Adrenal gland tumours. Different clinical presentations in three dogs: a case report

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ABSTRACT: Three dogs were evaluated due to the presence of unilateral adrenal gland masses with or without clinical signs. Case 1 showed a unilateral non-functional adrenocortical adenoma, discovered accidentally while Case 2 presented a unilateral cortisol-secreting adrenocortical adenoma; a pheochromocytoma was accidentally discovered in Case 3. The adrenalectomy was the treatment of choice in all cases. The development of diagnostic imaging techniques, mainly ultrasonography, and its application to routine abdominal examinations, have allowed the detection of adrenal gland masses more frequently. However, there is no pattern of echogenicity or architecture which would help in the differentiation in a functional tumour from a non-functional tumour, a pheochromocytoma, a metastatic lesion to the adrenal or a granuloma. A complete description of history, clinical signs, laboratory analysis and imaging studies is included. Moreover, a revision of the different types of adrenal gland tumours, with their clinical presentation, a standardised diagnosis protocol and options for treatment are discussed.

Keywords: incidentaloma; adrenal gland tumour; pheochromocytoma; hyperadrenocorticism; dog

CBC = cell blood count, ACTH = adrenocorticotropic hormone, 17-OH prog = 17-hydroxiprogesterone, LDDT = low dose dexamethasone suppression test

Primary adrenal gland tumours have been reported to account for 1–2% of all canine neoplasias (Bailey and Page 2007). Adenoma and adenocarcinoma are the most common, followed by pheochromocytoma, myelolipoma, aldosteronoma, deoxycorticosterone-secreting and sex hormone-secreting adrenal tumours (Reine et al. 1999; Lurye and Behrend 2001; Rijnberk et al. 2001; Syme et al. 2001; Tursi et al. 2005; Frankot et al. 2012).

The development of diagnostic imaging techniques has allowed the detection of adrenal gland masses more frequently. However, in order to establish the correct diagnosis and appropriate treatment, it is recommended to follow a structured protocol.

The following report describes three cases of dogs with different types of unilateral adrenal gland masses and the appropriated structured diagnosis protocol.

Case description

Case 1

A 12-year-old, castrated male (30 kg), Golden retriever was referred to the Endocrinology Service of the Veterinary Clinical Hospital at the University of Zaragoza (Spain), with a 6-week history of weight lost and lethargy. Clinically, the dog exhibited symptoms of polyphagia and reduced activity but polyuria-polydipsia were absent. On initial evaluation, the dog lied down all the time. Physical examination revealed a body condition score of 2/5 and hypertension (191 mmHg), while the rest of the exploration was normal.

A blood sample from the jugular vein was taken for routine cell blood count (CBC) and serum biochemical analyses and results were within reference limits. Leishmaniosis analysis was negative and the

proteinogram was normal. Moreover, basal serum total and free thyroxine (T_4) and serum cTSH concentrations were within reference limits. A urinary sample was obtained by cistocentesis and the results were normal.

Due to the fact that all previous analyses returned normal results, we decided to carry out a cardiac and abdominal ultrasonographic examination. The echocardiography did not reveal any pathology. However, a unilateral adrenal gland mass was identified with a size of the left-side of 1.67 cm which was causing pressure on the caudal vena cava. The right adrenal gland was echographically normal. The thoracic radiograph ruled out the presence of pulmonary metastasis.

An adrenocorticotropic hormone (ACTH) stimulation test was performed and cortisol and 17-hydroxiprogesterone (17-OH prog) were measured before and 90 minutes after administration of ACTH (Nuvacthén Depot®, Sigma-Tau, Spain) (0.25 mg, *i.m.*). Basal cortisol and post-ACTH cortisol concentrations were within reference intervals as were pre- and post-17-OH prog and basal ACTH concentrations. A low dose dexamethasone suppression test (LDDT) was proposed in order to complete the diagnosis but the owner declined further diagnostic tests.

Based on non-clinical signs compatible with hyperadrenocorticism, analytical results and abdominal ultrasonography, a diagnosis of unilateral non-functional adrenal gland tumour was made.

After discussing treatment options with the owner, adrenalectomy of the left-adrenal was performed. No intraoperative complications developed and postoperative recovery was completely normal. Histopathology diagnosed the adrenal tumour as an adrenocortical adenoma. At a 12-month follow-up examination, no problems had developed.

