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## Effectiveness of Nutritional Supplement (SKORA 33) in the Management of *Malassezia* dermatitis in Dogs

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### Abstract

*Malassezia* dermatitis is one of the common dermatological conditions in dogs, often requiring comprehensive therapeutic management. Ten dogs were diagnosed with *Malassezia* dermatitis based on clinical signs and confirmed through direct microscopic examination of swab smears stained with Loeffler's methylene blue. Treatment was carried out with itraconazole with omega-3 and omega-6 fatty acids (SKORA 33), antioxidants, vitamins, and trace minerals. Over a three-week treatment period, the supplemented group recorded marked clinical improvement, including enhanced hair regrowth.

**Keywords:** *Malassezia* dermatitis, Dog, nutritional supplement (SKORA-33).

### Introduction

Dermatological disorders are among the most frequently encountered clinical issues in small animal veterinary practice, with *Malassezia* dermatitis being a commonly reported in dogs. Although *Malassezia* species are part of the normal flora on the skin and mucous membranes of healthy dogs, their proliferation can become pathogenic under certain conditions. Successful case management of the condition is often dependent upon both treating yeast overgrowth with topical or systemic antifungal treatments, as well as identifying and correcting the predisposing factors. (Selvi *et al.*, 2024). SKORA-33 is a scientifically formulated plant-based blend fortified tablet with essential fatty acids, natural antioxidants, multi-vitamins, minerals (total 33 supplements) to optimize overall skin health of pets. The study has been conducted to assess the effect of SKORA-33 supplementation in dog with *Malassezia* dermatitis.

### Materials and Methods

A total of ten dogs, irrespective of breed, age and sex that were presented to the small animal outpatient unit, Department of Veterinary Medicine, Rajiv Gandhi Institute of Veterinary Education and Research, Puducherry, with history and clinical signs

suggestive of *Malassezia* dermatitis formed the study group. Dogs already receiving any systemic or topical skin supplements within the last 30 days were excluded from this study. A detailed history, physical, and dermatological examination, followed by collection of samples for cytological examination was obtained from all ten dogs included in the study. Cytology, haematological and biochemical evaluation were undertaken (Marsella *et al.*, 2000). Comparison between pre-treatment (0 day) and post-treatment (29 day) was done using a paired t-test. These ten dogs that were positive on direct microscopic/cytological examination were administered with oral Itraconazole (@ 5mg/kg b wt, sid) for two consecutive days in a week for four weeks as recommended by (Pinchbeck *et al.*, 2002), antifungal shampoo and dermatological supplement tablet (SKORA 33) for 10 to 20 days based on severity. Haematological and biochemical parameters were estimated using haematological analyzer (IDEXX ProCyt Dx) and serum biochemical analyzer (FGBCAOE M001-OEM).

### Results and Discussions

Clinical signs observed in the affected dogs are depicted in Table -1. These findings were in accordance with Selvi *et al.* (2024), who reported that pruritus, erythema, hyperpigmentation, malodour, and traumatic alopecia are the major clinical signs in dogs

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with *Malassezia* dermatitis (fig 1-5). No statistical significance between pre-treatment (0 day) and post-treatment (29 day) values of blood profile was noticed (Table 2). *Malassezia* organisms, appeared as small, oval to peanut- or footprint-shaped structures (Fig. 6).

Clinical response to supplement therapy was monitored on day 29 after 4 weeks of treatment using cytological evaluation and pruritus score. There was a considerable reduction in the mean yeast count and pruritus score post-treatment (Table 3).



At 0<sup>th</sup> day of Pre - treatment



At 29<sup>st</sup> day of Post - treatment

**Fig.1. Dog showing erythematous lesions with alopecia at sternal region**



At 0<sup>th</sup> day of Pre-treatment



At 29<sup>st</sup> day of Post-treatment

**Fig.2. Dog with hyperpigmentation with scaly lesions at ventral aspect of neck region**



At 0<sup>th</sup> day of Pre-treatment



At 29<sup>st</sup> day of Post-treatment

**Fig.3. Greasy rancid odour with erythematous lesions at face, neck and forelimbs.**



At 0<sup>th</sup> day of Pre-treatment



At 29<sup>st</sup> day of Post-treatment

**Fig.4. Lichenification at full ventral aspect of the body.**



At 0<sup>th</sup> day of Pre-treatment

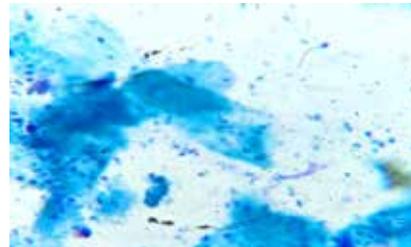


At 29<sup>st</sup> day of Post-treatment

**Fig.5. Otitis externa with Epidermal collarettes.**



At 0<sup>th</sup> day of Pre-treatment



At 29<sup>st</sup> day of Post-treatment

**Fig.6. Swab impression cytology showing budding yeast stained with Löffler's methylene blue.**

**Table 1: Clinical signs observed in dogs affected with *Malassezia* dermatitis.**

SI No.	Clinical signs	No of animals (n* = 10)	Percentage
1.	Pruritus	10	100
2.	Musty/ rancid odour	10	100
3	Erythema	08	80
4	Scales	07	70
5	Hyperpigmentation	06	60
6	Hyperkeratinization	06	60
7	Lichenification	05	50
8	Greasiness	04	40
9	Epidermal collarettes	03	30
10	Otitis	02	20

(\*n= sample size)

**Table 2: Comparison of hematological and serum biochemical values, pre and post treatment in *Malassezia* affected dogs.**

Sl No.	Parameter	Mean ± SE*		t value	p value
		Pre-treatment (0 <sup>th</sup> day)	Post-treatment (29 <sup>th</sup> day)		
1	Hb (g/ dL)	9.66 ± 0.42	9.29 ± 0.68	1.81	0.103
2	PCV%	32.59 ± 1.15	32.53 ± 2.03	-1.15	0.280
3	TLC (x 10 <sup>3</sup> / cmm)	14.04 ± <b>0.56</b>	14.30 ± 2.38	-0.57	0.586
4	Platelet ((x10 <sup>3</sup> /μL)	<b>289.7</b> ± 30.48	246.4 ± 62.9	1.32	0.220
5	ALT(U/L)	30.63 ± <b>1.47</b>	28.67 ± 4.5	0.61	0.554
6	Total protein (g/dl)	7.12 ± 0.26	6.99 ± 0.41	-0.74	0.479
7	Albumin (g/dl)	2.693 ± 0.10	2.83 ± 0.13	-0.76	0.467

\*\*P ≤ 0.05 = ns (not significant) (\*SE= Standard Error)

**Table 3: Comparison of Visual Analog Scale in *Malassezia* affected dogs.**

Sl. No	Parameter	Mean ± SE*		t value	p value
		Pre-treatment (0 <sup>th</sup> day)	Post-treatment (29 <sup>th</sup> day)		
1	Visual analog scale for pruritus	3.500 ± 0.2800	1.100 ± 0.1500	7.55	< 0.0001**
2	Yeast load (mean yeast organism per oil immersion field)	4.300 ± 0.6000	1.200 ± 0.1900	4.93	≈ 0.0008**

\*\* Significant at 0.01 level (P < 0.01), \*SE = Standard Error.

## Conclusion

The present study revealed that incorporating the nutritional supplement into standard antifungal therapy significantly improved the therapeutic outcome.

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## Haemato-biochemical changes in small ruminants with haemoprotozoan diseases

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### Abstract

Haemoprotozoan diseases is one of the important infectious ailments affecting small ruminants. Haemato-biochemical changes in small ruminants with haemoprotozoan diseases was carried out. Animals with fever, lymphadenopathy, haemoglobinuria, tick infestation were screened. 100 number of samples were taken up for the study during the period of November 2024 to November 2025. These blood samples were subjected to haematology and serum biochemistry analysis. Reduction in haemoglobin, packed cell volume, erythrocyte and increase in total leucocyte count and differential count were observed. Elevated serum total bilirubin, aspartate amino transferase, alkaline phosphatase and gamma glutamyl transferase and magnesium were observed. Reduced serum albumin, iron and copper were noticed in these small ruminants.

**Keywords:** Haemoprotozoan diseases, haematology, serum biochemistry

### Introduction

Parasitic infections represent one of the major limiting factors affecting the productivity and health of small ruminants, causing considerable economic setbacks, especially in developing and underdeveloped nations worldwide (Shah *et al.*, 2019). The study is undertaken to evaluate the haemato-biochemical changes in small ruminants infected with haem protozoan diseases.

### Materials and Methods

A total of 100 blood samples were collected from small ruminants, comprising 80 clinically infected (Group II) and 20 healthy animals (Group I) presented to the Large Animal Out-Patient Medicine Unit of the Madras Veterinary College Teaching Hospital, Vepery, Chennai during the period from November 2024 to November 2025. Haematological and serum biochemical parameters were analyzed using Mindray BC-2800 Vet Auto Hemoanalyzer and Auto biochemical analyser (A15 Biosystem Inc, Spain) respectively. Iron and copper were estimated using ProTech Serum Iron

and Copper Estimation Kit by spectrophotometrically at 630 nm and 578 nm respectively.

### Results and Discussion

The Mean  $\pm$  S.E. values of haematological parameters in small ruminants belonging to control healthy animals Group I (n=10) and animals infected with HPD Group II (n=80) are detailed in Table 1.

are in agreement with the reports of Razmi *et al.* (2019) and Jayalakshmi *et al.* (2022). The blood pictures are normocytic to microcytic anemia, it resulting from intraerythrocytic parasitism, hemolysis, and enhanced erythrophagocytosis as reported by Velusamy *et al.* (2015). A highly significant rise ( $p < 0.01$ ) recorded in total leukocyte count (TLC) and differential counts, reflect an active inflammatory and immunological response to parasitic invasion, corroborating the observations of Razmi *et al.* (2019). The Mean  $\pm$  S.E. values of various biochemical parameters in small ruminants belonging to control healthy animals Groups I (n=20) and animals infected with HPD II (n=80) are presented in Table 2.

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**Table 1: Mean  $\pm$  S.E. values haematological parameters**

Parameters	Group I (n=20)	Group II (n=80)	t value	p value
Haemoglobin (g/dl)	11.69 $\pm$ 0.35	7.36 $\pm$ 0.26	6.262	0.00**
PCV (%)	37.90 $\pm$ 0.31	20.98 $\pm$ 0.76	8.42	0.00**
RBC (millions/cmm)	14.08 $\pm$ 0.3	13.61 $\pm$ 0.59	0.301	0.76 <sup>NS</sup>
MCV (fl)	26.43 $\pm$ 0.68	16.29 $\pm$ 0.52	7.28	0.00**
MCH (pg)	8.24 $\pm$ 0.24	5.73 $\pm$ 0.16	5.717	0.00**
MCHC (g/dl)	30.52 $\pm$ 0.85	35.61 $\pm$ 0.59	-3.187	0.00**
WBC cells/cmm	5970 $\pm$ 301.75	20144.93 $\pm$ 1191.26	-4.502	0.00**
Platelets (cells/cmm)	345187 $\pm$ 5100.06	307989.57 $\pm$ 8667.31	1.619	0.10 <sup>NS</sup>
Neutrophils (cells/cmm)	2665.89 $\pm$ 147.67	11633.79 $\pm$ 946.82	-3.585	0.00**
Lymphocytes cells/cmm)	2970.52 $\pm$ 337.15	7525.57 $\pm$ 449.8	-3.813	0.00**
Monocytes (cells/cmm)	89.97 $\pm$ 16.61	754.89 $\pm$ 63.3	-4.038	0.00**

The low values of haemogram in parasitised animals

**Table 2: Mean  $\pm$  S.E. values of various biochemical parameters**

Parameters	Group I (n=20)	Group II (n=80)	t value	p value
Glucose (mg/dl)	62.10 $\pm$ 2.09	87.62 $\pm$ 5.03	-1.916	0.05*
Cholesterol (mg/dl)	110.07 $\pm$ 3.82	118.00 $\pm$ 4.6	-0.597	0.55 <sup>NS</sup>
Total Protein (g/dl)	7.19 $\pm$ 0.09	6.38 $\pm$ 0.16	1.877	0.06 <sup>NS</sup>
Albumin (g/dl)	3.15 $\pm$ 0.1	2.54 $\pm$ 0.07	2.885	0.00**
Total Bilirubin (mg/dl)	0.06 $\pm$ 0.01	0.83 $\pm$ 0.06	-4.231	0.00**
Direct Bilirubin (mg/dl)	0.02 $\pm$ 0.003	0.56 $\pm$ 0.06	-3.644	0.00**
BUN (mg/dl)	18.26 $\pm$ 1.01	28.03 $\pm$ 2.31	-1.597	0.11 <sup>NS</sup>
Creatinine (mg/dl)	1.14 $\pm$ 0.07	1.29 $\pm$ 0.14	-0.411	0.68 <sup>NS</sup>
AST (IU/L)	93.70 $\pm$ 3.88	126.92 $\pm$ 14.62	-2.017	0.05*
ALP (IU/L)	92.50 $\pm$ 7.21	214.08 $\pm$ 40.88	-2.675	0.01**
GGT (IU/L)	37.20 $\pm$ 1.82	42.92 $\pm$ 1.75	-2.253	0.03*
Sodium (mmol/L)	147.88 $\pm$ 2.06	140.03 $\pm$ 1.14	2.537	0.01**
Potassium (mmol/L)	4.36 $\pm$ 0.16	4.09 $\pm$ 0.1	1.039	0.30 <sup>NS</sup>
Chloride (mmol/L)	103.26 $\pm$ 1.41	105.95 $\pm$ 1.84	-0.549	0.58 <sup>NS</sup>
Calcium (mg/dl)	10.53 $\pm$ 0.16	9.85 $\pm$ 0.19	1.322	0.19 <sup>NS</sup>
Phosphorous (mg/dl)	4.82 $\pm$ 0.22	5.61 $\pm$ 0.32	-0.953	0.34 <sup>NS</sup>
Magnesium (mg/dl)	2.21 $\pm$ 0.05	4.82 $\pm$ 0.17	-5.897	0.00**

A highly significant reduction ( $p < 0.01$ ) in the mean serum albumin levels ( $2.54 \pm 0.07$ ) in small ruminants affected by haemoprotozoan diseases (HPD), are similar with results that were documented by Mahmoud *et al.* (2019) and Eliwa *et al.* (2021). The observed hypoalbuminemia in haemoprotozoan infections may be attributed to decreased hepatic synthesis due to liver dysfunction (Abdullah *et al.*, 2022).

Haemoprotozoans infected small ruminants exhibited significantly higher aspartate aminotransferase (AST) ( $126.92 \pm 14.62$ ), alkaline phosphatase (ALP) ( $214.08 \pm 40.88$ ) and gamma-glutamyl transferase (GGT) ( $42.92 \pm 1.75$ ), aligning with earlier observations by Haq *et al.* (2021). The concentration of iron and copper in healthy and HPD infected animals are detailed in Table.3.

**Table 3: Mean  $\pm$  S.E of micro minerals in small ruminants infected with HPD and control healthy groups**

S.No	Group I Control healthy Goats (n=10)	Group II Goats infected with HPD (n=68)	Group III Control healthy Sheep (n=10)	Group IV Sheep infected with HPD (n=12)	F value
Iron ( $\mu\text{g}/\text{dl}$ )	144.55 $\pm$ 5.62	148.72 $\pm$ 5.12	65.98 $\pm$ 8.99	55.47 $\pm$ 4.04	64.029**
Copper ( $\mu\text{g}/\text{dl}$ )	167.99 $\pm$ 6.05	105.12 $\pm$ 4.70	127.56 $\pm$ 5.69	81.83 $\pm$ 3.82	50.088**

Reduced serum iron and copper values in the diseases animal were in agreement with results documented by Sajid *et al.* (2023), who also reported lowered serum iron levels in sheep and goats affected with *Babesia*, *Theileria*, and *Anaplasma* infections. A decreased level of was observed in the infected animals when compared with the control group. Similar findings were described by Esmailnejad *et al.* (2012), who reported reduced serum copper concentration (hypocupremia) in *Babesia*-infected sheep and goats.

