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A brief insight into Canine Hypoadrenocorticism (Addison's Disease)

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Abstract
Hypoadrenocorticism is an endocrinopathy characterized by a deficiency of glucocorticoid and/or mineralocorticoid secretion from the adrenal cortex. Primary adrenocortical insufficiency(Addison's disease) with a deficiency of mineralocorticoidand glucocorticoid secretion is the most common form of the disease.Hypoadrenocorticism is typically a disease of young tomiddle-aged female dogs with a median age of 4 to 6 years(range, 2 months to 12 years).Signs vary from mild in chronic hypoadrenocorticism to severe andlife-threatening in an acute Addisonian crisis.One of the hallmark signs of hypocortisolism is impaired tolerance to stress, and clinical signs often become more pronounced when the animal is placed in stressful situations.Definitive diagnosis is by demonstration ofundetectable to low (<2µg/dL) baselineserum cortisol concentrations that fail toincrease above 2µg/dL following adrenocorticotropic hormone (ACTH)administration.Therapy includes glucocorticoid and mineralocorticoid supplementation, alongwith supportivetreatment.

Keywords: Hypoadrenocortvicism, Mineralocorticoid, Glucocorticoid, ACTH

Introduction

The adrenal glands are essential for life, being responsible for the minute-to-minute regulation of blood pressure, blood volume, water balance and vascular tone. Adrenal cortex is made up of three layers—zona glomerulosa, zona fasciculata, and zona reticularis and is responsible for mineralocorticoid synthesis, glucocorticoid and sex hormone synthesis, respectively.Hypoadrenocorticism is a deficiency of mineralocorticoids,glucocorticoids, or both.It is of three types; primary hypoadrenocorticism, atypical hypoadrenocorticism and secondary hypoadrenocorticism(Liftonet *al*. 1996; Thompson *et al*. 2007). It is most commonly seen in young to middle-aged female dogs. Great Danes, Portuguese water dogs, Rottweilers, Standard poodles and West Highland White Terriers appear to be at an increased risk of developing naturally occurring primary hypoadrenocorticism.The sodium/potassium ratio reflectschanges in these electrolyte concentrations in serum andfrequently has been used as a diagnostic tool to identify adrenal insufficiency. The normal ratio of Na/K varies between 27:1and 40:1. Values are often less than 27 and may be less than20 in animals with primary adrenal insufficiency. Results of an ACTH stimulation test confirms thediagnosis.

Types of hypoadrenocorticism:

- **Primary Adrenocortical Insufficiency (Addison's disease):**It is the result of atrophy or destruction of all layers of theadrenal cortex, usually resulting in glucocorticoid andmineralocorticoid deficiency. It is mostly associated with immune-mediated destruction of adrenal cortical tissue but may also occur due to iatrogenic administration of certain drugs, neoplasia, infection, or infarction of the adrenal glands.
- **Secondary Hypoadrenocorticism:** occurs due to central (anterior pituitary) deficiency of ACTH and leads to isolated glucocorticoid insufficiency. Mineralocorticoids are spared because ACTH does not directly influence their release (Lennon *et al*. 2005).It mainly results from abrupt discontinuation of long-term exogenous administration of corticosteroids or progesterone analogues and rarely, due to congenital defects of the pituitary gland (cystic Rathke's pouch, neoplasia, or trauma).
- **Atypical hypoadrenocorticism:**Seen in a small population of "atypical" dogs. Adrenal destruction is purported to spare the glomerulosa layer, resulting in an isolated glucocorticoid deficiency and so, do not progress to clinically significant mineralocorticoid deficiency. Isolated glucocorticoid insufficiency is more commonly seen in older dogs with vague gastrointestinal

signs, weight loss, normal electrolyte concentrations, hypoalbuminemia and hypocholesterolemia (Melian and Peterson 1996).

Clinical signs

No historical or clinical finding or set of clinical signs is pathognomonic for hypoadrenocorticism, therefore dogs with this disease are termed as "The Great Pretender".The most common clinical manifestations arerelated to alterations in the gastrointestinal tract and mentalstatus and include lethargy, anorexia, vomiting, and weightloss (Willard *et al*. 1982; Peterson *et al*. 1996).Weakness is a common client complaint. Additionalphysical examination findings may include dehydration, bradycardia,weak femoral pulses, and abdominal pain. Hyperkalemiaand hypoadrenocorticism should be suspected in ananimal with bradycardia and signs consistent with hypovolemia. Biochemical findings in mineralocorticoid insufficiency results in low sodium concentration and high potassium concentration resulting in hypovolemic shock, dehydration and collapse.If hyponatremia and hyperkalemia become severe, theresultant hypovolemia, prerenal azotemia, and cardiacarrhythmias may result in an addisonian crisis. Whereas glucocorticoid insufficiency results in vague signs such as lethargy, weakness, polyuria, polydipsia, vomition, diarrhea, abdominal pain, inappetence and weight loss. The Addisonian crisis is the life-threatening culmination of combined hormone deficiencies that can be fatal if not appropriately treated.