Case 2

An 11-year old intact female Poodle weighing 4.5 kg was brought to the Endocrinology Service due to non-pruritic bilateral symmetrical hair loss that developed over the trunk, caudal thighs and perineum, over the period of one year. On physical examination, no abnormalities were observed apart from the skin lesions.

Complete blood count and serum biochemical analysis were performed and while haematological parameters were within reference limits biochemical abnormalities included hypercholesterolaemia (9.72 mmol/l; reference range: 3.62–6.21 mmol/l), hypertriglyceridaemia (1.14 mmol/l; reference range: 0.56–1.12 mmol/l), increased sodium concentrations (158 mmol/l; reference range: 141–155 mmol/l) and hyperproteinaemia (76 g/l; reference range: 57–75 g/l). Serum cTHS, basal serum total and free T_4 concentrations were within reference ranges. The ACTH stimulation test was performed using 0.125 mg of synthetic ACTH. Blood for serum cortisol and 17-OH prog values was collected before and 90 min after ACTH administration. The basal cortisol concentration was normal (69 nmol/l, reference range: 29.8–215.2 nmol/l) and post-ACTH cortisol was within the normal reference range (186 nmol/l; reference range: 215 to 469 nmol/l) which suggested iatrogenic Cushing (reference range: < 215 nmol/l). Serum 17-OH prog concentrations pre- and post-stimulation were 8.18 nmol/l (reference range: < 1–1.9 nmol/l) and 8.81 nmol/l (reference range: 1–5.5 nmol/l) respectively. A low dose dexamethasone suppression test (Dexamethasone, Cortexona 2 mg/ml, Laboratorios Syva SA, Spain 0.01 mg/kg, *i.v.*) revealed no post- dexamethasone cortisol depression at four hours (89.4 nmol/l; reference range: < 41.1 nmol/l) and at eight hours (108 nmol/l; reference range: < 41.1 nmol/l). Moreover, a basal ACTH concentration of under 10 pg/ml (reference range: 20–50 pg/ml), suggested the presence of a functional adrenal gland tumour. The diagnosis was confirmed by performing an abdominal ultrasound which revealed an unilateral enlargement of the right adrenal gland (1.40 cm). The left adrenal gland was small (0.25 cm).

A unilateral secreting tumour of the adrenal cortex was diagnosed based on the dermatological clinical signs, abdominal ultrasonography images, low serum ACTH concentration and lack of suppression of serum cortisol concentration after LDDT.

The owner decided to treat surgically and a right adrenal gland adrenalectomy was performed without complications. The histological diagnosis was an adrenocortical adenoma.

After 48 h and since the clinical evolution of the patient was good, the patient was discharged from the hospital. Home treatment consisted of dexamethasone (0.06 mg/kg, *v.o.*, q 12 h). After four days, both cortisol concentrations pre- and post-ACTH stimulation were under 27.6 nmol/l, suggesting Addison disease. Also the 17-OH

prog was evaluated pre- (< 0.06 nmol/l; reference range: < 1–1.9 nmol/l) and post-ACTH stimulation (0.27 nmol/l; reference range: 1–5.5 nmol/l). Biochemical analysis showed sodium, potassium and chlorine concentrations within reference limits and arterial pressure was 110 mmHg.

Two weeks after the surgery, a new ACTH stimulation test and electrolyte analyses were performed, showing similar results to the previous ones. The abdominal ultrasonography showed a peritoneal reaction around the right kidney and the left adrenal gland could not be detected. Four weeks after the adrenalectomy, the bitch was hospitalised with haemorrhagic vomiting and weakness. Twenty-four hours later the dog developed a cerebral oedema and died.

On postmortem examination, fatty liver, intestinal haemorrhage and subacute renal infarcts over more of 50% of the renal area were observed. Histologically, a diagnosis of fatty liver degeneration with liver necrosis with diffused light hepatitis and renal infarcts due to thrombosis of arcuata arteries was established.

Case 3

A 45 kg 7-year-old intact cross-breed female was referred to the hospital for a pregnancy diagnosis. The bitch ran away during its oestral phase and the owner was suspicious that it was pregnant. Clinically, the dog was panting and neither polyuria-polydipsia nor polyphagia had been observed. Over the preceding three years, the dog experienced spring allergy episodes characterised by intensive itching which had been controlled with corticosteroids. No steroid treatment had been administered for the past six months. On physical examination, the dog presented a body condition of 3/5 and systemic hypertension (195 mmHg) determined by three repeated oscillometric measurements. The abdominal ultrasound examination confirmed a ten day old gestation as well as an encapsulated mass (1.5 cm) associated with the left adrenal gland. Neither renal invasion nor visceral metastases were identified and the right adrenal gland was normal. The owner decided to terminate the pregnancy, and to this end aglepristone (Alizin 30 mg/ml, Virbac, Spain) (10 mg/kg, *s.c.* q 24 h, two doses) was administered.