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## Assessment of economic impact of babesiosis in cattle in Andhra Pradesh

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### Abstract

The goal of the current study was to document the financial losses incurred by cattle infected with babesiosis between January 2023 and December 2024 in Chittoor district of Andhra Pradesh, India. Throughout the course of the study, the total average financial impact resulting from babesiosis was ₹31,60,464 and the average annual loss resulting from individual cattle was ₹15,049. When comparing the different variables causing the economic loss highest percent was due to mortality loss (39.87 percent), reproductive loss (37.79 percent), production loss (8.05 percent), treatment cost (7.64 percent) and for daily wages loss (6.65 percent).

**Keywords:** Cattle, babesia, financial loss, production loss

### Introduction

Babesiosis is a haemoprotozoan disease that is prevalent in tropical and subtropical regions, including in India which is transmitted by vectors (Salih *et al.*, 2015). It is caused by an intra-erythrocytic protozoan parasite that infects many domestic and wild animals. This disease has a significant economic impact because it results in mortality, morbidity, and a reduction in milk production. The disease causes INR 580.16 crore in losses every year in India (Narladkar, 2018). The present study was carried out to assess the economic productivity losses due to babesiosis in cattle in Chittoor district districts of Andhra Pradesh

### Materials and Methods

The study was conducted on the cattle presented to the various veterinary dispensaries in and around Chittoor district districts of Andhra Pradesh for a period of two years from January 2023 to December 2024. Cattle presented with pyrexia, anaemia, tick infestation, lymphadenopathy and haemoglobinuria were included in the present study. Selection of cattle were based on history of tick infestation, haemoglobinuria, pale mucus membranes, chronic emaciation and babesiosis was confirmed by stained blood smear examination and further confirmed by polymerase chain reaction (Sivajothi *et al.*, 2023). Cattle with babesiosis were treated with specific antiparasitic drugs (Singh *et al.*, 2021) along with supportive and symptomatic therapy. Assessment of economic losses due to babesiosis was carried out in 105 cattle with babesiosis by filling

questionnaire specially prepared for the study. The components under criteria were loss due to mortality, reproductive loss, production loss (Milk), treatment cost and daily wages losses.

### Results

Total number of 480 cattle were included based on the clinical signs suggestive of haemoprotozoans. Out of which, 105 cattle were confirmed for babesiosis based on the microscopic examination of stained blood smears and further confirmed by polymerase chain reaction assay, which targeted the 18s rRNA gene by creating an amplified product of 733 bp. Cattle which were positive only for babesiosis included in the present study and other haemoprotozoans and/or mixed parasitic infections were excluded from the study. The economic loss due to babesiosis in cattle was summarized in the Tables 1 and 2; Fig.1. During the study period of two years, 105 cattle with babesiosis were included and estimation of economic loss was done. Grossly economic loss was calculated into five major groups including loss due to mortality, reproductive loss, production loss (Milk), treatment cost and daily wages losses.

**A) Loss due to mortality:** The estimated loss due to average loss of adult cattle was ₹70,000 as per the value in the present geographical location. During the assessment period, 18 cattle died, causing an average loss of ₹12,60,000 (Range from 10,60,750 to 15,80,900).

**B) Reproductive loss:** The aggregative loss due to reproductive loss was ₹11,94,500 (range from 9,75,900 to 14,08,5800). Reproductive loss was again divided into loss due to abortions, increased calving interval

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and infertility issues in cattle with babesiosis. (B1)- Loss due to abortions: Abortions resulted in the loss of five male calves (each valued at ₹1,000) and 11 female calves (each worth ₹5,000) amounting to loss of ₹60,000 (Range from 55, 000 to 75,000). (B2) - Loss due to increased calving interval: 55 cows had increased calving interval. Delayed calving resulted in a loss of ₹ 10,39,500 (Range from 8,40,900 to 12,90,800). (6 months delay, 3liters/day loss for 180 days at ₹ 35/liter). (B3)- Loss due to infertility: 19 cattle had infertility and did not become pregnant even after fourth time of insemination. Infertility led to loss of ₹95,000 in (Range from 80,000 to 1,20,000) (₹5000/year per affected cow).

**C) Production loss (milk loss):** 71 cows had reduction in milk yield. The average reduction in milk yield per animal per day was 5.12 litre; the average milk loss period was 20 days; the average cost of one litre of milk was ₹35. The estimated economic loss due to average milk loss was ₹2,54,464 (Range from 2,35, 200 to 2, 89, 200).

**D) Treatment cost:** 105 cows were treated and the average expenditure for treatment of sick cattle (specific therapy ₹500; symptomatic therapy ₹600; supportive therapy ₹1200) was ₹2300. The estimated economic loss due to treatment cost was ₹2,41,500 (Range from 2,10,000to 2,78,000).

**E) Daily wages loss:** 105 farmers lost their working time to spend the for the treatment of cattle and the average loss was ₹2000; The estimated economic loss due to daily wages loss of ₹2,10,000 (Range from 1,56,700 to 2,59, 200).

**Total loss:** The cumulative financial impact due to babesiosis in cattle was ₹31,60,464 (Range from 26, 29,550 to 38, 93, 100) during the two years period of study in 105 cattle. An average loss due to individual cattle per year was ₹15,049.

In the present study average economic loss was estimated for 105 cattle with babesiosis during two years period and it was ₹31,60,464, out which the loss due to mortality was 12,60,000 (39.87%), productive loss was 11,94,500 (37.79%), production loss by milk was 2,54,464 (8.05%), treatment cost was 2,41,500 (7.64%), daily wages were 2,10,000 (6.65%). (Table-21, Figure-14, Plate 5). When comparing the different variables causing the economic loss highest percent was due to mortality loss (39.87%), reproductive loss (37.79%), production loss (8.05%), treatment cost (7.64%) and for daily wages loss (6.65%).

## DISCUSSION

According to Laha *et al.* (2012), babesiosis caused an average daily milk loss of 1.72 liters. However, over the course of 20 days, the average milk yield loss in this study was 5.12 liters per day. According to Banerjee *et al.* (2005), after 18 days of starting treatment, cows will continue to produce milk. According to a study, the estimated annual loss from tick-borne diseases (TBD) was 364 million USD, with the loss of milk production accounting for 6% of this total (Kiveria, 2006). Given the lack of reports of financial losses resulting from natural *Babesia* infection in Indian crossbred cows, this estimate will be useful to farmers.

A study on the financial impacts of nematode, trematode, and tick parasitism on beef cattle production was carried out by Strydom *et al.* (2023). They came to the conclusion that live weight, feed efficiency, calf yield, quality, and reproductive performance are all adversely affected by parasitic infections in beef cattle. In addition to being a primary reason for carcass condemnations, these infections may be a factor in the increase in greenhouse gas emissions. The quantity of meat available to satisfy the demands of an expanding population can be decreased by such production losses, which can be significant. A study on financial losses resulting from the foot and mouth disease outbreak in cattle in certain impacted areas of Bangladesh was carried out by Giasuddin *et al.* in 2021. They discovered that the largest percentage of losses occurred when the affected cattle died (63.47%), followed by veterinary expenses (10.71%), the weight loss of the fattening cattle (10.68%), the decrease in milk production (9.17%), and the loss of labor for caring for the affected cattle (5.98%).

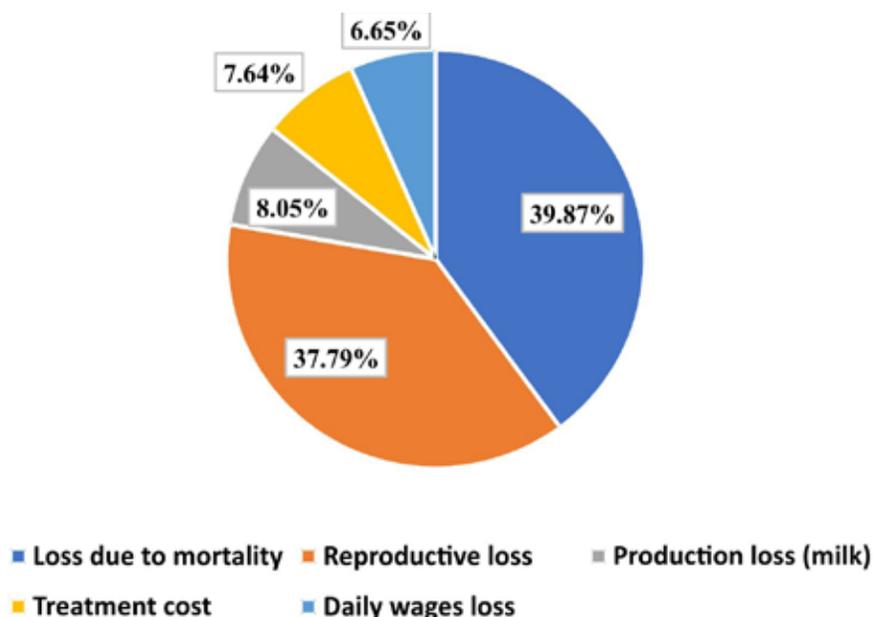
Over the course of the two-year study period, the total financial impact of babesiosis in these 105 cattle comes to ₹31,60,464. Over the course of the study, the average annual loss resulting from individual cattle was ₹15,049. The loss was ₹12,60,000 (39.87%) from mortality, ₹11,94,500 (37.79%) from productive loss (abortions, abnormal calving interval, infertility loss), ₹2,54,464 (8.05%) from milk production loss, ₹2,41,500 (7.64%) from treatment costs (specific, symptomatic and supportive therapy) and ₹2,10,000 (6.65%) from daily wages. During the study period, the average annual economic loss resulting from babesiosis in cattle was ₹15,049; this amount was made up of 39.87 percent from mortality loss, 37.79 percent from reproductive loss, 8.05 percent from milk loss and 7.64 percent from treatment cost.

**Table 1: Assessment of economic loss due to babesiosis in cattle (105)**

1.	Loss due to mortality	₹ 70,000 per cattle	70,000	18	12,60,000	10,60,750 to 15,80,900
2.	Reproductive loss				11,94,500	9,75,900 to 14,08,5800
	A) Abortions	Male calf- ₹ 1000 Female calf- ₹ 5000	Male calf- 1000 Female calf- 5000	16	60,000	55,000 to 75,000
	B) Calving interval	6 months increased, additional milk loss=3L×180D×35Rs	18900	55	10,39,500	8,40,900 to 12,90,800
	C) Infertility loss	calf a year (as per)	5000	19	95,000	80,000 to 12,0000
3.	Production loss (milk)	5.12ltr×20days×35Rs/L	3584	71	2,54,464	2,35,200 to 2,89,200
4.	Treatment cost	Specific therapy (₹ 500) Symptomatic therapy (₹ 600) Supportive therapy (₹ 1200)	2300	105	2,41,500	2,10,000 to 2,78,000
5.	Daily wages loss		2000	105	2,10,000	1,56,700 to 2,59,200
	<b>Total</b>				31,60,464	26,29,550 to 38,93,100

**Table 2: Percentage of different variables causing economic loss**

S. No	Parameters	Total loss (₹)	Total economic loss in percentage (%)
1.	Loss due to Mortality	12,60,000	39.87
2.	Reproductive loss	11,94,500	37.79
3.	Production loss (milk)	2,54,464	8.05
4.	Treatment cost	2,41,500	7.64
5.	Daily wages loss	2,10,000	6.65
		31,60,464	100.00



**Figure 1: Percentage of different variables causing economic loss.**

### Acknowledgements

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## Clinico Pathological and Ultrasonographic Evaluation of Dogs with Ascites

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### Abstract

Ascites is a common clinical manifestation of underlying hepatic pathology in dogs, often resulting from portal hypertension, cirrhosis, or hypoalbuminemia. The study was aimed to evaluate haematological, biochemical, electrolyte, ascitic fluid, and ultrasonographic alterations in 15 dogs with ascites. Haematologically mean values of Hb, PCV and TEC were significantly reduced while total leukocyte with neutrophil count were significantly increased. Biochemically, mean values of total protein and albumin were significantly reduced while mean values of ALT, AST, ALP, and BUN were significantly increased. On sonography increased parenchymal echogenicity, irregular liver margins, rounding of liver margins and diffuse nodular changes were noticed.

**Keywords:** Ascites, Portal hypertension, SAAG, Sonography

### Introduction

Ascites, defined as the abnormal accumulation of fluid within the peritoneal cavity. A wide variety of aetiologies can contribute to the development of ascites, including hepatic disorders, cardiac dysfunction, renal impairment, neoplastic processes, inflammatory and infectious conditions, or combinations. The present study was undertaken to characterize haemato-biochemical alterations, ascitic fluid biochemistry, and imaging features in dogs with ascites.

### Materials and Methods

The present study was conducted on 34 dogs presented to the Department of Veterinary Medicine, DGCN-COVAS, Palampur, Himachal Pradesh, exhibiting signs of abdominal distension. A detailed clinical history was recorded for each case, including age, breed, sex, and duration of illness. Peripheral blood samples were collected aseptically from the cephalic or saphenous veins. For haematological analysis, samples were drawn into sterile K<sub>3</sub>EDTA vials, while plain sterile vials were used for biochemical and electrolyte profiling. Abdominocentesis was performed under aseptic conditions at a site located approximately 2–3 cm caudal to the umbilicus and 1 cm lateral to the ventral midline

(either left or right side). Ascitic fluid was collected into sterile containers and subjected to biochemical analysis, including total protein and albumin concentration. Based on clinical findings, haematobiochemical parameters, effusion analysis, and imaging studies, 15 dogs with ascites were selected and included in this study.

### Results and Discussion

Out of the 15 dogs diagnosed with ascites, the majority (53.3%) were within the 1 to 3-year age group, followed by 4 to 8 years (33.3%) and less than 1 year of age (13.3%). Regarding sex wise distribution, 66.6% of the affected dogs were male (n = 10), while 33.3% (n = 5) were female indicating higher incidence in male dogs. Breed-wise, Golden Retrievers constituted the highest proportion (26.6%), followed by non-descript and Labrador Retrievers (20% each), German Shepherds (13.3%), and Shih Tzu, Pomeranian, and American Bully breeds (6.6% each). Clinical signs included inappetence (60%), lethargy (40%), and pale mucous membranes (40%). Less frequently, dogs presented with tarry faeces (melena) in 20% of cases, enlarged lymph nodes (13.3%), and vomiting or icteric mucous membranes, each in 6.6% of cases. These findings were consistent with that of James *et al.* (2009), and Sarvanan *et al.* (2014),

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**Table 1: Mean SE values of haematological parameters in hepatic origin ascitic dogs**

S.No.	Parameters	Healthy Control (n=10)	Diseased (n=15)
1.	Hb (g/dl)	12.87 ± 0.42	8.272*** ± 0.60
2.	PCV (%)	39.05 ± 0.96	22.272*** ± 1.54
3.	TEC (×10 <sup>12</sup> /L)	6.81 ± 0.25	4.116*** ± 0.33
4.	TLC (10 <sup>9</sup> /L)	10.69 ± 0.56	14.796* ± 1.32
5.	N (%)	79.68±0.85	82.436*** ± 1.21
6.	L (%)	17.08 ± 2.11	12.545 ± 1.26
7.	M (%)	3.94 ± 0.29	6.054* ±0.73
8.	E (%)	1.35±0.18	0.881 ± 0.73
9.	MCV (fl)	58.38 ± 0.87	55.354* ± 0.87
10.	MCH (pg)	19.02 ± 0.49	19.536 ± 0.47
11.	MCHC (g/dl)	31.63 ± 0.39	37.01 ± 4.15
12.	Platelets (10 <sup>9</sup> /L)	253.00 ± 20.57	181.363 ± 25.72

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

The mean values of Hb, PCV, MCV, and TEC were significantly reduced, while TLC and neutrophil counts were significantly increased (table. 1). These findings are in accordance with earlier studies by

Tantary *et al.* (2014), and Singh *et al.* (2019). Significant increase in TLC might be due to ongoing inflammation. Neutrophilic leucocytosis might be due to acute inflammatory conditions in hepatitis (Phom *et al.*, 2019).

