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Hyperglycemic Hyperosmolar Syndrome in a Lhasa Apso Dog

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Abstract

Hyperglycemic hyperosmolar syndrome is an infrequently described complication of canine diabetes mellitus, differing from diabetic ketoacidosis in certain aspects, including treatment. A nine-year-old Lhasa Apso was presented to the Madras Veterinary College Teaching Hospital with a history of polyuria and polydipsia for a period of one week, and sudden dullness and inappetence. Clinical examination revealed the presence of hyperglycemia (799 mg/dL). An arterial blood gas analysis revealed a primary respiratory alkalosis, hyponatremia and hypokalemia. The serum creatinine level was elevated. Based on the absence of acidosis and ketonuria, and the eventual development of obtundation, a diagnosis of hyperglycemic hyperosmolar syndrome was made. Chronic kidney disease was diagnosed by ultrasonography. Treatment was done with intravenous infusion of normal saline, soluble insulin as a constant rate infusion and potassium chloride. The animal died after four days of treatment. The prognosis is guarded to poor.

Keywords: hyperglycemic hyperosmolar syndrome, diabetes, dog

Introduction

Hyperglycemic Hyperosmolar Syndrome (HHS) is a rare complication of canine diabetes mellitus. It is an infrequently reported syndrome (Trotman *et al.,* 2013). HHS is characterized by severe hyperglycemia and dehydration. The nervous system is principally affected by hyperosmolality and neurologic dysfunction is the major clinical manifestation of severe hyperosmolality (Solomon, 2022). It is crucial to lower the serum sodium concentration slowly, in order to decrease the risk of fluid shifting from the extracellular compartment to the brain and the prognosis is guarded to poor (Gough *et al.,* 2024). The present case describes a case of Hyperglycemic Hyperosmolar Syndrome in a Lhasa Apso dog.

Case History and Observations

A nine-year-old intact female Lhasa Apso weighing 14 kilograms was presented to the Madras Veterinary College Teaching Hospital with a history of polyuria, polydipsia and weight loss for a period of one week. The animal was dull and was reported inappetent. Recurrent bilious vomiting was also seen over the past week. Clinical examination, haemato-biochemical examination, urinalysis, arterial blood gas analysis and abdominal ultrasonography were carried out. Heart rate was respiratory rate, and rectal temperature were 140 bpm, 180 breaths/minute and 102.5 °F respectively. Congested mucous membranes, dehydration. The popliteal lymph nodes were palpable. The. Mild dehydration was present. The systolic blood pressure was 110 mm Hg. Haematology revealed a hemoglobin of 12.2 g/dL, PCV of 35.7%, RBC count of 6.30 m/cmm, WBC count of 25,300/cmm, a platelet count of 3,81,000/cmm and Blood picture revealed neutrophilia. Serum biochemistry revealed hyperglycemia (>600 mg/dL), cholesterol of 158 mg/dL, triglycerides of 189 mg/dL, total protein 8.2 g/dL, albumin 2 g/dL, blood urea nitrogen 54.23 mg/dL, creatinine 4.36 mg/dL, ALT 34 U/L, ALP 198 U/L, total bilirubin 1.06 mg/dL, direct bilirubin 0.93 mg/dL, calcium 8.96 mg/dL and phosphorus 14.72 mg/dL. Urinalysis revealed an acidic pH, proteinuria, glucosuria and absence of ketonuria. Abdominal ultrasonography revealed a diffuse increase in echogenicity of the liver parenchyma (indicative of diabetic hepatopathy) and mild irregularity in the contour of both kidneys (indicative of chronic kidney disease). The arterial blood gas analysis results are tabulated in Table 1 and were indicative of a primary respiratory alkalosis.

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Table 1 – Arterial Blood Gas Analysis

Parameter	Day 1	Day 2	Day 3	Day 4
pH	7.567	7.470	7.417	7.358
pCO_2	25.1 mm Hg	26.4 mm Hg	21.7 mm Hg	16.6 mm Hg
pO_2	77.3 mm Hg	90.5 mm Hg	76.5 mm Hg	89.6 mm Hg
$cHCO_3^-$	22.8 mmol/L	19.2 mmol/L	14 mmol/L	9.3 mmol/L
BE (ecf)	0.7 mmol/L	-4.5 mmol/L	-10.6 mmol/L	-16.1 mmol/L
Na^+	114 mmol/L	133 mmol/L	136 mmol/L	144 mmol/L
K^+	3.2 mmol/L	3.5 mmol/L	3.6 mmol/L	5 mmol/L
Ca^{++}	0.96 mmol/L	0.93 mmol/L	0.84 mmol/L	0.76 mmol/L
Cl^-	73 mmol/L	105 mmol/L	109 mmol/L	128 mmol/L
AGap	20 mmol/L	10 mmol/L	14 mmol/L	8 mmol/L
AGapK	23 mmol/L	14 mmol/L	18 mmol/L	13 mmol/L
Glu	>700 mg/dL	>700 mg/dL	>700 mg/dL	689 mg/dL
Lac	3.61 mmol/L	2.39 mmol/L	5.03 mmol/L	6.46 mmol/L
BUN	71 mg/dL	61 mg/dL	80 mg/dL	80 mg/dL
Crea	6.64 mg/dL	5.62 mg/dL	7.81 mg/dL	7.81 mg/dL

Treatment and Discussion

Dog was administered with intravenous infusion of Potassium chloride (20 mEq) diluted in Normal Saline to combat the hyponatremia and hypokalemia. This was followed by an IV infusion of soluble insulin (@ of 0.1 IU/kg/hour mixed in Normal Saline). Pantoprazole and ondansetron injections were given intravenously. The animal was presented for the next three days in lateral recumbency, in progressively deteriorating condition. Hourly monitoring of vitals and blood glucose was done. The systolic blood pressure dropped to 90 mm Hg. Oxygen supplementation was given on day four as oxygen saturation dropped down to 89 per cent. The animal was discharged in the evening with a blood glucose of 300 mg/dL. The animal collapsed the next morning on the way to the hospital.

Hyperglycemia contributes substantially to the serum osmolality in diabetic animals (Feldman and Nelson, 2004). Glucose and sodium are important contributors to serum total and effective osmolality in diabetics. Hyperglycemia establishes an osmotic gradient between the extracellular and intracellular compartment of the brain resulting in cellular dehydration. This induces altered consciousness and may explain the dullness and recumbency seen in these patients (Doromal and Canter, 1973). Hyperglycemia stimulates endogenous insulin production and may in some cases cause intracellular translocation of potassium leading to hypokalemia (DiBartola and de Morais, 1992). The excretion of glucose by the kidneys also causes an osmotic diuresis and may cause depletion in potassium, sodium and phosphorus, thereby aggravating the hypokalemia (Evans, 1992). The hyperosmolarity of the serum and the absence of acidosis led to a diagnosis of HHS (Schermerhorn and Barr, 2006). Treatment involves addressing of dehydration, hyperglycemia, electrolyte abnormalities and concurrent disease. The presence of concurrent disease complicates recovery and the prognosis is guarded to poor.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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