After discovering the adrenal mass accidentally, we recommended a CBC, serum biochemistry analysis, ACTH stimulation test and serum basal ACTH concentration analysis. The haematological results were normal while biochemical abnormalities included increased serum cholesterol concentration (6.47 mmol/l; reference range: 3.62–6.21 mmol/l), increased triglyceride concentration (1.27 mmol/l; reference range: 0.56–1.12 mmol/l) and increased serum total protein concentration (76 g/l; reference range: 57–75 g/l). The results of the ACTH stimulation test showed serum cortisol and 17-OH prog pre- and post-ACTH within serum reference ranges. The serum basal ACTH concentration was under 10 pg/ml (reference range: 20–50 pg/ml). The diagnoses were completed by performing a LDDT. Serum cortisol pre- and post-LDDT were also within normal reference limits. No neoplastic masses were seen on the thoracic radiograph examination.

A diagnosis of a left adrenal mass without invasion of the caudal vena cava was made. Because the discovery of the adrenal mass was accidental and no clinical signs were observed, the owner declined to treat it surgically and decided for periodic control using ultrasonography and the ACTH stimulation test.

Six months later, the dog´s only clinical sign was panting. Systemic arterial pressure continued to be very high (196 mmHg) and the ultrasonography exam revealed a great increase in the left adrenal mass (2.5 cm). No obvious thrombus invasion of the caudal vena cava was detected. Cell blood count abnormalities included leucopoenia (4.8×10^9 cells/l; reference range: 6×10^9–17.3×10^9 cells/l). The cortisol pre-ACTH stimulation test was normal and cortisol post-ACTH was 149 nmol/l (reference range: < 215 nmol/l iatrogenic Cushing). Both 17-OH prog pre- and post-ACTH stimulation were within reference limits. The serum ACTH basal concentration was similar to those previously analysed (< 10 pg/ml; reference range: 20–50 pg/ml).

Although no clinical signs were observed, as there was a significant increase in the left adrenal gland mass and because the dog was in good health, surgery was performed. The adrenalectomy of the left adrenal gland was successful and no post-surgical complications appeared. The histopathological report corroborated the diagnosis of the adrenal gland mass as a pheochromocytoma.

Six weeks after surgery, the dog was evaluated physically; arterial pressure was measured (160 mmHg) and CBC and a ACTH suppression test were performed. Panting had practically disappeared and the dog was more active. Leucopoenia

persisted (5×10^9 cells/l; reference range: 6×10^9 to 17.3×10^9 cells/l), cortisol pre-ACTH stimulation test was within the reference limits and cortisol post-ACTH was low (185 nmol/l; reference range: < 215 nmol/l iatrogenic Cushing). Abdominal ultrasound revealed normal peritoneal reaction post-surgery and the size of the right adrenal gland was normal.

Six months after adrenalectomy no clinical signs were observed, physical examination was normal, systemic arterial tension had been normalised (130 mmHg) and CBC, basal serum ACTH concentration and ACTH stimulation test were performed. Leucopoenia was slightly improved (5.1×10^9 cells/l; reference range: 6×10^9–17.3×10^9 cells/l), basal serum ACTH concentration was high (77.5 pg/ml; reference range: 20–50 pg/ml) and cortisol pre- and post-ACTH stimulation test were within normal reference limits. The control ultrasound showed an increase in size of the right adrenal gland (1.04 cm).

A further check-up nine months post-surgery showed normal blood pressure values (120 mmHg), slight leucopoenia (5.5×10^9 cells/l; reference range: 6×10^9–17.3×10^9 cells/l), normal serum ACTH concentration (24.8 pg/ml; reference range: 20–50 pg/ml) and normal serum cortisol pre-ACTH (61.5 nmol/l; reference range: 29.8–215.2 nmol/l) and post-ACTH stimulation test (345 nmol/l; reference range: 215–469 nmol/l). The ultrasound exam did not show any abnormality. Panting was not observed, the dog was active and no clinical signs compatible with adrenal disease were observed.