**Table 2: Mean values of biochemical parameters in hepatic origin ascitic dogs**

S.No.	Parameters	Healthy	Diseased (n=15)
1	Total Protein (g/dl)	6.68 ± 0.12	4.78** ± 0.44
2	Albumin (g/dl)	3.71 ± 0.17	1.766*** ± 0.10
3	ALT (U/L)	36.12 ± 4.29	91.58* ± 16.38
4	AST (U/L)	31.27 ± 4.32	75.48** ± 10.29
5	ALP (U/L)	57.26 ± 8.23	161.45** ± 22.01
6	Total Bilirubin (mg/dl)	0.25 ± 0.04	0.707 ± 0.22
7	Glucose (mg/dl)	108.15 ± 2.82	105.89 ± 6.62
8	BUN (mg/dl)	14.47 ± 1.22	63.28* ± 17.01
9	Creatinine (mg/dl)	1.03 ± 0.15	1.39 ± 0.28

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

Mean values of ALT, AST, ALP, and BUN were significantly increased while total protein and albumin were significantly reduced compared to healthy group (table.2). These findings were in accordance with Pratibha *et al* (2022) and Patowary *et al.* (2024).

Tantary (2014) stated that elevations of plasma transaminases such as ALT and AST were indicative of altered hepatocellular membrane permeability, hepatocellular necrosis, and inflammation with degree proportional to number of injured hepatocytes. The

observed hypoproteinaemia and hypoalbuminemia may result from the liver's central role in synthesizing most plasma proteins. Liver dysfunction impairs both protein synthesis and degradation pathways. The decline in plasma oncotic pressure increases fluid accumulation and worsens ascites (Saravanan, 2014).

**Table 3: Mean values of serum electrolytes in hepatic origin ascitic dog**

S.No.	Parameters	Healthy	Diseased (n=15)
1	Sodium (mmol/L)	141.7 ± 1.45	139.86 ± 2.17
2	Potassium (mmol/L)	4.42 ± 0.21	4.529 ± 0.25
3	Chloride (mmol/L)	104.2 ± 1.18	106.03 ± 1.95
4	Calcium (mg/dl)	10.41 ± 0.89	10.398 ± 0.10

In the present study, serum electrolyte levels including sodium, potassium, chloride, and calcium did not show statistically significant alterations and remained similar to normal healthy group (table.3). Abdominal ultrasonography revealed the presence of anechoic ascitic fluid in all cases (100%). Three dogs (20%) presented with a small-sized liver, irregular margins, and marked hyper echogenicity, suggesting chronic liver pathology such as cirrhosis or advanced fibrosis. Rounding of the liver margins was observed in

nearly half (46.66%) of the cases in this study. Similar findings have been described by Elhiblu *et al.* (2015) and Tantary *et al.* (2014), linking margin irregularity and loss of lobulation with chronic parenchymal remodelling and fibrosis. Mean value of ascitic fluid total protein, and albumin stated in the above table 4 indicate that the fluid was transudative. The present study highlighted the diagnostic significance of integrated haematological, biochemical, ascitic fluid, and ultrasonographic assessments in dogs with ascites of hepatic origin.



(A)



(B)



(C)



(D)

**(A) rounding of liver margins with increased echogenicity; (B) irregular liver margins surrounded by anechoic ascitic fluid; (C) anechoic diffuse nodules in liver parenchyma; (D) large amount of free fluid containing echogenic debris and fibrin threads.**

**Table 4: Mean value of ascitic fluid total protein, albumin and SAAG**

Sample	Total protein (g/dl)	Albumin (g/dl)	SAAG
Ascitic Fluid	0.59 ± 0.14	0.16 ± 0.04	1.39 ± 0.42

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## Cutaneous Ulcerative Disease in a Star Tortoise

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### Abstract

A rescued Indian Star Tortoise (*Geochelone elegans*) was presented with shell deformities, limb weakness and ulcerative plastron lesions. Clinical evaluation, radiography and biochemical analysis were carried out. The plastron was abnormally soft and pliable, deviating markedly from the rigidity observed in healthy individuals. Deep ulcerative lesions were noted on the pectoral and abdominal scutes of the plastron. Hypocalcaemia with elevated phosphorus, parathyroid hormone and alkaline phosphatase levels was observed while reduced serum vitamin A and 1,25-dihydroxycholecalciferol concentrations. Microbiological culture of the ulcerative plastron lesions yielded *Escherichia coli* and *Staphylococcus* spp. Treatment included calcium and vitamin supplementation, fluid therapy and antimicrobial therapy, resulting in significant clinical improvement and shell remineralization within six weeks.

**Keywords:** Star tortoise, Cutaneous Ulcerative Disease

Reptilian species, particularly captive tortoises such as the Indian Star Tortoise (*Geochelone elegans*), are increasingly diagnosed with complex metabolic and infectious diseases resulting from improper husbandry and inadequate nutrition. This paper presents a case of a rescued *Geochelone elegans* (Indian Star Tortoise) exhibiting cutaneous ulcerative disease.

A male Indian Star Tortoise (*Geochelone elegans*) weighing approximately 800 grams was presented with a history of anorexia, stunted growth and restricted limb movement. Physical examination revealed multiple shell abnormalities, including irregular carapacial development with visible cracks (Fig. 1, 2) and inwardly collapsed scutes, most prominently in the caudal region (Fig. 3). The plastron was abnormally soft and pliable with deep ulcerative lesions on the pectoral and abdominal scutes of the plastron (Fig. 4). The plastrocarapacial bridge appeared vertically expanded. The pelvic limbs were splayed, with overgrown claws (Fig. 3). A cloacal prolapse was also evident upon examination (Fig. 5). The mucous membranes appeared dry and the tortoise exhibited clinical signs consistent with moderate to severe dehydration. Radiographic assessment revealed a generalized reduction in shell radiodensity, indicating decreased mineralization of both the carapace and plastron. Anaemia,

leukocytosis, hypocalcaemia, elevated phosphorus and alkaline phosphatase levels (Table 1) were noticed. Microbiological culture of the ulcerative plastron lesions yielded *Escherichia coli* and *Staphylococcus* spp., confirming secondary bacterial infection associated with necrotic shell regions. Serum vitamin A (retinol) and vitamin D<sub>3</sub> (1,25-dihydroxycholecalciferol) concentrations were reduced. The condition was diagnosed as Cutaneous Ulcerative Disease (SCUD).

Lactated Ringer's solution (@ 20 mL/kg subcutaneously bid for 3 days), calcium gluconate (100 mg/kg, intramuscularly, once daily 5days) were administered. Oral calcium carbonate supplementation (100 mg/kg, once daily), oral cholecalciferol drops (400 IU/kg, weekly), vitamin A supplementation (2000 IU/kg, orally, every other day for two weeks) were provided. Enrofloxacin (5 mg/kg, intramuscularly, once daily for 7 days) with topical wound care with diluted povidone iodine solution and mupirocin ointment was followed. After four weeks of treatment, the tortoise showed marked improvement in appetite, mobility and hydration status. Radiographic follow-up at six weeks revealed early signs of shell remineralization. Ulcerative lesions showed progressive epithelialization and serum calcium and phosphorus levels began to normalize, with a concurrent decline in PTH and alkaline phosphatase activity. The animal was deemed clinically stable and

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was transferred back to Forest Department for continued rehabilitation under improved husbandry conditions.

In reptiles, calcium and phosphorus homeostasis is critically dependent on adequate dietary ratios and vitamin D<sub>3</sub> metabolism. Vitamin D<sub>3</sub> synthesis through cutaneous photoconversion under UVB exposure is essential for calcium absorption and skeletal integrity (Mitchell and Perry, 2017). Inadequate exposure to natural or artificial UVB light, compounded by an imbalanced calcium and phosphorus intake, disrupts this homeostasis, resulting in chronic hypocalcaemia (Juan-Salles and Boyer, 2020). The resultant parathyroid hyperactivity promotes bone resorption and demineralization, producing classical manifestations such as shell softening, pyramidal scute formation and skeletal deformities (Music and Strunk, 2016). The observed decrease in serum vitamin A and 1,25-dihydroxycholecalciferol levels further evidence for the nutritional inadequacy responsible for this condition.

Consequently, ulcerative shell lesions frequently develop as secondary complications in animals with metabolic bone disease. Cutaneous Ulcerative Disease are largely avoidable through appropriate captive management. Ensuring optimal dietary calcium-phosphorus ratios, incorporating natural sunlight lighting, maintaining hygiene and routine veterinary monitoring are critical components in preventing recurrence.

#### Acknowledgement

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**Figure 1: Convex carapace with pyramidal growth**



**Figure 2: Cracked carapace (arrow)**



**Figure 3: Solid, cracked, inwardly collapsed scutes in caudal carapace with splay out legs**



**Figure 4: Deep ulcerative lesion on pectoral and abdominal scutes**



**Figure 5: Cloacal prolapse**

**Table 1: Blood profile in Indian Star Tortoise**

Parameters	Value
Haemoglobin	7.5 g/dl
PCV	23%
RBC	0.54 X 10 <sup>6</sup> /μL
Heterophils	39%
Eosinophils	6%
Lymphocyte	52%
Monocyte	3%
Glucose (mg/dL)	194
Total protein (g/dL)	4.93
Albumin (g/dL)	2.13
Globulin (g/dL)	2.80
BUN (mg/dL)	19.7
Creatinine (mg/dL)	1.1
Cholesterol (mg/dL)	115
Total bilirubin (mg/dL)	0.20
ALT (U/L)	6
AST (U/L)	23
ALP (U/L)	72
CK (U/L)	176
LDH (U/L)	356
Calcium (mg/dL)	7
Phosphorus (mg/dL)	11.8
Magnesium (mg/dL)	1.9
Vitamin A (Retinol) (mg/mL)	0.01
Vitamin D (nmol/L) (25 hydroxy vitamin D3)	12.9
T4 nmol/L	5.2 (0.46-3.15)

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## Arterial Blood Gas analysis in Labrador retriever puppies with parvoviral enteritis

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### Abstract

Labrador Retriever Puppies aged between 3 months to 5 months with parvoviral enteritis were selected and their Arterial Blood Gas (ABG) profile was analyzed. It revealed decreased pO<sub>2</sub> and elevated anion gap affected puppies. There was significant reduction in the mean concentrations for plasma total CO<sub>2</sub> (TCO<sub>2</sub>), partial pressure of carbon dioxide (pCO<sub>2</sub>) and HCO<sub>3</sub> values.

**Keywords:** Arterial Blood Gas (ABG) - Canine Parvovirus (CPV)

Canine parvo viral enteritis remains a common and important cause of morbidity and mortality in young dogs. The present study was designed to evaluate the nature of acid base disturbances in Labrador puppies with Canine Parvoviral (CPV) enteritis.

The study was undertaken with the clinical cases presented to the Small Animal Medicine Referral Clinics at Madras Veterinary College. From these cases, eight (n=8) puppies of Labrador Retriever aged between 3 to 5 months with the symptoms of parvoviral enteritis such as vomiting, bloody diarrhoea, fever, weakness, inappetence, lethargy etc., were randomly selected and included in the present study. Blood samples were collected from femoral artery in a heparinised syringe as per standard protocol for estimation of Arterial Blood Gas (ABG) and ABG Parameters were evaluated by using Blood gas analyzer (SIEMENS RAPIDLAB 348). Simultaneously, faecal samples were collected from the same puppies for detection of parvo viral antigen by polymerase chain reaction (PCR). For this, DNA was extracted from faecal samples by stool DNA extraction kit. The PCR was standardised for the primer set pCPV-

2ab and pCPV- 2b, as reported by Pereira *et al.* (2000) with slight modifications and samples were tested. Data were analyzed with the statistical program (SPSS, Chicago, Illinois, USA).

The Mean ± S.E. value for pO<sub>2</sub> (47.43±5.73mmHg) was very low (Table 1) when compared to the reference values (91.35-106.05mmHg) reported by Kules *et al.* (2015) and 106.2mmHg by Tamura *et al.* (2015). The observed values of Anion Gap was 17.0-26.4mmol/L, which was slightly higher when compared to the reference values (5.49-19.674mmol/L) reported by Kules *et al.* (2015). Dogs with CPV enteritis were observed to have a significant higher anion gap at admission, and a lower plasma concentration of Na<sup>+</sup>, K<sup>+</sup>, and Cl<sup>-</sup>. In a study by Neppart *et al.* (2002), blood gas analysis of puppies admitted with CPV enteritis were found to have a significant decrease in HCO<sub>3</sub><sup>-</sup> but a significant increase in blood pH. However such a change was not observed in this study. Rapid dehydration usually contributes to the metabolic acidosis in the pathophysiology of secretory diarrhea. Hence an early and effective fluid therapy with appropriate fluids will help in preventing these acid base and electrolyte alterations and prevent the occurrence of further complications.

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**Table 1: Mean ± S.E. values of Arterial Blood Gas (ABG)**

Parameter	Units	Observed Range	Mean±S.E.	Reference range*
pH	-	7.275-7.427	7.39±0.02	7.37-7.47
pCO <sub>2</sub>	mmHg	26.0-43.7	33.03±1.99	22.62-34.52
pO <sub>2</sub>	mmHg	19.9-74.5	47.43±5.73	91.35-106.05
Na <sup>+</sup>	mmol/L	138-152	145.00±1.54	125.10-146.10
K <sup>+</sup>	mmol/L	2.64-4.29	3.44±0.20	3.57-4.81
Cl <sup>-</sup>	mmol/L	101-112	106.25±1.59	96.60-115.60
HCl	%	25-46	36.00±2.41	-
HCO <sub>3</sub> act	mmol/L	16.7-21-21.3	19.46±0.63	14.06-22.10
HCO <sub>3</sub> std	mmol/L	18.4-22.6	20.29±0.54	-
BE (ecf)	mmol/L	(-7.6)-(-2.9)	-5.16±0.68	-12.05-1.61
BE (B)	mmol/L	(-6.7)-(-1.9)	-4.40±0.62	-9.7-0.46
Ct CO <sub>3</sub>	mmol/L	17.5-22.4	20.49±0.64	-
Anion Gap	mmol/L	17.0-26.4	22.48±1.08	5.49-19.67
O <sub>2</sub> SAT	%	65.7-95.6	81.49±4.03	-
O <sub>2</sub> CT	ml/dl	10.6-19.5	15.86±1.26	-
Ct Hb (est)	g/dl	8.7-15.8	12.26±0.82	8.46-16.61

\* Reference range Kules *et al.* (2015)

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## Salt Toxicity in a Buffalo Heifer

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### Abstract

Sodium chloride is an essential dietary component for cattle, but excessive intake or water restriction can cause salt poisoning and hypernatremia, leading to severe neurological dysfunction. This report describes a case of salt toxicosis in one year old buffalo heifer that developed acute neurological signs after ingesting 1.5 kg of common salt without access to drinking water. Diagnosis was based on history, clinical presentation, and elevated serum sodium and chloride levels. The heifer was successfully treated through gradual rehydration, isotonic fluid therapy, and supportive management.

