Canine hyperadrenocorticism – diagnosis

Dr Christine Griebsch DipECVIM-CA, University Veterinary Teaching Hospital Sydney, Australia, discusses diagnosis of canine hyperadrenocorticism in part one of this two-part series

Hyperadrenocorticism (HAC) is a common endocrine disease in older dogs caused by an increased production of cortisol by the adrenal cortex. Pituitary dependent hyperadrenocorticism (PDH) is the most common form of naturally occurring HAC, followed by HAC due to a functional adrenal tumour (FAT). Diagnosis is established based on the presence of typical clinical signs, clinicopathological abnormalities and supportive endocrine screening tests. Differentiation between PDH and FAT is based on results of endocrine differentiation tests and imaging. Because of the increased awareness of HAC by veterinarians, HAC is often investigated and detected at earlier stages when clinical signs are mild. In these cases, test results can often be equivocal and a diagnosis might not be straightforward.

AETIOLOGY AND PATHOGENESIS

ADRENAL ANATOMY AND PHYSIOLOGY

The adrenal glands are located craniomedial to the kidneys. They consist of the medulla and the cortex. The medulla produces catecholamines (epinephrine, norepinephrine). The cortex consists of three layers (from outside to inside): zona glomerulosa, which produces mineralocorticoids (aldosterone), and zona fasciculata and zona reticularis, which produce cortisol and androgens.

Under physiological conditions, corticotropin-releasing hormone (CRH) is released from the hypothalamus. CRH stimulates release of adrenocorticotropic hormone (ACTH) from the anterior lobe of the pituitary gland. ACTH stimulates steroid hormone production (mainly cortisol) in the adrenal glands. An increase in cortisol concentration decreases ACTH and CRH secretion through a negative feedback mechanism (Figure 1).

PATHOGENESIS OF HYPERADRENOCORTICISM

Between 80-85% of dogs have pituitary dependent (PDH) and 15-20% adrenal dependent HAC due to a FAT. PDH is more common in smaller breed dogs, whereas half of the dogs with FAT weigh >20kg. It seems that females might be more often affected than males. There is no breed predisposition. Iatrogenic HAC is caused by exogenous administration of glucocorticoids.

PDH is caused by excessive secretion of ACTH by a pituitary tumour. This causes excessive cortisol secretion in both adrenal glands and subsequently bilateral symmetric adrenal gland enlargement (Figure 2). In some cases, nodular hyperplasia can develop, which might be difficult to distinguish from adrenocortical tumours. Most pituitary tumours are adenomas, carcinomas are rare. The true prevalence of macroadenomas (>1cm diameter) versus

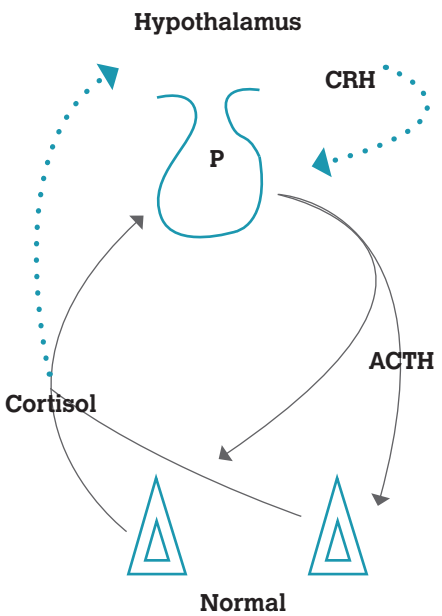

Figure 1: Physiologic hypothalamic pituitary adrenal axis (HPPA).

