (Fig. 6). Prednisolone was tapered gradually as no new lesions developed. Treatment with prednisolone was continued for a period of 4 weeks. A definitive diagnosis can be made by cytological exam and histopathology accompanied by typical

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complete blood count changes including leucocytosis, neutrophilia and normochromic-normocytic anaemia (Bassett *et al.*, 2005). The condition is often mistaken as cutaneous drug reactions or bacterial pyoderma (Bassett *et al.*, 2005). Pyogranulomatous inflammation with no micro-organisms is the main cytologic examination finding unless a secondarily infected area is sampled. If pursued, histopathology findings would show an epidermis that ranges from normal to acanthotic, sometimes with ulcerated lesions. The dermis typically contains multiple discrete or confluent granulomas and pyogranulomas, which are composed of nodular clusters of large epithelioid macrophages and neutrophils. These pyogranulomas are often arranged around hair follicles,

usually without invading the follicular walls, and may extend into the panniculus and subcutis (Bajwa, 2022). Cyclosporine with prednisolone has also been found effective in cases where inadequate response is seen to sole prednisolone; or adverse effects like polydipsia-polyuria are evident (Park *et al.*, 2010; Bajwa, 2022). If cytologic or clinical signs of secondary bacterial infections are present, bactericidal antibiotics such as cephalexin, cefadroxil, or amoxicillin-clavulanate should be administered concurrently. Relapses are uncommon as reported by Kumar *et al.* (2013). The case study emphasizes the need for accurate diagnosis and immediate immunosuppressive therapy for the better prognosis of the disease.



Fig. 1 Juvenile cellulitis having inflamed, swollen muzzle and ear pinnae on day-1



Fig. 2 Bilateral prescapular lymphadenopathy

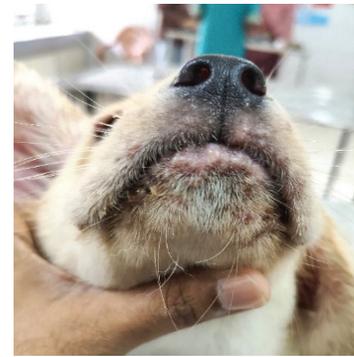


Fig. 3 Erythematous raised nodules at the mucocutaneous junction of lips



Fig. 4 Inflamed and occluded ear canal with pustules and ulceration

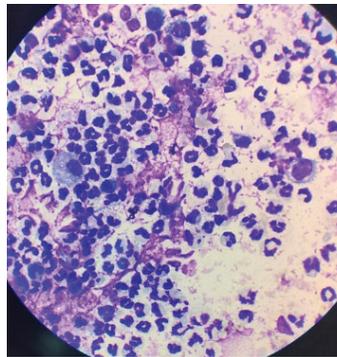


Fig. 5 Prescapular lymph node aspiration cytology suggestive of pyogranulomatous lymphadenitis

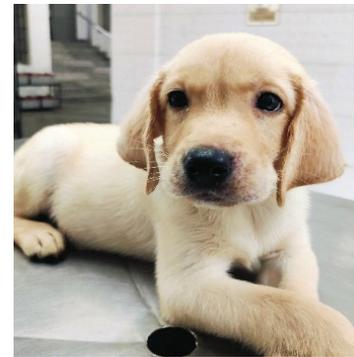


Fig. 6 Puppy post day-5 of glucocorticoid therapy

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Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author.

Animal Ethics Declaration

The study was performed in accordance with the Institutional Animal Ethics Committee and consent from the owner had been collected.

References

- Bajwa, J. 2022. Juvenile cellulitis (juvenile sterile granulomatous dermatitis and lymphadenitis) in a 9-week-old puppy treated with prednisolone-cyclosporine combination therapy. *Can. Vet. J.*, **63**: 313-316.
- Bassett, R.J., Burton, G.G. and Robson, D.C. (2005). Juvenile cellulitis in an 8-month-old dog. *Australian Vet. J.*, **83**: 280-282.
- Cohn, L. and Cote, E. 2019. Cote's Clinical Veterinary Advisor: Dogs and Cats. IV edn., Elsevier, Missouri, pp. 567-568.
- Kumar, A.A., Pillai, U.N. and Aipe, A.A. 2013. Clinical management of juvenile cellulitis in a dachshund pup. *Intas Polivet*, **14**: 234-235.
- Martens, S.M. 2016. Juvenile cellulitis in a 7-week-old golden retriever dog. *Can. Vet. J.*, **57**: 202.
- Miller, W.H., Griffin, C.E. and Campbell, K.L. 2011. Muller and Kirk's Small Animal Dermatology. VII edn., Elsevier, Missouri, pp. 708-709.
- Park, C., Yoo, J., Kim, H., Kang, B. and Park, H. 2010. Combination of cyclosporine A and prednisolone for juvenile cellulitis concurrent with hind limb paresis in 3 English Cocker Spaniel puppies. *Can. Vet. J.*, **51**: 1265-1268.

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Management of *Babesia gibsoni* infection in a Great Dane Dog

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Abstract

Canine babesiosis is a hemolytic disease which is caused by *Babesia canis* and *B. gibsoni*. In this case report, a two-year-old male Great Dane dog was presented with the history of inappetence, dullness and partial blindness for last 10 days. Animal was dull with elevated rectal temperature, slightly tachycardia and hyphema. Thrombocytopenia, elevated liver enzymes, anemia, leukocytosis and increased blood urea nitrogen were observed. Blood smear examination showed presence of intra-erythrocytic piroplasm organisms. Upon confirmatory diagnosis with *B. gibsoni*, the treatment was initiated with Atovaquone in combination with Azithromycin. Hematinic, antioxidant, platelet booster syrup and hemostatic drugs were given twice daily for 5 days. The animal had uneventful recovery following therapy.

Keywords: *Babesia gibsoni*, Blindness, Atovaquone, canine

Introduction

Canine babesiosis is one of the most significant and potentially fatal hemoprotozoan diseases transmitted by ticks to dogs. Babesiosis produced by *Babesia gibsoni* is less severe and persistent than *Babesia canis*. Clinical signs include anorexia, lethargy, icterus, vomiting, and loss of body condition. Although paraplegia, blindness, ocular bleeding, immune-mediated haemolytic anaemia are rare findings, but were noticed by some clinicians (Gonde *et al.*, 2017). This article describes clinical manifestation of Babesiosis and its management in a dog.

Case History and Observations

A 1.5-year-old male Great Dane dog was presented to Teaching Veterinary Clinical Complex, ICAR- Indian Veterinary Research Institute, Izatnagar with a history of fever, weakness, anorexia, dullness, tick infestation, blindness, dark yellow urine, tarry colored feces and erythematous skin lesions on the ventral abdomen. On clinical examination, the animal was having high temperature (105°F), increased heart rate (125 Beats/ minutes), and enlarged popliteal lymph nodes. Congested conjunctival mucus membrane (Fig. 1a), hyphema (Fig. 1b) and echymotic haemorrhagic lesions on the inguinal region (Fig. 2a) were noticed. Pupillary light reflex was sluggish in both eyes and

the animal was unable to see obstacles. Decreased haemoglobin, packed cell volume, erythrocytopenia, and thrombocytopenia, along with leucocytosis were observed. Increased levels of total protein, globulin, serum glutamic oxaloacetic transaminase (SGOT), alkaline phosphatase (ALP), blood urea nitrogen (BUN), and creatinine were observed (Table 1). Blood smears revealed the presence of intra-erythrocytic *Babesia gibsoni*, and few spherocytes (Fig.3). Confirmatory diagnosis was done through PCR which revealed *B. gibsoni* (Fig.4).

Treatment and discussion

The treatment regimen was initiated with Atovaquone (@13.3 mg/kg twice a day) in combination with Azithromycin (@10 mg/kg once a day, orally for 10 days). Supportive therapy included DNS (@ 25mg/kg b. wt. once daily IV for 3 days), hematinic (Iron, folic acid and vitamin B12- 8-10 ml orally), antioxidant (Ascorbic Acid @20mg/ kg PO), platelet booster syrup (extract of *Cariya papaya* leaves, *Tinospora cordifolia* and *Andrographis paniculate*- 10 ml PO), hemostatic drugs (Ethamsylate- 300 mg total dose PO BID twice daily for 5 days). Prednisolone (@2 mg/ kg b. wt. IM twice a week) was administered to manage immune-mediated haemolytic anaemia. There was significant improvement in clinical signs, improvement in vision and had complete recovery in 10 days.