**Keywords:** buffalo, salt toxicity

Salt (sodium chloride) plays a critical role in bovine nutrition by maintaining osmotic balance, blood viscosity, acid-base equilibrium, and gastric hydrochloric acid secretion (Sastry and Rama Rao, 2001). Cattle diets generally contain 0.5-1% salt. However, excessive intake, particularly under conditions of restricted water availability, can lead to salt toxicosis. Errors in feed formulation or inadequate mixing can also contribute to excessive dietary salt (Aiello, 2016). Although salt poisoning is well documented in pigs and poultry, it is less frequently reported in cattle and buffaloes (Radostits *et al.*, 2007; Smith, 2020). This article describes successful management of salt toxicosis in a buffalo heifer

A year old buffalo heifer from Daddiyan village, Amritsar of Punjab, India was presented with acute neurological signs, including lateral recumbency, profuse salivation and paddling of limbs (Fig. 1). According to the owner, the heifer had been drenched with 1.5 kg of common salt mixed in a small quantity of water in the morning. The animal remained confined in the shed without access to water until evening, when nervous signs became evident. This practice of salt drenching, locally believed to stimulate appetite and improve body condition, is common among farmers in Punjab. Clinical examination revealed dullness, depression, congested mucous membranes, severe dehydration, reduced reflexes, anuria, absence of defecation, intermittent opisthotonus and profuse salivation. Vital parameters recorded were rectal temperature 103.2 °F, heart rate 110/min, and respiration rate 44/min. The laboratory findings revealed elevated packed cell volume (52%),

serum sodium (168 mEq/L) and serum chloride (124 mEq/L). Based on history, clinical signs, and laboratory confirmation of hypernatremia, a diagnosis of salt toxicosis was established.



**Fig.4. Buffalo heifer in lateral recumbency exhibiting opisthotonus**

Intravenous isotonic fluids were administered along with dexamethasone sodium phosphate (5 ml), mannitol (100 ml) and vitamin B complex (5 ml), as described by Radostits *et al.* (2007) and Aiello (2016). The heifer was managed with frequent provision of small amounts of fresh, clean water at short intervals. Immediate administration of large volumes of water was avoided, as rapid correction of hypernatremia can cause cerebral oedema. Hence, a gradual hydration was practiced in the present case. The animal responded favourably and recovered uneventfully after five days of treatment and monitoring. Salt toxicosis in ruminants arises from excessive sodium chloride intake, water

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restriction, or a combination of both (Sharma *et al.*, 1993; Radostits *et al.*, 2007; Smith, 2020). While cattle may tolerate up to 900 g of salt per day if unlimited water is available (Smith *et al.*, 1972), water restriction precipitates intoxication. In the heifer, observed signs i.e. salivation, opisthotonus, and limb paddling were consistent with earlier report (Aiello, 2016).

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## Endoscopic retrieval of *Pentastomid* worm occlusion in a rescued Indian Rat Snake (*Ptyas mucosa*)

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### Abstract

A rat snake was presented with the history of rescue from a rural household. It was said to have some hit injury and was dull and reluctant to move. Bubbling of nasal discharge from the nostril, open mouth breathing and copious salivation were noticed. On auscultation deep breathing was observed; respiratory distress was noticed. The snake was medicated with ketamine hydrochloride and Diazepam and prepared for endoscopic assessment. A 2.8mm thin flexible endoscopy was used to assess the trachea, lungs and air sac area. In the middle of respiratory tract, occlusion with Pentastomid worm was noticed which was retrieved using grasping forceps. Dextrose Normal Saline was administered orally using feeding tubes and ivermectin was given subcutaneously. The snake recovered uneventfully after endoscopy and later rehabilitated and released in to a non-human habitat area.

**Keywords:** Indian rat snake, Flexible Endoscopy, Pentastome

Reptiles are susceptible to various infectious and parasitic diseases that can compromise their health in both wild and captive settings (Divers and Stahl, 2019). Pentastomiasis, caused by tongue worms (Pentastomida) is a significant parasitic disease in snakes, affecting their respiratory tract and occasionally other tissues. This article highlights the significance of endoscopic diagnosis of pentastomiasis in a rat snake.

A injured Indian Rat Snake (*Ptyas mucosa*) was presented by the Forest Department, Pattukottai Division to Veterinary College and Research Institute, Orathanadu, Thanjavur. It was dull and reluctant to move. Bubbling nasal discharge from the nostrils, open-mouth breathing, copious salivation and respiratory distress were observed. External injuries were noted in the proximal third region (Fig.1). Respiratory rate of 32 breaths per minute, heart rate of 84 beats per minute, and cloacal temperature of 34.2°C were recorded. Haematological and biochemical analyses revealed no

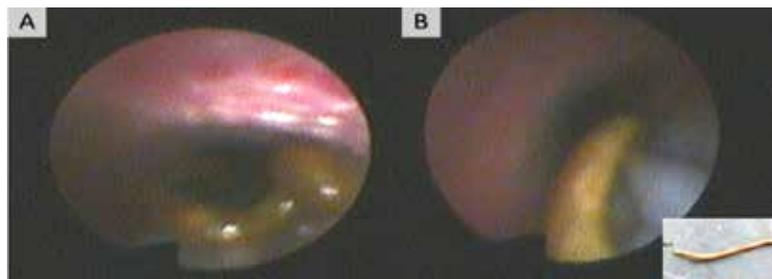
significant abnormalities (Table 1). Electrocardiographic assessment was performed and showed no abnormalities. Cloacal swabs were examined for parasitic ova, but no parasitic infestation was detected. Radiographic assessment showed no remarkable findings. The snake was manually restrained, and anaesthesia was induced using Ketamine (@ 20 mg/kg IM) and Diazepam (@ 1 mg/kg IM). A 2.8mm flexible endoscopy system (URF-P5 OES Flexible Uretero-Reno Fiberscope, Olympus) was gently introduced into the glottis and advanced under direct visual guidance.

The trachea, lungs, and air sacs were well visualized and appeared normal, except for a few hyperaemic spots. In the middle respiratory tract, a parasitic worm was visualized. Using grasping forceps, the parasite was successfully retrieved and identified as an adult pentastomid worm (*Porocephalus crotali*). Dextrose Normal Saline and B-complex syrup were administered using a feeding tube for three days. Subcutaneous administration of Ivermectin at 0.2 mg/kg was given. The snake showed progressive recovery and was released to a non-human habitat.

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**Table 1: Haematology and serum biochemical values of the rescued Indian rat snake**

Haematology parameters		Serum biochemical parameters	
Haematocrit	34 %	Total protein	6.24 g/dl
Haemoglobin	8.6 g/dl	Albumin	2.11 g/dl
Erythrocyte	$0.91 \times 10^6 / \mu\text{l}$	Glucose	84 mg/dl
Leukocyte	$9.2 \times 10^3 / \mu\text{l}$	AST	124 IU/L
Heterophils	56 %	ALT	40 IU/L
Lymphocyte	37%	ALP	110 IU/L
Monocyte	1%	CK	336 IU/L
Eosinophils	6%	BUN	7 mg/dl
Basophils	0	Creatinine	0.7 mg/dl

**Fig.1. The rescued snake (A) with the external injuries at proximal third region (B)****Fig.2. Endoscopic examination in the rescued Indian rat snake.****Fig.3. Endoscopic view performed in Indian rat snake****A: Hyperaemic spots with Pentastomid worm occlusion in the middle.****B: Endoscopic retrieval of Pentastomid worm occlusion in the rescued Indian rat**

Reptiles in the wild commonly harbour parasites, with pentastomes being a frequent finding (Rataj *et al.*, 2011). Although pentastome infections are often asymptomatic, heavy infestations can lead to clinical disease or even death. Diagnosis is typically achieved through the detection of characteristic calcified larvae on radiographs, post-mortem examination or repeated faecal and lung lavage tests (Drabick, 1987). Divers (2010) described an endoscopic approach to the lungs through the trachea. In this study, a 2.8 mm thin flexible endoscope with a working length of 70 cm provided significant clinical advantages by allowing near-complete visualization of the respiratory tract. This study highlights the effectiveness of non-surgical approaches, particularly flexible endoscopy, in diagnosing and extracting adult pentastomes from the respiratory tract of a snake.

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## Clinico pathological studies of Pericardial effusion in fourteen dogs

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### Abstract

Fourteen dogs presented to the Small Animal Outpatient Unit of Madras Veterinary College Teaching Hospital, Chennai, exhibiting clinical signs suggestive of pericardial effusion were subjected to detailed clinical, radiographic, electrocardiographic, ultrasonographic, and echocardiographic evaluations. Pericardiocentesis was performed for diagnostic and therapeutic purposes. Echocardiography proved to be a reliable diagnostic tool for confirmation and monitoring of pericardial effusion. Cytological evaluation aided in differentiation of idiopathic, neoplastic, and inflammatory causes.

**Keywords:** Pericardial effusion, Echocardiography, Pericardiocentesis, Cytology, Dog.

Pericardial effusion in dogs is a clinically significant condition characterized by abnormal accumulation of fluid within the pericardial sac, often leading to cardiac tamponade and impaired hemodynamics. The etiopathological diagnosis relies heavily on pericardial fluid analysis, which provides essential clues regarding underlying disease processes.

Dogs presented to the small animal outpatient unit of Madras Veterinary College Teaching Hospital, Chennai exhibiting dyspnoea, respiratory distress, weight loss, exercise intolerance and abdominal distension were taken up for the study. These dogs were subjected to detailed clinical, haematological and serum biochemical examinations, thoracic radiography, electrocardiography and echocardiography parameters were analysed. Of the fourteen dogs with pericardial effusion, seven dogs (50%) were diagnosed with idiopathic pericardial effusion, five dogs (36%) had neoplastic pericardial effusion, and two dogs (14%) were identified with inflammatory pericarditis. Among the five neoplastic cases, two were diagnosed as lymphoma, two as mesothelioma, and one as aortic body tumor. The common clinical signs observed were inappetence, lethargy, dyspnea, exercise intolerance, ascites, tachycardia, muffled heart sounds,

jugular distension, and pulsus paradoxus. In some cases, cough, pedal oedema, and syncope were noticed. Clinical manifestations noticed in affected dogs in the present agree with descriptions by Wann and Passen (2008) and Case *et al.* (2014), who stated that these signs primarily reflect right-sided cardiac tamponade due to impaired diastolic filling. Mild anemia was observed in some cases whereas all other hematobiochemical parameters were within the normal range.

Radiographic examination revealed a globoid cardiac silhouette (**Fig. 1**) and in some cases, pleural effusion was also noticed. Electrocardiography revealed reduced 'R' wave amplitude (**Fig. 2**) and electrical alternans. Abdominal ultrasonography showed ascites, hepatic venous congestion, and marked hepatomegaly without evidence of any specific hepatic pathology (**Fig. 3**). Radiographic findings in the present study, including a globoid cardiac silhouette and concurrent pleural effusion in some dogs, correspond to the observations of Ehrhart *et al.* (2002) and Vicari *et al.* (2001). The electrocardiographic features of low voltage QRS complexes and electrical alternans seen in the present investigation are also in agreement with Cobb *et al.* (1996) and Sidley *et al.* (2002), who reported these changes as typical of pericardial effusion and cardiac tamponade.

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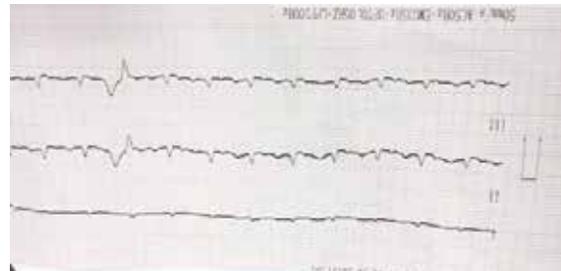
Echocardiographic examination confirmed the presence of pericardial effusion (**Fig. 4**). Echocardiographic M-mode parameters were within the normal range. Pulsed wave tissue doppler imaging at the mitral annulus showed mild to moderate diastolic impairment. (**Fig. 5**). The M-mode and two-dimensional echocardiographic findings of diastolic collapse of the right atrium and ventricle, along with echo-free space surrounding the heart, were consistent with previous reports by Jutkowitz (2008) and Olcott and Sleeper (2010).

In dogs with idiopathic pericardial effusion (n=7), cytological smears showed a mixed population of erythrocytes, macrophages, and reactive mesothelial cells with occasional small lymphocytes (**Fig. 7**). Macrophages frequently exhibited erythrophagocytosis, and mesothelial hyperplasia was evident in some samples, indicating chronic irritation of the pericardium. In cases with lymphoma (n=2), cytology was dominated by large atypical lymphoid cells exhibiting anisocytosis, coarse chromatin, and prominent nucleoli (**Fig. 8**). In mesothelioma (n=2), cytology revealed clusters

of reactive mesothelial cells arranged in papillary aggregates with distinct cell borders and basophilic cytoplasm, along with macrophages and lymphocytes. The inflammatory pericarditis cases (n=2) exhibited a predominance of macrophages and non-degenerate neutrophils, accompanied by a moderate number of reactive mesothelial cells and a few lymphocytes. Bacteriological examination of pericardial fluid samples collected from thirteen dogs revealed no evidence of bacterial organisms, either on direct staining or on culture using suitable media. Cytological examination revealed reactive mesothelial cells and macrophages with erythrophagocytosis in idiopathic effusion, large atypical lymphoid cells in lymphoma cases, and mesothelial clusters in mesothelioma, similar to the findings of Vasilatis and Vernau (2022) and Ojeda et al. (2015). The clinicopathological and imaging findings are consistent with those documented by Shaw and Rush (2007), Olcott and Sleeper (2010) and Vasilatis and Vernau (2022). Early echocardiographic diagnosis and etiological differentiation are essential for prognosis and management.



**Fig.1. Globoid enlarged heart**



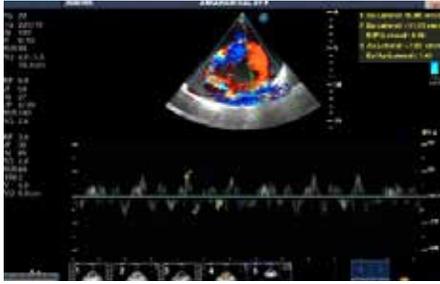
**Fig.2. Reduced 'R' amplitude and sinus tachycardia**



**Fig.3. Hepatic vein congestion and ascites**



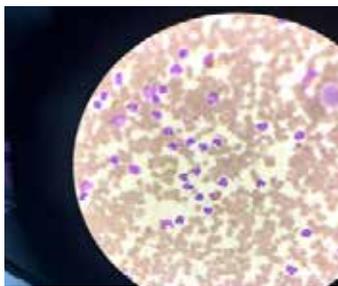
**Fig.4. 2D – Right parasternal long axis view- severe pericardial effusion with right atrial and mild degree of ventricular collapse**



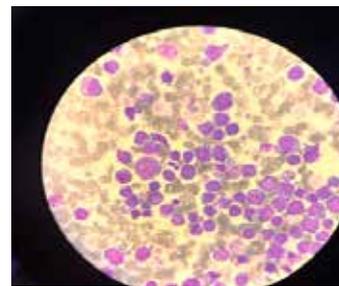
**Fig.5. PW - TDI left parasternal apical view at mitral annulus E/E' – 8.96 moderate diastolic dysfunction**



**Fig.6. Pericardiocentesis**



**Fig.7. Numerous RBC, neutrophils, haemosiderin crystals, few macrophages – Idiopathic pericardial effusion**



**Fig.8. Numerous lymphoblasts /reactive lymphocytes – suggestive of lymphoma**

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## Electrocardiographic changes in dogs with Mitral Valve Disease

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### Abstract

Mitral valvular disease is the most common acquired cardiac disease in dogs, which is characterised grossly by nodular distortion of the valve leaflets, as well as by thickening and lengthening of the chordae tendineae. The study was conducted in six dogs with mitral valve disease. Confirmation of mitral valve disease was carried out using echocardiography. Fine atrial fibrillation was the major electrocardiographic findings in dogs diagnosed with mitral valvular disease. Even though, no statistical significance could be established between control and diseased dogs, duration of P wave, QRS, PR, ST and QT interval were increased in diseased dogs.