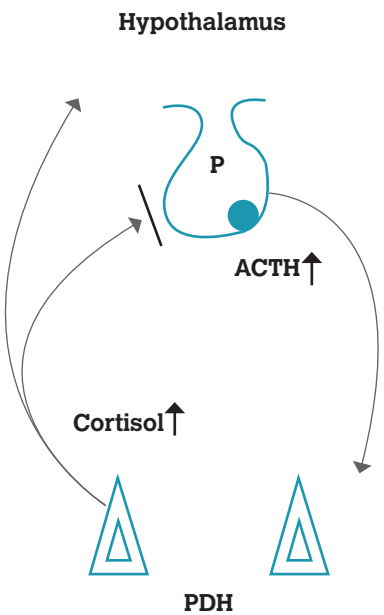

Figure 2: Simplified scheme of the HPPA in dogs with pituitary dependent HAC (PDH).

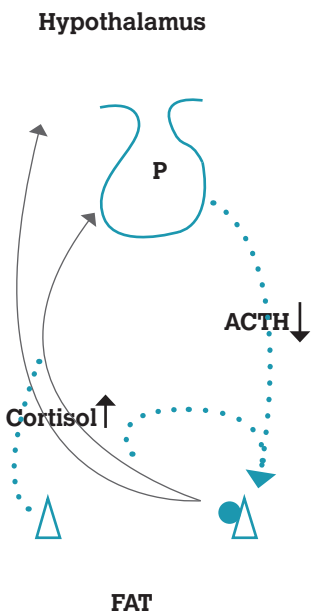

Figure 3: Simplified scheme of the HPPA in dogs with HAC due to a functional adrenal tumour (FAT).

Legend: ACTH = adrenocorticotropic hormone; CRH = corticotropin releasing hormone; P= pituitary gland.

Figure 4: Muscle wasting, distended abdomen and diffuse alopecia in a16-year-old male neutered Maltese with PDH.

microadenomas at time of diagnosis is unknown.
HAC due to FAT is caused by adrenocortical adenomas and adenocarcinomas that secrete excessive amounts of cortisol autonomously, and independently of pituitary control. Hypercortisolaemia suppresses hypothalamic CRH and circulating plasma ACTH concentrations. The result of this chronic negative feedback is cortical atrophy of the unaffected adrenal gland and of non-neoplastic cells in the affected adrenal gland (Figure 3). It is difficult to distinguish between adenomas and carcinomas even on histopathology. Features of malignancy are adrenal tumors >2cm and vascular invasion. Calcification is not a feature of malignancy. Malignancy is confirmed if distant metastasis appears similar on histopathology.
Other causes of HAC are less common. In dogs with concurrent PDH and FAT, an adrenal mass and hyperplasia of the contralateral adrenal gland can be seen. The concurrent presence of PDH and/or FAT and pheochromocytoma is also possible.
Ectopic ACTH secretion has been described as a cause of HAC in human medicine. There has been one case report of a dog with ectopic secretion of ACTH from an abdominal neuroendocrine tumour. In this case, endogenous (eACTH) concentration was increased, bilateral adrenal gland hyperplasia was present and hypophysectomy did not control clinical signs of HAC. The dog responded well to trilostane treatment.[1] ACTH independent food-dependent HAC was described in another dog and was thought to be caused by enhanced adrenal responsiveness to gastric inhibitory polypeptide (GIP). In this case, eACTH concentration was low in the presence of bilateral adrenal gland hyperplasia, there was, at least, a doubling in the urine cortisol to creatinine ratio (UCCR) in response to food and this increase in cortisol could be prevented by administration of octreotide.[2]
'Atypical' or 'occult' hyperadrencorticism is defined as a syndrome in which a dog appears to have HAC based on history, physical examination and clinicopathological findings but the low-dose dexamethasone suppression test (LDDST), urine cortisol: creatinine ratio (UCCR) and ACTH stimulation test (ACTH-ST) fall into currently accepted reference ranges. The proposed pathomechanism is secretion of cortisol precursors and sex hormones instead of cortisol.[3] The true existence of atypical hyperadrencorticism is widely debated.[4]

DIAGNOSIS

Establishing a diagnosis of HAC is centred around a high index of suspicion based on history, the presence of typical clinical signs and clinicopathological changes in conjunction with endocrine testing supporting the diagnosis. It should be emphasised however that due to increased awareness of HAC, dogs commonly undergo investigation of HAC earlier in the course of disease and changes maybe more subtle.

