

Evaluation of the low-dose dexamethasone suppression test and ultrasonographic measurements of the adrenal glands in cats with diabetes mellitus

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Summary

The objectives of the study were to evaluate the low-dose dexamethasone suppression (LDDS) test and the size of the adrenal glands via ultrasonography in cats with diabetes mellitus. Twenty-two cats were enrolled in the study. In 19 cats, suppression of cortisol concentrations below 5.5 nmol/litre occurred four and eight hours after intravenous administration of dexamethasone (0.1 mg/kg). In one other cat, the cortisol concentration was also below 5.5 nmol/litre at eight hours but was 11.0 nmol/litre at four hours. The results were in agreement with those of healthy cats in a previous study. The cortisol concentrations four and eight hours after administration of dexamethasone did not differ between cats with good glycemic control (n = 8) and those with moderate to poor control (n = 12). The adrenal glands of the diabetic cats were not enlarged compared with those of healthy cats. In two diabetic cats, the LDDS test results were abnormal. One cat had a pituitary adenoma and adrenal glands of normal size as determined by ultrasonography. The size of the adrenal glands of the other cat clearly differed; histological examination of the larger adrenal gland revealed an adrenocortical adenoma. Based on our findings, the results of the LDDS test using 0.1 mg/kg of dexamethasone are normal in cats with diabetes mellitus independent of the quality of glycemic control. In addition, diabetes mellitus does not lead to a measurable increase in the size of the adrenal glands in cats. Further studies are needed to evaluate if the dexamethasone dosage used in this study is useful to diagnose mild form of hypercortisolism.

Keywords: cat, diabetes mellitus, dexamethasone test, ultrasonography, hypercortisolism

Untersuchung des niedrig-dosierten Dexamethason-Suppressionstests und ultrasonographische Messungen der Nebennieren bei Katzen mit Diabetes mellitus

Die Ziele der Arbeit waren, den Ausfall des niedrig-dosierten Dexamethasonstests (LDDS-Test) und die ultrasonographisch ermittelte Grösse der Nebennieren bei Katzen mit Diabetes mellitus zu untersuchen. Es wurden 22 Katzen in die Studie aufgenommen. Bei 19 der Katzen lag die Kortisolkonzentration 4 und 8 Stunden nach der Applikation von Dexamethason (0.1 mg/kg) unter der Messgrenze des Assays (< 5.5 nmol/l). Bei einer weiteren Katze betrug die Kortisolkonzentration nach 4 Stunden 11.0 nmol/l und lag nach 8 Stunden unter der Messgrenze. Diese Resultate stimmen überein mit denjenigen, die in einer vorangegangenen Studie bei gesunden Katzen erzielt wurden. Katzen mit guter (n = 8) und mässiger-schlechter (n = 12) Stoffwechselkontrolle zeigten keine Unterschiede hinsichtlich der Kortisolkonzentrationen nach 4 und 8 Stunden. Die Nebennieren der Katzen mit Diabetes mellitus waren nicht grösser als diejenigen gesunder Katzen. Die Resultate des LDDS-Tests waren abnormal bei 2 Katzen mit Diabetes mellitus. Eine der beiden Katzen hatte ein Adenom des Hypophysenvorderlappens, die ultrasonographische Untersuchung ergab normale Grössen der Nebennieren. Bei der anderen Katze waren die beiden Nebennieren ultrasonographisch unterschiedlich gross, die histologische Untersuchung ergab das Vorhandensein eines adrenokortikalen Adenoms in der grösseren der beiden Nebennieren. Zusammenfassend lässt sich sagen, dass der LDDS-Test bei Verwendung von 0.1 mg/kg Dexamethason bei Katzen mit Diabetes mellitus normal ausfällt, unabhängig von der Qualität der Stoffwechselein- stellung. Darüber hinaus wurde festgestellt, dass ein Diabetes mellitus bei Katzen nicht zu einer messbaren Vergrösserung der Nebennieren führt. Anhand weiterer Studien muss nun herausgefunden werden, ob die verwendete Dosis von Dexamethason geeignet ist, auch milde Formen eines Hyperkortisolismus zu diagnostizieren.

Schlüsselwörter: Katze, Diabetes mellitus, Dexamethason- test, Ultrasonographie, Hyperkortisolismus

Introduction

Diabetes mellitus is one of the most frequently diagnosed endocrine disorders in cats. It is currently thought that the majority of cats suffer type 2 diabetes, as occurs in human beings (Rand and Marshall, 2005). Some diabetic cats, however, have concurrent underlying hypercortisolism, which may cause variable degrees of insulin resistance (Hoenig, 2002; Feldman and Nelson, 2004; Rand and Marshall, 2005). Because the clinical signs of both diseases are similar (polyuria/polydipsia [PU/PD], polyphagia, lethargy, weakness), the diagnostic work-up for hypercortisolism in diabetic cats is usually not pursued until insulin resistance becomes apparent. The same screening tests that are used in dogs are carried out to differentiate cats with and without hypercortisolism. Because of the low sensitivity of the ACTH stimulation test, the low-dose dexamethasone suppression (LDDS) test and the urine cortisol-to-creatinine ratio (UCCR) are the preferred screening tests in cats (Feldman and Nelson, 2004; Gunn-Moore, 2005). However, for determination of the UCCR, urine must be collected from the cat at home and this may not be feasible in every case (Zimmer and Reusch, 2003). Thus, the LDDS test is currently the most important screening test in cats. In dogs, several studies have addressed the possibility of over-activity of the hypothalamus-pituitary adrenal axis, leading to false positive test results. It has been shown that chronic illness can lead to adaptive changes in the axis, which inhibit dexamethasone suppression (Chastain and Nichols, 1984; Kaplan et al., 1995; Van Liew et al., 1997). Diabetes mellitus is considered one of the diseases that may potentially influence the results of the LDDS test, and abnormal as well as normal results have been reported in diabetic dogs (Chastain and Nichols, 1984; Zerbe et al., 1988). In cats, comparable data on the LDDS test are sparse. Only one study investigated the effect of diabetes mellitus and found that diabetic cats had significantly more variation in basal and post-dexamethasone plasma cortisol concentrations than healthy or ill non-diabetic cats. However, those authors used the combined dexamethasone suppression-ACTH stimulation test, and cortisol was only measured two hours after dexamethasone administration (Zerbe et al., 1987). Therefore, it is not known whether diabetes mellitus can lead to abnormal results of the LDDS test when carried out as a single test. In addition to endocrinological testing, ultrasonographic examination of the adrenal glands has become an important diagnostic tool not only in dogs but also in cats with hypercortisolism (Feldman and Nelson, 2004). To our knowledge, the ultrasonographic appearance of the adrenal glands in cats with diabetes mellitus has not been reported. It is therefore not known whether adrenal gland size increases because of the stress of di-