Mitral valvular disease is the most common acquired cardiac disease in dogs, which is characterised grossly by nodular distortion of the valve leaflets, as well as by thickening and, sometimes, lengthening of the chordae tendineae. The present study reports the electrocardiography changes in six dogs with mitral valve disease.

Dogs brought to general health check-up and vaccination were selected as the control animals. Confirmation of mitral valve disease in six dogs was carried out using echocardiography. Standard single channel six-lead ECG was recorded using the BPL-CARDIART-6108T ECG machine. The analysis included measurement of P wave, QRS complexes, PR interval, ST segment, T wave QT interval in Lead II, and evaluation for the presence of arrhythmias. The data were analysed statistically using software package – IBM SPSS Statistics v24 .

Mean values of electrocardiography of control and diseased groups are presented in Table II. Fine atrial fibrillation was the major electrocardiographic findings in dogs diagnosed with mitral valvular disease (Fig1.). Apart from fine atrial fibrillation, intermittent notched P wave and T wave were observed in an eight year old Labrador. Intermittent atrial fibrillation, notched P and T wave and occasional ventricular premature complexes of left ventricle origin could be found out in a 6 year

old Golden retriever. Varying P wave morphology with notched P wave and ventricular premature complexes of left ventricle origin could be recorded in a five year old male Labrador presented with chronic hind limb oedema. Right bundle branch block could be recorded in five year old obese Dachshund presented with dyspnoea. Notched P wave could be detected in a nine-year-old mini-Pom presented with severe dyspnoea and syncope. Even though, no statistical significance could be established between control and diseased dogs, duration of P wave, QRS, PR, ST and QT interval were increased in diseased dogs. The corresponding values of MVD dogs were  $0.050 \pm 0.007$ ,  $0.039 \pm 0.004$ ,  $0.124 \pm 0.008$ ,  $0.099 \pm 0.009$  and  $0.160 \pm 0.019$  sec respectively. Other parameters of dogs with mitral valve disease were similar to or less than the control mean values.

Electrocardiography study in the animals of MVD revealed that all parameters were within the normal range and there was no statistical difference between control and mitral valve disease group. This was further supported by Sisson (1995) who opined that dogs with cardiomegaly and mitral regurgitation in radiography and echocardiography respectively usually showed normal electrocardiographic findings. The predominant morphological changes recorded in ECG were fine atrial fibrillation and notched P waves. This could be correlated with the atrial stretching due to haemodynamically significant volume overload in mitral valvular disease (Ettinger and Feldman, 2017).

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**Table 1: Mean values of ECG of apparently health dogs and dogs with MVD**

Variables	Control	MVD	t-value	P-value
P duration	0.041 ± 0.005	0.050 ± 0.007	1.085 <sup>NS</sup>	0.294
P height	0.460 ± 0.228	0.281 ± 0.033	0.692 <sup>NS</sup>	0.499
PR interval	0.110 ± 0.005	0.124 ± 0.008	1.429 <sup>NS</sup>	0.172
QRS duration	0.037 ± 0.003	0.039 ± 0.004	0.358 <sup>NS</sup>	0.725
QRS height	1.245 ± 0.287	1.550 ± 0.331	0.699 <sup>NS</sup>	0.495
Q dip	0.195 ± 0.072	0.100 ± 0.045	1.054 <sup>NS</sup>	0.307
ST interval	0.098 ± 0.008	0.099 ± 0.009	0.063 <sup>NS</sup>	0.950
QT interval	0.131 ± 0.008	0.160 ± 0.019	1.387 <sup>NS</sup>	0.197



**Fig.1. Lead II ECG tracing of a dog with mitral valve disease showing fine atrial fibrillation. The arrow indicates undulations of baseline(25mm/sec, 10mm/mV).**

### Acknowledgments

The authors would like to thank the authorities of Kerala Veterinary and Animal Sciences University for the facilities provided

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## Management of Acute-on-Chronic Kidney Injury in a Golden Retriever

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### Abstract

A four-year-old Golden Retriever bitch weighing 26 Kg was presented with the history of inappetence for one week. Clinical examination revealed emaciated, pyrexia, pale mucus membrane, hypertension (170 mmHg). splenomegaly and generalized lymphadenopathy. Laboratory evaluation showed hypochromic anemia with thrombocytopenia, hyposthenuria with proteinuria with elevated blood urea nitrogen and creatinine levels. Nephrosonogram revealed normal parenchyma with good corticomedullary differentiation, with irregular border and increased cortical echogenicity. Polymerase chain reaction (PCR) confirmed *Babesia gibsoni* infection.. Targeted antiprotozoal therapy comprised a 28-day course of triple antibiotic protocol with doxycycline, clindamycin, and metronidazole along with hematinic and thrombopoietic support. Therapeutic management included fluid therapy, antiemetics, proton pump inhibitors, antihypertensives, phosphate binders, renal-specific diet, and supportive care. Marked clinical improvement was observed during follow-up, with progressive normalization of renal parameters, restoration of appetite, and improvement in body condition score. .

**Keywords:** *Babesia gibsoni*, Dog, Kidney injury, Triple therapy

*Babesia gibsoni* is an intraerythrocytic protozoan parasite widely recognized as a causative agent of canine babesiosis, particularly in Asia, including India, where its endemic presence has been increasingly reported (Kumar *et al.*, 2023; Sivajothi and Reddy, 2018). Acute kidney injury (AKI) or progression to acute-on-chronic kidney injury (ACKI) has been observed in severe cases, necessitating timely diagnostic and therapeutic interventions (Preethi *et al.*, 2024). This case report presents a successful management of *Babesia gibsoni*-associated acute-on-chronic kidney injury in a Golden Retriever.

A four year old Golden Retriever bitch weighing 26 kg with a Body Condition Score of 3/9 was presented with inappetence for one week. Increased body temperature (103.5°F) with pale roseatte mucus membrane, hypertension (170 mmHg), generalized lymphadenopathy and splenomegaly were observed. Hypochromic anemia, thrombocytopenia, azotemia, isosthenuria, proteinuria, urine protein creatinine ratio (UPC) of 4.8, elevated BUN and creatinine values were also noticed (Table- 1). Screening for haemoparasites revealed *Babesia gibsoni* genome organism on nested PCR. Normal cortico-medullary differentiation, slightly

increased cortical echogenicity irregular renal borders, with the interlobar artery revealing Resistive Index (RI) of 0.60 and Pulsality index (PI) of 1.33 were observed under ultrasonography (Fig 2). Based on the clinical, laboratory and ultrasonographic findings the case was diagnosed as *Babesia gibsoni* induced acute on chronic renal failure.

Treatment was initiated with triple therapy with doxycycline (@ 10mg/Kg bwt IV), clindamycin (@ 11mg/Kg bwt IV) and metronidazole (@ 15 mg/Kg bwt IV). Ringers Lactate (@ 20 ml/Kg bwt twice daily IV), pantoprazole (@ 1mg/Kg bwt IV), ondansetron (@ 0.5mg/Kg IV), N- Acetyl Cysteine (@ 40 mg/Kg), darbepoietin (@ 0.8 mg/Kg subcutaneously) were administered. Benazapril (@ 0.5 mg/Kg bwt twice daily), renal diet, phosphate binder (@ 20 mg/Kg PO BID), syrups containing omega 3, 6 fatty acids, haematinic and thrombopoietic support were given to the dog. The hematology and serum biochemistry values were monitored periodically (Table 1). The dog had an uneventful recovery with reduction of proteinuria and blood pressure to normal limit and dog is now under management with renal diet and antihypertensive.

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**Fig.2a. Kidney with irregular border with normal cortico-medullary differentiation, Fig 2b Diameter of aorta at renal artery Fig 2c Doppler studies of the interlobar artery of left kidney measuring Resistive Index (RI) -0.60 and Pulsatility Index (PI) 1.33.**

**Table 1: Haematobiochemical parameters**

Parameter	Day 1	Day 3	Day 10	Day 17	Day 24	Reference Range
Haemoglobin (g/dL)	9.3	7.3	9.1	9.4	9.9	11.9- 18.9
PCV (%)	27.9	22.6	24	27.6	29.4	35 - 57
RBC (m/cmm)	5.06	3.9	4.54	4.97	5.2	4.95 – 7.87
WBC (/cmm)	8400	8400	10,300	11,400	12,800	5 – 10.1
Platelets (/cmm)	81,000	1,40,000	3,68,000	4,29,000	4,69,000	2,11,000 -6,21,000
Glucose (md/dL)	75	53.0	87	93	69	76 - 119
Total Protein (g/dL)	7.2	7.4	7.5	7.90	5.70	5.4 – 7.5
Albumin (g/dL)	1.8	1.3	1.5	2.40	1.90	2.3 – 3.1
Urea (mg/dL)	103.17	94.88	101.16	17.38	19.07	8 - 28
Creatinine (mg/dL)	9.03	8.27	4.68	1.44	0.80	0.5 – 1.6
ALT (U/L)	41	44.0	73	37	34	10-109
ALP (U/L)	615	892.0	782.0	110	193	1- 114
Sodium (mmol/L)	152	154.0	151	149	140.2	142 -152
Chloride (mmol/L)	111	115.4	113	111	109.7	110 -124
Potassium (mmol/L)	5.45	5.39	4.89	4.7	3.63	3.9 -5.1
Calcium (mg/dL)	9.22	9.59	13.38	8.96	10.68	9.1– 11.7
Phosphorous (mg/dL)	14.00	7.75	9.51	4.32	6.15	2.9 – 5.3

In the present case, the Golden Retriever exhibited hallmark clinical signs of babesiosis including inappetence, fever, pallor, lymphadenopathy, and splenomegaly, consistent with earlier reports (Barta *et al.*, 2021; Sivajothi and Reddy, 2018). In the present case, azotemia, proteinuria, and ultrasonographic alterations in renal architecture (increased cortical echogenicity and irregular renal margins) were suggestive of acute-on-chronic kidney injury (ACKI). This aligns with findings by Yamasaki *et al.* (2020), who reported

similar nephrosonographic changes in experimental *B. gibsoni* infections. Triple therapy with doxycycline, clindamycin, and metronidazole was employed as an alternative to the Atovaquone-azithromycin protocol. Although the latter is considered superior in efficacy, financial and availability constraints limit its routine use in India (Ravindran *et al.*, 2023; Shakya *et al.*, 2021). Fluid therapy, antiemetics, phosphate binders, antihypertensives, renal-specific diets, omega-3 fatty acid supplementation, and hematopoietic support facilitated

renal recovery and overall clinical improvement. Serial monitoring of hematological and biochemical parameters indicated progressive normalization of renal indices. This outcome reaffirms that even cases with significant azotemia and systemic involvement can demonstrate reversible renal dysfunction if managed promptly and comprehensively (Baneth *et al.*, 2020)

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## Clinical and molecular evaluation of *Proteus mirabilis* in a family pet dog with urolithiasis

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### Abstract

Urinary tract infections are a major concern in family pet dogs. Golden Retriever dog was found to have urinary incontinence, haematuria, stranguria and foul-smelling urine. Radiography and ultrasonography confirmed multiple nephroliths in the right kidney and urethral calculi measuring 5.28 mm. Elevated BUN, creatinine, phosphorus, anemia, haematuria, proteinuria, and struvite crystalluria were noticed. The isolation and characterization of *Proteus mirabilis*, using both conventional and molecular methods, including 16S rRNA gene sequencing, confirmed its association with urolithiasis. Urethral calculi were relieved by retrograde urohydropropulsion, and the dog was treated with Amoxicillin-Clavulanate along with dietary and supportive therapy. Post-treatment urinalysis by the fourth week showed no abnormalities, indicating clinical improvement.

**Keywords:** *Proteus mirabilis*, dog, struvite crystals.

Urinary tract infections (UTI), occur in approximately 14% of dogs in their lifetime (Decome *et al.*, 2020), particularly in individuals with anatomical or functional urinary tract abnormalities, urolithiasis or long-term indwelling urinary catheters (Warren *et al.*, 1982; Smarick *et al.*, 2004). The most common uropathogens isolated from dogs are *Escherichia coli*, *Staphylococcus* spp., *Enterococcus* spp., and *Proteus* spp (Wong *et al.*, 2015 ; Aurich *et al.*, 2022). The emergence of multidrug-resistant (MDR) *Proteus mirabilis* has been described in both human and veterinary medicine (Harada *et al.*, 2014). This study documents the *Proteus mirabilis*-associated urolithiasis in a pet dog.

A two-and-a-half-year-old Golden Retriever dog was presented with complaints of urinary incontinence, haematuria, stranguria, passing turbid and malodorous urine, and inappetence. The dog had a previous history of urethral and cystic calculi and an emergency cystotomy

to remove the calculi. The dog appeared dull, depressed, and lethargic, with a rectal temperature of 103°F, tachycardia, tachypnea, and a congested conjunctiva and mucous membrane, suggesting the clinical status of the infections. Similar findings were reported earlier by Subapriya *et al.* (2020) and Chauhan *et al.* (2025). The abdominal palpation revealed a distended bladder and evinced pain. Decreased levels of RBC, PCV, and Hb (Hemoglobin Level of 8.2 g/dL, PCV of 29%, RBC count of 3.8 x 10<sup>9</sup>/cmm) were noticed. Total leukocyte count of 27500/cmm, Neutrophil Count of 22000/μL, Lymphocyte Count of 4400/μL, monocyte count of 1100/μL, and platelet count of 98000/cmm were observed. The radiography showed multiple nephroliths in the right kidney with cystic calculi (Fig.1) while abdominal ultrasonography revealed urethral calculi measuring 5.28 mm (Fig.2a) and multiple nephroliths in the right kidney (Fig. 2b) with hydroureter.

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**Fig.1. Multiple nephroliths in the right kidney with Cystic calculi**



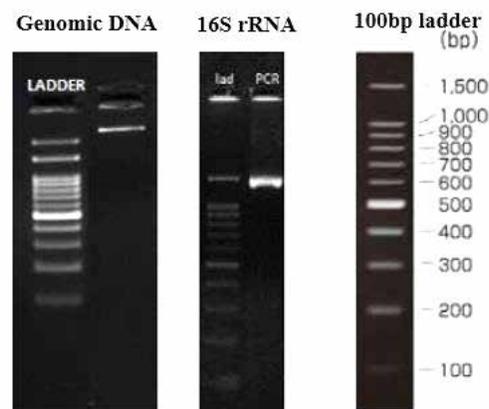
**Fig.2. Kidney -Urinary Bladder (KUB) Ultrasonography**

**Fig.2a. Urethral calculi measuring 5.28 mm**

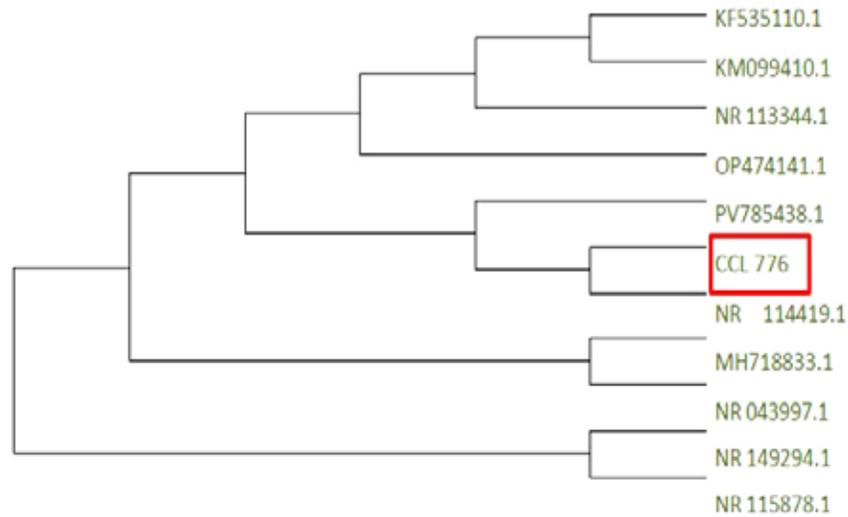
**Fig.2a. Right kidney with multiple nephroliths**

An increased leukocyte count and neutrophilia observed in this case, agreed with the findings of Devipriya *et al.* (2018) and Sonu *et al.* (2019). Elevated blood urea nitrogen (104 mg/dl), creatinine (3.56 mg/dl), ALT (141 IU/L), ALP (219 IU/L), Calcium (10.50 mg/dl) and Phosphorus (8.9 mg/dl) were observed. The urine sediment cultured on Brain Heart Infusion agar showed a thin, filmy, widespread growth of bacteria across the entire surface, rather than individual, isolated colonies. On characterization of the isolated bacteria, it was identified as Gram-negative, motile, catalase-positive, urease-positive, producing hydrogen sulphide,

and oxidase-negative. Based on the biochemical tests, it was tentatively identified as *Proteus* spp. A fragment of the 16S rRNA gene of the isolate with an amplicon size of 1500 bp was amplified by PCR (Fig. 3). On nucleotide BLAST analysis, the partial sequence of the 16S rRNA gene of the isolate showed 100% percent identity with 16S rRNA gene sequences of *Proteus mirabilis* strain ATCC 29906. The phylogenetic analysis revealed that the isolate was closely related to *Proteus mirabilis* and distantly related to other species in the genus *Proteus* (Fig. 4).