HISTORY

Important information to obtain includes administration of any medication that can cause clinical signs and clinicopathological changes consistent with HAC (such as phenobarbital, exogenous or topical glucocorticoids), information on water intake and urination, information on appetite, ability to jump, breathing pattern and lethargy.

CLINICAL SIGNS

Typical clinical signs of HAC include polyuria (PU), polydipsia (PD), polyphagia, panting, abdominal distension/pot-bellied appearance, hepatomegaly, muscle weakness and skin changes (alopecia, hyperpigmentation, thin skin, comedones, poor hair regrowth, pyoderma). Lethargy, weight gain, urinary incontinence/urine leakage, testicular atrophy, persistent anoestrus and unilateral or bilateral facial nerve palsy are less common clinical signs.
Hypertension has been described in 50-80% of dogs with HAC.[5] Uncommon complications of HAC include insulin-resistant diabetes mellitus, thromboembolism, ligament rupture due to ligament laxity, pseudomyotonia and calcinosis cutis. A cause and effect relationship between HAC and gall bladder muoceles has been proposed[6] but has yet to be definitively documented.[3] Similarly, a causal relationship between sudden acquired retinal degeneration syndrome (SARDS), which causes acute blindness and HAC, remains controversial.[7]
Hypercoagulability can cause thromboembolism and acute respiratory distress in case of pulmonary thromboembolism. Diabetes mellitus can develop in 5-10% of dogs with HAC and is often insulin resistant. Calcinosis cutis is an uncommon but characteristic dermatologic condition in dogs with HAC. Myotonia is a myopathy characterised by persistent active muscle contraction. Myotonia typically affects the pelvic limbs and causes a stiff gait.
Central nervous system (CNS) symptoms such as inappetence, anorexia, stupor, circling, aimless wandering, pacing, seizures, behaviour changes and depression can occur secondary to a large space-occupying pituitary tumour (pituitary macrotumor syndrome). Adrenocortical carcinomas might invade blood vessels and can cause a tumour thrombus causing hindlimb paresis, retroperitoneal or intra-abdominal haemorrhage and abdominal pain.

CLINICOPATHOLOGICAL FINDINGS

Typical findings on haematology are a stress leukogram (neutrophilia, monocytosis, lymphopaenia, eosinopenia), thrombocytosis and a mild erythrocytosis. On biochemistry the most pathognomonic finding is an increase of the alkaline phosphatase (ALP) activity (often >1000U/l) due to increase of the steroid isoenzyme. The alanine aminotransferase (ALT) activity can be mild to moderately increased (often <400U/l). A mild fasting hyperglycaemia is common. A mild to moderate increase in cholesterol and triglycerides is present in about 50% of affected dogs. About 30% of affected dogs can have a mild increase in bile acids and a positive bile acid stimulation test. The BUN concentration is decreased in half of affected dogs due to increased diuresis.

Most dogs will have a decreased urine specific gravity (USG) <1015. About 50% of dogs with HAC will have a urinary tract infection (UTI) on initial presentation. Proposed pathomechanisms include immunosuppression, decreased bactericidal properties of dilute urine and urinary retention due to PU. Most UTIs are asymptomatic. Recurrent UTIs could potentially cause pyelonephritis. Proteinuria is present in more than half of affected dogs.[5] A proposed pathomechanism is development of glomerulosclerosis. The UPC is typically <6. Proteinuria does not always resolve with treatment of HAC. Glucosuria might be present in dogs with concurrent diabetes mellitus. Euthyroid sick syndrome with total T4 concentration below the reference range is present in half of affected dogs. Thyroid stimulating hormone (TSH) concentration is typically decreased or normal in these patients. Increased canine pancreatic lipase (cPLI) results should be interpreted with caution in dogs with HAC as they have higher cPLI concentrations than healthy dogs.[8]

