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THERAPEUTIC MANAGEMENT OF DIABETIC KETOSIS IN A LABRADOR DOG – A CASE REVIEW

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ABSTRACT

Diabetic Ketosis (DK) is a metabolic complication of diabetes mellitus in dogs due to inadequate production of insulin from the endocrine pancreas or increased resistance of the body tissues to insulin. A five-year-old intact female Labrador dog was presented with a history of inappetence, weight loss, polyuria and polydipsia. Haemato-biochemistry revealed hyperglycaemia (810mg/dl) and elevated liver enzymes (alanine aminotransferase, aspartate aminotransferase and alkaline phosphatase). Urine analysis indicated glucosuria and ketonuria. The case was diagnosed as diabetic ketosis and successfully managed with daily administration of insulin along with dietary recommendations.

KEYWORDS: Diabetic ketosis, hyperglycaemia, ketonuria, insulin, dog.

INTRODUCTION

Diabetes mellitus is a common endocrinopathy in dogs with an estimated prevalence between 0.005 and 1.5 per cent (Qadri *et al*., 2015). Canine diabetes has been compared with human type 1 diabetes (Insulin dependent diabetes mellitus - IDDM) and is multifactorial, having both genetic and environmental influences, in both humans and dogs (Davidson *et al.*, 2005). Hypoinsulinemia, a characteristic feature of IDDM, along with elevated levels of counter regulatory hormones, especially glucogon, leads to the mobilization of peripheral fat by lipolysis and the formation of ketone bodies such as acetoacetate, beta-hydroxybutyrate and acetone ultimately resulting in diabetic ketosis (Hume *et al.*, 2006) and is a life threatening complication. The condition is mostly seen in dogs previously not diagnosed with diabetes mellitus due to failure of the owners to recognize the signs of uncomplicated diabetes mellitus early (Kasabalis *et al.*, 2015). Common clinic-pathological abnormalities include hyperglycaemia, anaemia, elevated liver enzymes, hyperlipidaemia and electrolyte imbalances. The present paper describes the clinical findings, diagnosis, treatment and outcome of a case of diabetic ketosis in a female Labrador dog.

MATERIALS AND METHODS

A five-year-old intact female Labrador dog was presented to the Department of Veterinary Medicine, Small Animal Unit, Veterinary Clinical Complex, Rajiv Gandhi Institute of Veterinary Education and Research, Puducherry with a history of inappetence, polydipsia, polyuria, fatigue and weight loss. The dog was vaccinated and dewormed regularly. Clinical examination revealed obese on appearance, dull, anorexia, elevated rectal temperature (103°F), congested conjunctival mucus membrane, palpable lymph nodes, dehydration, rough hair coat, heart and respiration rate were 156 beats/min and 67 breaths/min respectively. Blood sample was collected from cephalic vein and transferred to EDTA vial and clot activator for haematology and serum biochemistry examinations. Animal had normal haematological values and serum biochemistry showed hyperglycaemia (810 mg/dl) and elevated liver enzymes-alanine aminotransferase (168 U/L), aspartate aminotransferase (94 U/L) and alkaline phosphatase (777 U/L) (Table I). BUN and creatinine were within normal range. The qualitative urine analysis showed positive for Benedict's test (Fig. 1a) and Rothera's test (Fig. 1b) indicating glucosuria and ketonuria respectively.

Table I: Haematological and Biochemical Parameters of a dog affected with diabetes ketosis.

Parameter	DAY 0	DAY 45	Reference range*	Key Findings
Hb (g/dL)	11.8	11.5	12-19	Hyperglycaemia and elevated liver enzymes
TLC (10^3/mm^3)	8.2	14.1	5.0-14.1	
Neutrophils (%)	77	80	58-85	
Lymphocytes (%)	15	13	8-29	
Eosinophils (%)	02	04	0-9	
Monocytes (%)	06	03	5-11	
Basophils (%)	00	00	0-4	
Platelet count (10^3/mm^3)	301	569	2.11-6.21	
Total Protein (g/dL)	6.1	7.5	5.4-7.5	
Albumin (g/dL)	3.2	4.0	2.3-3.1	
Globulin (g/dL)	2.9	3.50	2.4-4.4	
Random Glucose (mg/dL)	**810.0**	**383.0**	76-119	
ALT (U/L)	**168.0**	51.0	10-109	
AST (U/L)	**94.0**	44.0	13-15	
ALP (U/L)	**777.0**	**213.0**	1-114	
BUN (mg/dL)	7.47	19.0	8-28	
Creatinine (mg/dL)	0.75	0.7	0.5-1.7	

(***Source*:** Haematological and serum biochemical reference ranges, 11th edition The Merck Veterinary Manual)

