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Feline Acromegaly: An Underdiagnosed Endocrinopathy?

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Background: Feline acromegaly has been reported infrequently in the veterinary literature and current knowledge of this endocrinopathy is based on limited numbers of animals with relatively advanced clinical signs.

Hypothesis: This study was undertaken to screen diabetic cats for the presence of acromegaly.

Animals: Diabetic cats with variable control examined by general practitioners in the United Kingdom.

Methods: Blood samples were screened for the possible presence of acromegaly with basal serum concentrations of insulin-like growth factor 1 (IGF-1) and, when available, feline growth hormone (fGH). In patients with markedly increased IGF-1 concentrations intracranial computed tomography (CT) was offered, and in selected cats additional imaging was performed.

Results: IGF-1 was determined in 184 variably controlled diabetic cats; 59 cats had markedly increased IGF-1 concentrations (>1,000 ng/mL; reference interval, 208–443 ng/mL). Eighteen cats subsequently were examined, and acromegaly was confirmed in 17 cats. Notable findings included absence of a detectable pituitary mass lesion in some affected cats regardless of whether CT or magnetic resonance imaging (MRI) was used. Hypertension was not found to be a complication in the evaluated cats and respiratory stridor was more prevalent than previously reported.

Conclusions and Clinical Importance: Measurement of IGF-1, growth hormone (GH), or both is useful in the diagnosis of acromegaly in cats.

Key words: Cat; Diabetes; Growth hormone; Pituitary adenoma; Somatotrophic adenoma.

Acromegaly in cats is thought to be rare, but its true prevalence is unknown and only 30 cases have been reported.[1,2] Acromegaly in cats is caused by a functional somatotrophic adenoma in the pars distalis of the anterior pituitary gland resulting in excessive growth hormone (GH) secretion.[3] Clinical signs are the result of the catabolic and diabetogenic effects of GH, the anabolic effects of IGF-1, and the space-occupying effect of the pituitary macro-adenoma. A GH-induced postreceptor defect in insulin action at the level of target tissues is thought to explain why most cats with acromegaly have concurrent diabetes mellitus.[3,4] High IGF-1 concentrations induce excessive soft tissue growth with bony remodelling and thickening. Cats in previous reports typically were middle-aged to older, castrated male mixed breed cats with insulin-resistant diabetes mellitus,[1,3,5–9] but additional cats must be evaluated to determine if this signalment is typical.[2]

Confirmation of acromegaly can be difficult because of the disorder's insidious onset, the cost of imaging procedures, and the lack of a readily available fGH assay.[2,3] Because of the limited availability of fGH assessment, IGF-1 has been used as an aid in the diagnosis of acromegaly.[6] However, increased IGF-1 concentrations have been reported not only in acromegalic cats but also in nonacromegalic insulin-resistant diabetic cats,[10,11] suggesting the possibility of false positive results. In addition, Norman and Mooney[6] found normal IGF-1 concentrations at initial presentation in 1 cat later diagnosed with acromegaly (although increased serum IGF-1 concentration eventually was documented on measurement).

Although the duration, amplitude, and frequency of the pulsatile release of GH are increased in human patients with acromegaly,[12] solitary serum GH measurements have proved unreliable in confirming the diagnosis.[13,14] Nevertheless, a high probability of acromegaly exists in humans in whom single GH concentrations are >10 ng/mL.[13] In acromegalic cats in which a single serum GH measurement was performed, all had increased serum fGH,[6,8,15–18] confirming the value of such an assay in the diagnostic process.

Although CT has proved useful in demonstrating a mass lesion in the region of the pituitary gland in suspected acromegalic cats,[17,19] the sensitivity of contrast-enhanced CT studies to detect pituitary abnormalities in acromegalic cats has not been fully assessed. For example, Peterson et al[5] reported 1 cat in which a pituitary mass was not evident on CT imaging.

The present study was undertaken to screen diabetic cats for the possible presence of acromegaly and further determine the likelihood of the disorder in suspect cases. In addition, the relative value of IGF-1, fGH, CT, and MRI in the diagnostic process was further defined.

Materials and Methods

Recruitment

Serum fructosamine measurements on diabetic cats (both insulin-sensitive and insulin-resistant) were offered to veterinary practices throughout the United Kingdom. Excess serum from submitted blood samples was used to determine basal serum IGF-1 concentrations. Basal serum GH concentrations were determined in selected cats, depending on assay availability, in both acromegalic and nonacromegalic diabetic cats.