DISCUSSION AND CONCLUSIONS

Abdominal ultrasonography has become the test of choice for determining the origin of hyperadrenocorticism in dogs. Moreover, adrenal tumours can frequently be detected, and bilateral adrenal enlargement can be found in dogs with pituitary-dependent hyperadrenocorticism. Furthermore, abdominal ultrasonography may enable the detection of liver metastasis or invasion of the caudal vena cava in adrenal carcinomas (Hoffmann 2003; Peterson 2007; Guillaumont et al. 2012).

In many cases, as documented also here in Cases 1 and 3, the discovery of the adrenal gland mass is accidental and no clinical signs compatibles with adrenal pathology are found. Those adrenal masses discovered accidentally during diagnostic imaging performed for indications other than adrenal diseases are known as incidentalomas, most of them are endocrinologically silent (Singh and Buch 2008; Terzolo et al. 2009; Galac et al. 2010). In contrast, Case 2 showed dermatologic manifestations commonly observed in dogs with hyperadrenocorticism, and the adrenal gland mass was found during routine diagnoses. The majority of canine adrenal gland tumours are cortisol-secreting and sex hormone-secreting (Syme et al. 2001; Galac et al 2010), as was the case here.

At any rate, after discovering an adrenal gland mass it is necessary to investigate its characteristics (non tumoural, benign or malignant tumour), its origin (medullar, cortical or adrenal metastases), and if it is endocrinologically silent or hormonally active (secreting cortisol, aldosterone or 17-OH prog or others). Unfortunately, there is no pattern of echogenicity or architecture which would help to differentiate a functional adrenocortical tumour from a non-functional tumour, a pheochromocytoma, a metastatic lesion to the adrenal gland or a granuloma. Therefore, it is recommended to follow a structured protocol in order to establish a correct diagnosis and the appropriate treatment (Terzolo et al. 2009).

Usually, the initial assessment includes a complete history and clinical examination of the patient focusing on features of the adrenal hyperfunction. Moreover, a CBC, serum biochemical analysis including electrolytes, urinalysis and blood pressure measurement are essential for establishing a differential diagnosis. The evaluation of functional status and risk of malignancy should be assessed by means of adrenal function tests and imaging procedures.

Adrenal cortical tumours can be functional or endocrinologically silent. The most frequent adrenocortical tumours secrete cortisol resulting in the development of Cushing's syndrome. Others have the potential of secreting aldosterone, leading to Conn's syndrome, or other steroids or hormonal precursors like 17-OH prog. Adrenocortical tumours traditionally have been assumed to secrete a single hormone. However, different reports have found adrenal gland tumours secreting more than one hormone (Behrend et al. 2005; Machida et al. 2008; Frankot et al. 2012).

Cortisol-secreting adrenocortical tumours are responsible for around 15–20% of dogs with naturally occurring Cushing's syndrome, which in dogs takes place with a frequency of 1–2/1000 dogs/year (Willeberg and Priester 1982; Feldman and Nelson 2004). Most of them are unilateral solitary lesions

(90%) occurring in dogs of middle and old age without breed or sex predilection (Galac et al. 2010; Beuschlein et al. 2012). The three cases described here had a unilateral adrenal mass and their ages ranged between 7 and 12 years old. The typical clinical symptoms of a functional cortisol-secreting adrenal tumour are those of glucocorticoid excess, with polyuria, polydipsia, polyphagia, abdominal obesity, weight gain, fatigue, panting at rest, muscle atrophy and skin changes such as thin coat, alopecia or skin atrophy (Galac et al. 2010). The extent of clinical signs present can change depending on adrenal gland secretion of cortisol. Thus, some dogs with adrenal tumours show only moderate symptoms. In this case report, non-pruritic symmetrical alopecia was observed in Case 2, clinical signs in Case 1 were unspecific (lethargy, weight loss and hypertension), while panting was the only symptom found in Case 3.

