Haryana Vet. (December, 2024) **63(2),** 262-264

Short Communication

OCCULT DIABETIC KETOACIDOSIS IN LABRADOR RETRIEVER: DIAGNOSIS AND MANAGEMENT

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Received: 06.07.2023; Accepted: 24.02.2024

SUMMARY

The objective of this study is to report a complicated combination of enteropathies leading to life threating condition in a Labrador Retriever. A five-year-old Labrador Retriever dog primarily having history of hypothyroidism was presented at Intensive Care Unit of Veterinary Clinical Complex, Vety. College, Lala Lajpat Rai University of Veterinary and Animal Sciences, Hisar with persistent vomiting and lethargy. The dog was diagnosed with hyperglycemia along with ketonuria i.e., diabetic ketoacidosis. Dog was successfully managed with treatment including intravenous fluid resuscitation, intravenous insulin therapy and antibiotic.

Keywords: Hyperglycemia, Hypothyroidism, Insulin, Ketonuria, Lipase

How to cite: Yadav, A., Kumar, T., Gupta, S., Sharma, M., Agnihotri, D. and Sindhu, N. (2024). Occult diabetic ketoacidosis in Labrador Retriever: Diagnosis and management. *Haryana Vet.* **63(2)**: 262-264.

Diabetic Ketoacidosis (DKA) is a serious complication of diabetes mellitus type1that occurs most commonly in dogs and cats with previously undiagnosed diabetes (Claus *et al.*, 2010). Biochemical triads that characterize this condition are acidity, ketosis and hyperglycemia (O'Brien, 2010). Main triggering factor is relative or absolute insulin deficiency associated with increase counter-regulatory hormones causing ketone bodies overproduction (Durocher *et al.*, 2008). Less frequently, DKA happens in an insulin-treated diabetic dog or cat that is receiving an insufficient dose of hormone, which frequently happens with underlying inflammatory, viralor hormonal condition that is insulin-resistant. Intravenous fluid resuscitation, insulin administration, correction of acid-base and electrolyte imbalances and identification and treatment of any associated illnesses are all part of DKA therapy (Nelson, 2015).

CASE DESCRIPTION

A five-year-oldmale Labrador retriever dog weighing 50 kilograms, primarily having history of hypothyroidism was presented at Intensive Care Unit of Veterinary Clinical Complex, Hisar with persistent vomiting and lethargy. History revealed that problem started 2 days before with melena, polyuria, polydipsia, vomition and dark colored urine. Clinical examination revealed hyperthermia, lethargy and dehydration. Pet was immediately shifted to intensive care ward. Routine clinical examination, hemato-biochemical and urine analysis along with abdominal ultrasonography was performed. Blood samples were analyzed in automated hematology cell counter (MS4S, Melet Schlosing Lab.) and serum samples were analysed using automated random access clinical chemistry (EM-200, ERBA Diagnostics Mannheim GmbH). Urine sample was collected aseptically by catheterization and processed microscopically for various cells, crystals and casts. Routine urinalysis was done with the help of urine strip reader Laura Smart®.

RESULTS AND DISCUSSION

Non-specific clinical signs like polyuria, lethargy, vomiting, anorexia in the affected dog were noticed that might be due to acidemia secondary to ketonemia. Both glucose and ketones are osmotically active, leading to polyuria, polydipsia and subsequent dehydration with risk of hypovolaemia. Hematological analysis depicted relative neutrophilia and lymphopenia (Table 1) with other parameters within reference range. Pancreatic enzymes i.e. amylase, thyroid function parameters i.e. free T4 or TSH and serum electrolytes like sodium, potassium, chloride were found to be within normal range (Table 2). Forthe past one year, the animal has continued to receive levothyroxine at a dose of 0.02 mg/kg twice daily and there was significant hyperglycemia (421 mg%) with elevated serum alanine aminotransferase (ALT), pancreatic lipase and creatinine (2.62 mg%) as shown in table 2. Routine urinalysis revealed acidic pH, significant glucosuria (+3) and ketonuria (+4) (Table 3).

Hyperglycemia and glucosuria resulted from glucose's inability to enter cells to produce adenosine triphosphate (ATP) in affected dog. Abdominal ultrasonography interpreted mild hydronephrotic changes in left kidney while other organs had no alterations. Based on haemato-biochemical analysis and clinical signs, pet was diagnosed to be affected with diabetic ketoacidosis and

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Table 1. Hematological findings in affected dogs pre and 48-hour post therapy

Parameters	Variations in value pre and post therapy	
	0 day	After 48hr post therapy
Hemoglobin	14.7	16.50
Packed cell volume (PCV)	38	48.3
Total erythrocyte count (TEC)	5.48	6.96
Total leucocyte count (TLC)	13.6	11.73
Neutrophil (%)	89	79
Lymphocyte (%)	7	11
Monocyte (%)	4	08
Total platelet count (thousand/cumm)	158	169

Table 2. Biochemical parameters alteration in affected dogs on 0, 24 and 48 hour post therapy

Parameters	Variations in value pre and post therapy		
	0 day	24 hr post therapy	48 hr post therapy
Sodium (mEq/l)	133.3	143.8	145
Potassium (mmol/l)	3.81	4.41	4.4
Chloride (mmol/l)	109.1	101.6	106
Free T4 (ng/dl)	1.02	-	-
TSH (mIU/l)	32.15	-	-
Glucose (mg%)	483	427.7	421
SGPT/ALT (IU/l)	364.6	284.3	116.4
SGOT/ AST (IU/l)	359.7	276.4	103.8
Blood urea (mg/dl)	38.5	-	-
Creatinine (mg/dl)	2.62	-	-
Amylase (IU/l)	639	-	-
Lipase (IU/l)	881.7	-	-