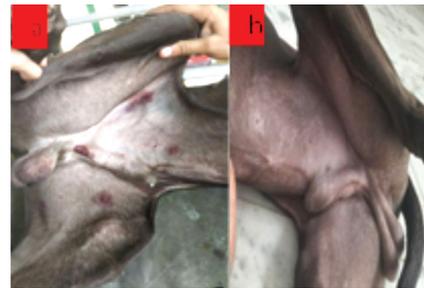
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Table 1. Haemato-Biochemical values

Parameters (%)	Day of presentation (before therapy)	10 days post-treatment	Reference range
Hb (g/dl)	10	11	12-18
PCV (%)	29.5	34	37-55
TEC ($\times 10^6 / \mu\text{l}$)	3.8	4.5	5.5-8.5
TLC ($\times 10^3 / \mu\text{l}$)	19.7	14.6	6-17
Neutrophils (%)	70	71	60-77
Lymphocytes (%)	22	13	12-30
Monocytes (%)	08	06	3-10
Eosinophil (%)	01	00	3-10
Basophil (%)	00	00	0-1
Platelets ($\times 10^6 / \mu\text{l}$)	42	210	150-450
Total protein (g/dl)	9.6	7.9	5.4-7.5
Albumin (g/dl)	2.8	3.1	2.3-3.1
Globulin (g/dl)	6.8	2.9	2.3-3.1
SGPT(IU/L)	43.5	92	10-109
SGOT(IU/L)	102.5	48	13-15
ALP(IU/L)	385	109	76-119
BUN (mg/dl)	88	32	8-28
Creatinine(mg/dl)	1.14	1.10	0.5-1.7



**Fig. 1: a Congested conjunctiva
b Hyphema c After therapy**



**Fig. 2: a Ecchymotic haemorrhage on inguinal region.
B No Ecchymotic haemorrhage on inguinal region**

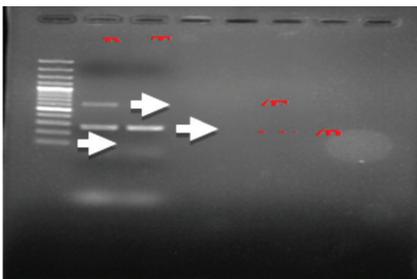


Fig. 3: Confirmation of *B. gibsoni* in the blood sample of a dog by PCR and 2D gel electrophoresis. L: Ladder, PC: Positive Control, TS: Test Sample

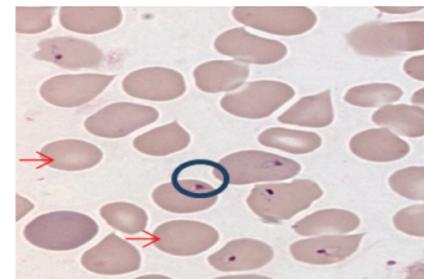


Fig. 4: Identification of *B. gibsoni* by microscopical examination. Blue circle: *B. gibsoni*; RBC with Red arrow: Spherocytes.

Decreased Hb and RBC levels might be due to direct mechanical disruption caused by parasite as it leaves red blood cells, intravascular hemolysis, and immune-mediated or non-immune mediated destruction of red blood cells or due to severe anemia (Venkatesakumar *et al.*, 2018). Rise in level of ALP could be due to damage or dysfunction of biliary system. Increased activities of ALT were might be due to escape of these enzymes from the damaged hepatic parenchymal cells either because of necrosis or changes in membrane permeability, indicating impaired hepatic function (Reddy *et al.* 2014). Atovaquone selectively block protozoan mitochondrial electron transport causing inhibition of pyrimidine and ATP synthesis whereas azithromycin is a macrolide antibiotic that binds to the 50S subunit of the prokaryote ribosome and inhibits the translation of mRNA and bacterial protein synthesis (Baneth, 2018). Additionally, Prednisolone acetate has been administered to mitigate auto-immune haemolysis.

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References

- Baneth, G. 2018. Antiprotozoal treatment of canine babesiosis. *Vet. Parasitol.*, **254**, 58-63.
- Furlanello, T., Fiorioa, F., Caldina, M., Lubasb, G. and Solano Gallegoa, L. 2005. Clinicopathological findings in naturally occurring cases of babesiosis caused by large form *Babesia* from dogs of northeastern Italy. *Vet. Parasitol.*, **134**:77-85.
- Gonde, S. U. R. E. S. H., Chhabra, S., Singla, L. D. and Randhawa, C. S. 2017. Clinico-haemato-biochemical changes in naturally occurring canine babesiosis in Punjab, India. *Malaysian .J Vet Res.*, **8**: 37-44.
- Reddy, B.S., Sivajothi, S., Reddy, L.S.S. and Raju, K.G.S. 2014. Clinical and laboratory findings of Babesiainfection in dogs. *J. Parasit. Dis.*, **92**:268-272. 12.
- Venkatesakumar, E., Kumar, V. and Ramprabhu, R. 2018. Diagnosis and Management of Concurrent Ehrlichiosis and Babesiosis in a Dog. *Intas Polivet*, **19(2)**:267-268.
- Yogeshpriya, S., Sivakumar, M., Saravanan, M., Venkatesan, M., Veeraselvam, M., Jayalakshmi, K. and Selvaraj, P. 2018. Clinical, haemato-biochemical and ultrasonographical studies on naturally occurring *Babesia gibsoni* infection in dogs. *J. Entomol. Zool. Stud.*, **6**: 1334-1337.

Management of Dilated Cardiomyopathy in a Labrador

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Abstract

Dilated cardiomyopathy is the most common form of cardiomyopathy in canines which is characterized by progressive ventricular dilation and loss of myocardial contractility. Four year old, male Labrador was presented with complaint of tachypnea, activity intolerance, inability to walk and inappetence for a period of 15 days to Veterinary Clinical Complex, CVSc., Rajendranagar, Hyderabad. ECG showed deep Q wave and elevated R-wave with VPCs which were also confirmed on 24 hour Holter. Elevated urea, uric acid and SDMA levels were noticed. Thoracic radiograph and echocardiography revealed biventricular dilation, globoid appearance of heart, systolic dysfunction with reduced ejection indices, thus diagnosing the case as DCM. The dog was treated with benazepril pimobendan and furosemide and there was significant clinical improvement after one month of therapy.

Keywords: Dilated Cardiomyopathy, Labrador, diagnostic imaging

Introduction

Dilated cardiomyopathy (DCM) is a progressive myocardial disease in dogs, characterised by ventricular dilation, impaired systolic function, and reduced cardiac contractility, ultimately leading to congestive heart failure and increased risk of sudden cardiac death (Freeman & Rush, 2022). The etiology of canine DCM is multifactorial. Clinically, affected dogs may remain asymptomatic for an extended period before developing signs such as exercise intolerance, respiratory distress, syncope, and ascites (Tidholm & Häggström, 2023). Early detection is crucial, as a significant proportion of cases present with acute heart failure requiring immediate intervention, and sudden cardiac death remains a major concern, especially in predisposed breeds (Tidholm & Häggström, 2023). In the present case report, successful management of dilated cardiomyopathy (DCM) in a dog is reported.

Case history and observation

Four year-old male Labrador Retriever was presented with complaint of dyspnea, reduced exercise tolerance, lethargy and inappetence for the past two weeks. Thoracic radiographs revealed cardiomegaly, pulmonary congestion and edema, tracheal elevation, increased sternal contact, with an increased vertebral heart score of 13.5 (Fig.1). ECG revealed a deep Q wave (0.8 mV), ventricular premature complexes, and an

elevated R wave amplitude (3 mV), findings consistent with biventricular enlargement whereas, continuous ECG monitoring using a Holter device revealed sinus arrhythmia, episodes of sinus bradycardia and sinus tachycardia, alterations in P wave morphology, and intermittent pauses lasting up to 2.3 seconds (Fig 2-3). Haematobiochemical analysis indicated normal hepatic function, elevated blood urea (107.5 mg/dL), blood urea nitrogen (BUN, 50.3 mg/dL), and symmetric dimethylarginine (SDMA, 15.2 µg/dL). NT-pro BNP assay demonstrated a markedly elevated concentration (5391.4 pmol/L), indicative of significant cardiac stress. Two-dimensional B-mode long-axis imaging revealed marked dilation of both the left and right cardiac chambers, as well as thinning of the inter-ventricular septum. In the short-axis view, the left ventricle appeared globoid with a dilated lumen. There was increased left atrial-to-aortic root ratio (LA/Ao) of 2.20, consistent with left atrial enlargement. Fractional shortening (FS) of 12% (reference range: 33.6–38.93%) and an ejection fraction (EF) of 12% (reference range: 55–70%) were observed indicative of severe systolic dysfunction. Based on these findings, along with echocardiographic evidence of severe cardiac chamber dilation and systolic dysfunction, it was diagnosed with overt dilated cardiomyopathy.

Treatment and discussion

The dog was administered with benazepril @ 0.5 mg/kg body weight orally twice daily), pimobendan

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(@ 0.3 mg/kg body weight, orally twice daily), furosemide (@ 2 mg/kg body weight, orally twice daily), and levocarnitine was prescribed for a duration of one month. After one month of therapy, dog was re-evaluated. Electrocardiographic assessment revealed resolution of ventricular premature complexes and normalization of the Q wave and thoracic radiographs showed with no evidence of pulmonary congestion or edema. Echocardiographic parameters, including ejection fraction (EF) and fractional shortening (FS), showed modest improvement. Clinical improvement in the dog was observed.

Dilated cardiomyopathy (DCM) is one of the common acquired cardiac disease in dogs, accounting for approximately 10% of all canine cardiac diagnoses (Freid *et al.*, 2021). The existence of an occult phase, during which affected dogs may exhibit minimal or no overt clinical signs despite progressive myocardial

dysfunction, further complicates timely diagnosis (Calvert *et al.*, 1997). This highlights the importance of routine cardiac screening including echocardiography, electrocardiography, and biomarker analysis in at-risk populations for early detection and intervention (Freid *et al.*, 2021). Once diagnosed, dogs with DCM require lifelong therapy with a combination of medications such as inodilators (e.g., pimobendan), diuretics (e.g., furosemide), and ACE inhibitors (e.g., benazepril or enalapril) to manage symptoms, delay disease progression, and improve quality of life (Keene *et al.*, 2019). Recent advances in pharmacotherapy, including the introduction of angiotensin receptor-neprilysin inhibitors (ARNi), have shown promise in improving cardiac function and renal hemodynamics in experimental and early clinical studies, suggesting a potential future role in the management of canine DCM (Porciello *et al.*, 2022).