To determine if an adrenal mass is functional, all dogs should be evaluated for their hormone hyperfunction. The ACTH stimulation test has been commonly used as a screening test for hyperadrenocorticism in dogs. An exaggerated response 90 min after ACTH administration is observed in dogs with hyperadrenocorticism. However, a false positive result can be found in dogs with chronic stress produced by a non-secreting adrenal mass. Moreover, the ACTH stimulation test has a low sensitivity in dogs with adrenal gland tumours because of the lack of ACTH receptors (Feldman and Nelson 2004). Cortisol pre- and post-ACTH stimulation were within normal limits in Case 1 and 3. The low-dose dexamethasone suppression test is considered by many to be the test of choice for the diagnosis of hyperadrenocorticism in dogs (Behrend et al. 2002). Hypersecretion of cortisol by adrenocortical tumours cannot be suppressed when administrating dexamethasone. Compared with the ACTH stimulation test, the LDDT is much more sensitive in confirming hyperadrenocorticism since the results are diagnostic in virtually all dogs with cortisol-secreting adrenal tumours and in 90–95% of dogs with pituitary-dependent hyperadrenocorticism (Feldman 1983). The results of this test in Case 2, with no cortisol suppression after four and eight hours, confirmed the diagnosis of an adrenal tumour, although no serum cortisol increase was observed after ACTH stimulation. However, normal serum cortisol suppression was observed in Case 3, ruling out the diagnosis of a cortisol-secreting adrenal tumour. The owner of Case 1 declined to carry out this test.

To complete the diagnosis, serum basal ACTH concentration is a reliable means of determining the cause of hyperadrenocorticism once the diagnosis has been confirmed. Endogenous ACTH concentrations are normal to high in dogs with pituitary-dependent hyperadrenocorticism whereas ACTH values are usually low or undetectable in dogs with adrenal tumours or with iatrogenic hyperadrenocorticism. The basal ACTH concentration of Case 2 was under 10 pg/ml (reference range: 20–50 pg/ml) which was suggestive of a glucocorticoid-secreting adrenal gland tumour. However, the hystopathological analysis of the adrenal gland in Case 3 resulted in the diagnosis of a pheochromocytoma, in spite of a ACTH concentration under 10 pg/ml. In contrast, Case 1 had a normal basal ACTH concentration.

It is important to remember that adrenal tumours may also secrete other adrenocortical hormones in excess like sex hormones (Syme et al. 2001; Hill et al. 2005) or aldosterone (Javadi et al. 2003; Behrend et al. 2005; Davies et al. 2008; Machida et al. 2008).

Some dogs with adrenal tumours and clinical signs of hyperadrenocorticism have normal ACTH stimulation and LDDT results. These results are compatible with atypical hyperadrenocorticism. In these animals, serum cortisol concentrations could be normal but an excessive sex hormone production like 17-OH prog, oestradiol or testosterone can be found (Syme et al. 2001; Hill et al. 2005). Because of this, 17-OH prog was evaluated before and after ACTH administration, being within normal limits in Case 1 and 3, but not in Case 2.

Primary aldosteronism is rare in dogs (Rijnberk et al. 2001; Feldman and Nelson 2004; Behrend et al. 2005; Johnson et al. 2006; Machida et al. 2008). The excessive activation of mineralocorticoid receptors leads to the hypersecretion of aldosterone by the adrenocortical tumour. Hyperaldosteronism includes signs of polyuria, polydipsia, nicturia and episodic weakness (Behrend et al. 2005; Rijnberk and Kooistra 2010; Frankot et al. 2012). The most consistent laboratory abnormality detected includes hypokalaemia. In some cases, there may be hypophosphataemia, hypomagnesaemia and increased serum alkaline phosphatase concentration. Systemic hypertension is usually observed. A diagnosis of hyperaldosteronism should ideally be made on the basis of high serum aldosterone concentrations in the presence of hypokalaemia and serum renin concentration within or under the reference range. However, due to laboratory limitations, there is a need to rule out all second-

ary causes of hyperaldosteronism, which include cardiovascular and kidney failure and severe generalised hepatocellular dysfunction (Behrend et al. 2005; Rijnberk and Kooistra 2010; Frankot et al. 2012). None of the dogs in this report showed hypokalaemia.