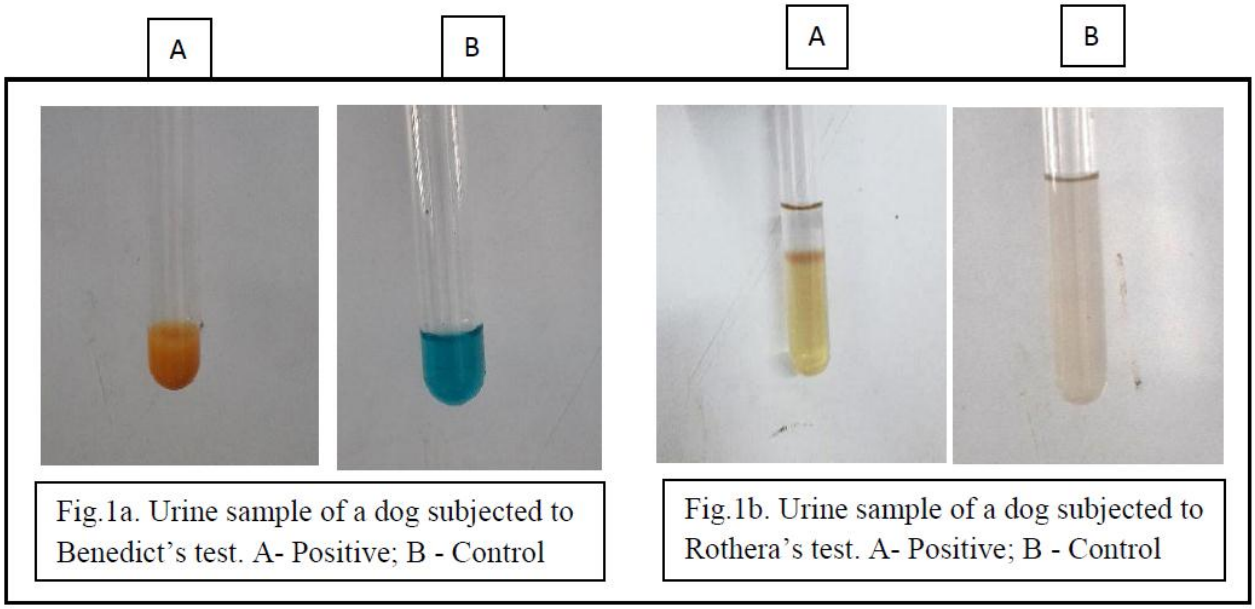

Fig.1a. Urine sample of a dog subjected to Benedict's test. A- Positive; B - Control

Fig.1b. Urine sample of a dog subjected to Rothera's test. A- Positive; B - Control

Based on the history, clinical findings, laboratory investigation and urine analysis, the case was diagnosed as diabetic ketosis. The dog was stabilised with Inj. Ringer's lactate 250ml IV and short acting insulin (INSUGEN - R™) @ 0.5 IU/kg BW, BID administered intravenously for 3 days followed by subcutaneous injections and liver protectant syrup (LIV.52™) @ 10ml daily PO. On day three of treatment, the dog stopped urinating. Tab. Furosemide (LASIX™) @ 1 mg/kg BW PO, and angiotensin converting enzyme (ACE) inhibitor, enalapril maleate (ENVAS 10™) @ 0.25 mg/kg BW, PO, SID was given for 10 days. After two weeks of treatment, random blood glucose was 630 mg/dl and dose of insulin was adjusted to 1.0 IU/kg BW, SC, BID. After a month, the animal was subjected to complete hemato-biochemical investigation and revealed the blood glucose level as 383mg/dl. It was advised to continue lifelong insulin therapy along with a high fibre diet and complex carbohydrates in small portions through the day to reduce postprandial elevation of the blood glucose levels.

RESULTS AND DISCUSSION

Canine diabetes mellitus is classified as Insulin dependent diabetes mellitus (Type 1) and Non-Insulin dependent diabetes mellitus (Type 2) with the former being more predominant in dogs. The aetiology of the disease is not clear and is multifactorial. A rise in the human cases of type 1 diabetes mellitus has been reported (Onkamo *et al.*, 1999) which is suggestive that since humans and dogs share the same environment, similar factors may be responsible for the development

of the disease in both species. Destruction of the beta cells of the pancreas leads to insulin deficiency will cause hyperglycaemia due to hepatic gluconeogenesis and glycogenolysis, decreased glucose uptake by tissues and increased protein and lipid catabolism (Das *et al*., 2019). Persistent hyperglycaemia, greater than the renal tubular threshold level (>180-220 mg/dl), causes glucosuria. Continued lipid catabolism results in hepatic lipidosis, production of ketone bodies causing ketoacidosis, will damage the endothelium and immunosuppression (Chen *et al.,* 2012). Hess (2013) reported that the condition is diagnosed by detecting the presence of hyperglycemia, ketonemia, ketonuria, and glucosuria with or without metabolic acidosis which is in concurrence with the present study. Samoyeds, Miniature Schnauzer, Poodles and Pugs are at higher risk of developing diabetes whereas German Shepherd, Golden Retriever and American Pit Bull Terriers were at lower risk (Hess*et al.,* 2000). Diabetes is more prevalent in bitches than male dogs due to the progesterone-dominant phase of diestrum and release of growth hormone into the circulation causing insulin resistance (Pöppl *et al.,* 2017). Heeley *et al.,* (2020) reported that the commonly affected age group is between 5 – 12 years and the incidence of diabetes mellitus is greater in winter season in dogs (Davidson *et al.,* 2005). These findings are in agreement with the present study. Das *et al.,* (2019) reported that biphasic isophane insulin @ 0.5 IU/kg BW SC OD was effective in the treatment of diabetes ketosis in a mongrel dog. In the present study, short acting insulin (INSUGEN R™) @ 1 IU/kg BW BID SC was found to be effective.

CONCLUSION

Diabetic ketosis is a complication of Type 1 diabetes mellitus characterized by hyperglycemia, glucosuria, ketonemia and ketonuria with or without metabolic acidosis. Dogs affected with diabetic ketosis/Type 1 diabetes mellitus require lifelong administration of insulin to maintain the blood glucose levels along with dietary management. The prevention and control of the condition is centred around a healthy diet and adequate physical exercise aimed at maintaining a normal body weight.

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