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A case of diabetic ketosis in a mongrel dog and its therapeutic management

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Abstract

A 9-year-old female mongrel dog was presented to the Teaching Veterinary Clinical Complex, College of Veterinary Sciences and Animal Husbandry, Selesih with a history of polyurea, polydipsia, polyphagia, fatigue, weakness, weight loss and diminished eye vision. Detailed physical and clinical examination revealed mild cachexia, rough hair coat, dehydration, congested conjunctival mucous membrane, tachycardia and tachypnoea. Ophthalmological examination revealed mild bilateral cataract. Haematological findings were within the normal range. Serum biochemistry revealed hyperglycaemia (526 mg/dL) and increased liver-specific enzymes (aspartate aminotransferase and alanine aminotransferase). Qualitative urinalysis revealed glucosuria and ketonuria with very high specific gravity. Based on the physical, clinical and laboratory findings, the case was diagnosed as diabetic ketosis. The case was successfully managed with insulin therapy along with strict dietary suggestions. The blood glucose level came to normal range within 7 days and remained stable till 14 days of post therapy.

Keywords: Mongrel, hyperglycaemia, glucosuria, ketonuria, diabetic ketosis, Insulin

1. Introduction

Diabetes mellitus (DM) is one of the common metabolic disorders affecting middle-aged to geriatric dogs characterized by hyperglycaemia, glucosuria and weight loss, resulting from an absolute or relative deficiency of insulin. The incidence of canine diabetes is also increasing day by day globally due to various factors such as obesity, alteration in feeding habits, owner inattention, lack of awareness and advancement in disease diagnosis. Canine diabetes mellitus (CDM) is mostly type 1 (insulin dependent diabetes mellitus-IDDM), with the underlying cause of destruction or loss of pancreatic β -cells as a result of an inflammatory process in the exocrine or endocrine tissues ^[1]. IDDM is characterized by hypoinsulinemia and failure to establish glycaemic control in response to diet ^[2]. Hormonal imbalance of hypoinsulinemia and increased counterregulatory hormones like glucagon, cortisol, catecholamines and growth hormone may contribute to increased peripheral lipolysis and ultimately to the production of the ketone bodies such as acetoacetate, beta-hydroxybutyrate and acetone resulting in a condition known as diabetic ketosis ^[3]. Diabetic ketosis is characterised by combination of hyperglycaemia and ketosis. Dogs with diabetic ketosis are usually having IDDM. Diabetic ketoacidosis is a severe life threatening complication of diabetes mellitus. The present paper describes a case of diabetic ketosis in a female mongrel dog and its successful therapeutic management.

2. Case history and diagnosis

A 9-year-old female mongrel dog was presented to the Teaching Veterinary Clinical Complex, College of Veterinary Sciences and Animal Husbandry, Central Agricultural University, with a history of polyurea, polydipsia, polyphagia, fatigue, weakness, weight loss and diminished eye vision. The dog was properly dewormed and vaccinated. Detailed physical and clinical examination revealed mild cachexia, rough hair coat, dehydration, congested conjunctival mucous membrane, tachycardia (142 bpm) and tachypnoea (65/min). Ophthalmological examination revealed mild bilateral cataract (Fig.1 A). Whole blood and serum samples were collected for haematology and serum biochemistry. Urine sample was collected by using urinary catheterization technique. Haematological findings were within the normal range and

serum biochemistry revealed hyperglycaemia (526 mg/dL) and increased liver-specific enzymes (aspartate aminotransferase and alanine aminotransferase) (Table 1). Benedict's test and Rothera's test of urine sample revealed presence of glucose and ketone bodies, respectively (Fig.1 B and Fig.1 C). Qualitative urinalysis using commercially available (URS-10 HI) strip also showed presence of glucose and ketone bodies in urine with high specific gravity (Fig.1 D) (Table 2). Based on the physical, clinical and laboratory findings, the case was diagnosed as diabetic ketosis.

3. Treatment and Discussion

Initially the animal was stabilised using intravenous administration of Inj. NS @ 250 ml IV, Inj. B-complex @ 1 ml IV. After the confirmatory diagnosis, the specific treatment was started with biphasic isophane insulin (50:50) (Human MixtardTM) @ 0.5 IU/kg BW, SC, OD and the dose was standardized to 0.5 IU/kg BW, SC, BID after continuous blood glucose monitoring. Along with insulin therapy, multivitamins supplement (Multistar PetTM) at 1 tsp/10 kg BW, PO, BID and liver protective silymarin (Silybon[™]) at 1 tsp/10 kg BW, PO, BID for 2 weeks. The dietary management was suggested as per earlier reports ^[4]. Routine blood glucose level was monitored using automated glucometer so that hypoglycaemia should not develop. The blood glucose level came to normal range within 7 days and remained stable till 14 days of post therapy. It is also advised to continue recommended diet and insulin treatment for life time.

