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# ADDISONIAN CRISIS: A PRACTICAL APPROACH

Addison's disease is often missed at first, and subsequently becomes an urgent Addisonian crisis. This lecture has 2 goals: Firstly preventing patients to be missed earlier in the course of the disease (Refresh the symptoms associated with Addison's; How to screen for Addison's; How to achieve a diagnosis). The second part focuses on the treatment of the Addisonian crisis: What, when, why, how? What about the timing of glucocorticoid and mineralocorticoid administration?

Addison's disease is a life-threatening endocrine disorder. Lack of gluco- and/(or) mineralocorticoids leads to systemic signs secondary to hypovolemia, poor tissue perfusion, electrolyte and acid-base disturbances. Unfortunately, most Addisonian patients presented to an emergency department have a medical history compatible with Addison's disease. Therefore it seems the 'emergency' could have been avoided. The most common clinical signs of Addison's are GI-signs such as anorexia, vomiting and occasional diarrhoea, and polyuria/polydipsia. Other more vague symptoms (regurgitation secondary to a megaoesophagus; collapse due to hypoglycaemia) are reported. In fact, few clinical signs have not been linked with Addison's disease.

Any dog with recurrent GI-disease without an obvious cause that clinically looks 'poor', deserves a further work-up such as abdominal imaging and a minimal database. A rule out for Addison's disease can definitely be considered in this scenario. Leukopenia and an inversed stress-leukogram, can be further hints to consider Addison's in this scenario, and demand a rule out for sure.

Similarly, every dehydrated dog with PU/PD, 'suspected' of chronic renal disease, but responding 'amazingly' to fluid therapy, is suspected of Addison's disease. If the same patient represents few days later with a relapse and history repeats itself, Addison's should be on top of your list (perhaps together with hypercalcemia).

Every time Addison's disease is on your list of differentials (believe me, it often will be there!), check whether sodium and potassium were measured and look for evidence of hyponatremia and hyperkalaemia. Look for signs of an inversed stress leukogram and look for the presence of low glucose and calcium levels. As not all patients with an inversed stress leukogram, hyponatremia and hyperkalaemia, hypocalcaemia and hypoglycaemia have Addison's disease, and inversely not all patients with a normal blood work don't… these findings do not allow to rule out or diagnose Addison's disease… Therefore, after having considered the signalment, history, clinical examination and minimal database, you will either consider Addison's to be unlikely (yet not excluded), or highly suspected (yet not diagnosed).

In order to rule out Addison's, basal cortisol levels must be assessed. Illness causes basal cortisol levels to rise, almost always above 2µg/dL, allowing you to rule out Addison's disease. If the basal cortisol concentration however is below 2µg/dL, you did NOT rule out Addison's, and the patient suddenly is strongly suspected off Addison's disease… To definitively diagnose Addison's disease, the gold standard remains the ACTH-stimulation test. A dose of 5µg/kg IV one hour before the second sample suffices to provoke a maximal stimulation of the adrenal glands. A post-ACTH cortisol <2µg/dL is diagnostic for Addison's disease.

Addison patients mostly succomb to cardiac arrest due to hyperkalaemia, due to misdiagnosis and unjustified euthanasia for chronic renal disease or GI-ulceration and only rarely due to metabolic acidosis and multisystemic organ failure or aspiration pneumonia. First aim at treating hyperkalaemia. Fluid therapy will prevent potassium levels to rise, but in sevrely hyperkalemic patients (>8mmol/L) more direct action is required. An easy first option is the administration of glucose boluses, possible associated with short acting insulin mixtures to promote the shift of potassium across the cell membrane into the cell. In bradycardic patients,or very severe hyperkalemia (>10mmol/L), calciumgluconate is concurrently administered to reset the electrical threshold of the cardiomyocytes. This is a 'patch-method', and does not alter potassium concentrations, so all other treatments should be diligently performed and monitored to avoid tragedies later on. Therefore, electrolytes and pH (severe acidosis may require sodium bicarbonate administration before potassium concentrations decrease) should be regularly reassessed. As soon as the patient is up, eating and drinking, mineralocorticoids can be administered and their effect should be monitored regularly over the first year of treatment, and annually thereafter.

## ENDOCRINOLOGY

Administer higher dosages of glucocorticoids during hospitalization and taper at home. During stress moments (recheck visits, New Year's Eve), higher glucocorticoid dosages may be warranted. The prognosis for Addisonian patients is excellent. There are hardly any more exciting endocrine patients, yet the follow-up is much easier than for instance the diabetic patient. At rechecks, always screen for signs of hypercortisolism, good appetite and drinking behaviour, perform a routine urinalysis and check the electrolytes. Patients may gain a lot of weight after diagnosis and treatment, which may require dosage adjustments. In dogs that at first only have a glucocorticoid deficiency, electrolytes must be monitored regularly as Addison's disease typically is caused by an immune mediated destruction of the adrenal glands, and therefore mineralocorticoid deficiencies can still develop (but usually do so within the first six months after diagnosis).