SCREENING TESTS

The positive predictive value (PPV) and negative predictive value (NPV) – true positive and true negative results – of a test depends on the prevalence of disease in the tested population. Therefore, screening tests should only be performed if there is a high index of suspicion for HAC. In a dog with high index of suspicion, a single negative screening test does not exclude HAC and an alternative test should be performed. If all tests are negative an alternative diagnosis should be considered. Alternatively, hormone testing can be repeated three to six months later if clinical signs progress. All screening tests can be influenced by nonadrenal illness and stress. If possible, hormone testing should therefore be delayed in a dog with concurrent illness until this has resolved. Testing should also be delayed in dogs that have been treated with oral or topical glucocorticoids as these can suppress the hypothalamic-pituitary-adrenal-axis (HPAA). The duration of suppression of the HPAA is unknown however and depends on the duration of use, dose, administration route, form of steroid (long- or short-acting) and individual sensitivity.[3]

It should be emphasised that there are different specialist opinions about the best test to use in dogs with suspected HAC. Furthermore, reference ranges and cut-off values might differ between different laboratories hence are not provided here.

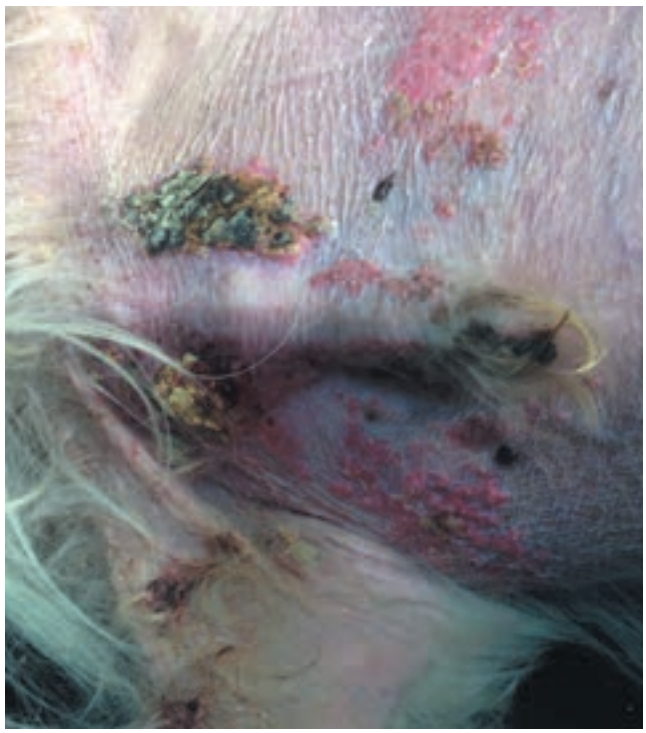

Figure 5: Thin skin, prominent subcutaneous blood vessels, calcinosis cutis and "rat tail" in a 16-year-old male neutered Maltese with PDH.

URINE CORTISOL: CREATININE RATIO

The UCCR offers good sensitivity (75-100%) but poor specificity (24-77%). It is, therefore, a good test to rule out HAC. A negative UCCR excludes HAC. A positive UCCR can be a consequence of stress or nonadrenal illness. Therefore, it is important to collect urine at home in a non-stressful environment. Urine collection should also be delayed for at least two days after a visit to a veterinarian. UCCR results are not affected by collection time of day, however morning urine might be preferred as it represents several hours of urine production.