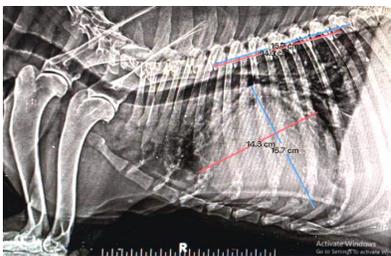


Fig. 1: Right lateral radiograph showing enlarged cardiac silhouette and VHS of 13.5.



Fig. 2: Electrocardiography (ECG) tracing depicting deep Q wave (circle), ventricular premature complexes (arrows) and elevated R

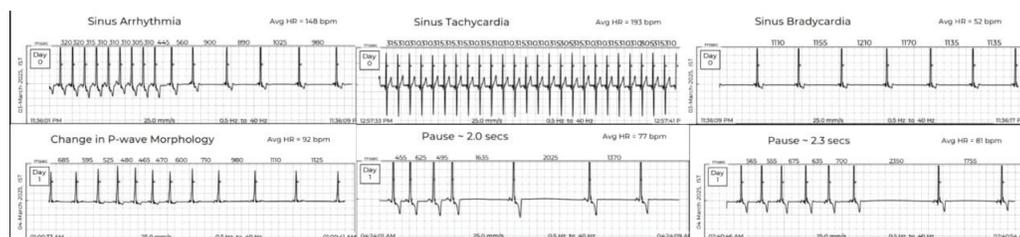


Fig. 3 : Holter ECG- sinus arrhythmia, episodes of sinus bradycardia and sinus tachycardia, alterations in P wave morphology, and intermittent pauses lasting up to 2.3 seconds

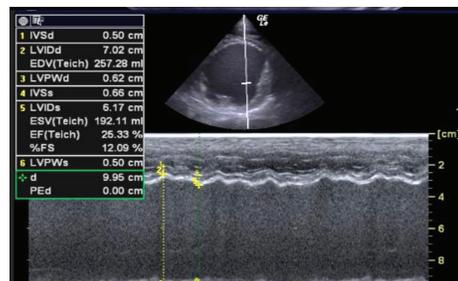
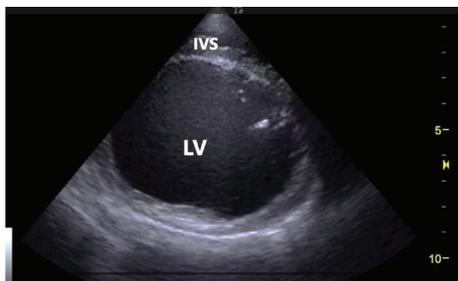


Figure 4: B-mode short axis view-dilation of left ventricle, thinning of inter-ventricular septum (IVS). M-mode echocardiograph- dilated left ventricle lumen and reduced EF

References

- Calvert, C. A., Jacobs, G. J., & Pickus, C. W. 1997. Occult dilated cardiomyopathy in Doberman Pinschers: Prevalence, incidence, and implications. *J. Am. Vet. Med. Assoc.*, **210(4)**: 505–511.
- Freeman, L. M. and Rush, J. E. 2022. Dilated cardiomyopathy in dogs: Etiology, diagnosis, and treatment. *J. Vet. Cardiol.*, **40**: 1–15.
- Freid, K., Daminet, S., Smets, P. and Chetboul, V. 2021. Incidence of canine dilated cardiomyopathy, breed and age distributions, and impact on lifespan. *J. Vet. Cardiol.*, **35**: 1–10.
- Keene, B. W., Atkins, C. E., Bonagura, J. D., Fox, P. R., Häggström, J., Fuentes, V. L. and Stepien, R. L. 2019. ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs. *J. Vet. Internal Med.*, **33(3)**: 1127–1140.
- Porciello, F., Guglielmini, C., & Baron Toaldo, M. (2022). Angiotensin receptor-neprilysin inhibitor (ARNi) in veterinary medicine: A new therapeutic option for canine heart failure? *Veterinary Sciences*, 9(5), 251.
- Tidholm, A., & Häggström, J. 2023. Epidemiology and clinical features of canine dilated cardiomyopathy. *Vet. Rec.*, **192(4)**: 123–129.

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Ebstein's Anomaly in a Golden Retriever puppy

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Abstract

A 3-month-old Golden retriever pup was presented with complaints of abdominal distension, lethargy and inappetence. On clinical examination pale mucous membrane, tachycardia and cardiac murmurs were observed. Haemato-biochemical examination revealed anaemia, leukocytosis, hypoalbuminaemia and elevated serum alkaline phosphatase. Radiographic findings indicated cardiac enlargement and ascites. Increased P wave duration and atrial tachycardia were the major findings on electrocardiography. Echocardiography revealed enlarged right chambers, collapsed left chambers and posteriorly placed tricuspid valves along with regurgitant flow diagnostic of Ebstein's anomaly. Treatment was initiated with antibiotics, diuretics, ACE inhibitors and cardiac supportive. Pup succumbed to death the next day.

Keywords: Tricuspid dysplasia, Ebstein's anomaly, Echocardiographic changes

Introduction

Ebstein's anomaly is a special case of tricuspid dysplasia in which tricuspid valve leaflets were apically displaced compared to the level of mitral annulus and there was a division of right ventricle (RV) into the functional and atrialized RVs leading to enlarged right atrium (RA) and tricuspid regurgitation ultimately leading to right sided heart failure signs (Dearani, *et al.*, 2015). Ebstein's anomaly in a Golden retriever puppy is presented in this article.

Case History and Observations

A 3-month-old male Golden retriever was referred to Teaching Veterinary Clinical Complex, Pookode due to unresolved ascites. Owner reported that the animal was having reduced feed intake and distended abdomen for one month and was treated with amoxicillin, pantoprazole and furosemide but there was no improvement. The animal was lethargic and ascites was noticed on physical examination (Fig.1). Clinical examination revealed blanched mucous membrane, with rectal temperature of 102.6°F, tachycardia and 5/6 holosystolic murmurs on auscultation. No parasitic ova could be detected on faecal sample examination. Clinico-pathologic data revealed mild anaemia (4.9×10^6 erythrocytes/ μ L), leukocytosis ($28.64 \times 10^3/\mu$ L), mild hypo-albuminaemia (2.05 g/dL), and increase in serum alkaline phosphatase (606.17U/L). There was cardiac enlargement, and ascites (Fig. 2). Atrial tachycardia,

increased P wave duration (0.08 sec), short QT interval and increased R wave amplitude (3.4 mV) were observed in electrocardiography (Fig 3). Echocardiography revealed displaced tricuspid valve (Fig 4 & 5), marked dilatation of right heart chambers (Fig 6), and a defect at interventricular septum (turbulence on colour doppler). Considering all these findings animal was diagnosed as having tricuspid valve dysplasia/ Ebstein's anomaly. Treatment was initiated with amoxicillin-sulbactam, pantoprazole, furosemide and amino acids as injections. Advised to continue amoxicillin-sulbactam, pantoprazole, furosemide orally along with cardio-supportive tabs. Animal succumbed to death the next day. The owner denied conducting a necropsy of the dog

Discussion

Tidholm (1997) reported that Ebstein's anomaly is uncommon in dogs and cats. Most commonly affected breed is Labrador retriever (Andelfinger *et al.*, 2003). Frequently observed structural abnormalities in Ebstein's anomaly encompass atrial septal defect, patent foramen ovale, pulmonary stenosis or atresia, and ventricular septal defect. The diagnosis of tricuspid valve dysplasia relies on clinical indicators and supplementary tests. According to Kittleson (1998), significant enlargement of the right atrium in a young animal exhibiting a right apical systolic murmur is typically indicative of severe tricuspid dysplasia. Echocardiographic assessment typically reveals a significantly enlarged right atrium accompanied by right ventricular volume overload

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(Chetboul *et al.*, 2004). Additionally, the left heart chambers are frequently smaller than usual. Tricuspid leaflets may exhibit limited mobility, appear oversized, or adhere to either the interventricular septum or papillary muscles. Furthermore, turbulent regurgitant flow into the right atrium during systole is noticed

(Kittleson, 1998). In human medicine, the primary approach to treating Ebstein's anomaly involves surgical intervention, complemented by medical management targeting signs of right-sided heart failure. Conversely, surgical intervention is not commonly pursued in veterinary practice due to technical limitations.



