CANINE ACROMEGALY CASE STUDY

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Vicki Brown details investigations of the symptoms of a suspected case of canine acromegaly, with concurrent diabetes mellitus, along with subsequent results and treatment.

AN eight-year-old entire female Border collie cross presented with polydipsia, polyuria, polyphagia, exercise intolerance, excessive panting and inspiratory stridor over the preceding few months.

Her last season was approximately five months ago; they were regular at every six months.

Clinical examination

The bitch (Figure 1) was overweight at 17kg, the heart rate was 150bpm and there was enlargement of the gingivae and widening of the interdental spaces, especially between the incisors. She looked and felt potbellied; her temperature and respiration rate were unremarkable.

Investigation

Full biochemistry and haematology were measured (Table 1).

Haematology revealed a mild haemoconcentration and eosinophilia. Biochemistry revealed a mild hepatopathy, hyperglycaemia, and pre-renal azotaemia.

Urinalysis of a voided sample showed a glycosuria and a specific gravity of 1.070 (concentrated),
and other urine parameters were normal. Radiographs of the head, thorax and abdomen were performed under general anaesthesia, and showed increased bone growth, cardiomegaly, hepatomegaly and renomegaly.

These results raised the suspicion of acromegaly, with concurrent diabetes mellitus.

**Diagnosis**

Growth hormone (GH) elevation – characteristic of acromegaly – is difficult to assess, as GH is labile and has to be sent for analysis on ice by guaranteed next-day delivery, and there are few laboratories offering a commercial assay in Europe.

Demonstration of elevated serum plasma insulin growth factor (IGF-1) is a good indicator of elevated GH levels. It is a simple test to perform, as only one sample is required and IGF-1 is stable in the post. This test was performed on the patient.

Plasma IGF-I concentrations in normal dogs are a function of body size. The IGF-1 level in the patient was 735ng/ml – approximately seven times the mean level expected in an average, normal Border collie.

A diagnosis was made of acromegaly, with concurrent diabetes mellitus, caused presumptively by the antagonism of insulin by elevated GH levels.

**Treatment**

Ovariohysterectomy was performed in order to remove endogenous progestogens causing insulin antagonism and inducing GH excess.

A spot blood glucose test, taken three days postoperatively, showed that the blood glucose levels had already begun to fall.

This confirmed the suspicion that the diabetes mellitus was secondary to the elevated GH, causing insulin antagonism. Glucose levels continued to fall and, within two weeks, blood glucose levels were within normal limits (Table 2).

**Discussion**

GH is produced by the anterior lobe of the pituitary gland. It directly influences protein, carbohydrate and lipid metabolism, and controls the rate of skeletal and visceral growth in growing puppies. The hormone acts on tissues via peripheral factors.
Chronic hypersecretion of GH in the adult dog results in acromegaly, an insidious condition associated with connective tissue and bone overgrowth. In dogs, acromegaly is caused by exogenous progestogen therapy (for example, proligestone, megestrol acetate or medroxyprogesterone acetate), commonly for suppression of oestrus; or by endogenous progesterone produced during the metoestrous phase of the oestrus cycle. The progesterone-induced GH excess originates from hyperplastic ductular epithelium in the mammary gland, not from the pituitary gland. In cats, acromegaly is caused by a pituitary tumour that secretes excess GH; this cause is uncommon in dogs.

CT/MRI scanning may be helpful in demonstrating a pituitary mass, and failure to respond to progesterone removal (either by progestogen therapy withdrawal or by ovariohysterectomy) would certainly arouse suspicion of a pituitary tumour.

Clinical signs of acromegaly develop slowly in middle-aged to older intact bitches that are either cycling normally or being given regular treatment with progestogens to prevent oestrus.

Initially, there is increased soft tissue swelling around the head and neck, and this may result in excessive panting, inspiratory stridor and exercise intolerance.

In this patient’s case, the coat was thick, making it easy to miss the soft-tissue swelling; however, it could be appreciated on deep palpation. Excess skin folding around the distal extremities may also be seen, and increased interdental spacing, especially of the incisors, is a hallmark of the condition.

Some bitches may collapse during exertion due to thickening of the oropharyngeal tissues, and may lose consciousness. This may be the only presenting complaint, leading to a false suspicion of epilepsy.

Since GH antagonises the effects of insulin, mild glucose intolerance – or, as in this case, overt diabetes mellitus – will develop, causing polyuria and polydipsia.

Diabetes mellitus induced by GH may be permanent or reversible, depending on the circulating concentration and duration of the excessive GH secretion. Initially, hyperglycaemia is associated with increased insulin concentrations, but, as the disease progresses, beta-cell exhaustion may result in lowered insulin secretion.

Most cases of acromegaly present with insulin-resistant diabetes mellitus, and patients require abnormally high doses of exogenous insulin to control blood-glucose concentrations. Fortunately, in this case, the diagnosis of acromegaly was made at the same time as that of diabetes.

As an aside: researching this article, I read that, in one study, one-in-three cats with uncontrolled diabetes mellitus were suffering from concurrent acromegaly; pituitary irradiation is the treatment of
choice.

Successful treatment of acromegaly in the bitch involves the withdrawal of progestogen therapy and ovariohysterectomy for oestrus control.

Insulin requirements may decrease dramatically – or even cease – following reduction in progesterone levels, with a concurrent reduction in GH levels.

Therefore, if patients are on insulin therapy, they should be monitored closely to avoid insulin over-dosage and the development of hypoglycaemia.

References