

Introduction to Canine Hypoadrenocorticism

Pathophysiology

Spontaneous hypoadrenocorticism (*Addison's disease*) is caused by failure of adrenal gland secretion of glucocorticoids and mineralocorticoids. The underlying pathology is usually immune mediated destruction of the adrenal cortices. Spontaneous central (*pituitary mediated*) hypoadrenocorticism occurs much less commonly and results from inadequate pituitary ACTH secretion. "Central hypoadrenocorticism" is most commonly iatrogenic.

Signalment and Clinical Signs

Various breeds are predisposed including standard poodles, West Highland white terriers, Great Danes and bearded collies. Young-middle aged dogs (*males and females*) are typically affected (*average age 4 years*). The history is frequently vague, with intermittent clinical signs and variable response to symptomatic therapies. Anorexia, vomiting, diarrhoea, weakness, exercise intolerance, polydipsia and polyuria are usual. Gastrointestinal haemorrhage is not uncommon. Clinical signs may be precipitated by a stressful event such as kennelling, veterinary attention or domestic changes.

Diagnostic Tests

Hyperkalaemia, hyponatraemia, hypochloraemia, hypercalcaemia, azotemia, metabolic acidosis, hypoglycaemia, lymphocytosis and eosinophilia are typical. A mild normocytic normochromic anaemia may occur and there may be evidence of anaemia secondary to GI blood loss. A useful clue can be the presence of an inappropriately "*normal*" or increased lymphocyte count in a clinically ill dog due to reduced circulating glucocorticoids. Radiographic findings relate to hypovolaemia. Urinalysis generally demonstrates inappropriately dilute urine. This can lead to a misdiagnosis of primary renal dysfunction. ECG abnormalities generally reflect the electrolyte disturbances. Confirmation requires an ACTH stimulation test which demonstrates inadequate adrenocortical reserve capacity. Aldosterone measurement can be used to retrospectively confirm Addison's disease in dogs that have already been started on therapy with glucocorticoids. Endogenous circulating ACTH concentrations are increased in primary Addison's disease but not so in central Hypoadrenocorticism.

Treatment

Treatment of the acute hypoadrenal crisis is a true medical emergency. Therapy should be primarily directed at restoring circulating blood volume, correcting electrolyte and acid-base status, and glucocorticoid supplementation. Life-threatening hyperkalaemia can be treated with iv glucose solution, sodium bicarbonate administration, soluble insulin therapy or calcium gluconate administration. These therapies must be very carefully controlled: doses and protocols are reported elsewhere. Please contact the lab for specific advice if required. Chronic therapy of the stable Addisonian dog consists of permanent mineralocorticoid therapy (*fludrocortisone acetate, starting dose 0.015mg/kg/day*). Dogs may or may not also require concurrent glucocorticoid therapy. Initially dogs should be medicated with 0.5 mg/kg prednisolone, on a tapering regime. In some cases glucocorticoid therapy can be stopped, but others require a low dose (approx 0.2 mg/kg/day) for life.