Fig 1. Golden retriever puppy with ascites

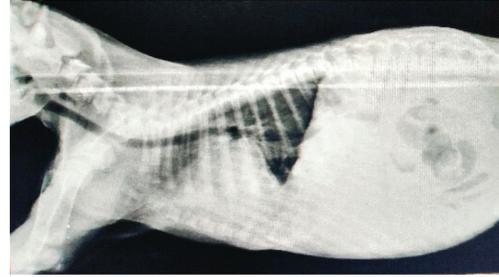


Fig 2. Radiography- Ascites

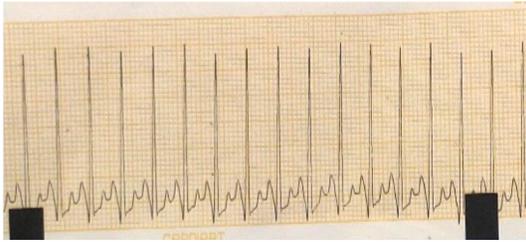


Figure 3. ECG of the puppy suggesting atrial tachycardia. 25mm/s paper speed

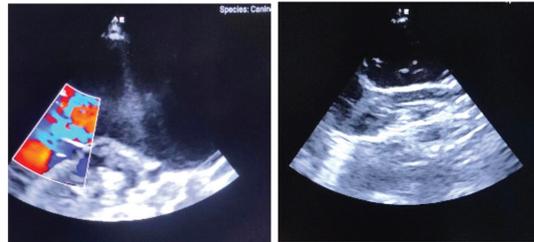


Fig 4 and 5. Echocardiography showing regurgitation and displaced tricuspid valves



Figure 6. Echocardiography showing severely dilated right atrial chamber

References

- Andelfinger, G., Wright, K.N. and Lee, H. 2003. Canine tricuspid valve malformation, a model of human Ebstein anomaly, maps to dog chromosome 9. *J. Med. Genet.*, **40**: 320-324.
- Chetboul, V., Tran, D. and Carlos, C. 2004. Congenital malformations of the tricuspid valve in domestic carnivores: a retrospective study of 50 cases. *Schweiz. Arch. Tierheilkd.*, **146**: 265-275.
- Dearani, J.A., Mora, B.N., Nelson, T.J. Haile, D. T. and O'Leary, P. W. 2015. Ebstein anomaly review: what's now, what's next? *Expert Rev. Cardiovas. Ther.*, **13**: 1101-1109.
- Kittleson, M.D. 1998, Congenital abnormalities of the atrioventricular valves. In: *Small Animal Cardiovascular Medicine*. Bonagura, J.D. and Twedt, D.C. (eds.), Mosby, Saint Louis, pp. 273-281.
- Sangwan, T., Saini, N. and Kataria, D. 2022. Ebstein's anomaly in a French bulldog. *Vet. Res. Forum*, **13**: 615-619.

- Sousa, M.G., Gerardi, D.G., Alves, R.O. and Camacho, A.A., 2006. Tricuspid valve dysplasia and Ebstein's anomaly in dogs: case report. *Arquivo Brasileiro de Medicina Veterinária e Zootecnia*, **58**:762-767.
- Takemura, N., Machida, N., Nakagawa, K., Amasaki, H., Washizu, M. and Hirose, H. 2003. Ebstein's anomaly in a beagle dog. *J. Vet. Med.*, **65**: 531-533.
- Tidholm, A. 1997. Retrospective study of congenital heart defects in 151 dogs. *J. Small Anim. Pract.*, **38**(3):94-98.

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Cerebral Babesiosis in a Geriatric Pomeranian- A Case Report

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Abstract

Canine babesiosis is a globally significant haemoprotozoan disease caused primarily by *Babesia gibsoni* and *Babesia canis*, with clinical presentations ranging from subclinical carriers to fulminant, life-threatening illness. While hematological and systemic manifestations are well documented, central nervous system (CNS) involvement is an uncommon and often overlooked presentation, particularly in geriatric animals, where clinical signs may be non-specific. The current case describes a ten-year-old female Pomeranian exhibiting progressive ataxia, systemic lethargy, and prolonged anorexia in the absence of overt haematologic crisis. Peripheral blood smear examination revealed intraerythrocytic piroplasms consistent with *Babesia gibsoni*. Treatment with atovaquone and azithromycin led to significant clinical improvement, supported by hepatoprotective and metabolic therapy.

Keywords: Canine- *Babesia gibsoni* - cerebral babesiosis

Introduction

Babesiosis is a clinically significant vector-borne protozoan infection in domestic dogs, with *Babesia gibsoni* being a prominent causative agent. Classic clinical signs include pyrexia, lethargy, hemolytic anemia, icterus, and thrombocytopenia. However, in chronic or atypical cases, especially those involving elderly or immuno-compromised animals, the disease may progress insidiously and present with vague systemic or neurological abnormalities. This case report describes a rare instance of subclinical cerebral babesiosis in a geriatric Pomeranian, where the diagnostic challenge was compounded by the absence of visible ectoparasites and the lack of hallmark hematologic derangement. This present case describes the neurological manifestation of babesiosis in Pomeranian dog.

Case History and Observations

A ten-year-old, unspayed female Pomeranian weighing 9.5 kg was presented to the Veterinary Clinical Complex at ANDUAT with a history of progressive neurological deterioration, including hind limb ataxia and impaired motor coordination, accompanied by systemic signs such as persistent anorexia, intermittent low-grade fever, and marked lethargy. The duration of the clinical signs extended over nearly three months, during which the dog exhibited gradual weight loss, reduced voluntary

movement, and increasingly impaired proprioception. The owner reported that the dog had not consumed solid food in the previous three days and had only ingested a single biscuit. The patient had no known exposure to ticks in recent months, and her vaccination and deworming records were complete and up to date. There was no history of trauma, recent travel, toxin exposure, or prior systemic illness. Clinical examination revealed a dull and depressed demeanor, mild dehydration, poor body condition, and a rectal temperature of 101.6 °F. The mucous membranes were pink with adequate capillary refill time, and no icterus or petechiae were observed. Neurological assessment revealed generalized ataxia, delayed proprioceptive positioning, and reduced awareness of environmental stimuli, suggestive of central nervous system involvement. The absence of spinal pain or asymmetry indicated that the signs were unlikely to be due to orthopedic or spinal cord pathology. Initial considerations included the neurological form of canine distemper, tick-borne encephalitis, ehrlichiosis, intervertebral disc disease (IVDD), hepatic encephalopathy, and cerebral Babesia. Canine distemper was ruled out based on the current vaccination, absence of characteristic signs such as myoclonus, ocular/nasal discharge, or hyperkeratosis, and lack of history of exposure. IVDD or spinal trauma was considered less likely because of the symmetrical limb deficits, absence of spinal pain, and no focal neurological deficits.

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Metabolic encephalopathies, such as hypoglycemia and hepatic encephalopathy, were ruled out following normal blood glucose levels and the absence of hepatic insufficiency signs. A complete blood count revealed mild anemia (Hb: 10.2 g/dL) and moderate neutrophilic leukocytosis (78%), consistent with ongoing inflammatory or infectious processes. Biochemical parameters indicated mild elevations in SGPT (112 IU/L) and blood urea nitrogen (26 mg/dL). Giemsa-stained peripheral blood smear revealed small,



Fig 1. --

Treatment and Discussion

Following confirmation of the diagnosis, treatment was initiated with a combination of Atovaquone and Azithromycin, a regimen known for its high efficacy against *Babesia gibsoni*. Atovaquone was administered orally at a dose of 13.3 mg/kg every eight hours, while Azithromycin was administered at 10 mg/kg once daily for ten days. Intravenous isotonic fluids (Lactated Ringer's solution) were administered to correct dehydration and support renal perfusion. Nutritional supplementation, including energy-dense palatable diets and B-complex vitamins, was provided to counteract anorexia and support hematopoiesis. Within 48 h of treatment initiation, the patient exhibited noticeable improvements in mentation and appetite. By the fifth day, proprioceptive reflexes improved, and the animals were ambulatory with reduced ataxia. By the tenth day, neurological signs had fully resolved, and peripheral blood smears were negative for parasitemia.

This case underscores the increasingly recognized but underreported phenomenon of neurological babesiosis in dogs, particularly in geriatric patients. The pathogenesis of cerebral babesiosis is believed to involve the sequestration of parasitized erythrocytes within the cerebral microvasculature, resulting in microthrombi formation, localized hypoxia,

intraerythrocytic ring-form piroplasms morphologically consistent with *Babesia gibsoni*, establishing the final diagnosis of subclinical cerebral babesiosis. The diagnostic challenge was compounded by the absence of thrombocytopenia, overt hemolysis, or visible tick burden, which are typically expected in acute babesiosis. However, the neurological signs combined with haematological and parasitological findings confirmed a case of cerebral involvement by *B. gibsoni*, manifesting in a subacute, atypical form.

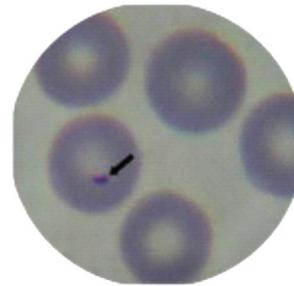


Fig 2. Giemsa-stained smear intraerythrocytic piroplasms

and perivascular inflammation. Multiple treatment options have been proposed for *Babesia gibsoni*, including traditional agents such as Diminazene aceturate and imidocarb dipropionate, both of which have shown limited efficacy in persistent or neurologically involved cases. Multi-drug regimens, such as clindamycin-metronidazole-doxycycline (CMD) and PCR-monitored protocols involving doxycycline with fluoroquinolones or rifampin, have also been employed with inconsistent success and prolonged treatment durations. In contrast, the combination of Atovaquone and Azithromycin has emerged as a superior therapeutic strategy owing to its dual mechanisms of action and favorable tissue penetration, including in the central nervous system.