ACTH STIMULATION TEST

The ACTH stimulation test (ACTH-ST) has an 80-95% sensitivity and 80-90% specificity to diagnose PDH. If there is a high index of suspicion, a positive ACTH-ST is diagnostic for HAC. A negative ACTH-ST however does not exclude the presence of HAC. In dogs with FAT, sensitivity is 57-63%, hence only half of dogs with FAT will have a positive ACTH-ST. Glucocorticoids, progestagens and ketoconazole suppress the HPAA (Figure 1) and decrease response to ACTH. Fasting is not recommended unless lipaemia affects the cortisol assay used. The ACTH-ST is performed by administration of 250ug/dog or 5ug/kg of synthetic ACTH IV or IM.[9] Blood is collected before (basal) and 60 minutes after administration of ACTH. Synthetic ACTH can be stored in plastic syringes in the freezer at -20C for up to six months and hence one vial can be used for multiple patients.[10] A recent study suggests that low-dose synthetic ACTH (1ug/

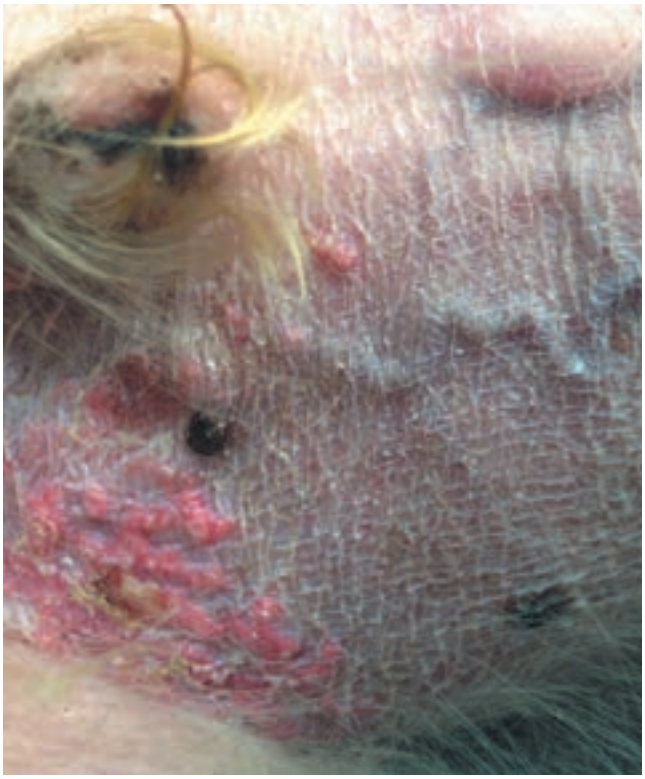

Figure 6: Simplified scheme of the HPPA in dogs with HAC due to a functional adrenal tumor (FAT).

kg IV) can be used to effectively monitor treatment of PDH with trilostane or mitotane, however is not recommended for diagnosing HAC.[11] Similarly, accidental perivascular, instead of IV injection of ACTH, does not affect results.[12]
A depot ACTH preparation can be used at a dose of 5ug/kg IM; however, should not be given intravenously. Blood samples for determination of cortisol should be taken immediately before and three hours after depot ACTH administration to diagnose HAC.[13]
The ACTH-ST can also be used to diagnose 'atypical' HAC by measuring cortisol precursors. Particularly measurement of 17-hydroxyprogesterone (17-OH) has been proposed.[14] 'False' positive or negative results are possible.

LOW-DOSE DEXAMETHASONE SUPPRESSION TEST

The LDDST has a high sensitivity (85-100%) but low specificity (44-73%). A negative LDDST almost certainly excludes HAC. A positive LDDST does not confirm the diagnosis. A LDDST can be false positive in dogs with nonadrenal illness or due to stress. Stress should therefore be avoided while performing the LDDST. In very stressed dogs this test is of little use. Feeding should be avoided during the test, fasting for the test is not necessary unless lipaemia influences the cortisol assay used.[3] The author uses this test in dogs with a low index of suspicion for HAC to exclude the disease. The test is performed measuring cortisol before and four and eight hours after administration of dexamethasone 0.01 mg/kg IV. The eight-hour cortisol concentration is used to diagnose HAC and the test is positive if there is a lack of suppression (cortisol above the laboratory cut-off or >50% of the basal cortisol concentration). In a recent study, lack of suppression had a positive and negative predictive values (PPV) of 94% for diagnosis of HAC whereas partial suppression (<50% suppression) had a PPV of 70%.[15] In one study, a LDDST with 'inverse' results was described. In this study, 5/80 dogs with HAC had a complete suppression at eight hours but no suppression at four hours post dexamethasone administration. All of these dogs were diagnosed with PDH later.[16] An 'inverse' result had a PPV of 37% in a recent study.[15]
Clinical signs and biochemical abnormalities in dogs treated with phenobarbital may be similar to those in dogs with HAC. Occasionally phenobarbital treated dogs may not show suppression on LDDST. Therefore, the LDDST might be difficult to interpret in dogs receiving phenobarbital treatment.[3]
An extended LDDST with additional blood collection after 10 and 12 hours could not differentiate between healthy dogs and dogs with suspected atypical HAC.[17]