**Fig.3. Agarose gel electrophoresis showing amplified 16S rRNA gene of *Proteus* sp**



**Fig.4. Phylogenetic analysis of isolates based on the 16S rRNA gene sequences. The dendrogram was built from a 1500 bp-based alignment of nucleotide sequences by the Neighbor-joining method.**

Based on laboratory investigation and imaging studies, the present case was diagnosed as a complicated urinary tract infection due to *Proteus mirabilis* and its associated recurrent urolithiasis, nephroliths, and hydroureter syndrome. Similarly, complications of urolithiasis due to *Proteus mirabilis* have been reported by Singh *et al.* (2011) and Decome *et al.* (2020). Based on clinical findings, treatment was initiated with

Amoxicillin-clavulanic acid for 4-6 weeks. A similar approach was used by Weese *et al.* (2019). After the treatment, urine samples were collected for urinalysis and culture studies, which did not reveal any abnormalities over a period of one month. The owner was advised to provide special diets for better renal health and to review health every month.

**Table 1: Details of *Proteus sp* used in phylogenetic analysis**

Description	Query Cover	Per. Identity	Accession
<i>Proteus mirabilis</i> strain BAB-199	100%	99.80%	KF535110.1
<i>Proteus mirabilis</i> strain TCR46	100%	99.80%	MH718833.1
<i>Proteus mirabilis</i> strain PS01	100%	99.80%	OP474141.1
<i>Proteus mirabilis</i> strain MNNIT PN40	100%	99.80%	KM099410.1
<i>Proteus mirabilis</i> strain CX05	100%	99.80%	PV785438.1
<i>Proteus mirabilis</i> strain ATCC 29906	99%	100.00%	NR_114419.1
<i>Proteus mirabilis</i> strain NCTC 11938	99%	99.73%	NR_043997.1
<i>Proteus mirabilis</i> strain JCM 1669	98%	99.93%	NR_113344.1
<i>Proteus terrae subsp. cibarius</i> strain JS9	99%	99.12%	NR_149294.1
<i>Proteus vulgaris</i> strain ATCC 29905	99%	98.99%	NR_115878.1

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## Radiographic, Electrocardiographic and Echocardiographic Evaluation in dogs with ascites

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### Abstract

Dogs presented with the history of abdominal distension, anorexia, respiratory distress and pedal oedema were selected for study. Radiography, ultrasonography and echocardiography were used for evaluation of these dogs. Radiographic findings were characteristic ground glass appearance. Ultrasonographic findings were hepatomegaly, hyperechoic, hypoechoic and mixed echogenicity of liver parenchyma etc. Echocardiographic findings were dilated cardiomyopathy, congestive heart failure, pericardial effusions.

**Keywords:** Ascites, Radiography, Ultrasonography, Echocardiography.

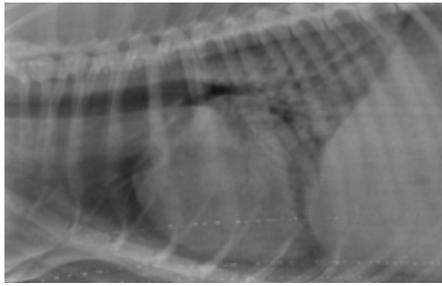
Ascites is defined as collection of serous or serosanguinous fluids in the peritoneal cavity (Ettinger and Feldman, 2005). It can be a secondary symptom of cardiac, hepatic, renal and various systemic diseases in dogs (Ihedioha *et al.*, 2013) and also secondary to hypoalbuminemia and portal hypertension (Center, 2015).

The dogs presented to the Veterinary Clinical Complex, College of Veterinary Science, Tirupati with the clinical signs of abdominal distension, dyspnoea, exercise intolerance and limb oedema were screened. Physical examination findings were pyrexia, pale conjunctival mucus membrane, fluid thrill up on abdominal palpation. Imaging techniques like radiography, ultrasonography and echocardiography were used for evaluation. A total of 2948 dogs were brought to veterinary clinical complex, College of Veterinary Science, Tirupati during the study period from May 2024 to November 2024. Among them ascites was diagnosed in 28 dogs. Out of 28 dogs, 15 dogs had ascites due to hepatic origin, 4 dogs had ascites due to cardiac origin, 5 dogs had ascites due to mixed origin, 3 dogs had ascites due to neoplastic origin and 1 dog had

ascites due to hypoproteinaemia.

Out of 23 dogs, in radiography ground glass appearance and loss of serosal details were observed in 21 dogs each (91.30%), floating intestines in 11 dogs (47.82%), gas filled intestines in 10 dogs (43.47%). Hepatomegaly with rounding of liver edge was observed in four dogs (17.39%), shrunken liver with irregular liver margins in two dogs (8.69%) and splenomegaly were recorded in three dogs (13.04%). The current findings align with the reports of Bhatti (2020). Radiographic examination of thorax revealed cardiomegaly and pericardial effusions in five dogs each (21.73%), globoid heart, elevated trachea and pleural effusions in four dogs each (17.39%) while consolidation of lungs and shrunken cranial lung lobe were observed in three dogs each (13.04%). Obscured cardiac silhouette, elevated carina, interstitial pattern of lung lobes were observed in two dogs each (8.69%) while mixed lung pattern and bronchial pattern was observed in one dog each (4.34%). Biatrial enlargement, right atrial enlargement was observed in three dogs each (13.04%) and left atrial enlargement was observed in two dogs (8.69%). These findings were consistent with the observations made by Kocaturk *et al.* (2016) and Bhatti (2020).

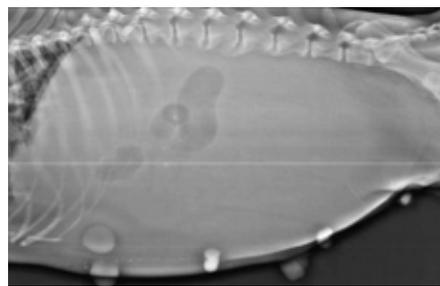
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**Left lateral thorax radiograph -enlargement of the heart (VHS- 11.5)**



**Left lateral thorax radiograph- pleural effusion and pericardial effusion**



**Lateral abdominal radiograph showing classical ground glass appearance-ascites**

The abdominal ultrasound examination in these demonstrated the presence of anechoic peritoneal fluid in all the 28 cases. Hepatomegaly was observed in five dogs (17.85 %), and the liver parenchyma was hyperechoic, hypoechoic and mixed echogenic in 11 dogs (39.28%), one dog (3.57%) and four dogs (14.28%), respectively. The liver margins were irregular in four dogs which corresponds to 14.28 per cent of the ascites affected dogs and round edges of the liver were recorded in five dogs (17.85%). Engorged and distended hepatic vasculature and space occupying lesion was present in

one dog each (3.57%). The results obtained align with the studies conducted by Gupta *et al.* (2020). Out of 23 dogs, mitral regurgitation, tricuspid regurgitation, left atrial enlargement, left ventricular enlargement was observed in six dogs, six dogs, seven dogs and two dogs, respectively. Similarly pericardial effusion was recorded in six cases (26.08%), pleural effusion, dilated cardio myopathy and congestive heart failure was recorded in two cases each (8.69%). The results obtained are in concurrence with the earlier research conducted by Bhatti (2020).



**Sonogram of abdomen depicting fibrin strands in the anechoic fluid**



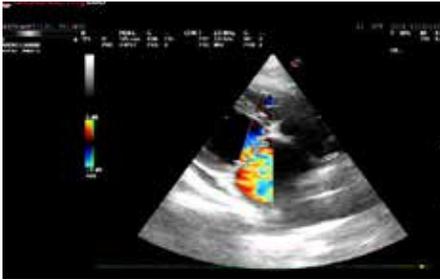
**Hepatosonogram depicting hepatomegaly and anechoic fluid in the abdominal cavity**



**Hyperechoic liver parenchyma with irregular margins in cirrhosis**



**Gall bladder with mucocele**



**Right parasternal long axis left ventricular outflow tract view**  
**Left: Mitral valve regurgitation in a dog with ascites Right: Pericardial effusions**



**Left atrial enlargement (La/Ao=2.88)**



**Dilated cardiomyopathy in dogs with ascites**  
**(Right parasternal short axis view)**

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## Successful Management of *Pemphigus vulgaris* in dogs - A Case Report

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### Abstract

Two dogs were presented to Small Animal Clinics Out-Patient Dermatology Unit at Madras Veterinary College Teaching Hospital (MVCTH) with oral ulcers and erosions on gums and palate. The oral lesions and erosions were positive for direct Nikolsky's sign and the samples from the lesions were negative for bacterial and fungal infections. Direct impression smears from the ulcers and erosions of the two animals were subjected to cytology which showed the presence of acantholytic cells surrounded by neutrophils. Based on the cytology evaluation and distribution of lesion it was confirmed as *Pemphigus vulgaris* (PV). Both the animals were treated with prednisolone at 0.5 mg/kg body weight for a month along with supplements viz omega fatty acids and vitamins. Improvement was observed in two cases but one dog showed recurrence of lesions after 2 weeks of discontinuation of prednisolone.

**Keywords:** *Pemphigus vulgaris*, Cytology, Prednisolone

### Introduction

*Pemphigus* group is one of the most common blistering autoimmune skin diseases of dogs. *Pemphigus* group is classified into *Pemphigus vulgaris* (deep vesicle) and *Pemphigus foliaceus* (Shallow vesicle) based on the depth of lesion that form within the skin (Rory, 2008). Molla *et al.* (2016) reported that *Pemphigus* was characterized by the production of autoantibodies that disrupted intercellular adhesion within the epidermis, pemphigus that led to acantholysis and blister formation. Successful management of pemphigus in dogs is presented in this article.

### Case History and Observations

Two dogs (a six-year-old non-descriptive dog and a five-year-old Spitz) were presented to the Small

Animal Clinics Out-Patient Dermatology Unit at Madras Veterinary College Teaching Hospital with a history of oral ulcerations vesicles and erosive lesions on the gums and palate. Gentle pressure applied with the thumb over the lesions resulted in bleeding from the erosions, indicating epidermal fragility and a positive direct Nikolsky's sign. Clinical signs such as anorexia, dysphagia and drooling of saliva were recorded. Both the dogs had vesicles, erosions and ulcers within the oral cavity and cytological findings revealed clusters of acantholytic cells surrounded by neutrophils. These cases were negative for pyoderma and dermatophytosis. Based on the nature, depth and distribution of the lesions (Fig.1 and 4) and cytological evaluation (Fig.5) the diagnosis was made as *Pemphigus vulgaris*.

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**Fig.1. Severe ulcerative lesion involving the upper lip and gingiva, with marked tissue erosion**



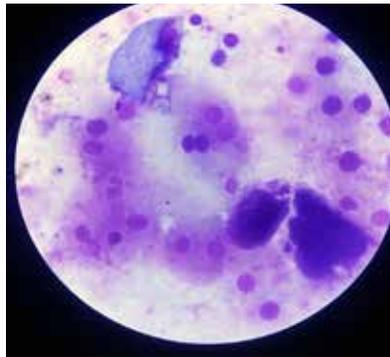
**Fig.2. After recovery**



**Fig.3. Erosion and ulcers over gums**



**Fig.4. After recovery but recurrence is noticed after treatment discontinued**



**Fig.5 Clusters of acantholytic cells**

### Treatment and Discussion

The animals were given prednisolone at a dosage of 0.5 mg/kg body weight orally for a month along with omega fatty acids and vitamin supplements. During the treatment period, the values of complete blood counts and serum biochemistry were normal. Clinical improvement was noted in two cases after a month (Fig.2 and 4) however, one dog experienced lesion recurrence following the discontinuation of prednisolone after two weeks, while the other remained relapse-free.

Olivry (2004) reported that Pemphigus vulgaris was a rare but clinically important autoimmune blistering disease in dogs, predominantly affecting mucosal surfaces. Foster and Foil (2003) mentioned that intraepidermal blistering, most commonly observed in the oral cavity and mucocutaneous junctions in this disease. The location of the ulcers in the present study also agreed with Foster and Foil (2003). Molla *et al.* (2016) stated that all Pemphigus variants share this immunopathogenic mechanism but they differ in antigen specificity and the level of epidermal cleavage: *Pemphigus foliaceus* affects the superficial epidermis,

while pemphigus vulgaris involves deeper layers. Our study also concurred with the findings of Molla *et al.* (2016). Further, identification of acantholytic keratinocytes, often surrounded by neutrophils by cytological evaluation confirmed that the cause is *Pemphigus vulgaris*. Gregoriou *et al.* (2015) found that corticosteroids remain the gold standard treatment for Pemphigus vulgaris. Hence the present study also used prednisolone as the first line treatment for PV. Almela and Chan (2021) mentioned that the prognosis for dogs with Pemphigus is fair. however, recurrence is common.

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## Cobra Envenomation in a Doberman Dog – A Case Report

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### Abstract

A two-year-old male Doberman dog was presented in a recumbent state with a history of snake bite and clinical manifestations of dullness, depression, respiratory distress and frothy salivation. Clinical examination revealed two fang marks on the lateral aspect of the left shoulder with oozing of blood. Haematological evaluation showed leukocytosis and reduced packed cell volume. Based on the clinical signs and history, the condition was diagnosed as cobra envenomation. The dog was treated with polyvalent anti-snake venom along with atropine sulphate, antibiotics, B-complex, fluid therapy and antiseptic wound dressing. Uneventful recovery was observed within three days.

**Keywords:** Cobra bite, *Naja naja*, Dog, Anti-snake venom, Neurotoxic envenomation.

### Introduction

Snakebite is a frequently encountered emergency in small animal practice in India, where dogs often come into close proximity with venomous snakes. Both cobra (neurotoxic) and viper (hemotoxic) envenomation are commonly reported, with multiple authors documenting year-round incidence in different regions (Vigneswari *et al.*, 2020; Vijayakumar *et al.*, 2019). Cobra (*Naja naja*) venom exerts potent postsynaptic neurotoxic effects that interfere with neuromuscular transmission, resulting in respiratory distress, muscle weakness, hypersalivation and rapid collapse (Sooryadas, 2012). Viper envenomation typically produces hemorrhage, coagulopathy, renal dysfunction and significant local tissue damage (Arun *et al.*, 2021; Saravanan *et al.*, 2017). The present case report describes the clinical presentation and successful management of cobra envenomation in a Doberman dog.