Table 3. Routine urinalysis findings in affected dog pre and 24 hr post therapy

Parameters	Variations in value pre and post therapy	
	0 Day	24 hr post therapy
Colour	Dark yellow	Pale yellow
pH	6	7.5
Protein	2+	1+
Glucose	3+	2+
Pus cells	Rare	rare
RBC	25-30/ hpf	3-4/hpf
Epithelial cells	5-7/hpf	few
Crystals/ casts	Nil	Nil
Bacteria	Nil	Nil
Ketone bodies	4+	2+

management was initiated. The pet was given intravenous fluid of one-liter normal saline IV over 1 hour at the rate of 20 ml/kg along with injection meropenem 1 g (@ 20 mg/kg body weight) 24 hourly and antiemetics 12 hourly. Regular insulin 10 units at the rate of 0.2 unit/kg was givenin 100 ml of NS IV followed by 5 units of regular Insulin intramuscularly after 3 hours of first dose. Patient was advised to be kept on nil per oral and regular monitoring of blood glucose was advised. Haemato-biochemical examination after 24 and 48 hr post therapy showed decrease in blood glucose (483 mg/dl to 421 mg/dl) with marked clinical improvement. Urinalysis also showed significant decrease in ketonuria and glucosuria after 24h of therapy. This study reflects the effective management of DKA in critically ill patient but suffers from limitation of not monitoring hourly blood glucose level.

In present study, affected dog also found to have concurrent hypothyroidism. Cooper *et al.* (2015) also reported that vast majority of patients with DKA (reportedly 70% of dogs and 90% of cats) have concurrent disease process. Concurrent infection leads to further increase in stress hormones (e.g. glucagon and cortisol) which counteract insulin effects resulting further exaggerating ketone production (Hume *et al.*, 2006).

Initial stabilization included fluid resuscitation and correction of electrolyte and acid base abnormalities. Sodium chloride (NaCl 0.9%) has traditionally been the recommended fluid choice in these patients because higher sodium content was thought to reduce fluid shifts and therefore cerebral oedema risk (Gant, 2019). Continuous low dose regular insulin intravenously @ 0.05-0.1 IU/kg followed by intramuscular regular insulin @ 0.1-0.4 to combat hyperglycemia.

Insulin treatment aims to support cellular glucose uptake, decrease hepatic glucose production and interrupt ketogenesis promoting ketone metabolism and clearance (Macintire, 1993). Antiemetic i.e. ondansetron @ 0.1-0.2 mg/kg slow intravenously and prochlorperazine @ 0.5 mg/kg intramuscularly given to control emesis. Antibiotics and supportive therapy are given to combat secondary bacterial infections. Patient showed improvement following seven days of therapy. Table 1, 2 and 3 showed haemato-biochemical and urinalysis finding in affected dog pre and post therapy depicting significant improvement. Dog was doing well and therapy was continued for 7 days with significant clinical improvement. Patient owner was advised for at least biannual routine health checkup with lifelong insulin therapy.

REFERENCES

Claus, M.A., Silverstein, D.C., Shofer, F.S. and Mellema, M.S. (2010). Comparison of regular insulin infusion doses in critically ill diabetic cats: 29 cases (1999-2007). *J. Vet. Emerg. Critical Care.* **20(5)**: 509-517.

Cooper, R.L., Drobatz, K.J., Lennon, E.M. and Hess, R.S. (2015). Retrospective evaluation of risk factors and outcome predictors in cats with diabetic ketoacidosis (1997-2007): 93 cases. *J. Vet. Emerg. Critical Care.* **25(2)**: 263-272.

Durocher, L.L., Hinchcliff, K.W., DiBartola, S.P. and Johnson, S.E. (2008). Acid-base and hormonal abnormalities in dogs with naturally occurring diabetes mellitus. *J. Am. Vet. Med. Assoc.* **232(9)**: 1310-1320.

Gant, P. (2019). Diabetic ketoacidosis in cats and dogs. *Vet. Ireland J.* **9(10)**: 549-554.

Hume, D.Z., Drobatz, K.J. and Hess, R.S. (2006). Outcome of dogs with diabetic ketoacidosis: 127 dogs (1993-2003). *J. Vet. Int. Med.* **20(3)**: 547-555.

Macintire, D.K. (1993). Treatment of diabetic ketoacidosis in dogs by continuous low-dose intravenous infusion of insulin. *J. Am. Vet. Med. Assoc.* **202(8)**: 1266-1272.

Malerba, E., Cattani, C., Del Baldo, F., Carotenuto, G., Corradini, S., Golinelli, S., Drudi, I. and Fracassi, F. (2020). Accuracy of a flash glucose monitoring system in dogs with diabetic ketoacidosis. *J. Vet. Int. Med.* **34(1)**: 83-91.

Nelson, R.W. (2015). Diabetic ketoacidosis In: Feldman EC, Nelson RW, Reusch CE, Scott-Moncrieff JC, Behren EN, Eds. Canine and Feline Endocrinology. (4th Edn.), St Louis, MO: Elsevier Saunders; pp. 315-347.

O'Brien, M.A. (2010). Diabetic emergencies in small animals. *Vet. Clinics: Small Anim. Practice.* **40(2)**: 317-333.

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