The CDM can be classified into two major types: type 1insulin dependent diabetes mellitus (IDDM) and type 2-non insulin dependent diabetes mellitus (NIDDM) or insulin resistant diabetes mellitus ^[1]. Most of the dogs suffer from IDDM. The cause of CDM has been poorly understood and is multi-factorial in nature. Destruction of β cells of pancreas due to inciting factors such as genetic predisposition, obesity, immune-mediated mechanisms, infection, insulin-antagonistic diseases and drugs, and inflammation resulting in a condition known as hypoinsulinemia. Hypoinsulinemia in turn results in impaired transport of blood glucose into most cells, increase in hepatic gluconeogenesis and glycogenolysis which results in hyperglycaemia ^[2, 5].

The classical clinical signs of diabetic dogs are polyuria, polydipsia and polyphagia and similar signs were also observed in the present case ^[6]. The high level of glucose in urine induces osmotic diuresis. Polydipsia is a compensatory mechanism of correction of dehydration due to polyurea. Mild bilateral cataract was also noticed in the present case and it may be due to osmotic changes in the lens, glycosylation of structural proteins and decreased concentration of antioxidants ^[7]. Increase in the liver specific enzymes in the present case can be because of hepatic lipidosis [8]. In DM, there is accelerated protein and fat metabolism that is responsible for hepatic changes ^[7, 9]. Gluosuria and ketonuria were the major findings of urinalysis. Increased activity of counterregulatory hormones may contribute toperipheral lipolysis and ultimately to the production of the ketone bodies like acetoacetate, beta-hydroxybutyrate and acetone resulting in a condition known as diabetic ketosis ^[10]. Decrease in the body weight is probably due to catabolic stage induced by diabetic ketosis.

The specific treatment was started with biphasic isophane insulin and the dose was standardised after continuous blood glucose monitoring. IDDM requires exogenous insulin administration to maintain the glycaemic balance. The treatment of CDM aims to alleviate clinical signs and improve quality of life, avoiding acute complications, such as diabetic ketoacidosis and iatrogenic hypoglycaemia and limiting chronic complications such as infections and cataracts ^[5, 11, 12]. The supportive treatment comprising multivitamin and hepatoprotective supplements along with prescription diet. Proper nutrition and preventive care prolongs the lifespan of the dog ^[13]. It is advisable to feed the dog within four hours of exogenous insulin administration, but, if the dog doesn't take the feed it may result in hypoglycaemic condition. In the present case, the exogenous insulin was administered to the dog immediately after feeding to avoid practical problem of iatrogenic hypoglycaemia.

Parameters	Day 0	Day 14	Reference range*	Key findings
Hb (g/dL)	12.9	12.2	12-19	
TEC (10 ⁶ /mm ³)	5.54	5.42	5.0-7.9	
TLC (10 ³ /mm ³)	12.1	11.51	5.0-14.1	
Neutrophils (%)	61	60	58-85	
Lymphocytes (%)	29	28	8-29	
Monocytes (%)	06	07	5-11	
Basophils (%)	01	00	0-4	
Eosinophils (%)	03	05	0-9	• Uymanalyzanamia
Platelets count (lakhs/mm ³)	3.28	2.98	2.11-6.21	 Hypergrycaenna Ingrosped liver specific enzymes
Total protein (g/dl)	5.45	5.52	5.4-7.5	 Increased river specific enzymes
Albumin (g/dl)	2.51	2.49	2.3-3.1	
Globulin (g/dl)	2.94	3.03	2.4-4.4	
Random Blood Sugar (mg/dl)	526	109	76 – 119	
ALT (U/L)	143	98	10 - 109	
AST (U/L)	52	25	13 – 15	
BUN (mg/dl)	16.4	16.8	8-28	
Creatinine (mg/dl)	1.12	1.09	0.5 - 1.7	

Table 1: The haemato-biochemical parameters of dog on day 0 and day 14

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

Parameter	Before treatment	After treatment
Urobilinogen	Nil	Nil
Bilirubin	Nil	Nil
Ketone	++++	+
Blood	Nil	Nil
Protein	++	Trace
Nitrite	Nil	Nil
Leukocytes	Nil	Nil
Glucose	++++	+
Specific gravity	1.060	1.030
pH	6.5	6.5



Fig 1: Clinico-pathological changes noticed in a dog with diabetic ketosis A. Mild bilateral cataract B. Benedict's test of urine showed positive for glucose C. Rother's test of urine showed positive for ketone bodies D. Qualitative urinalysis using URS-10 HI strip showed presence of glucose and ketone bodies in urine with high specific gravity

4. Conclusion

The present case describes diabetic ketosis in a female mongrel dog and its successful therapeutic management with biphasic Isophane insulin.

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6. Conflict of interest

The authors declare that they don't have any conflict of interest.

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