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D Akilkumar

PG Scholar, Department of
Veterinary Clinical Medicine, Ethics
and Jurisprudence, College of
Veterinary and Animal Sciences,
Mannuthy, Kerala Veterinary and
Animal Sciences University, Pookode,
Kerala, India

Shyma VH

Assistant Professor, Department of
Veterinary Epidemiology and
Preventive Medicine, College of
Veterinary and Animal Sciences,
Mannuthy, Kerala Veterinary and
Animal Sciences University, Pookode,
Kerala, India

Manju K Mathew

Assistant Professor, Department of
Veterinary Clinical Medicine, Ethics
and Jurisprudence, College of
Veterinary and Animal Sciences,
Mannuthy, Kerala Veterinary and
Animal Sciences University, Pookode,
Kerala, India

Arun George

Assistant Professor, Department of
Veterinary Clinical Medicine, Ethics
and Jurisprudence, College of
Veterinary and Animal Sciences,
Mannuthy, Kerala Veterinary and
Animal Sciences University, Pookode,
Kerala, India

Madhavan Unny N

Professor and Head, Department of
Veterinary Clinical Medicine, Ethics
and Jurisprudence, College of
Veterinary and Animal Sciences,
Mannuthy, Kerala Veterinary and
Animal Sciences University, Pookode,
Kerala, India

Corresponding Author:

D Akilkumar

PG Scholar, Department of
Veterinary Clinical Medicine, Ethics
and Jurisprudence, College of
Veterinary and Animal Sciences,
Mannuthy, Thrissur, Kerala
Veterinary and Animal Sciences
University, Pookode, Kerala, India

A case report on diabetic ketosis in a labrador retriever and its management

**D Akilkumar, Shyma VH, Manju K Mathew, Arun George and
Madhavan Unny N**

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Abstract

Diabetes mellitus is an endocrine metabolic disorder in dogs with persistent hyperglycemia and glucosuria. Diabetic ketosis is a complication of undiagnosed or uncontrolled diabetes mellitus. A seven-year-old female labrador retriever was brought to medicine unit with the history of vomiting and hyporexia for the past three days. Animal also had polydipsia and polyuria. On observation, the animal was dull and had bilateral cataract. Complete blood count revealed anemia. Fasting serum glucose was 600 mg/dl. Urinalysis revealed proteinuria, ketonuria and glucosuria. Urine sediment examination revealed the presence of few red blood cells and pus cells. Blood gas analysis revealed primary respiratory alkalosis. Treatment was initiated with Inj. Insulin mixtard with regular monitoring, amoxicillin clavulanate and Pantoprazole per os. On day two, fasting blood glucose was found to be 356 mg/dl. Hypokalemia was also recorded. It was advised to continue subcutaneous insulin twice a day with food and oral potassium chloride was also added. On day five, marked clinical improvement was noticed. The details of the case will be discussed in this report.

Keywords: Hyperglycemia, cataract, ketonuria, insulin, alkalosis

Introduction

Diabetes mellitus is an endocrine disorder resulting from relative or absolute deficiency of insulin. Insulin dependent diabetes mellitus is the most commonly occurring form of diabetes mellitus among dogs (Ettinger *et al.*, 2017) ^[1]. Insulin deficiency may arise from reduced synthesis from the beta cells of islet of Langerhans in endocrine pancreas. Reduced insulin results in reduced glucose uptake in cells which leads to hyperglycemia. Reduced glucose uptake by the cells increases gluconeogenesis in liver which cause weight loss and stimulate the satiety center in hypothalamus which causes increased appetite/polyphagia. Glucosuria will be evident when the blood glucose exceeds 180 – 200 mg/dl in dogs. The presence of glucose in the glomerular filtrate causes osmotic diuresis, leading to polyuria which in turn cause polydipsia. The above mechanisms cause the classical signs of polyuria, polydipsia, polyphagia and weight loss in clinical diabetes mellitus (Feldman *et al.*, 2014) ^[2]. Uncontrolled diabetes mellitus will result in the synthesis of ketone bodies which cause ketonemia. Ketone bodies will be readily reabsorbed in kidneys for energy synthesis. If the ketonuria exceeds the renal threshold, ketonuria will occurs (Kaneko *et al.*, 2008) ^[3] and this is termed as diabetic ketosis. Ketonic bodies are acidic in nature. Progressive ketonemia in diabetic ketoacidosis is life threatening (DiFazio and Fletcher, 2016) ^[4].