All veterinarians were made aware of the IGF-1 results and in cats in which IGF-1 concentrations were >1,000 ng/mL, CT was

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offered to the cats' owners. If the CT proved unremarkable, MRI of the brain was performed. Postmortem examination permission was requested at time of death and postmortem examination was performed when granted.

In accordance with previous recommendations,[1,2] cats were considered acromegalic in this study if they had insulin-resistant diabetes mellitus, IGF-1 concentration exceeded 1,000 ng/mL, and evidence of a pituitary gland abnormality could be established on the basis of CT, MRI, or postmortem examination.

In order to assess the use of fGH estimation to differentiate between the acromegalic diabetic and nonacromegalic diabetic state, fGH concentrations (when available) were compared between confirmed acromegalic and unlikely acromegalic diabetic cats. Cats were deemed unlikely to be acromegalic if IGF-1 concentrations were <1,000 ng/mL. However, to avoid inclusion of acromegalic cats with borderline increases in IGF-1 concentrations, only fGH concentrations of diabetic cats with IGF-1 concentrations <800 ng/mL were used.

Laboratory Methods

All blood samples were collected via venipuncture into serum gel tubes after ≥6 hours of fasting and before insulin administration. Samples arrived at the laboratory within 24 hours of collection. Previous studies have indicated that normal collection and transport minimally affects fGH levels when blood is collected into serum gel tubes and samples are separated and frozen within 24 hours of collection.[20]

Fructosamine was measured by a colorimetric assay, based on the ability of ketoamines to reduce nitrotetrazolium blue to formazans in an alkaline medium,[a] and a reference interval was previously established by the Royal Veterinary College (RVC) laboratory (reference interval, 205–322 μM).

Total serum IGF-1 was measured by a commercially available radioimmunoassay system (RIA)[b] developed for humans and previously validated for the cat.[10,21,c] Values >1,000 ng/mL were considered consistent with acromegaly in accordance with the laboratory's guidelines. Basal fGH was measured by an ovine GH RIA system previously validated for use in the cat.[20]

Imaging

Eighteen cats were presented for additional investigation. All 18 cats underwent complete physical examination, and blood was collected for routine CBC and biochemistry. Systolic blood pressure was determined indirectly (Doppler) in 14 cats and urinalysis was performed in 8. Echocardiographic examinations were performed on all cats with audible cardiac abnormalities on physical examination.

Intracranial imaging was performed under sedation with ketamine[d] (5 mg/kg) and midazolam[e] (0.25 mg/kg) IV or propofol[f] (4–5 mg/kg) IV with oxygen supplementation for CT examination or general anesthesia (propofol induction and isoflurane[g] maintenance) for MR imaging. The CT equipment available was a single slice spiral CT.[h]

If no pituitary abnormality was seen on plain CT images, 1 mL/kg of iodinated contrast agent (iopromide[i]) was administered IV and the study was repeated. Contrast also was administered when an abnormality was seen and sedation allowed additional postcontrast imaging. Precontrast and postcontrast (gadopentetate dimeglumine[j]) imaging was performed in all cats undergoing MR imaging. All images were evaluated by a board-certified radiologist (PM).

Assessment for Hypertension

Doppler systolic blood pressure measurement was performed in 14 cats and was considered normal if <160 mmHg. Measurements were repeated until 3 subsequent readings were obtained with <10 mmHg variation. Ocular fundus examination also was performed in all cats.

Other Diagnostic Tests

In cats in which acromegaly was thought to be the most likely diagnosis (13 of 18), pituitary-adrenal-axis testing was not performed. A corticotrophin (ACTH) stimulation test was performed in the remaining cats and urine cortisol : creatinine ratio was calculated if additional assessment was considered necessary. For similar reasons, serum total thyroxine (TT4) concentration only was measured in selected cats.

Serum cortisol and TT4 concentrations were measured by RIA methods validated for use in cats. For ACTH stimulation testing, blood for serum cortisol determination was collected before and 1 and 2 hours after the IM or IV injection of 0.125 mg of tetracosactide hexaacetate.[k]

Urinalysis with or without urine culture was performed in selected cats at the discretion of the attending clinician.