Diagnosis

The disease is diagnosed on the basis of combination of several parameters

- Compatible history and clinical signs (lethargy/depression, decreased appetite/anorexia, vomiting, weakness, weight loss, diarrhea, waxing/waning illness, dehydration, hypothermia, shaking/shivering, weak pulses, polyuria/polydipsia, melena,painful abdomen)
- Laboratory abnormalities includes complete blood count, serum biochemistry panel, and urinalysis (hemogram findings mayinclude eosinophilia, lymphocytosis, mild anaemia, high haematocrit, serum biochemical and electrolyte abnormalities results in hypoalbuminemia, hypercholesterolaemia, hypoglycaemia, azotaemia hyperkalemia, hyponatremia and hypercalcemiawhereasurinalysis commonly reveals isosthenuria)
- Diagnostic imaging
- ACTH stimulation test: The gold standard for diagnosis of all forms of hypoadrenocorticism; it should be performed in any patient suspected of having the disease. A post-sample of < 2.0 µg/dL (55 nmol/L) is consistent with clinical diagnosis of hypoadrenocorticism. A baseline serum cortisol level > 2 µg/dL can be used to rule out hypoadrenocorticism, while a cortisol level ≤ 2 µg/dL necessitates an ACTH stimulation test (Burkitt *et al*. 2007).

Treatment

The main goals of therapy include:

- Restoration of blood volume
- Correction of electrolyte/acid-base disorders
- Fluid therapy: is always instituted prior to use of adrenal steroid replacement therapy and can be performed whilethe patient is undergoing the ACTH stimulation test.
- Starting with aggressive IV fluid therapy with isotonic crystalloids (0.9% sodium chloride, Ringer's lactate solution)
- The mineralocorticoid desoxycorticosterone pivalate (DOCP) is administered at 2.2 mg/kg, IM or SC, every 25–28 days and electrolytes should be measured at 3 and 4 weeks after the first few injections to determine the duration of action.
- Alternatively, fludrocortisone acetateis administered PO at 10–30 mcg/kg/day. Serum electrolytes should be monitored weekly until the proper dose is determined.
- Some dogs (especially dogs on DOCP) also require daily oral glucocorticoid therapy to adequately control clinical signs.

Conclusion

Primary hypoadrenocorticism results from the destruction of>90% of the adrenal cortex mainly due to an immune-mediated process. Young to middle-age dogs are predisposed, with increased breed predisposition for West Highland White Terriers, and GreatDanes.Addison's disease is known as the "Great Pretender" because nonspecific signs such as lethargy,decreased appetite, and weight loss predominate.Hypoadrenocorticism is often tentatively diagnosed on thebasis of the history, physical examination findings, clinicopathologicfindings, and, in the case of primary adrenalinsufficiency, identification of appropriate electrolyte abnormalities. Results of an ACTH

stimulation testconfirms the diagnosis. Treatment protocol includes glucocorticoid and mineralocorticoid supplementation, alongwith intravenous fluids and supportivetherapy.

Bibliography

Adamantos, S. andBoag, A. (2008). Total and ionised calcium concentrations in dogs with hypoadrenocorticism. Vet Rec.**163(1):**25–26.

Adler, J.A., Drobatz, K.J., Hess, R.S. (2007). Abnormalities of serum electrolyte concentrations in dogs with hypoadrenocorticism. J VetIntern Med.**21:**1168–1173.

Herrtage, M.E. (2000). Hypoadrenocorticism. In: Ettinger SJ, ed. Textbook of Veterinary Internal Medicine, 5th edn. Philadelphia:Saunders, , pp. 1612–1622.

Lathan, P., Moore, G.E., Zambon, S., Scott-Moncrieff, J.C. (2008). Use of a low-dose ACTH stimulation test for diagnosis of hypoadrenocorticismin dogs. J VetIntern Med.**22:**1070–1073.

Lifton, S.L., King, L.G., Zerbe, C.A. (1998). Glucocorticoid deficient hypoadrenocorticism in dogs: 18 cases. J Am Vet Med Assoc.**209:**2076–2081.

Melian, C., Peterson, M.E. (1996). Diagnosis and treatment of naturally occurring hypoadrenocorticism in 42 dogs. JSAP**37:**268–275.

Peterson, M.E., Kintzer, P.P., Kass, P.H. (1996).Pretreatment clinical and laboratory findings in dogs with hypoadrenocorticism: 225 cases. J Am Vet Med Assoc.**208(1):**85–91.

Schaer, M., Chen, C.L. (1983). A clinical survey of 48 dogs with adrenocortical hypofunction. JAAHA.**19:**443–452.

Thompson, A.L., Scott-Moncrieff, J.C., Anderson, J.D. (2007). Comparison of classic hypoadrenocorticism with glucocorticoid-deficienthypoadrenocorticism in dogs: 46 cases. J Am Vet Med Assoc.**230:**1190–1194.

Willard, M.D., Schall, W.D., McCaw, D.E., Nachreiner, R.F. (2010). Canine hypoadrenocorticism: Report of 37 cases and review of 39 previouslyreported cases. J Am Vet Med Assoc.**180:**59–62.

Burkitt, J.M. et al. (2007). Relative adrenal insufficiency in dogs with sepsis. J Vet Intern Med.**21**:226.

Lennon, E.M. et al. (2007). Use of basal serum or plasma cortisol concentrationsto rule out a diagnosis of hypoadrenocorticism in dogs: 123cases. J Am Vet Med Assoc.**231:**413.

Wenger, M. et al. (2010). Ultrasonographic evaluation of adrenal glands indogs with primary hypoadrenocorticism or mimicking diseases. Vet Rec.**167:**207.