References

- Irwin, P.J. 2009. Canine babesiosis: from molecular taxonomy to control. *Parasit Vectors*. **2(Suppl 1)**: S4. <https://doi.org/10.1186/1756-3305-2-S1-S4>
- Solano-Gallego, L., Baneth, G. 2011. Babesiosis in dogs and cats—expanding parasitological and clinical spectra. *Vet Parasitol.*, **181(1)**:48–60. doi.org/10.1016/j.vetpar.2011.04.023
- Boozer, A.L., Macintire, D.K. 2003. Canine babesiosis. *Vet. Clin. North Am. Small Anim. Pract.*, **33(4)**:885–904. [https://doi.org/10.1016/S0195-5616\(03\)00041-1](https://doi.org/10.1016/S0195-5616(03)00041-1)

- Zygner, W., Gójska-Zygner, O., Bartosik, J., Wedrychowicz, H. 2007. Cerebral babesiosis in a dog. *Pol. J. Vet. Sci.*, **10(3)**:171–5.
- Jefferies, R., Ryan, U.M., Irwin, P.J. 2007. PCR-RFLP detection of *Babesia gibsoni* and other canine erythrocytic parasites. *Vet Parasitol.*, **144(1–2)**:20–7. doi.org/10.1016/j.vetpar.2006.09.040
- Lin, M.Y., Huang, H.P., Hsieh, L.E., Lin, C.N. 2012. Therapeutic efficacy of atovaquone and azithromycin for treating *Babesia gibsoni* in dogs. *Vet. Parasitol.*, **186(3–4)**:159–64. doi.org/10.1016/j.vetpar.2011.11.054
- Baneth, G., Harrus, S., Ohnona, F.S., Aroch, I. 2004. Long-term efficacy of atovaquone and azithromycin combination therapy for canine babesiosis caused by *Babesia gibsoni*. *Vet Parasitol.*, **119(1)**:25–33. https://doi.org/10.1016/j.vetpar.2003.10.013
- Chakrabarti, A. 2013. *Textbook of Clinical Veterinary Medicine*. Vol. 2. Ludhiana: Kalyani Publishers; 2013. p. 211–7.
- Bhatia, B.B and Pathak, K.M.L. 2016. *Textbook of Veterinary Parasitology*. 3rd ed. New Delhi: CBS Publishers, pp. 210–6.
- Sanyal, P.K. 2013. *A Textbook of Veterinary Parasitology*. 2nd ed. New Delhi: Indian Council of Agricultural Research 2013. p. 189–96.
- Plumb, D.C. 2018. *Plumb's Veterinary Drug Handbook*. 9th ed. Stockholm, WI: PharmaVet Inc.; pp. 104–106, 190–193.
- Ettinger, S.J. and Feldman, E.C. 2017. *Textbook of Veterinary Internal Medicine: Diseases of the Dog and Cat*. 8th ed. St. Louis: Elsevier, Vol. 2, pp. 759–767.
- Kiran, N.K., Ananda, K.J., Sunitha, S., Prasad, H.L.K. 2009. Subclinical babesiosis in a Labrador dog: A case report. *Vet. World*, **2(12)**:478–9.

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Therapeutic management of Trypanosomiasis with bilateral corneal opacity in a dog

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Abstract

Two year old male mongrel dog was brought to the Teaching Veterinary Clinic at College of Veterinary Sciences and A.H., Jalukie, Nagaland, India, having markedly pale mucous membrane, corneal opacity and inappetance. On the basis of the history and clinical signs, a tentative diagnosis of canine trypanosomiasis was made followed by its confirmation with Giemsa stained blood smear examination. The animal was successfully treated with single dose of diminazene aceturate at the dose rate of 3.5 mg/kg body weight, intramuscularly along with the supportive therapy.

Keywords: Corneal opacity, Diminazine aceturate, Trypanosomiasis

Introduction

Trypanosomiasis is a haemoprotozoan disease which is caused by various species of *Trypanosoma* sp. It affects a wide range of hosts such as camels, horses, cattle, and buffaloes and dogs. The illness is spread by biting flies, specifically Tsetse, Tabanus, Stomoxys, and Culicoides (Green, 2006). However, dogs might acquire sick by consuming the carcass of a diseased animal. Two forms of Trypanosomiasis occurs in dogs viz. American trypanosomiasis (Chagas disease) caused by *T. cruzi* and African trypanosomiasis (surra or sleeping sickness) produced by *T. evansi*. However, on the Indian subcontinent, dog trypanosomiasis is mostly caused by *T. evansi* (Eloy and Lucheis 2009), which is deadly in dogs and second only to horse trypanosomiasis. The condition is often acute in dogs, with clinical symptoms including hind leg oedema, anorexia, intermittent fever, ocular opacity, lethargy, dehydration, pale mucous membranes, fever, and weight loss (Eloy and Lucheis 2009).

Case History and Observations

A male mongrel dog of 2 years age weighing 13 kg with improper deworming and vaccination history was presented to the Teaching Veterinary Clinic at College of Veterinary Sciences and A.H., Jalukie, Nagaland, India, with history of anorexia, bilateral corneal opacity (Fig.1) and dullness for a week. On clinical examination, pyrexia (103°F), markedly pale mucous membrane, enlarged submandibular lymph node, bilateral corneal

opacity and generalized debility was observed. So, on the basis of history and clinical observations, a tentative diagnosis of canine trypanosomiasis was made followed by confirmation with Giemsa stained blood smear (Coles, 1986). Examination revealed the presence of *Trypanosoma* organism outside the RBCs (Fig.2). Haematology revealed normocytic normochromic anemia with neutropenia and lymphocytosis (Table 1).

Table 1: Preliminary Blood Report

Blood Parameters	Normal Range	Result
1. Hb g/dL	12-18	9.0
2. PCV %	37-55	25.4
3. RBC m/mm ³	5.5 – 8.5	4.05
4. WBC m/mm ³	6 – 17	8.16
5. Platelets m/mm ³	120 – 600	349
6. Neutrophils %	50 – 80	36.5
7. Lymphocytes %	8 – 38	62.7

Treatment and discussion

Therapeutic regimen was comprised of Diminazine aceturate-Phenazone combination (@ 3.5 mg/kg deep IM; Rani and Suresh, 2007). Supportive treatment with hepatic protectant and iron supplement was given as 5 ml twice daily, Meloxicam with Paracetamol 150 mg/ml (5 mg +150 mg/ml @ 0.4 mg/kg BW IM BID for 3 days) was also given. Improvement with respect to corneal opacity took 7 days time (Fig.3).

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The dog recovered clinically, within a week's time. The reported clinical signs were in agreement with the findings of Dakhane *et al.*, 2024, Howes *et al.* (2011), Rani and Suresh (2007), Saurabh Kumar (2017). A single dose of diminazene aceturate @3.5 mg/ Kg body weight had been successful in treating the dog with trypanosomiasis and similar finding was also reported

by Ramesh *et al.* (2016). In conclusion, trypanosomiasis in dogs causes gradual unthriftiness, ocular and renal involvement. Clinical symptoms along with blood smear examination and complete blood count (CBC) can be used to diagnose the disease. A single dose of diminazene aceturate can be successfully used to treat the disease.

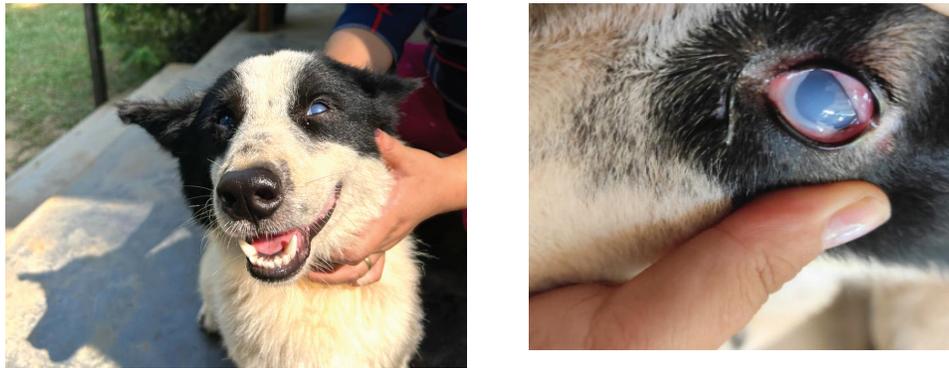


Fig.1. Picture showing bilateral corneal opacity

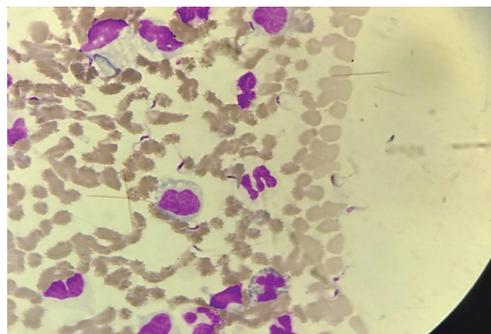


Fig.2. Giemsa Stained Blood Smear of Dog (X100) showing Trypanosomes.