ORAL LDDST AND UCCR

An oral LDDST has been described. After collection of two morning urine samples at 8am on two consecutive days at home, an oral dose of 0.01mg/kg dexamethasone is administered after obtaining the second urine sample. The bladder is emptied at 12pm. A third urine sample is collected at 4pm, eight hours after administration of dexamethasone. A decrease in the third UCCR >50% of the mean of the basal values excludes HAC. Dogs with HAC should not have suppression of the UCCR.[18]

DIFFERENTIATION BETWEEN PDH AND HAC DUE TO FAT

Differentiation tests should only be performed once a diagnosis of HAC has been established.

ENDOGENOUS ACTH CONCENTRATION

Measurement of endogenous ACTH concentration (eACTH) has a very high sensitivity (100%) and specificity (95-100%) to differentiate between PDH and FAT when measured with a two-site solid-phase chemiluminescent immunometric assay. eACTH is normal to increased in dogs with PDH and undetectable in dogs with FAT.[19] Samples for eACTH have to be collected into chilled, silicon-coated glass or plastic tubes containing EDTA, centrifuged within 15 minutes, the plasma transferred into plastic tubes and frozen immediately.

LDDST AND HDDST

In dogs with FAT cortisol secretion cannot be suppressed by dexamethasone administration. If suppression occurs the dog likely has PDH.
Using results of LDDST about 50% of dogs with PDH will have suppression of the four-hour cortisol concentration below the laboratory cut-off or <50% of the basal cortisol concentration. The high dose dexamethasone suppression test (HDDST) is performed similarly to the LDDST, however, the dose of dexamethasone is 0.1mg/kg IV. PDH is present

if there is suppression of the four- or eight-hour cortisol concentration below the laboratory cut-off or <50% of the basal cortisol concentration. At least 75% of dogs with PDH meet at least one criterion for suppression on either the LDDST or HDDST. If no suppression is present, differentiation between PDH and HAC due to FAT is not possible.

ORAL HDDST AND UCCR

An oral HDDST has been described. After collection of a morning urine sample on two consecutive days at home, three oral doses of 0.1mg/kg dexamethasone are administered at six- to eight-hour intervals. A third urine sample is collected the next morning. A decrease in the third UCCR <50% of the mean of the basal values is consistent with PDH. Lack of suppression does not confirm FAT. In one study the UCCR suppressed in 72% of dogs with PDH.[20]

IMAGING

RADIOGRAPHS

Abdominal ultrasound is a better imaging modality for further workup of HAC than radiographs. Common changes on radiographs are abdominal distension, good contrast because of abdominal fat deposition, hepatomegaly, and mineralisation of bronchi, pulmonary interstitium, dermal tissue and subcutaneous tissue (calcinosis cutis). Calcium-containing uroliths are occasionally identified. An adrenal tumour may be visualised because of its mass effect or tumoral calcification. Possible metastases of a malignant FAT might be visible on chest radiographs.[3]

ABDOMINAL ULTRASOUND

The size of the adrenal gland is assessed by measuring the width of the caudal pole of the adrenal gland. A limit of 7.4mm has long been used as the upper limit of normal. Newer studies suggest, however, that the reference range threshold varies by breed and body size and these must be considered when interpreting adrenal gland measurements.[21]