### Case History and Observation

A two-year-old male Doberman was presented in a recumbent state (Fig 1) to District Veterinary Hospital, Karimnagar with history of snake bite and sudden onset of dullness, depression, respiratory difficulty and frothy salivation (Fig 2). Physical examination revealed two distinct fang marks on the lateral aspect of the left shoulder with mild swelling and oozing of blood (Fig 3). Mucous membranes were congested, and temperature, respiration, pulse rates were recorded as 102.3 F, 18/min, 42/min respectively. Blood sample

was collected to evaluate the blood clotting time and haematology. The haematological parameters revealed decreased haemoglobin concentration (9.6 g/dl) and packed cell volume (27%) and increased total leukocyte count ( $31 \times 10^3/\mu\text{L}$ ) with normal blood clotting time of 12 minutes (20 WBCT method). Based on history of the owner, clinical signs and laboratory results, the case was diagnosed as cobra envenomation and therapeutic protocols were initiated immediately

### Treatment and Discussion

The dog was administered with polyvalent anti-snake venom (10ml) diluted in 100ml Normal Saline intravenously over a period of one and half hour. Atropine sulphate (@ 0.02mg/kg IM), ceftriaxone (@20mg/kg IM), tetanus toxoid (@ 0.5 ml IM), Vitamin B1, B6 and B12 (@ 2ml IV) were administered. The bite site was thoroughly cleaned with antiseptic solution (KMno4) and dressed with povidone iodine. Antibiotic and supportive therapy were continued for 5 days. Clinical improvement was noticed within 12–18 hours of treatment. Frothy salivation diminished and respiratory distress reduced significantly. By second day, the dog was alert and in sternal recumbency, and appetite gradually returned. By third day, the dog achieved complete recovery with stable vital signs, normal gait and resolution of neurological signs. Clinical signs such as salivation, dullness, muscular weakness with abnormal gait observed in the present case have also been observed by Ananda *et al.* (2009). These clinical signs can be attributed to the enzymatic and non-enzymatic compounds in the snake venom, according to Klaassen

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**Fig.1. Dog presented in recumbent state with dullness, depression.**



**Fig.2. Frothy salivation from mouth**



**Fig.3. Fang marks on the lateral aspect of the left shoulder.**

(2008). The present case exhibited typical neurotoxic signs of cobra envenomation, including respiratory distress, hypersalivation, recumbency and muscular weakness which are consistent with previous reports of cobra envenomation in Indian dogs (Sooryadas, 2012; Abinaya *et al.*, 2019; Dhillon *et al.*, 2020). The rapid and complete recovery observed in present case corresponds with the outcomes documented in both cobra and viper envenomation cases (Saravanan *et al.*, 2017; Vijayakumar *et al.*, 2019). Cobra envenomation in dogs is a life-threatening emergency requiring rapid diagnosis and early administration of polyvalent anti-snake venom. In the present case, timely intervention combined with comprehensive supportive therapy resulted in complete recovery within three days.

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## Urolithiasis in a Dog with Diabetes Mellitus - A Case Report

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### Abstract

A 7-year-old female Beagle, diagnosed with diabetes mellitus six months earlier and on insulin therapy, presented with haematuria, dysuria, and abdominal discomfort. Diagnostic workup included complete blood count, blood smear, mean blood glucose estimation, serum amylase and lipase, rapid Snap 4DX test, Babesia spp. antigen tests, radiography, and ultrasonography. All infectious disease tests (*E. canis*, *Anaplasma spp.*, *Dirofilaria immitis*, *Borrelia burgdorferi*, *Babesia canis*, *B. gibsoni*) were negative. Complete blood count showed a platelet count of 399,000/ $\mu$ L. Random blood glucose was 600 mg/dL, reduced to 224 mg/dL post-insulin. Imaging revealed a 3.52 cm hyperechoic, radio-opaque structure in the lower abdomen, consistent with cystic calculi, along with a thickened urinary bladder wall (0.61 cm), suggestive of cystitis likely associated with diabetes and uroliths. Surgery was performed, and a 24.42 g, yellowish, apricot seed-shaped calculus (3.1  $\times$  2.4  $\times$  1.3 cm) was removed.

**Keywords:** Cystitis, Diabetes mellitus, Dog, Hematuria, Urolithiasis

### Introduction

Diabetes mellitus is one of the most common endocrinopathies observed in middle-aged to older dogs, primarily characterized by hyperglycemia, glycosuria, and weight loss due to an absolute or relative deficiency of insulin (Audrey, 2012). Diabetic animals are more prone to bacterial and fungal infections and are predisposed to chronic or recurrent infections such as cystitis, prostatitis, bronchopneumonia and dermatitis. The present article describes cystic calculi in a dog with diabetes mellitus.

### Case History and Observations

A 7-year-old female Beagle was presented to the Veterinary Clinical Complex, Bihar Veterinary College, Patna, with a primary complaint of haematuria. The animal had been diagnosed with diabetes mellitus six months earlier and was receiving insulin therapy. The owner reported associated signs including polyuria, polydipsia, lethargy, reduced appetite, and bilateral cataract formation. On clinical examination revealed severe haematuria. A complete blood count (CBC)

performed using an automated hematology analyzer (Nihon Kohden) revealed hemoglobin 10.7 g/dL, RBC  $4.98 \times 10^6/\mu$ L, hematocrit 33.5%, MCV 67.3 fL, MCH 21.5 pg, MCHC 31.9 g/dL, WBC  $82 \times 10^3/\mu$ L (neutrophils 79.8%, lymphocytes 16%, monocytes 3.8%, eosinophils 0.3%), and platelet count  $3.99 \times 10^5/\mu$ L. Serum biochemical analysis using a dry chemistry analyzer (Fuji DRI-CHEM NX600V) indicated marked hyperglycemia (600 mg/dL), elevated blood urea nitrogen (48 mg/dL), serum creatinine (2.1 mg/dL), alanine aminotransferase (100 IU/L), amylase (623 IU/L), and lipase (392 IU/L). Rapid lateral flow assays (SNAP 4Dx Plus and Babesia Rapid Test Kits) and Giemsa-stained blood smear examination were negative for *Ehrlichia canis*, *Anaplasma spp.*, *Dirofilaria immitis*, *Borrelia burgdorferi*, *Babesia canis*, and *Babesia gibsoni*. Abdominal ultrasonography revealed a hyperechoic mass with acoustic shadowing within the urinary bladder. Radiographic examination confirmed the presence of radio-opaque calculi within the urinary bladder. Although the urolith was surgically removed, the dog, which was also diagnosed with diabetes mellitus, later developed severe epilepsy before death.

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**Fig. 1: Ultrasound examination of urinary bladder showed hyperchoic mass with acoustic shadowing**



**Fig 2: Surgical removal of cystolith of yellowish, apricot seed-shaped, measuring 3.1 x 2.4 x 1.3 cm.**

### Treatment and Discussion

Immediate intervention was initiated to manage the uncontrolled hyperglycemia, including administration of isophane insulin at a dose of 1 U/kg body weight twice daily via subcutaneous injection, along with a prescription of a commercial diabetic diet. Subsequent biochemical analysis revealed an improvement in blood glucose levels, reducing to 220 mg/dL. Surgical intervention was performed to remove the urolith, which was identified as a 24.42 g, yellowish, apricot seed-shaped calculus, measuring 3.1 x 2.4 x 1.3 cm (Fig 2). Post-operatively, the patient received antibiotics (amoxicillin clavunate @10mg/kg IV), fluid therapy (Ringers lactate@ 10mg/kg IV) and isophane insulin ( 0.5 U/kg body weight, subcutaneously twice daily). In this case, clinical signs of haematuria, dysuria, and abdominal discomfort were attributable to irritation of the bladder mucosa and partial obstruction of the urinary bladder by the calculus, as previously described by Fromsa and Saini (2019). The slight reduction in hemoglobin and erythrocyte count observed may have been due to blood loss associated with hematuria (Tion *et al.*, 2015). Biochemical alterations, including elevated blood urea nitrogen and creatinine, indicated compromised renal function, likely associated with post-renal azotemia caused by partial urinary tract obstruction (Vijayakumar *et al.*, 1999). The persistently high blood glucose concentration confirmed inadequate glycemic regulation. In this case, ultrasonography demonstrated a large hyperechoic structure within the urinary bladder,

consistent with reports by Fromsa and Saini (2019). The coexistence of diabetes mellitus and urolithiasis in this dog aligns with the findings of Mircean *et al.* (2006), who reported a predisposition to urinary tract disorders in diabetic dogs due to altered urine composition and increased susceptibility to infection. In conclusion, this case highlights the complex interplay between diabetes mellitus, urolithiasis, and cystitis in dogs, where persistent hyperglycemia predisposes to urinary tract complications and infection.

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## Therapeutic Management of Anaplasmosis in a Kid

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### Abstract

A non-descript male kid about 4 months old was presented with complaint of anorexia, lethargic and tick infestation. Clinical examination revealed elevated temperature (104.6°F), pale conjunctival mucous membrane, anaemia and enlarged lymph nodes. Peripheral blood smear showed the presence of Anaplasma organism in the RBCs. Whole blood was collected and subjected to PCR, in which sample shown positive for *Anaplasma ovis* infection. The kid was administered with oxytetracycline @ 20 mg/kg body weight I/M, Sharkoferrol vet (@ 5 gm daily) and B- complex injection (@ 1 mL I/M) at an interval of 48h. Significant clinical improvement was noticed after five days of treatment.

**Keywords:** Anaplasmosis, PCR, Oxytetracycline, Small ruminants

### Introduction

Anaplasmosis is an infectious, non-contagious disease caused by *Anaplasma ovis*, a rickettsial organism that exists as an obligate intra-erythrocytic pathogen (CabezasCruz *et al.*, 2019). The disease is usually mild and subclinical, but it may advance to an acute stage when aggravated by stress, severe mite infestation, inadequate nutrition, high environmental temperatures, or concurrent infections (Stuen *et al.*, 2003). This article reports anaplasmosis in a kid.

### Case History and Observation

A non-descript male kid about 4 months old was presented with complaint of anorexia, lethargy (Fig.1) and tick infestation (Fig.2). On clinical examination, pyrexia (104.6°F), pale conjunctival mucous membrane (Fig.3) and enlarged lymphnodes were noticed Peripheral blood smear stained with Giemsa stain, showed the presence of *Anaplasma* organism in the RBCs. For further confirmation whole blood was subjected to PCR as described by Torina *et al.* (2008)



Fig.1. Animal was dull and lethargy

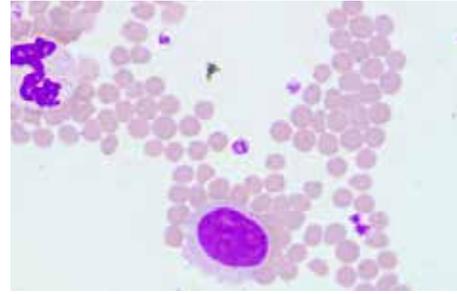


Fig.2. Severe Acariasis

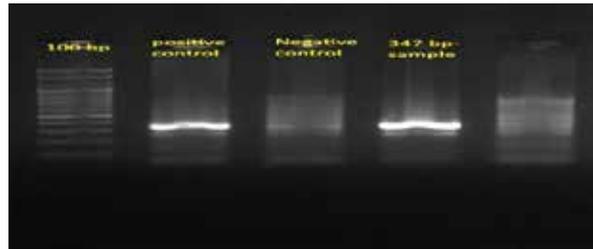
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**Fig.3. Pale conjunctival mucous membrane**



**Fig.4. RBCs infected with *Anaplasma* organism**



**Fig.5. PCR- amplicon size of *Anaplasma ovis* is 347bp**

### Treatment and Discussion

The kid was administered with oxytetracycline @ 20 mg/kg body weight I/M, Sharkoferrol vet (@ 5 gm daily) and B- complex injection (@ 1 mL I/M) at an interval of 48h. Significant clinical improvement was noticed after five days of treatment. *Anaplasma ovis* is

the principal species responsible for the disease in sheep and goats (Smith *et al.*, 2009). In this study microscopic examination suggested for *Anaplasma* spp and this was further confirmed by PCR. Smith *et al* (2009) reported that Imidcarb dipropionate might provide better treatment outcome in these cases.

**Table 1: Serum biochemistry of *Anaplasma* infected kid**

PARAMETERS	OBSERVED VALUE	NORMAL RANGE
Glucose (mg/dl)	68	45-75
Total protein (g/dl)	6.9	6.1-7.5
Albumin (g/dl)	3.3	2.3-3.6
Calcium (mg/dl)	9.8	9.0-11.7
Phosphorous (mg/dl)	4.7	3.7-9.7
ALT (U/L)	43	11-40
AST (U/L)	86	66-230
GGT (U/L)	14	20-50

**Table 2: Complete blood count of Anaplasma infected kid**

PARAMETERS	OBSERVED VALUE	NORMAL RANGE
Haemoglobin (g/dl)	6.2	8.0–12.0
Packed cell volume (%)	20	22-38
RBC (m/cmm)	3.8	8.0-18.0
WBC/ cmm	12,000	4.0-13.0
PLATELETS /cmm	4,00,000	3.0-6.0
Neutrophils (%)	40	30-48
Lymphocytes (%)	57	50-70
Monocytes (%)	2	1-4
Eosinophils (%)	1	3-8

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## Tetanus in a calf - A case report

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### Abstract

A one-day old female Crossbred Jersey (CBJ) calf was presented to the Referral Veterinary Polyclinic (RVP), ICAR-IVRI with the complaint of dullness, inappetence and dribbling of urine from the umbilical cord since birth. Physical examination revealed high temperature (104.5°F), difficulty in drinking milk, erect ears, muscle rigidity, hindlimb stiffness and difficulty in walking. Based on these classical findings, the present case was diagnosed as Tetanus. The calf was treated with Anti Tetanus Serum (ATS), procaine penicillin, and fluid therapy for 5 days. The animal showed uneventful recovery following therapeutic management.

**Keywords:** Tetanus, *Clostridium tetani*, Calf, Anti tetanus serum (ATS)

### Introduction

Tetanus is a non-contagious bacterial disease caused by *Clostridium tetani*, Gram positive bacteria, spore forming obligate anaerobic bacteria. Among these, umbilical infection is the primary cause for tetanus in young animals like calves, sheep and foals. (Das et al., 2011; Popoff, 2020 and Saravanan et al., 2021). Neonatal tetanus can occur with contamination of umbilical cord due to certain insanitary conditions such as parturition (Constable et al, 2017). The present case describes about tetanus in a calf.

### Case History and Observation

Day old female CBJ calf weighing around 28 kg was presented to the Referral Veterinary Polyclinic (RVP), ICAR-Indian Veterinary Research Institute with the history of inappetence, dribbling of urine from the umbilical cord. High temperature (104.5°F), difficulty in drinking milk, muscle rigidity, stiffness in the hindlimb (Fig 1), absence of stifle joint flexion and erect ears carrying backwards (Fig 2) with no abnormality in the forelimb were noticed. The calf showed difficulty in walking and jumping gait was observed while walking. Blood sample was taken for haematological examination. Fecal sample was taken for parasitic endoparasites. Faecal sample was negative for endoparasites. Haematological analysis was within the normal range.



**Fig.1. Calf in lateral recumbency with hindlimb stiffness and erect ears**



**Fig.2. Calf shows erect ears carrying backwards**

### Treatment and Discussion

Based on these findings, the calf was diagnosed with Neonatal Tetanus. The calf was treated with Fortified procaine penicillin (@ 6000 IU/kg IM), tetanus toxin, anti-tetanus serum (@ 1500 IU was given subcutaneously :3 doses), Normal saline (@ 10 ml/kg IV), diazepam (@ 0.5 mg/kg IV) and vitamin B1 B6 B12 (@ 2ml IV) was given for the period of 7 days. By

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the 3<sup>rd</sup> day of treatment, the calf showed improvement in the milk intake and significant reduction in the stiffness of hindlimb was observed. Complete recovery was noticed on the 7<sup>th</sup> day of treatment.



