CASE REPORT

Concurrent Diabetic Mellitus with Cushing Syndrome in a Dog

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Cortisol is the principal glucocorticoid released by the adrenals in dogs and cats. Prolonged exposure to inappropriately elevated plasma concentrations of free cortisol leads to devolpment of Cushing's syndrome or hyperadrenocorticism (HAC) (Rijnberk *et al*., 2010). HAC is typically a chronic and progressive disease, encountered in middle aged to older dogs. These dogs are usually in a good condition with excellent appetite. The clinical manifestations include polyuria, polydipsia, increased appetite, weight gain, abdominal enlargement, and cutaneous lesion. Laboratory findings include neutrophilia, monocytosis, increased alanine aminotransferase and alkaline phosphatase, increased triglycerides and cholestrol. Screening tests for HAC includes ACTH stimulation test, Low dose dexamethasone suppression test and Urinary cortisol : creatinine ratio. Diagnosis of HAC is confirmed by ultrasonography, Computed Tomography, Magnetic Resonance Imaging (Rijnberk *et al*., 2010). Laproscopic adrenalectomy is surgical procedure of choice in veterinary medicine or by the use of adrenocorticolytic and adrenocorticostatic drugs (Rijnberk *et al*., 2010). HAC is occasionally associated with fasting hyperglycemia, overt diabetes mellitus and ketoacidosis. The incidence of DM with HAC in dogs is approximately 16.6-22.0 % (Poppl *et al*., 2016). The clinical signs for DM are almost common to those of HAC (Polyuria, polydipsia, weight gain, hepatomegaly) (Hess *et al.,* 2000). This communication puts on record a case of concurrent diabetic mellitus with Cushing syndrome in a dog.

Case History and Observations

An 11-year old Miniature Pinscher dog named Bruno, weighing 3.9 kg was presented to Medicine Unit of VCRI Hospital, Namakkal. Bruno came with the history of polyuria and polydipsia. Physical examination revealed distension of abdomen (Fig. 1a). On abdominal palpation, the urinary bladder was found to be flaccid and the estimated glucose concentration by glucometer was 396 mg/dL. Urine strip revealed glucosuria and traces of presence of protein and ketone. Specific gravity of urine was found to be 1.060. Serum biochemistry revealed hypertriglyceridemia and hyperchloestrolemia (1684 mg/dL). Low dose dexamethasone suppression test was performed. Dexamethasone sodium phosphate @ 0.0l5 mg/kg was administered intravenously and blood sample was collected for cortisol at 0, 4 and 8 h interval, which revealed the values of 4.72, 6.05 and 4.32 µg/dL, respectively. This rise in serum cortisol at 4 h and partial suppression at 8 h confirmed HAC. Ultrasonography revealed the hypoechoic structure in the cranial pole of the left kidney which was the left adrenal gland measuring 5.7 mm (Fig. 1b), however, right adrenal gland was normal (3.2 mm). This unilateral adrenomegaly confirmed the diagnosis of adrenal dependent HAC.

Treatment and Discussion

The dog was treated with Insulin @ 1 IU/kg S/C for Diabetes mellitus and Tab- Trilostane @ 30 mg/kg P/O, SID for 2 months. Animal was recovering during the course of treartment. About 15-20 % of dogs with naturally occurring HAC have an adrenocortical tumor that autonomously secretes excessive amounts of cortisol (Ettinger *et al*., 2024). Most adrenocortical tumors are unilateral solitary lesions (Melian *et al*., 2021). But bilateral tumors occur in about 10% of cases (Rijnberk *et al*., 2010). Common clinical signs of HAC are polyuria and polydipsia (Mohanambal *et al.,* 2023). Dogs with HAC present disturbances in lipid metabolism such as hypertriglyceridemia or hypercholesterolemia or both (Jerico *et al*., 2009; Mohanambal *et al.,* 2023). HAC is usually associated with alterations in glucose metabolism, thus resulting in DM. Insulin's effects are antagonized by glucocorticoids, which results in a sustained increase in the levels of circulating glucose and thus results in DM (Di Dalmazi *et al*., 2012). DM is due to resistance to insulin or limited insulin reserve in the case of HAC. Though the clinical signs of DM and HAC are common, it is important to rule out HAC in diabetic dogs, if the dog has resistance to exogenous insulin

therapy in lowering glucose levels. The dog must be stabilized with insulin for diabetes screening or before proceeding to treatment for Cushing's syndrome. Dogs having both HAC and DM will have weight gain, once DM is treated, the dog will have a weight loss (Castillo *et al.,* 2008) due to anabolic effect of insulin (Dimitriadis *et al*., 2011). After stabilizing the animal with insulin, it should be treated for HAC either with surgical adrenalectomy or therapy using mitotane or trilostane. Life expectancy shortens in the diabetic dog with HAC (Peterson *et al*., 1981). ACTH stimulation test performed using Consytropin, was suggestive of HAC.

In brief, concurrent DM and HAC are quite common in dogs. This case suggest that, in clinical practice dogs presented with DM should also be screened for HAC to rule out comorbid condition. This report will help for future researchers for managing concurrent issues.

Fig. 1(a): Miniature pinscher dog with abdominal distension.

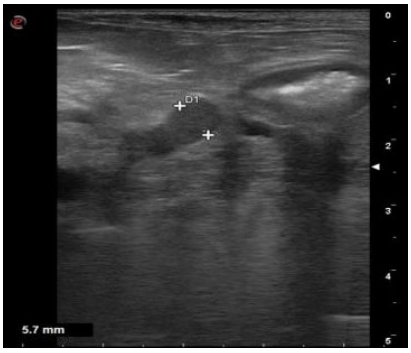

Fig 1(b): Ultrasonography showing enlarged left adrenal gland measuring (5.7 mm).

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