Fig.3. Clearance of Corneal opacity

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Reference

- Agrawal, H., Jaiswal, M. and Tripathi, A.K. 2020. Successful management of trypanosomiasis in a dog. *Indian J. Vet. Med.*, **40(2)**:35-36.
- Coles, E.H. 1986. *Veterinary Clinical Pathology*. 4th edn. WB Saunder's Company, Philadelphia, USA. 53-56.
- Dakhane, P.S., Suryawanshi, A.A., Thorat, G.C. and Gimmvanekar, S.S. 2024. Therapeutic management of Canine Trypanosomiasis: A case report. *International Journal of Veterinary Sciences and Animal Husbandry*, **9(3)**: 277-278.
- Eloy, L.J. and Lucheis, S.B. 2009. Canine trypanosomiasis: Etiology of infection and implications for public health. *J. Venom Anim. Toxins Incl. Trop. Dis.*, **15(4)**:589–611.
- Green, C.E. 2006. *Infectious diseases of dogs and cats*. 3rd. edn. Elsevier Inc. pp. 676- 680.
- Habila, N., Inuwa, M.H., Aimola, I.A., Udeh, M. U and Haruna, E. 2012. Pathogenic mechanisms of *Trypanosoma evansi* infections. *Res. Vet. Sci.*, **93**:13–17.
- Howes, H., Da Silva, A.S., Athayde, C.L., Costa, M.M., Corrêa, M.M.B., Tavares, K.C.S., Miletti, L.C., Lopes, S.T.A., Amaral, A.S and Schmidt, C. 2011. A new therapeutic protocol for dogs infected with *Trypanosoma evansi*. *Acta. Sci. Vet.*, **39(3)**:988–991.
- Nongo, N.N., Tion, M.T., Apan, T.T., Ogunro, B.N. 2015. A case of Canine Trypanosomosis with epistaxis in a two-year old Alsatian dog. *J. Agric. Vet. Sci.*, **8(11)**:68–72.
- Ramesh, P., Chowdary, S.R.C.H., Chaitanya, Y. 2016. Diagnosis and treatment of canine Trypanosomiasis - A case study. *Int. J. Sci. Environm.. Technol.*, **5**: 3387-93.
- Rani, N.L. and Suresh, K. 2007. Canine trypanosomiasis. *India Vet. J.*, **84**: 186-87.
- Saurabh, K. 2017. Trypanosomosis in dog – A Case Report. *Explor. Anim. Med. Res.*, **7(2)**: 220-222.
- Taylor, T.K, Boyle, D.B. and Bingham, J. 2008. Development of a TaqMan PCR assay for the detection of *Trypanosoma evansi*, the agent of surra. *Vet. Parasitol.*, **153**:255-264.

Management of Traumatic Brain Injury in Juvenile Golden Jackal (*Canis aureus*)

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Abstract

A juvenile male Golden Jackal was rescued from a village. Anamnesis suggested possible injury due to automobile accident. Initial observation and physical examination revealed lateral recumbency and hindlimb stiffness along with dorsal curvature of the back. A thorough neurological as well as orthopedic examination led to neuro-localization of the lesion approximately in the cerebellar region. Radiographic examination concluded that there was no detectable skeletal abnormality. Leucocytosis, neutrophilia and significant elevation in the SGPT and ALP values were noticed. Defecation and urination were voluntary. Initial therapeutic management included administration of a combination of steroidal drugs and supportive therapy which was continued for three days along with supportive fluid therapy. Additionally, infrared therapy was initiated to improve the ambulatory response, coupled with physiotherapy. The pup was fed a protein rich diet along with oral Syrup containing mecobalamin vitamins and lycopene for the duration of rehabilitation. Appropriate ambulation with reduced incoordination was exhibited by the puppy. After a rehabilitation period of around 45 days, the puppy was released back in to the wild.

Introduction

The Golden jackal (*Canis aureus*) is a wild canid found in a varied number of habitats ranging from tropical forests to deserts. In India, the golden jackal is listed in Schedule III of the Wildlife Protection Act (1972). Golden jackals are opportunistic and often venture into human habitations at night to feed at garbage dumps, or scavenge on livestock carcasses. This generally leads to susceptibility of these animals to vehicular trauma due to speeding automobiles. Little retrospective or prospective clinical data exist pertaining to the treatment of canine and feline head trauma patients (Mukherjee, 2004.) The following case report records the therapeutic management of a case of a juvenile male Golden Jackal puppy which was rescued and presented to Jivdaya Charitable Trust, Ahmedabad.

Case History and Observations

A juvenile, male Golden Jackal was presented to the hospital after being rescued from the road by a wildlife rescue team. The jackal was reported to have hit by an automobile and was found lying on roadside. Initial clinical observation, revealed that the puppy was ataxic and exhibited a typical decerebrate rigidity posture. Heart rate and respiration rate were marginally elevated with normal rectal temperature. All the cranial nerve reflexes such as the dazzle, palpebral, menace reflexes were positive for both eyes. Cutaneous trunci responses were present. The Modified Glasgow Coma Scale score

was recorded as 13 indicating a guarded prognosis. Radiographic examination was non-significant. Urination and defecation were voluntary. Leucocytosis, neutrophilia and significant elevation in the SGPT and ALP values were noticed. Neurolocalisation identified the CNS lesions approximately in the cerebellar region.

Treatment and Discussion

Butorphanol was administered intravenously (@ 0.2mg/kg) to induce slight sedation as well as analgesia, ensuring stress free treatment experience. Furosemide (@ 4mg/kg) followed by mannitol (@ 0.25 mg/kg) was administered over a period of 25 minutes. Dextrose (25% @ 4mL/kg) was administered intravenously to correct the fluid deficit. Vitamin B and cefotaxime sodium (@ 30mg/kg) were administered. This therapeutic regimen was repeated twice a day along with physiotherapy which included walking exercises in a pelvic sling and exposure to infrared light for a period of 15 minutes. Massage, stretching, passive range of motion (PROM), assisted standing, weight shifting was also a part of the physiotherapy regimen. A significant improvement in the mentation and gait was exhibited by the puppy after a week from presentation and initiation of therapy. Assisted walking and infrared therapy was continued for a period of two weeks. Supplementation of mecobalamin vitamins and lycopene was done for a period of one month. A significant reduction in ataxia and rapid walking movements were recorded. Exhibition of aggression such as display of canines and growling was exhibited. The patient was released back in the wild

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after extensive rehabilitation for a period of two months under intimation to forest department.

Jackal is successful hunter, especially of rodents although they are scavenger. They tolerate human presence more readily than the wolf (*Canis lupus*) and thus can be commonly seen around human settlements. Due to growing urbanization, roads are constructed to connect villages, has led to increase chance of road accident in wild animals (Ojha 2017). Modified MGCS which is adapted from a human GCS is used to suggest prognosis of a canine TBI patient (Platt *et al.*, 2001). The patient's total MGCS score is calculated by summing the scores of each category. A patient with an MGCS of 3 to 8 (score category I) indicates a grave prognosis, 9 to 14 (score category II) guarded prognosis, and 15 to 18 (score category III) a good prognosis. Spinella (2022) reported that massage improved circulation and lymphatic drainage and could be used to decrease edema and pain while providing stimulation that encourages nerve regeneration. Gibb (2014) reported that massage also promoted neuroplasticity and was beneficial in improving motor performance and deficits. In the current scenario, administration of cortico-steroids and medications to reduce intra-cranial pressure combined with physiotherapy and rehabilitation proved to be a utilitarian strategy in the management of traumatic brain injury in the Golden Jackal Puppy.

References

- Gibb, R.L., Gonzalez, C.L.R., Wegenast, W. and Kolb, B.E. 2010. Tactile stimulation promotes motor recovery following cortical injury in adult rats. *Behav. Brain Res.*, **214(1)**:102-107. <https://doi.org/10.1016/j.bbr.2010.04.008>
- Mukherjee, S., Goyal, S. P., Johnsingh, A. J. T. and Pitman, M. L. 2004. The importance of rodents in the diet of jungle cat (*Felis chaus*), caracal (*Caracal caracal*) and golden jackal (*Canis aureus*) in Sariska Tiger Reserve, Rajasthan, India. *Journal of Zoology*, **262(4)**: 405-411.
- Ojha, A. P., Sharma, G., and Rajpurohit, L. S. 2017. Ecology and conservation of golden jackal (*Canis aureus*) in Jodhpur, Rajasthan. *Journal of Applied and Natural Science*, **9(4)**, 2491-2495.
- Platt, S.R., Radaelli, S.T. and McDonnell, J.J. 2001. The prognostic value of the modified Glasgow Coma Scale in head trauma in dogs. *J. Vet. Intern. Med.*, **15(6)**:581-4. doi: 10.1892/0891-6640(2001)015<0581:tpvotm>2.3.co;2. PMID: 11817064
- Spinella, G., Bettella, P., Riccio, B. and Okonji, S. 2022. Overview of the current literature on the most common neurological diseases in dogs with a particular focus on rehabilitation. *Vet Sci.*, **9(8)**:429.
- Sharma, D. and Holowaychuk, M. K. 2015. Retrospective evaluation of prognostic indicators in dogs with head trauma: 72 cases (January–March 2011). *Journal of Veterinary Emergency and Critical Care*, **25(5)**, 631-639.

Hyperglycemic Hyperosmolar Syndrome in a Lhasa Apso Dog

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Abstract

Hyperglycemic hyperosmolar syndrome is an infrequently described complication of canine diabetes mellitus, differing from diabetic ketoacidosis in certain aspects, including treatment. A nine-year-old Lhasa Apso was presented to the Madras Veterinary College Teaching Hospital with a history of polyuria and polydipsia for a period of one week, and sudden dullness and inappetence. Clinical examination revealed the presence of hyperglycemia (799 mg/dL). An arterial blood gas analysis revealed a primary respiratory alkalosis, hyponatremia and hypokalemia. The serum creatinine level was elevated. Based on the absence of acidosis and ketonuria, and the eventual development of obtundation, a diagnosis of hyperglycemic hyperosmolar syndrome was made. Chronic kidney disease was diagnosed by ultrasonography. Treatment was done with intravenous infusion of normal saline, soluble insulin as a constant rate infusion and potassium chloride. The animal died after four days of treatment. The prognosis is guarded to poor.