Statistical Analysis

Depending on the presence or absence of normal distribution, fructosamine and GH concentrations were compared by Student's *t*-test or Mann-Whitney *U*-test, respectively, and results were considered significant at $P < .05$. Commercial statistical software was used.[l]

Results

IGF-1 was determined in 184 variably controlled diabetic cats (mean fructosamine concentration ± SD, 486 ± 157 μM; range, 156–795 μM). Fifty-nine cats had markedly increased IGF-1 concentrations (>1,000 ng/mL; reference interval, 208–443 ng/mL). Mean fructosamine concentrations were significantly higher in diabetic cats with IGF-1 concentrations >1,000 ng/mL than in those with IGF-1 concentrations <1,000 ng/mL (mean ± SD, 525 ± 142 μM versus 459 ± 159 μM, $P < .001$). Eighteen of the 59 cats with markedly increased IGF-1 concentrations were then examined and acromegaly was confirmed in 17.

Signalment and History

Of the 59 cats with IGF-1 concentrations >1,000 ng/mL, 52 were domestic short hair cats, 3 domestic long hair, and 4 of unknown breed. Forty-seven were neutered males; 6, neutered females; 5, intact males; and 1 of unknown sex. Their age ranged from 6 to 17 years (median, 11 years); body weight ranged from 3.5 to 9.2 kg (median, 5.8 kg), and median insulin dosage was 7 U q12h (range, 1–35 U q12h).

Fifteen of the 17 later confirmed acromegalic cats were neutered males and 2 were neutered females. All were domestic short hair cats, and their body weight ranged from 4.0 to 7.9 kg (mean ± SD, 5.9 ± 1.1 kg). The cats ranged in age from 6 to 15 years (mean ± SD, 10.1 ± 2.5 years).

Duration of the clinical signs for the 17 cats ranged from 2 to 42 months (mean ± SD, 11.2 ± 11.4). The earliest clinical signs included polyuria and polydipsia (n = 17), polyphagia (n = 16), and weight gain (n = 10). Some cats then developed lameness (n = 5) and

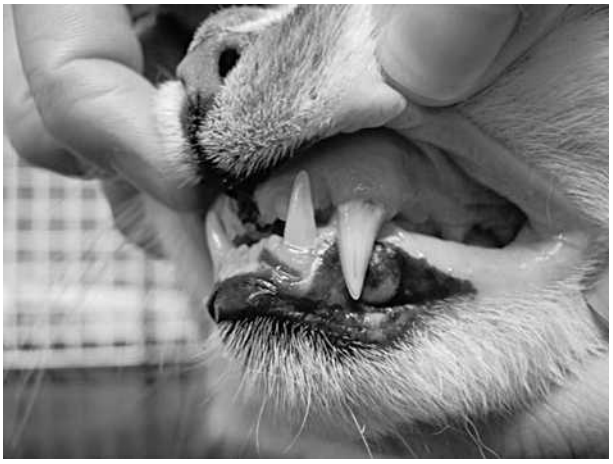

Fig 1. Prognatia inferior in an acromegalic cat.

Fig 2. Clubbed appearance of the front paw of a confirmed acromegalic cat.

eventually suspected or confirmed neurologic signs (n = 2: lethargy; n = 1: impaired vision, circling to the right, vocalizing; n = 1: confusion, right-sided Horner's syndrome) and presumed peripheral diabetic neuropathy (n = 1: plantigrade stance in hind limbs). Owners had noted increased size of the paws in 2 cats, broader facial features in 1 cat, and increased abdominal size in 1 cat. All cats were being treated with insulin and all experienced difficulties in glycemic control, necessitating administration of increasing amounts of insulin. The median insulin dosage of the 17 acromegalic cats was 10 U q12hr (range, 2–35 U q12h) compared with 3 U q12h for cats with IGF-1 <1,000 ng/mL.

Physical Examination in Confirmed Acromegalic Cats

On physical examination, there was abdominal organomegaly (liver and kidneys) (n = 15), prognathia inferior with increased distance between upper and lower canine teeth (n = 8; Fig 1), immature bilateral cataracts (n = 2), clubbed paws (n = 3; Fig 2), broad facial features (n = 14; Fig 3), systolic cardiac murmur (n = 4), gallop rhythm (n = 1), respiratory stridor (n = 9), multiple limb lameness (n = 5), impaired vision, dilated pupils, reduced pupillary light reflex, reduced menace response, decreased facial sensation on the left and decreased postural reactions on the left, circling to the right, vocalizing (n = 1), decreased menace response bilaterally, confusion, right-sided Horner's syndrome (n = 1), and plantigrade stance of the hindlimbs (n = 1). One cat experienced periods of open-mouth breathing and tachypnea when stressed.

