

Emergency therapy for dogs with an acute hypoadrenocortical (*Addisonian*) crisis

Overview:

Hypoadrenocorticism (*Addison's disease*) results from deficient production of glucocorticoids and/or mineralocorticoids from the adrenal glands. This is most commonly the result of immune-mediated destruction of the glands (*idiopathic*) but can also occur as a result of adrenal necrosis/haemorrhage/neoplasia or following treatment with mitotane. Atypical hypoadrenocorticism is occasionally reported and involves deficiency of glucocorticoids only.

The adrenal glands produce glucocorticoids and aldosterone. Aldosterone is a mineralocorticoid, which is produced by the zona glomerulosa of the adrenal cortex and promotes sodium, chloride and water resorption from the renal tubules. Deficiency leads to hypotension, reduced glomerular filtration rate and cardiac arrhythmias. Glucocorticoids are produced by the zona fasciculata and reticularis and promote gluconeogenesis. Deficiency leads to an inability to deal with stress, anorexia, vomiting and abdominal pain.

Hypoadrenocorticism tends to occur more commonly in young to middle-aged female dogs. Standard Poodles and Bearded Collies are over-represented.

Clinical signs:

- Collapse
- Shock
- Relative bradycardia
- Abdominal pain
- Melaena
- Vomiting

Common ClinPath Findings:

- Hyponatraemia and hypochloraemia
- Hyperkalaemia
- Azotaemia
- +/- hypercalcaemia

A diagnosis of hypoadrenocorticism should always be confirmed using an ACTH stimulation test! This should be performed prior to administration of steroids.

Treatment Objectives:

- [1.] Restore circulating blood volume
- [2.] Control hyperkalaemia and cardiac arrhythmias
- [3.] Provide glucocorticoids
- [4.] Provide mineralocorticoids

1. Restore circulating volume - Rapid infusion rates of 0.9% sodium chloride (40-60ml/kg/hr) required. Aim to rehydrate the animal over 2-4 hours. Fluids should then be continued at lower infusion rates until biochemical parameters have normalised.

2. Control hyperkalaemia - Fluid administration described above will help reduce potassium levels by encouraging renal excretion, however if hyperkalaemia is life threatening then additional measures will be required (see table below). Calcium gluconate antagonises the myocardial effects of hyperkalaemia, but its actions are short-lived and it does not lower serum potassium levels. Glucose solutions stimulate insulin release and result in movement of potassium intracellularly. Sodium bicarbonate also lowers potassium levels by moving K ions into cells, but can result in alkalaemia so should be used with care. Fluid therapy and glucose administration is often all that is required to lower serum potassium levels.

Treatment of Life-Threatening Hyperkalaemia:

- 10% calcium gluconate as a slow intravenous bolus (0.5-1ml/kg) over 20 minutes whilst monitoring ECG.
 - 50% glucose (1-2ml/kg) intravenously.
 - Sodium bicarbonate (1-2mmol/kg) intravenously over 5-15 minutes.
 - Regular insulin (0.25U/kg) intravenously in conjunction with dextrose (2g per unit of insulin).
 - 0-100mls of 50% dextrose should be added to each 1litre of maintenance fluids to prevent hypoglycaemia.
- 3. Provide glucocorticoids** - Dexamethasone 0.5-2mg/kg is administered intravenously. Once the animal is stable and eating, oral prednisolone can be used (0.2-0.5mg/kg/day) and the dose is gradually tapered.
- 4. Provide mineralocorticoids** - Mineralocorticoid supplementation is not essential in the management of an acute crisis. Hydrocortisone sodium phosphate (Efcortisol) has some mineralocorticoid activity and can be administered intravenously (0.625mg/kg/hr) to enhance renal potassium excretion. Once the animal is eating oral fludrocortisone therapy should be initiated.

Monitoring:

- Check hydration status every 1-4 hours and adjust fluid rates accordingly
- Check serum potassium every 4-6 hours until stable
- Check urea and creatinine every 24 hours