Dogs with PDH typically have bilaterally enlarged adrenal glands whereas dogs with FAT have one adrenal mass and atrophy of the contralateral adrenal gland (adrenal width <4-5mm). Abdominal ultrasound can however not differentiate between a FAT, pheochromocytoma, aldosteronoma or nonfunctional adrenal mass. The presence of bilateral adrenal tumours is possible. Distinguishing macronodular hyperplasia from FAT can be difficult with ultrasonography. An adrenal gland width of >4cm, vascular or soft tissue invasion and metastasis are signs of malignancy. Finding normal adrenal glands does not rule out HAC. Normal size adrenal glands can be seen in 25% of dogs with PDH.[22] Abdominal ultrasound is also useful to identify sequelae from HAC such as urinary calculi or gallbladder mucocele.

CT/MRI

A CT scan of abdomen and chest is indicated if surgical removal of an adrenal mass is planned. It can provide more information about features of malignancy such as vascular invasion or metastasis.

In dogs with PDH, imaging of the pituitary gland with MRI or CT gives valuable information especially if radiation therapy or hypophysectomy are considered. Just over half (56%) of dogs with PDH have a normal-appearing pituitary gland using CT.[23] MRI is more sensitive to identify pituitary microadenomas. Not all dogs with macroadenoma (>1cm diameter) show neurological signs. A pituitary tumour and FAT can occur simultaneously. Therefore, the author recommends including CT of the pituitary gland when abdominal and chest CT are performed before adrenalectomy in a dog with FAT.

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READER QUESTIONS AND ANSWERS

1: WHICH IS THE MOST COMMON CAUSE OF NATURALLY OCCURRING HYPERADRENOCORTICISM (HAC) IN DOGS?

A Functional adrenocortical tumour
B Functional tumour of the adrenal medulla
C Pituitary tumour
D Food-dependent HAC
E Ectopic ACTH secretion

2: WHICH OF THE FOLLOWING CLINICAL SIGNS IS NOT A COMMON CLINICAL SIGN OF HYPERADRENOCORTICISM?

A PU/PD
B Weight loss
C Polyphagia
D Panting
E Muscle weakness

3: COMMON ABNORMALITIES ON BIOCHEMISTRY REPORTED IN PATIENTS WITH HYPERADRENOCORTICISM INCLUDE (ONE CORRECT ANSWER):

A Hyperkalaemia, hyponatraemia, azotaemia
B Increased ALP activity, increased ALT activity (ALP>ALT), mild hyperglycaemia, hyperlipidaemia
C Hyperbilirubinaemia, hypercholesterolaemia, increased ALP activity
D Hypoglycaemia, hypoalbuminaemia, low BUN concentration
E Increased ALP activity, increased ALT activity (ALP<ALT), hyperbilirubinaemia

4 WHICH OF THE FOLLOWING STATEMENTS IS CORRECT

A The UCCR is a good test to confirm a diagnosis of HAC
B Measurement of endogenous ACTH is helpful as a screening test for HAC
C The ACTH-ST will always be positive in dogs with FAT
D A negative LDDST result excludes the presence of HAC
E Suppression of the four-hour cortisol concentration on LDDST is consistent with the presence of FAT

5: WHICH OF THE FOLLOWING PROTOCOLS CAN NOT BE USED TO PERFORM AN ACTH-ST AS A SCREENING TEST FOR HAC?

A Synthetic ACTH 250ug/dog IV with blood collection after 60 minutes
B Synthetic ACTH 5ug/kg IM with blood collection after 60 minutes
C Depot ACTH 5ug/kg IV with blood collection after 60 minutes
D Synthetic ACTH 5ug/kg IV with blood collection after 60 minutes
E Depot ACTH 5ug/kg IM with blood collection after three hours

ANSWERS: C, B, D, C