Laboratory Results

IGF-1 concentrations in the 17 confirmed acromegalic cats ranged from 1105 to >2,000 ng/mL. Table 1 presents fructosamine and GH concentrations determined in the confirmed acromegalic and 34 presumed nonacromegalic diabetic cats with IGF-1 concentrations <800 ng/mL. GH concentrations in the latter group were significantly lower than those in 9 of the 17 confirmed acromegalic cats ($P < .001$). Eight of the 34 nonacromegalic diabetic cats had fGH concentrations that exceeded the upper limit of the reference interval. Only 2 of these 8 had fGH concentrations that exceeded 10 ng/mL.

Hematologic abnormalities of the 17 confirmed acromegalic cats included increased red blood cell count (n = 1), eosinophilia (n = 1), decreased mean corpuscular volume (n = 2), decreased mean corpuscular hemoglobin concentration (n = 3), increased hematocrit (n = 1), decreased hematocrit (n = 1), decreased hemoglobin concentration (n = 1), increased hemoglobin concentration (n = 1), lymphopenia (n = 3), and monocytosis (n = 1).

In the confirmed acromegaly group, serum biochemistry abnormalities included hyperglycemia (n = 17; range, 12.2–37.3 mM; mean ± SD, 24.2 ± 6.6 mM), increased total protein concentration (n = 8; 82–93 g/L; reference interval, 54–82 g/L), decreased sodium concentration (n = 4), decreased chloride concentration (n = 2), increased potassium concentration (n = 1), increased total bilirubin concentration (n = 1), increased amylase activity (n = 4), increased lipase activity (n =

Fig 3. Broadening of facial features in a confirmed acromegalic cat as compared with photographs taken 5 years previously.

Table 1. Endocrinologic data of confirmed acromegalic and presumed nonacromegalic diabetic cats.

	Fructosamine (μM) (median and range)	fGH (ng/mL) (median and range)
Diabetics with IGF-1 <800	439; 219–790 (n = 34)	4.05; 2.22–16.7 (n = 34)
Confirmed acromegalics	553; 440–733 (n = 17)	16.1; 9.0–33.7 (n = 9)

fGH, feline growth hormone; IGF-1, insulin-like growth factor 1; n, number of samples.

1), mildly increased blood urea nitrogen (BUN) concentration without concurrent increase in creatinine concentration (n = 2), increased globulin concentration (n = 1), increased cholesterol concentration (n = 1), increased alkaline phosphatase (ALP) activity (n = 2), and increased alanine amino transferase (ALT) activity (n = 3).

Urinalysis was performed in 8 cats. Glycosuria was present in all 8 cats, and proteinuria in 2, bilirubinuria in 1 and traces of blood in another. Ketonuria was not detected. Urine cultures performed in 5 cats yielded a growth of *Escherichia coli* in 1 cat and was negative in the remaining cats.

ACTH stimulation test results were within normal limits in all 5 cats evaluated. The urine cortisol : creatinine ratio in 1 of these 5 cats also was normal. Serum TT4 concentrations were low in 5 cats.

Echocardiography and Electrocardiography

Electrocardiography was normal in all 17 confirmed acromegalic cats, whereas echocardiography in 5 cats was variable including no structural abnormalities, an enlarged left atrium, right atrial enlargement, generalized thickened interventricular septum, focal hypertrophy of the basal area of the interventricular septum, relaxation abnormalities on Doppler tissue imaging and mitral inflow assessment, systolic anterior motion of the mitral valve (SAM), mild mitral valve insufficiency and mild increase in left ventricular outflow tract velocity at higher heart rates, mild increase in left ventricular outflow tract velocities without SAM, mild left ventricular free wall hypertrophy with thinning toward the apex, poor left atrial wall motion, spontaneous echo contrast, and a restrictive pattern of pulmonary venous flow.