Keywords: hyperglycemic hyperosmolar syndrome, diabetes, dog

Introduction

Hyperglycemic Hyperosmolar Syndrome (HHS) is a rare complication of canine diabetes mellitus. It is an infrequently reported syndrome (Trotman *et al.*, 2013). HHS is characterized by severe hyperglycemia and dehydration. The nervous system is principally affected by hyperosmolality and neurologic dysfunction is the major clinical manifestation of severe hyperosmolality (Solomon, 2022). It is crucial to lower the serum sodium concentration slowly, in order to decrease the risk of fluid shifting from the extracellular compartment to the brain and the prognosis is guarded to poor (Gough *et al.*, 2024). The present case describes a case of Hyperglycemic Hyperosmolar Syndrome in a Lhasa Apso dog.

Case History and Observations

A nine-year-old intact female Lhasa Apso weighing 14 kilograms was presented to the Madras Veterinary College Teaching Hospital with a history of polyuria, polydipsia and weight loss for a period of one week. The animal was dull and was reported inappetent. Recurrent bilious vomiting was also seen over the past week. Clinical examination, haemato-biochemical examination, urinalysis, arterial blood gas analysis and abdominal ultrasonography were carried out. Heart rate

was respiratory rate, and rectal temperature were 140 bpm, 180 breaths/minute and 102.5 °F respectively. Congested mucous membranes, dehydration. The popliteal lymph nodes were palpable. The Mild dehydration was present. The systolic blood pressure was 110 mm Hg. Haematology revealed a hemoglobin of 12.2 g/dL, PCV of 35.7%, RBC count of 6.30 m/cmm, WBC count of 25,300/cmm, a platelet count of 3,81,000/cmm and Blood picture revealed neutrophilia. Serum biochemistry revealed hyperglycemia (>600 mg/dL), cholesterol of 158 mg/dL, triglycerides of 189 mg/dL, total protein 8.2 g/dL, albumin 2 g/dL, blood urea nitrogen 54.23 mg/dL, creatinine 4.36 mg/dL, ALT 34 U/L, ALP 198 U/L, total bilirubin 1.06 mg/dL, direct bilirubin 0.93 mg/dL, calcium 8.96 mg/dL and phosphorus 14.72 mg/dL. Urinalysis revealed an acidic pH, proteinuria, glucosuria and absence of ketonuria. Abdominal ultrasonography revealed a diffuse increase in echogenicity of the liver parenchyma (indicative of diabetic hepatopathy) and mild irregularity in the contour of both kidneys (indicative of chronic kidney disease). The arterial blood gas analysis results are tabulated in Table 1 and were indicative of a primary respiratory alkalosis.

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Table 1 – Arterial Blood Gas Analysis

Parameter	Day 1	Day 2	Day 3	Day 4
pH	7.567	7.470	7.417	7.358
pCO ₂	25.1 mm Hg	26.4 mm Hg	21.7 mm Hg	16.6 mm Hg
pO ₂	77.3 mm Hg	90.5 mm Hg	76.5 mm Hg	89.6 mm Hg
cHCO ₃ ⁻	22.8 mmol/L	19.2 mmol/L	14 mmol/L	9.3 mmol/L
BE (ecf)	0.7 mmol/L	-4.5 mmol/L	-10.6 mmol/L	-16.1 mmol/L
Na ⁺	114 mmol/L	133 mmol/L	136 mmol/L	144 mmol/L
K ⁺	3.2 mmol/L	3.5 mmol/L	3.6 mmol/L	5 mmol/L
Ca ⁺⁺	0.96 mmol/L	0.93 mmol/L	0.84 mmol/L	0.76 mmol/L
Cl ⁻	73 mmol/L	105 mmol/L	109 mmol/L	128 mmol/L
AGap	20 mmol/L	10 mmol/L	14 mmol/L	8 mmol/L
AGapK	23 mmol/L	14 mmol/L	18 mmol/L	13 mmol/L
Glu	>700 mg/dL	>700 mg/dL	>700 mg/dL	689 mg/dL
Lac	3.61 mmol/L	2.39 mmol/L	5.03 mmol/L	6.46 mmol/L
BUN	71 mg/dL	61 mg/dL	80 mg/dL	80 mg/dL
Crea	6.64 mg/dL	5.62 mg/dL	7.81 mg/dL	7.81 mg/dL

Treatment and Discussion

Dog was administered with intravenous infusion of Potassium chloride (20 mEq) diluted in Normal Saline to combat the hyponatremia and hypokalemia. This was followed by an IV infusion of soluble insulin (@ of 0.1 IU/kg/hour mixed in Normal Saline). Pantoprazole and ondansetron injections were given intravenously. The animal was presented for the next three days in lateral recumbency, in progressively deteriorating condition. Hourly monitoring of vitals and blood glucose was done. The systolic blood pressure dropped to 90 mm Hg. Oxygen supplementation was given on day four as oxygen saturation dropped down to 89 per cent. The animal was discharged in the evening with a blood glucose of 300 mg/dL. The animal collapsed the next morning on the way to the hospital.

Hyperglycemia contributes substantially to the serum osmolality in diabetic animals (Feldman and Nelson, 2004). Glucose and sodium are important contributors to serum total and effective osmolality in diabetics. Hyperglycemia establishes an osmotic gradient between the extracellular and intracellular compartment of the brain resulting in cellular dehydration. This induces altered consciousness and may explain the dullness and recumbency seen in these patients (Doromal and Canter, 1973). Hyperglycemia stimulates endogenous insulin production and may in

some cases cause intracellular translocation of potassium leading to hypokalemia (DiBartola and de Moraes, 1992). The excretion of glucose by the kidneys also causes an osmotic diuresis and may cause depletion in potassium, sodium and phosphorus, thereby aggravating the hypokalemia (Evans, 1992). The hyperosmolality of the serum and the absence of acidosis led to a diagnosis of HHS (Schermerhorn and Barr, 2006). Treatment involves addressing of dehydration, hyperglycemia, electrolyte abnormalities and concurrent disease. The presence of concurrent disease complicates recovery and the prognosis is guarded to poor.

Acknowledgement

The authors wish to thank the authorities of TANUVAS for the facilities provided.

Conflict of Interest

The authors declare that there is no conflict of interest.

References

- DiBartola, S.P. and de Moraes, A. 1992. Disorders of potassium: hypokalemia and hyperkalemia. In: Fluid therapy in small animal practice. Ed. S.P. DiBartola and W.B. Saunders, Philadelphia, pp. 89-115.
- Doromal, N.M. and Canter, J.W. 1973. Hyperosmolar hyperglycemic nonketotic coma complicating

- intravenous hyperalimentation. *Surgery, Gynecology and Obstetrics*, 136: 729-732.
- Evans, M. 1992. Hyperglycemia during nutrition support. *Crit. Care Nurse*, **12**: 64-70.
- Feldman, E.C. and Nelson, R.W. 2004. *Canine and feline endocrinology and reproduction*, 3rd edition. Philadelphia: WB Saunders; 589 pp.
- Gough, S.M., Thevelein, B., Brainard, B.M. and Koenig, A. 2024. The sodium correction factor for dogs undergoing treatment for a hyperglycemic crisis is a 1.6 mEq/L decrease in sodium per 100 mg/dL increase in glucose. *J. Am. Vet. Med. Assoc.*, **262(8)**: 1069-1075.
- Schermerhorn, T. and Barr, S.C. 2006. Relationships between glucose, sodium and effective osmolality in diabetic dogs and cats. *J. Vet. Emerg. Crit. Care*, **16(1)**: 19-24.
- Solomon, B. 2022. A case of hyperglycemic hyperosmolar syndrome in a pug dog. *Can. Vet. J.*, **63**: 1061-1064.
- Trotman, T.K., Drobatz, K.J. and Hess, R.S. 2013. Retrospective evaluation of hyperosmolar hyperglycemia in 66 dogs (1993-2008). *J. Vet. Emerg. Crit. Care*, **23(5)**: 557-564.

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Intravenous Lipid Therapy in the Management of Ivermectin Toxicity in a Cat

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Abstract

A 6-month-old female domestic short-haired kitten weighing about 1 kg was presented to Veterinary College and Research Institute, Namakkal with ataxia and lateral recumbency for 10 hours following accidental ingestion of a 10 mg over-the-counter ivermectin tablet. Mydriasis, absence of menace response, sluggish pupillary reflexes, tachycardia and dyspnoea were noticed. Ivermectin toxicity was diagnosed based on history and clinical findings. Treatment involved intravenous administration of lipid with normal saline. Significant clinical improvement was noted within 24 hours and by the third day, the kitten showed complete recovery.

Keywords: Ivermectin toxicity, kitten, lipid emulsion

Introduction

Ivermectin is one of the commonly used drug in veterinary medicine, which is effective in the management of both ecto and endo parasites (Gonzalez *et al.*, 2009). Siroka *et al.* (2013) reported that poisoning of over-the-counter drugs is a common problem especially in drugs like ivermectin due to improper dosing or while used in non-target species. Although toxicity and treatment of ivermectin are well documented in dogs, only a few reports of toxicity and treatment in cats and kittens have been reported so far. This article describes management of ivermectin toxicity in a kitten.