**Fig.3. After treatment**

Anti Tetanus Serum @ 1500 IU SC SID (2 doses) was given for the effective neutralization of toxin as given by the previous reports (Bhikane *et al.*, 2005; Das *et al.*, 2011; Khan *et al.*, 2016). The significance of Anti tetanus serum (ATS) is to neutralize the unbound toxin in the body of the animal outside the central nervous system. Normal saline was given to restore the hydration status. Administration of Anti Tetanus serum (ATS) in the earlier stage of Tetanus infection is highly effective in successful improvement of the condition.

#### **Acknowledgement**

The authors are thankful to the Director, ICAR-Indian Veterinary Research Institute, Izatnagar for providing necessary facilities for this research work.

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## Successful management of Babesiosis in a Dog – A Case Report

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### Abstract

A three-year-old intact male Labrador Retriever was presented to the Small Animal Clinic, Veterinary College Hospital, Bengaluru, with a history of pyrexia, anorexia, lethargy, and ascites. Clinical examination revealed pale mucous membranes, fever, generalized lymphadenopathy, and mild halitosis. Haematobiochemical analysis revealed anaemia, thrombocytopenia, neutrophilic leucocytosis, lymphocytopenia, hypoproteinaemia and elevated creatinine and ALT values. Peripheral blood smear examination revealed piroplasm-like structures, later confirmed as *Babesia gibsoni* by polymerase chain reaction (PCR). Abdominal ultrasonography demonstrated moderate hepatomegaly, ascites, and splenomegaly. The dog was successfully treated with a triple therapy regimen consisting of doxycycline, clindamycin, and metronidazole. Supportive care included protein supplements, antacids, haematinics, hepatoprotectants, antioxidants, and B-complex vitamins.

**Keywords:** Babesiosis, Haemolytic Anaemia, labrador

### Introduction

Canine babesiosis is a significant tick-borne haemoprotozoan disease commonly seen in tropical and subtropical regions due to high tick density. It is caused by intraerythrocytic protozoa of the genus *Babesia*, most notably *Babesia gibsoni* and *Babesia canis*. Severe infections may result in disseminated intravascular coagulation (DIC), renal dysfunction, and shock (Schoeman *et al.*, 2007; Zygnier *et al.*, 2015). A case of babesiosis in a dog and its successful management is placed on record.

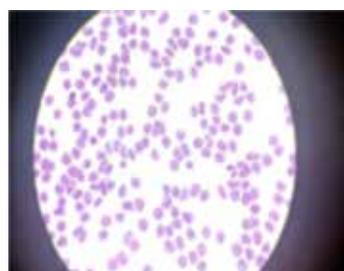
### Case History and Observations

A three-year-old intact male Labrador Retriever was presented to the Small Animal Clinic, Veterinary College Hospital, Bengaluru, with a history of anorexia, fever, lethargy, abdominal distension, and

decreased urination. Clinical evaluation revealed pale mucous membranes (Fig 1.1), pyrexia, generalized lymphadenopathy, halitosis, fine crackles on thoracic auscultation, and tachycardia. Haematobiochemical findings indicated moderate anaemia, thrombocytopenia, neutrophilic leukocytosis, lymphocytopenia, and hypoproteinaemia (Table 1.1). Peripheral blood smear revealed piroplasm-like organisms (Fig1.2) and spherocytes (Fig 1.3) confirmed as *Babesia gibsoni* by PCR. A positive saline agglutination test corroborated by a Coombs test, indicated immune-mediated haemolysis. Coagulation testing revealed prolonged PT (23.6s) and aPTT (36s). The electrocardiogram revealed a tall and narrow QRS complex with ST slurring and absence of consistent P waves (Fig 1.4). Abdominal ultrasonography showed moderate hepatomegaly and splenomegaly (Fig 1.5).

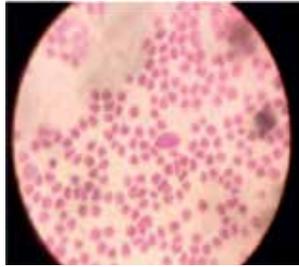


**Fig. 1.1: Pale conjunctival mucous membrane**

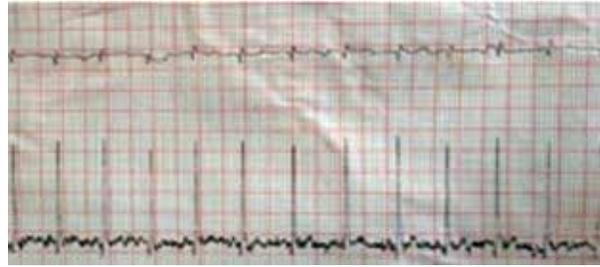


**Fig 1.2: Piroplasm of *Babesia gibsoni***

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**Fig 1.3: Spherocytes indicating oxidative stress**



**Fig 1.4: Atrial Fibrillation**

**Table 1.1: Haemato-biochemical parameters of the patient before and after therapy**

Parameter	Day 0	Day 7	Day 21	Reference range
RBC count (millions per mm <sup>3</sup> )	2.84	3.67	4.84	5-7.9
Haemoglobin (g/dl)	7.2	8.4	10.84	12-19
WBC count (cells/mm <sup>3</sup> )	23600	14500	13200	5000-14100
Platelet count (lakhs/mm <sup>3</sup> )	1,21,000	2,45,000	4,46,000	2,10,000-6,20,000
Neutrophils (%)	85	60	71	58-85
Lymphocytes (%)	18	22	25	32-36.3
Monocytes (%)	2	3	4	0-5
Total protein (g/dl)	4.1	4.9	6.1	5.4-7.5
Albumin (g/dl)	2.2	2.2	2.8	2.3-3.1
Globulin (g/dl)	2.0	2.7	3.3	2.4-4.4
BUN (mg/dl)	56	48	18	8-28
Creatinine (mg/dl)	3.87	2.65	1.47	0.5-1.7
ALT (IU/L)	187	102	89	10-109
ALP (IU/L)	212	158	89	15-156
TBIL (mg/dL)	1.1	0.89	0.6	0 – 0.3

#Reference range: Kahn, C.M. (2010). The Merck Veterinary Manual, (10th edn.). Merck & Co., USA

### Treatment and Discussion

Doxycycline (5mg/kg BID IV), Clindamycin (20 mg/kg BID IV) & Metronidazole (15 mg/kg bid IV) were used. Haematinics, oral prednisolone (2mg/kg/day P.O.) oral antacid (Gelusil 5mL P.O. bid), Pantoprazole (1mg/kg IV OD), acetyl cysteine (50mg/kg IV) and crystalline intravenous amino acids were used (Hermin @ 1mL/kg IV) were administered. Fluid therapy with crystalloid LRS, DNS, with furosemide (1 mg/kg IV) was also administered. The aforementioned intravenous triple-drug therapy was administered continuously for three days. On the fourth day, the dog exhibited

notable improvement in appetite, and oral hydration was initiated. The triple-drug therapy was continued orally for 21 days, along with haematinics and other supportive supplements. Hematobiochemical evaluations were performed on days 0, 7, 14, and 21 to monitor clinical improvement. According to Glaharn *et al.* (2018), complications associated with babesiosis—including DIC, acute kidney injury, hepatic dysfunction, cardiac anomalies, cerebral involvement, and acute respiratory distress syndrome—arise from both parasitic and host immune responses. Zygnier *et al.* (2023) reported that

anaemia in canine babesiosis is largely driven by host immune responses rather than direct parasitic damage.

### Acknowledgement

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## Cystoisosporosis in a puppy

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### Abstract

A two-month-old male German Shepherd pup was presented to the small animal outpatient unit, Teaching Veterinary Clinical Complex, Pookode, Wayanad with a history of malodorous dark tarry diarrhoea, intermittent vomiting and weakness for five days. The condition was diagnosed by microscopic examination of the faecal sample as cystoisosporosis. The treatment was initiated with intramuscular injection of sulphadiazine trimethoprim @ 20mg/Kg body weight for three days followed by sulphamethoxazole trimethoprim tablets for the subsequent four days. The animal made an uneventful recovery.

**Keywords:** *Cystoisosporosis* Sulphadiazine-Trimethoprim

### Introduction

Cystoisosporosis (previously known as isosporosis) is noticed in puppies aged few weeks to a few months old, and might be associated with stress, transport or change in diet or immunosuppression. Dauschies *et al.* (2000) reported that depending upon the age of the animal, immune status and the parasitic burden, severe dehydration and death can occur. Successful management of cystoisosporosis in a dog is reported in this article.

### Case History and Observation

A two-month-old male German Shepherd pup weighing about 3.4Kg was presented with a complaint of malodorous dark tarry diarrhoea, intermittent vomiting and weakness (Fig.1). Owner reported a decreased feed and water intake. The pup was vaccinated against Canine distemper and Parvovirus. The animal was dewormed 1 month back with an oral suspension containing Pyrantel embonate and Febantel.



**Fig.1. Dull and depressed animal in lateral recumbency**



**Fig.2. Photograph of *cystoisospora* spp. oocyst detected from a puppy (a, b) immature oocyst containing two sporoblast. (a) – Length of oocyst (b) - Width of oocysts**

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The animal was emaciated, dull and depressed on general examination. On clinical examination, the animal showed a body temperature of 101.3°F. The conjunctival mucous membranes were pale roseate in colour. Rapid thready pulse (168/min) and tachycardia (168 beats/minute) were also observed. Distended abdomen, serous nasal discharge and malodorous dark tarry faeces were observed. Complete Blood Count revealed mild leucocytosis ( $19.23 \times 10^3/\mu\text{L}$ ), neutrophilia ( $14.72 \times 10^3/\mu\text{L}$ ) and anaemia ( $2.52 \times 10^6 \text{RBC}/\mu\text{L}$ ) with a haemoglobin concentration of 8g/dL. Faecal sample examination revealed the presence of sporulated oocyst of *Cystispora* spp. (++++) (Fig.2). The microscopic image of the sporulated oocyst was acquired and analysed by LAS 4.10 Software (Leica Microsystem, Germany). The oocyst was oval without micropyle and measured 18.902µm to 22.551µm.

#### Treatment and Discussion

The treatment was initiated with sulphadiazine trimethoprim (@ 20 mg/Kg body weight twice intramuscularly). Supportive treatments include fluid therapy with Dextrose Normal Saline (@ 10mL/kg body weight) and B-complex vitamins (0.5ml intramuscularly) for three days. The animal was advised with Sulphamethoxazole Trimethoprim (PO @ 20mg/Kg), B-complex vitamins and Multivitamins orally for

four days. The case was reviewed after seven days and no parasitic ova could be detected from the faecal sample. There was complete recovery from dark tarry diarrhoea and the animal showed normal defecation and food intake. The pup became active and healthy. Daughies *et al.* (2000) and Lappin (2010) reported that diarrhoea among the puppies is a major health hazard encountered which can affect the normal development of the animal along with immunologically compromising it for various diseases and vaccination failure. [Garanayak](#) *et al.* (2017) reported that trimethoprim and sulphamethoxazole @ 40 mg/kg body weight in combination with metronidazole @ 10 mg/kg body weight twice daily for 5 days provided effective management for isospora infection in dogs and cats.

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## Successful Management of Oesophageal Obstruction in a Jersey Crossbred Cow

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### Abstract

A Jersey crossbred non-pregnant cow was presented with a history of anorexia and dysphagia. The cow was unable to swallow anything. Physical examination and oesophageal palpation revealed an enlargement in the left lateral neck region. An attempt to pass a stomach tube failed, indicating oesophageal obstruction. After sedation and manual intervention, a foreign body composed of groundnut cake and feed material was retrieved. The condition of the cow improved following the removal of the obstruction.

**Keywords:** oesophageal obstruction, foreign body, groundnut cake

### Introduction

Oesophageal foreign bodies in cattle are uncommon but potentially life-threatening if unattended. Ingested foreign bodies can result in dysphagia, anorexia, bloat which in turn leads to difficulty in respiratory and dehydration. Cattle are prone to swallowing foreign materials owing to their indiscriminate feeding habits, making esophageal obstruction an emergency condition in large animal practice (Constable *et al.*, 2017). Oesophageal obstruction is considered one of the more serious conditions due to the risk of dehydration, electrolyte imbalances, and bloat (Singh and Tyagi, 2012). Cases of esophageal obstruction in livestock have been documented in various species (Mohanambal *et al.*, 2018; Ravi *et al.*, 2018; Ammu *et al.*, 2019). Early diagnosis and timely intervention are crucial to prevent further complications, such as aspiration pneumonia and oesophageal rupture (Wilson and Mallinson, 2017). This case report discusses the successful removal of a feed-related foreign body from the esophagus of a Jersey crossbred cow and stresses the importance of proper feed management to prevent future occurrences.

### Case History and Observation

A Jersey crossbred non-pregnant cow was presented to the large animal medicine unit of veterinary clinical complex, Namakkal with a history of not taking feed and water for a period of 24 hours. The owner reported that the cow had been unable to swallow feed and water since the previous day. Attempts to offer feed and water were unsuccessful, as the cow appeared

to struggle with swallowing and showed signs of discomfort while drinking water. There was no prior history of trauma. Profuse salivation was present and the cow exhibited repeated attempts to swallow, which were unsuccessful. Physical palpation of the neck revealed enlargement on the left lateral aspect of the cervical esophagus (Fig 1). Attempts to pass a stomach tube were unsuccessful, as it could not advance beyond the mid-esophagus, suggesting an obstruction. Rumen showed mild distention and no other abnormalities were detected. Rectal temperature, pulse and respiratory rates were within normal limits.

### Treatment and Discussion

The cow was diagnosed to have oesophageal obstruction (choke) due to a foreign body, based on the history of inappetence, dysphagia and inability to pass a stomach tube. Cow was sedated using xylazine @ 0.1 mg/kg body weight intramuscularly. A Gunther's mouth gag was used. Hand was inserted into the oral cavity while external pressure was applied on the oesophagus, pushing the obstruction retrograde from the neck to the mouth. After several attempts, the foreign body, consisting of groundnut cake mixed with feed material, was successfully removed (Fig. 2). The cow was administered intravenous fluid (inj. RL @ 10 ml/kg, IV) was provided to rehydrate the cow and correct any electrolyte imbalances. The cow showed marked improvement and owner was advised to be fed only soft, easily digestible feed for the next few days to prevent further oesophageal irritation.

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**Fig. 1. Swelling on the left lateral aspect of the cervical esophagus**



**Fig. 2. Obstructed foreign body - groundnut cake**

Oesophageal obstruction in cattle, especially by foreign bodies, can present with clinical signs such as dysphagia, salivation, and anorexia. The obstruction in this case, caused by groundnut cake and feed material, which was successfully managed through sedation and manual extraction. Similar interventions are described by Ravi *et al.* (2018), where a linear rope was removed from the epiglottis of a Holstein Friesian cow, and by Mohanambal *et al.* (2018), where a sewing needle with a thread extending to the rumen was endoscopically retrieved from a goat kid. Foreign bodies lodged in the oesophagus can vary widely in both material and size, with some requiring endoscopic techniques for safe removal. However, for feed-related obstructions, manual retrieval under sedation often proves effective, as demonstrated in this case. Sedation and manual intervention is a common approach in large animal practice and has shown successful outcomes in similar cases (Ammu *et al.*, 2019; Kumar and Suthar, 2018; Ravi *et al.*, 2018 and Raju, 2020).

### Conclusion

The case highlights the importance of early diagnosis and appropriate management of oesophageal foreign body obstruction in cattle. Preventive measures such as meticulous feed material preparation are essential in reducing the risk of future obstructions.

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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 Poult. 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 figure, 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 coloured 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 <sup>3</sup>
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 <sup>2</sup>	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 use din 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.

#### **SHORT COMMUNICATION**

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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**DECLARATION**  
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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: 15<sup>th</sup> December, 2025

**Dr. G. Vijayakumar**