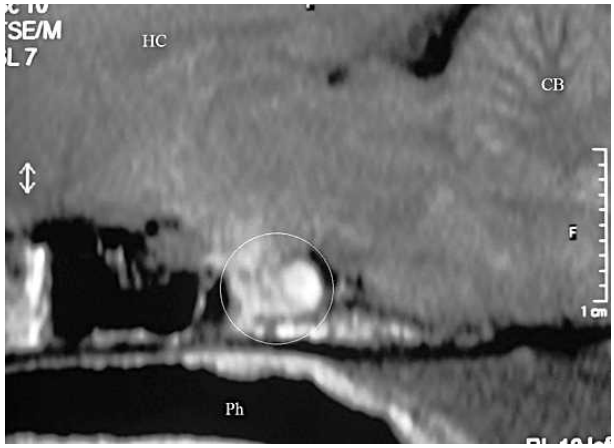

Fig 4. No pituitary lesion could be visualized in this acromegalic cat by CT imaging. On MRI evaluation, however, irregular contrast-uptake was evident at the level of the pituitary gland (white circle). Ph, pharynx; CB, cerebellum; HC, hippocampus.

Intracranial Imaging

CT was performed on 16 cats. Ten had pituitary lesions identified on non–contrast-enhanced images. In 3 cats, a mass lesion was only evident on the postcontrast views, and in 2 cats the mass was only detectable afterwards on MRI examination (Fig 4). In 1 cat, both CT and MRI were negative and 1 suspected acromegalic cat did not have intracranial imaging performed because of technical difficulties. In both latter cats, intracranial assessment was made at postmortem examination (see Postmortem Examination and Histopathology section).

Blood Pressure

Blood pressure was normal (<160 mmHg) in 12 of the 14 cats in which it was assessed. Blood pressure in the 2 remaining cats was 163 and 173 mmHg, respectively. No abnormalities were detected on ocular fundic examination in any of the confirmed cats (17/17).

Postmortem Examination and Histopathology

Postmortem examination and histopathology on the cat with both negative CT and MRI identified acidophil proliferation in the pituitary gland. Approximately one half of the cross-section of the tissue was composed of 3 solid areas of acidophils. Nearly one third was made up of normal anterior pituitary tissue with scattered acidophils throughout, and approximately one sixth of the tissue resembled pars intermedia (Fig 5).

The pancreas of this same cat appeared grossly enlarged and firm with 2 small cysts, and histopathology indicated diffuse hyperplasia with fibrous tissue and numerous lymphoid follicles in the interstitium. The islet cells were markedly vacuolated. A 2-cm cystic mass was detected adjacent to the larynx. Histopathologic evaluation of this mass was consistent with an undifferentiated malignancy, possibly squamous cell carcinoma, with spread to a local lymph node. Liver histopathology was consistent with chronic cholangiohepatitis. Microscopic examination of the kidneys indicated glomerulonephritis with diffuse thickening of the glomerular basement membrane, thickening of Bowman's capsule and periglomerular fibrosis. The tubules revealed fatty and hydropic change with epithelial degeneration and regeneration.

One cat with an IGF-1 concentration of 1,188 ng/mL, which did not have intracranial imaging performed due

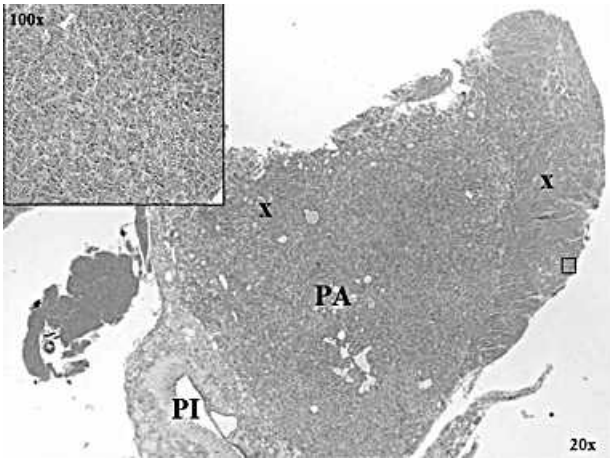

Fig 5. Postmortem examination and histopathology on the cat with negative CT and MRI indicated acidophil proliferation in the pituitary gland. The main image (stain, hematoxylin and eosin; original magnification 20x) depicts 2 solid areas of proliferation of acid-staining cells (indicated by "x") within the adenohypophysis (PA). The inset represents original magnification ×100 of the area within the black rectangle, more clearly depicting the proliferating acidophils. PI: pars intermedia.

to equipment failure, was euthanized and postmortem examination disclosed no pituitary abnormalities.

