ADDISON'S DISEASE

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In the last issue I attempted to explain the problems associated with a condition called Cushing's Disease which results from an over active adrenal gland. In this issue I shall attempt to explain the problems associated with an under active adrenal gland, known as Addison's Disease.

Addison's Disease is not as common as Cushing's Disease but probably does occur more commonly than is recognised for reasons which will become apparent later.

Addison's Disease (or hypoadrenocorticism if you must!) is caused by the destruction of those cells of the adrenal gland which produce substances called mineralocorticoid and glucocorticoid. The commonest cause for this is thought to arise by damage caused by the dog's own immune system which destroys these important cells for reasons which need not concern us here. Once about 95% of the cells have been destroyed, clinical disease will manifest. Another cause can be the destruction by the excessive use of a drug called Mitotane which is used for the treatment of Cushing's Disease and this is the reason why Cushing's Disease must be so carefully monitored as described in the last issue.

Further potential causes can be destructive lesions such as benign tumours in those parts of the brain which influence adrenal gland function via a chemical called Adrenocorticotrophic Hormone or ACTH for short. The condition can also arise after the cessation of prolonged glucocorticoid (steroid) therapy during which time ACTH producing cells have been suppressed and become lazy and atrophied. These forms of the disease are known as secondary hypoadrenocorticism as it is not failure of the adrenal gland itself which is the cause of the problem.

Loss of mineralocorticoids and glucocorticoids leads to an inability to conserve sodium and water and a failure to excrete potassium. The deficiency of sodium will lead to symptoms of dehydration such as lethargy; depression and nausea, low blood pressure and decreased blood flow through the heart and kidneys. The excess of potassium will lead to muscle weakness and worsening of the performance of the heart. Decreased tolerance of stress, loss of appetite and anaemia can result from glucocorticoid deficiency.

Some breeds seem to be more commonly affected that others, particularly Standard Poodles, Bearded Collies and Leonbergers that would seem to suggest a hereditary factor. In contrast to Cushing's Disease it is mainly young and middle aged dogs which are more commonly affected with approximately 70% of cases being female.

In the acute form of the disease, animals are often found in shock and collapsed or collapse when stressed. Other signs can include a weak pulse, slow heartbeat, abdominal pain, vomiting, diarrhoea, dehydration and hypothermia. The condition can deteriorate rapidly and become life threatening.

In the chronic form of the disease the signs are less dramatic and can be vague and non-specific with episodes of lethargy and depression, weakness and collapse, loss of appetite and vomiting. Episodes of illness can be interspersed with periods of apparent good health. Sometimes some of the symptoms of the acute form of the disease such as dehydration, slow heart beat and weak pulse may be seen. Occasionally bleeding into the bowel can cause chronic and profound anaemia.

As I hope you can appreciate, the symptoms of this disease are non specific and can easily be mistaken for other conditions which cause weight loss, weakness, loss of appetite, lethargy and vomiting and diarrhoea, and this is the reason Addison's Disease may be under diagnosed. Of course not all cases will display all of the symptoms and many different combinations of symptoms will be shown by different cases to further complicate the diagnosis. Many of the laboratory blood tests which can be routinely performed in practise nowadays will also mimic the characteristics of several other possible causes of the clinical symptoms. There is one test however which will almost always give a strong clue if the condition is Addison's Disease,

and that is the measurement of the sodium/potassium blood ratio which in 90% of cases of Addison's Disease will commonly be below the normal level. This is a very good indicator of the presence of Addison's and if detected further tests specific to adrenal gland function can be performed to confirm or deny the suspicion.

Treatment of the condition requires urgent correction of the dangerous dehydration which the mineral imbalance has brought about. Response to rehydration is often rewardingly dramatic. Glucocorticoid replacement is also vitally important and once the animal has been stabilised with intravenous fluids and glucocorticoids, replacement therapy with mineralocorticoid can be instigated. This mineralocorticoid replacement therapy will be ongoing throughout the animal's life and in many cases is the only treatment required but glucocorticoids may be necessary if the animal appears unwell or is in a stressful situation such as kennelling. Salt supplementation can be used initially to reverse the sodium deficiency but is not usually required long term although dogs needing high doses of mineralocorticoid may be able to lower this requirement with additional salt in their diets.

The prognosis for successful treatment is generally very good once diagnosis has been established and any acute phases managed promptly, but owner's of dogs with Addison's Disease should be aware of potential flare ups and destabilisation of the condition in times of stress.

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