Case history and observations

A seven-year-old intact female labrador retriever was brought to the Medicine unit of Teaching Veterinary Clinical Complex, Mannuthy with the complaint of vomiting and hyporexia for the past three days. Animal had history of polyuria and polydipsia for the past one month. On observation, animal was dull, had bilateral cataract and panting. Animal had rectal temperature of 101.2°F, pale roseate conjunctival mucous membrane and normal lymph nodes. Estimation of peripheral blood glucose undetectable with glucometer even though the

animal was anorectic for 2 days. Complete blood count, serum creatinine, blood urea nitrogen and glucose were estimated and are listed in table 1. Urine was collected by catheterization of urinary bladder and results of urinalysis are as given in table 2. Microscopic examination of urine cytology revealed few red blood cells and pus cells. Venous blood was collected in a heparinized vial for blood gas analysis and it revealed respiratory alkalosis.

Treatment and Discussion

Therapy was initiated with Inj. Insulin mixtard at 0.5 IU/kg IM with regular monitoring. Owner was advised to give Inj.

Insulin mixtard with food, Tab. Amoxicillin clavulanate @ 12.5 mg/kg PO BID and Tab. Pantoprazole @ 1 mg/kg PO OD. On day 2, fasting blood glucose was 356 mg/dL. Serum potassium was found to be low at 3.3 mmol/L. Oral potassium chloride supplementation @ 2mEq/4.5kg PO BID with food was recommended. Clinical improvement was reported from day three. On day 5, blood glucose was found to be 96 mg/dL after two hours of insulin administration with food. Glucosuria and ketonuria was resolved on day 5 of starting the therapy. Results of urinalysis on day 5 is given in table 2. Subcutaneous insulin therapy and diet change are helped to maintain the glycemic status of the animal.

Table 1: Results of complete blood count, serum biochemistry and venous blood gas analysis

Parameters	Present Case	Reference Values*
Total leucocyte count ($10^3/\mu\text{L}$)	9.4	6 - 17
Absolute granulocyte count ($10^3/\mu\text{L}$)	6	4.4 - 12.6
Absolute lymphocyte count ($10^3/\mu\text{L}$)	2.3	0.7 - 5.5
Absolute monocyte count ($10^3/\mu\text{L}$)	0.3	0.2 - 1.7
Red blood cell count ($10^6/\mu\text{L}$)	6.44	5.5 - 8.5
Hemoglobin (g/dL)	10.5	12 - 18
Hematocrit (%)	43	37 - 55
Platelet count ($10^3/\mu\text{L}$)	178	160 - 525
Serum Creatinine (mg/dL)	1.1	0.8 - 1.4
Blood Urea Nitrogen (mg/dL)	22	10 - 28
Serum Glucose (mg/dL)	600	65 - 118
pH	7.565	7.35-7.45
pCO ₂ (mmHg)	28.4	40 - 45
pO ₂ (mmHg)	50.1	83 - 100
cHCO ₃ ⁻ (mmol/l)	25.7	19 - 24
Base excess (mmol/l)	3.6	-5 - +5
cSO ₂ (%)	90	94 - 98

*Reference values from Merck's Veterinary manual

Diagnosis of diabetes mellitus is usually made by establishing persistent hyperglycemia and glucosuria along with clinical signs. Diabetic ketosis was established when ketonuria was present. Serum fructosamine and glycosylated hemoglobin are measured to estimate the long-term glycemic status of the animal and mostly used in assessing the glycemic status in animals on insulin therapy (Behrend *et al.*, 2018) [5]. Healthy animals with diabetic ketosis or ketoacidosis can be treated with usual intramuscular crystalline insulin until ketonuria resolves (Nelson and Couto. 2014) [6]. Animals with severe form of ketoacidosis need emergency therapy inclusive of restoration of fluid, acid base and electrolyte imbalance along with intravenous insulin therapy (Hume *et al.*, 2006) [7].

Respiratory alkalosis as recorded in this case was also reported by Duarte *et al.*, (2012) [8]. Respiratory alkalosis in dogs due to hyperventilation caused by pulmonary diseases, severe anemia, psychogenic/neurological disturbances, stress and pain (Kaneko *et al.*, 2008) [3]. Arterial blood gas analysis could provide more accurate results than venous blood. Animal responded well to the subcutaneous insulin therapy in this particular case and ketonuria got resolved in urinalysis on day 5. Animal had hypokalemia on day 2 due to the uptake of glucose by cells along with potassium after insulin therapy. Animals with diabetes mellitus are susceptible to recurrent bacterial cystitis as in the present case was treated with Amoxicillin clavulanate. Reduction in polyuria and polydipsia was reported by the owner. Animal was maintained under glycemic control with insulin therapy.

Table 2: Results of Urinalysis on day 1 and day 5.

Parameters	Day 1	Day 5
pH	5	5
Specific gravity	1.015	1.005
Blood	+	-
Bilirubin	-	-
Glucose	++++ (2 g/dL)	-
Protein	+	-
Ketone bodies	+	-
Leucocytes	+	-

Conflict of Interest

Not available

Financial Support

Not available

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