A pancreatic biopsy in another confirmed acromegalic cat disclosed multifocal ectasia of intralobular ducts, acinar cell hyperplasia, atrophic lobules, with mild interstitial chronic-active inflammation and fibrosis. Only occasional islet cells were evident in the biopsy specimens.

Discussion

The current study identified a high frequency of markedly increased IGF-1 concentrations in the evaluated population of cats, and in the 18 cats available for more detailed examination acromegaly was confirmed in 17. The serum samples for this study were obtained by offering free fructosamine assays, and this procedure could have biased veterinarians to submit samples from poorly regulated diabetics, increasing the prevalence of acromegaly within the assessed population. On the other hand, veterinarians were asked to submit samples from all diabetics, regardless of glycemic control, and the wide range of fructosamine concentrations (range, 156–795 μM) suggested the samples included both well and poorly regulated diabetic cats.

The typical diabetic signalment of a middle-aged to older, neutered male mixed breed cat of above average weight was further confirmed in this study.[1,2,5] Because cats included in this study were selected from the diabetic population on the basis of serum IGF-1 concentrations, no conclusion about the prevalence or existence of nondiabetic acromegalic cats can be made from this data.

Insulin-resistant diabetes mellitus was a prominent finding in the history of the described acromegalic cats, as has been previously reported.[5,6] As expected in any poorly controlled diabetic cat, polyuria, polydipsia, and polyphagia were prominent in the history. Polyphagia, although subjective and difficult to quantify, frequently was described as extreme. Also, weight gain, rather than weight loss, frequently was reported. In general, owners found it difficult to comment on possible changes in appearance, even in morphologically obvious acromegalic cats, which stresses the insidious onset of this disease.

Respiratory stridor has been reported in humans and dogs and occurs as a result of diffuse soft tissue growth of the tongue and surrounding oropharyngeal structures. Although such morphologic changes have been reported previously,[5] to the authors' knowledge, this clinical sign previously has been documented in only 3 cats.[3,6] In this study, the frequency of this finding both historically and on physical examination was approximately 53%, suggesting that it is more common in acromegalic cats than previously recognized.

Physical examination findings were variable, ranging from the typical previously described features of prognathia inferior and increased size of abdominal organs, head and paws, to cats with very subtle changes. Some cats in this study, despite a high index of suspicion, were phenotypically indistinguishable from normal cats.

Acromegalic cats in this study had hematologic and biochemical findings similar to those in previous reports except for the absence of hyperphosphatemia, an abnormality detected in one third of the reported acromegalic cats in 1 study.[2] Growth hormone-induced renal phosphate reabsorption may explain this finding. Hyperproteinemia was recognized in 9 of 14 cats described by Peterson and others[5] and also was a common finding in the present study. Many of the other laboratory findings (eg, hyperglycemia, increases in ALT, ALP, and BUN) presumably reflected unregulated diabetes and subclinical dehydration. Previously described erythrocytosis and biochemical evidence of nephropathy[2] were not common in the present study. Urinalysis results were similar to those previously reported.[6]

Hypertension has been regarded as common in acromegaly.[2,6] It may result in ocular hemorrhages, sudden blindness, or neurologic signs. Of interest, evidence of hypertension was not present in any of the 17 cats in this study

Although CT and MRI both have been recommended as part of the diagnostic evaluation for feline acromegaly,[2,6] this study suggests contrast-enhanced CT will detect most but not all pituitary mass lesions. In addition, even negative MRI findings do not preclude a diagnosis of acromegaly, as evidenced by the 1 cat with a postmortem examination diagnosis of acromegaly despite absence of CT and MRI evidence of pituitary abnormalities. The postmortem findings in this cat were interesting, because no evidence of a well-demarcated adenoma was found, and histopathology was more consistent with hyperplasia.

Evidence of pancreatic pathology was present in the 2 cats that underwent postmortem examination. Of interest, Gunn-Moore[1] also reported possible pathology of the pancreas in 2 of 10 cases. It is unknown if these

changes represent a secondary effect of insulin-resistant diabetes or direct effects of GH and IGF-1.

Echocardiography and electrocardiography results were similar to those previously reported, but 1 cat had no echocardiographically detectable abnormalities.