Case History and Observations

A 6-month-old female kitten weighing one about kilogram was presented to Veterinary College and Research Institute, Namakkal with ataxia for the past 10 hours. The owner reported that she had been given one

tablet of ivermectin (10 mg) bought over the counter in the previous night to control ticks. The dosage of the drug was 50 times the recommended dosage of 0.2 mg/kg. Generalized ataxia, lateral recumbency, mydriasis, tachycardia and dyspnoea were noticed. There was absence of menace and sluggish pupillary reflex.

Treatment and Discussion

Intravenous administration of lipid emulsion (@ 1.5mL/kg) was infused initially and followed by constant infusion (@ 0.25 mL/kg/min). Next day of presentation, the kitten was normothermic (101.6 °F) with heart rate of 110 bpm and normal respiration rate. Fluid therapy with normal saline (@ 20 ml/kg, IV) was given. Mydriasis got resolved and the pupillary and menace reflex were regained and the vision returned to normal. On 3rd day of review, the kitten was active and showed no signs of ataxia with complete recovery.

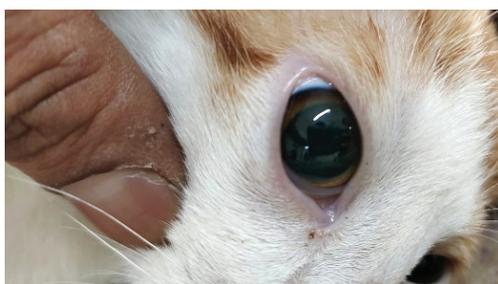


Fig. 1: First day of treatment- mydriasis of eye



Fig. 2: Third day of treatment – normal eyes

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Increased use of over-the-counter drugs by the pet parents without dose calculation can have a detrimental effect on the health of the pet. Gonzalez *et al.* (2009) reported that the signs of ivermectin toxicity included mydriasis, blindness, ataxia, weakness, recumbency, coma and even death. Muhammad *et al.* (2004) reported that neostigmine methyl sulphate @ 25µg and 5% dextrose I/V showed transient improvement from signs of ivermectin toxicity. Intravenous administration of lipid emulsion in adult cats showed complete recovery without any clinical signs according to Jourdan *et al.* (2015). The lipid-soluble nature of the ivermectin was used as an advantage for the removal of the drug using intravenous administration of the lipid emulsion.

References

- Jourdan, G., Boyer, G., Raymond-Letron, I., Bouhsira, E., Bedel, B. and Verwaerde, P. 2015. Intravenous lipid emulsion therapy in 20 cats accidentally overdosed with ivermectin. *J. Vet. Emerg. Crit. Care.*, **25**(5): 667-671.
- Gonzalez-Canga, A., Sahagun-Prieto, A. M., Diez-Liebana, M. J., Martinez, N. F., Vega, M. S, and Vieitez, J. J. 2009. The pharmacokinetics and metabolism of ivermectin in domestic animal species. *Vet J.*, **179**(1): 25–37. doi:10.1016/j.tvjl.2007.07.011.
- Muhammad, S., Saqib, M. and Ahmad, M. 2004. Use of Neostigmine in Massive Ivermectin Toxicity in Cats. *Vet. Hum. Toxicol.*, **46**(3): 132–134.
- Siroka Z and Svobodova Z. 2013. The toxicity and adverse effects of selected drugs in animals – overview. *Pol. J. Vet. Sci.*, **16**(1): 181–194.

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Abstract, Keywords, Introduction, Materials and Methods, Results, Discussion, Acknowledgement, if any, references, Tables, Figures

Title: Papers should be headed with full title, the initials and surname(s) of the author(s) and address of the Institution where the work was carried out. A shortened version of the title should also be supplied for running headlines. The serial titles are not acceptable, so each paper should have an individual title.

Abstract: This should not exceed 300 words and should outline briefly the purpose of the study, important findings and conclusions. Repetition and generally known information should be avoided.

Keywords: Important and relevant 4-6 keywords be mentioned

Introduction: This part should state briefly the nature and purpose of the work together with the important findings of previous workers.

Materials and Methods: The author(s) should describe materials, methods, apparatus, experimental procedure and statistical methods in detail to allow other workers to reproduce the results. Sub-heading may be used in this part.

Results: The experimental data should be presented clearly and concisely. Information presented in tables and figures should not be repeated.

Discussion: This should focus the interpretation of experimental findings. Do not repeat data presented in the introduction or information given in the result. References in this part should be cited as follows.

as observed by Kumar et al. (1984) or in parentheses were found (Dwivedi et al., 1983; Singh and Singh, 1984)

Acknowledgement(s): This should be short. Grants and technical helps provided should be acknowledged.

References: All publications cited in the text should be presented in the form of a list of references arranged alphabetically according to authors' surnames. Don't give serial numbers. Use the following system for arranging the references.

For periodicals: Name(s) and initials of author(s) year of publication, title of the paper, abbreviated title of the journal (in conformity with the World list of Periodicals), volume number (bold), colon, first and last page numbers.

a. For periodicals:

Bartley, E.E., Wheatcroft, K.L., Claydon, T.J., Fountaine, F.C. and Parrish, D.V. 1951. Effect of feeding saureomycin to dairy calves. *J. Anim. Sci.* **10**: 1036-1038.

b. For books:

Snedecor, G.W. and Cochran, W.G. 1994. *Statistical Methods*. VIII edn. Iowa State University Press, Iowa, USA, pp.287-192.

c. For chapter in a book:

Thomas, J.R. and Charles, C.C. 1997. Calcium regulating hormones and diseases of abnormal mineral metabolism. In: *Clinical Biochemistry of Domestic Animals*. Kaneko, J.J., Harvey, J. W. and Bruss, M.L. (eds) V. edn. Academic Press, London, pp. 619-702.

d. For thesis:

Singh, S.K. 1998. Studies on clinico-biochemical changes in Downer cow syndrome. M.V.Sc .thesis, Punjab Agriculture University, Ludhiana, India.

e. For proceedings of symposia/conference:

Shah, R.L., Kataria, J.M., Arya, S.C. and Verma, K.C. 1996. Study on inclusion body hepatitis in broiler chicks. *Proc. XX World Poultry Congress* held on Sept. 2-5, 1996, New Delhi, Vol. IV, pp. 313-314.

Tables: These should be as few as possible and typed on separate sheets and numbered in Roman numerical. Each table should have a brief and self-explanatory title.

Figures: Only good quality, unfolded and unmounted glossy prints of half-tone illustrations and clear lines drawings in India ink are accepted. The number of figures, the author's name and top of figure should be indicated lightly on the back by soft pencil. Legends to the figures should be typed on a separate sheet of manuscript. All the figures should be referred to in the text and their approximate place be indicated on the margin. A statement of the magnification of illustrations should be given wherever applicable. The colored illustration are also accepted.

Abbreviations and Symbols: Metric system should be followed in the text. The quantities should be expressed in SI units. Contributor(s) are requested to use the following abbreviations.

Body weight	b wt	Litre	l	Calory	cal
Meter	m	Centimeter	cm	Microlitre	μl
Counts per minute	cpm	Milligram	mg	Cubic centimeter	cm ³
Millilitre	ml	Degree centigrade	°C	Minute(s)	min
Degree Fahrenheit	°F	Once a day	od	Decilitre	dl
Parts per million	ppm	Gram	g	Percent	%
Hour(s)	hr	Picogram	pg	Inch	in
Revolution per min	rpm	Intramuscular	im	Seconds(s)	sec
Intraperitoneal	ip	Square centimeter	cm ²	Intravenous	iv
Subcutaneous	sc	Kilo calories	kcal	Thrice a day	tid
Kilogram	Kg	Year(s)	yr	Twice a day	bid
Volts	V				

All other abbreviations should be spelled out when first used in the text.

Footnotes: These should be used only when absolutely essential. When used, they should be numbered in text, indicated by superscript numbers and kept as short as possible.

CLINICAL ARTICLES

Clinical case reports of interesting and rare nature are published under this heading. The article sent for publication under this head, should not contain more than three typed pages including references and illustrations and should be marked 'Clinical Article' at the right upper corner of the first page of manuscript. An abstract of the case is necessary along with keywords. The manuscript should contain history and important clinical observations of the case, tentative diagnosis and its confirmation, line of treatment used and fate of the case. At last, it should have a brief discussion on the line of treatment and conclusion. All these can be given in separate paragraphs sequentially and sub-heading are not required.

The acknowledgement, if necessary, may be given but it should be as short as possible and should not bear subheadings. The references should be given as per format for the research articles.

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They should be in the same general format as full length papers, but should not exceed a maximum of three typed pages including tables and illustrations. An abstract of the case is necessary along with keywords. The subheading, except for acknowledgement and references, should not be written in the manuscript. The manuscript for this head should be clearly marked 'Short Communication' at the right corner on the top of the first page of manuscript.

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I, Dr. G. Vijayakumar, Department of Veterinary Clinical Medicine, Tamil Nadu Veterinary and Animal Sciences University, Chennai-600 007, Tamil Nadu hereby declare that the particulars given above are true to the best of my knowledge and belief.

Dated: 30th June, 2025

Dr. G. Vijayakumar