Pheochromocytomas are catecholamine-producing tumours of the adrenal medulla. Approximately 50% of pheochromocytomas are apparently inactive and discovered incidentally in dogs with no clinical signs. They are considered malignant tumours, whose potential consequences include invasion into local blood vessels, haemorrhage around the adrenal glands, pressure on surrounding abdominal structures and metastatic spread (Spall et al. 2011). The majority of clinical signs related to catecholamine excess are believed to occur as a consequence of systemic hypertension. However, depending on the pattern of catecholamine release, hypertension can be constant or paroxysmal. Case 3 was histopathologically diagnosed as a pheochromocytoma and showed hypertension and panting as the only clinical signs. The cell blood count and biochemical findings of this dog were non-specific. Hypercholesterolaemia can occur secondary to catecholamine-induced lipolysis and subsequent conversion of fatty acids in the liver (Gilson et al. 1994). Moreover, the clinical signs, weakness, episodic collapse, tachypnoea, panting, tachycardia, pacing, polyuria, polydipsia, hypertension, vomiting and diarrhoea, and diagnostic test interpretation are vague and non-specific, making diagnosis challenging (Kook et al. 2007, 2010; Quante et al. 2010). In humans, the diagnosis of a pheochromocytoma is based mainly on biochemical detection of excessive production of the secretory products of the tumours. Widely used tests in this regard, are aimed at measuring urinary and plasma catecholamine concentrations or their metabolites, metanephrine and normetanephrine. Recent studies have shown high urine normetadrenaline/creatinine ratio concentrations in dogs with pheochromocytoma compared with healthy dogs (Kook et al. 2010). Moreover, a marked increase in plasma normetadrenaline has been observed in a dog with pheochromocytoma (Gastelow and Syme 2010). Unfortunately, availability, technical difficulty and expense have limited the use of these measurements in veterinary medicine.

Therefore, definitive diagnosis of pheochromocytoma relies upon histopathology of adrenal tissue, often following adrenalectomy or post-mortem examination. Nowadays, surgery is the only definitive curative treatment for local pheochromocytoma in dogs. However, it is important to evaluate the history of the patient, because the perioperative mortality is high, ranging from 20–40% (Kyles et al. 2003). The owner of Case 3 decided for surgical removal of the adrenal gland because of the significant increase in adrenal gland size. After the surgery, clinical signs progressively disappeared and no pharmacological treatment was subsequently required.

The development of new medical treatments for canine pheochromocytoma has been a subject of investigation. The administration of ^{131}I metaiodobenzylguanidine to a dog with an inoperable pheochromocytoma resulted in clinically stable disease for four months (Bommarito et al. 2011).

Non-functioning adrenal incidentalomas need further evaluation to assess malignancy risk. As in Case 1, an adrenal gland mass was considered non-functional when no clinical signs were observed and adrenal hormone concentrations were in the reference range (Melian and Perez 2008). Diagnostic imaging is of great assistance in determining the choice of therapy and to evaluate the prognosis. Massari et al. (2011) found that dogs with an adrenal tumour with a major axis length ≥ 5 cm, metastasis or vein thrombosis have a worse prognosis. However, in the absence of metastasis or invasion of adjacent structures, differentiating between an adrenocortical adenoma and a carcinoma is often difficult (Reusch and Feldman 1991; Feldman 2000). As in human medicine, adrenal carcinomas are significantly larger than adenomas (Labelle et al. 2004; Lang et al. 2011). The difference in tumour size may serve as an indicator of malignancy. It appears that smaller adrenal tumours are less invasive into surrounding tissues; there is less intraoperative haemorrhage and fewer postoperative complications. Changes in calcification, necrosis and haemorrhage are rarely seen in benign adrenal tumours (Lang et al. 2011; Massari et al. 2011). The correct diagnosis of adrenal gland tumours therefore relies on integrating the clinical signs, laboratory analysis and abdominal ultrasonographic findings with the histological evaluation of the tumour (Labelle et al. 2004; Davis et al. 2012).

The management of adrenal masses may vary depending on the results of the aforesaid analytical and imaging tests. When diagnostic outcome has revealed no metastasis and it is likely that there is a adrenal gland tumour, the treatment of choice is

an adrenalectomy. However, it is necessary to assess the clinical history, physical exam, basic laboratory analysis, hormonal functional status and imaging studies as a whole before making a decision in each case (Kyles et al. 2003; Schwartz et al. 2008; Lang et al. 2011; Massari et al. 2011).

In this paper we reported on three dogs with unilateral adrenal gland masses. The clinical presentation of each case was different as was the etiology. For dogs with an adrenal gland mass discovered accidentally or during routine diagnoses of adrenal diseases, it is important to establish a structured protocol of diagnosis. The emphasis of the diagnostic tests should be on excluding autonomous hormone overproduction and assessing malignancy risk.

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Received: 2013–02–11
Accepted after corrections: 2013–07–30

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