As part of the validation process for a new fGH assay, fGH concentrations in 19 confirmed acromegalic cats (including some of the presented cats) were significantly different from those of normal control cats and there was no overlap.[20] The present study further emphasizes the likelihood of acromegaly in cats with fGH concentrations >10 ng/mL. However, 2 of the 34 diabetic cats with IGF-1 <800 ng/mL also had fGH concentrations >10 ng/mL, indicating that measurement of fGH should not be the sole criterion for the diagnosis of acromegaly. These 2 cats were not subjected to intracranial imaging or postmortem examination to determine the presence or absence of acromegaly.

Evaluating total serum IGF-1 concentration proved helpful in screening for acromegaly because 17 of 18 cats with markedly increased IGF1 concentrations were acromegalic. This finding is consistent with those in previous publications.[6,c] However, increased IGF-1 concentrations previously were detected in nonacromegalic diabetic cats,[10,11] as was the case in 1 cat in the present study. No evidence of a pituitary abnormality was found on histopathology during postmortem examination of this latter cat. The cat was difficult to stabilize and was receiving 5 U of intermediate-acting insulin twice daily at the time of euthanasia. However, a glucose curve with a nadir of 8.6 mM suggested that this cat may not have been severely insulin resistant. Abdominal ultrasound examination had suggested the possibility of concurrent pancreatitis. Postmortem examination of the pancreas was unremarkable, but autolytic changes precluded precise histopathologic evaluation. This case raises additional concern over the possibility of false-positive increases in IGF-1 concentrations and emphasizes that increased IGF-1 concentrations warrant further diagnostic evaluation and not necessarily a diagnosis of acromegaly.

In an attempt to minimize the confounding effect of the pulsatile basal secretion of GH, glucose-induced GH suppression testing commonly is undertaken in people with suspected acromegaly.[13] Although 1 report describes the use of this test in an acromegalic cat,[15] another suggests no suppression was seen in 4 healthy cats,[22] and other investigators have suggested that this test is unlikely to be of benefit in cats.[6]

Both acromegaly and hyperadrenocorticism cause insulin resistance, are associated with pituitary macrotumors, and have a strong association with diabetes mellitus in cats.[3] Consequently, both will result in clinical signs related to poorly controlled diabetes mellitus. In the present study, tests to rule out hyperadrenocorticism were not performed in all cats for financial, time, and practical reasons. Although hyperadrenocorticism is notoriously difficult to exclude in cats, acromegaly and not hyperadrenocorticism was thought to be present in the 12 cats that did not have pituitary-adrenocortical axis testing performed because the clinical picture was not consistent with hyperadrenocorticism.

The relative ease with which cats with acromegaly were recruited to this study suggests that this endocrinopathy may be underdiagnosed. In addition, cats with borderline high IGF-1 concentrations (but <1,000 ng/mL) were not evaluated, further suggesting underestimation of this disease in the UK cat population.

In summary, the present study suggests there is no completely effective solitary diagnostic test for acromegaly in cats. Until other diagnostic tests have been developed, a combination of serum concentrations of IGF-1 and fGH and CT or MRI should be considered to establish an antemortem diagnosis. The prevalence of acromegaly in this study also suggests that acromegaly always should be considered a possible explanation for insulin-resistant diabetes in cats because many confirmed acromegalic cats in this study did not demonstrate the typical phenotype. Respiratory stridor was a prominent and common clinical finding, whereas hypertension, erythrocytosis, and hyperphosphatemia were less commonly observed than in previous reports.

Footnotes

[a] ABX Diagnostics Fructosamine 360/600, ABX Diagnostics-Parc Euromedecine, P 7290-34187 Montpellier-France, Validated at the Royal Veterinary College laboratory
[b] Cambridge Specialist Laboratories Services Ltd, Cambridge, UK
[c] Berk RIM, Nelson RW, Feldman EC, et al. Serum insulin-like growth factor-1 concentration in diabetic and acromegalic cats. JVIM 2006;20:725 (Abstract)
[d] Ketavet, Pharmacia-Upjohn, Sandwich, UK
[e] Hypnovel, Roche Products Limited, Welwyn Garden City, UK
[f] Rapinovet, Mallinckrodt Veterinary Ltd, Uxbridge, UK
[g] Isoba, Merial Animal Health, Harlow, UK
[h] Picker PQ 5000, Picker International, Stevenage, Herts, UK
[i] Ultravist, Schering Health Care Ltd, West Sussex, UK
[j] Magnevist, Schering Health Care Ltd, West Sussex, UK
[k] Synacthen, Novartis, Frimley, Surrey, UK
[l] SPSS 14.0 for Windows, Chicago, IL

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