abetes mellitus, which would then complicate the diagnosis of concurrent hypercortisolism. In a previous study, reference ranges for the LDDS test and the size of the adrenal glands, determined ultrasonographically, were established in healthy cats (Zimmer et al., 2000).

The objectives of the present study were to evaluate the LDDS test and to measure the size of the adrenal glands in cats with diabetes mellitus.

Animals, Material and Methods

Criteria for selection of cases

This study was conducted prospectively between 2002 and 2004 at the Clinic for Small Animal Internal Medicine, University of Zurich. Cats with diabetes mellitus were selected for inclusion in the study provided that they did not have other significant concurrent diseases such as chronic renal failure, heart failure or hyperthyroidism. In addition, cats that had been treated with glucocorticoids or progestagens within six weeks prior to presentation were not included. Diagnosis of diabetes mellitus was based on characteristic clinical signs, including PU/PD and weight loss, hyperglycemia (fasting blood glucose > 9.5 mmol/litre), glucosuria and elevated serum fructosamine concentrations (> 340 µmol/litre).

Study design

On initial presentation, a thorough physical examination and history taking, complete blood count, biochemical profile (including fructosamine and total T4 concentrations), urinalysis and urine culture were carried out. Additionally, all cats underwent ultrasonographic examination of the abdomen during which the adrenal glands were evaluated. Insulin therapy was started with intermediate-acting insulin (porcine insulin) (Caninsulin, Intervet Inc, Boxmeer, The Netherlands) at a dosage of 0.25 to 0.5 units/kg twice daily. Re-evaluations were scheduled one, three, six, and 12 weeks after the first evaluation and included a detailed updated history, physical examination and measurement of total protein, albumin and fructosamine concentrations. A blood glucose curve was obtained by measuring blood glucose concentrations every two hours starting one to two hours after insulin had been injected at home. Insulin therapy was adjusted based on the glucose nadir: when the nadir was < 5 mmol/litre, 5 to < 9 mmol/litre, ≥ 9 mmol/litre, the insulin dosage was decreased (0.5 to 1 U/cat), left unchanged and increased (0.5 to 1 U/cat), respectively. Six weeks after initial presentation, a LDDS test was carried out. At that time, glycemic control was evaluated based on the clinical signs (PU/PD, polyphagia,

weight loss) and the fructosamine and blood glucose nadir concentrations. Cats were classified as either well regulated (clinical signs resolved, fructosamine \leq 400 $\mu\text{mol/litre}$, blood glucose nadir $<$ 9 mmol/litre) or moderately to poorly regulated (persistence of clinical signs, fructosamine $>$ 400 $\mu\text{mol/litre}$, blood glucose nadir \geq 9 mmol/litre).

Ultrasonographic examination of the adrenal glands

The cats were not sedated for ultrasonographic examination. The same approach and ultrasonographic equipment (Acuson Sequoia 512, Acuson, Mountain View, CA, USA) were used as described previously (Zimmer et al., 2000). In all cats, the maximum length and width of the adrenal glands were measured. Each measurement was repeated three times in succession, and the mean was calculated. The measurements were compared with those of healthy cats in a previous study (Zimmer et al., 2000).

Low-dose dexamethasone suppression test

The LDDS test was carried out by collecting blood samples from the jugular vein before and four and eight hours after injection of dexamethasone (0.1 mg/kg intravenously) (Dexadreson, Virbac, Küsnacht, Switzerland), as described previously (Zimmer et al., 2000). Serum cortisol concentrations were measured using a chemiluminescence immunoassay (ADVIA Centaur® System, Kentauer, Bayer AG, Zurich, Switzerland). The test results were compared with those of healthy cats in a previous study (Zimmer et al., 2000).

Statistical analysis

The results were analysed by means of non-parametric statistical methods using a computer software program (SPSS/PC, version 10.0, base manual, SPSS Inc, Chicago, Ill, USA). Ranges and median values are given. The Mann-Whitney *U* test was used to determine differences. For cortisol concentrations that were below the level of detection of the assay (5.5 nmol/litre), a number of 5.5 nmol/litre was used for comparison. The level of significance was set at $P < 0.05$.

Results

Patient and glycaemic status

Twenty-two cats that ranged in age from 2.4 to 15.2 years (median 11.2 years) and weighed between 3.5 and 8.4 kg (median 6.0 kg) met the inclusion criteria for the study. There were 16 neutered male and 6

spayed female cats; breeds included domestic shorthair (18), Carthusian (1), Burmese (1), Siamese (1) and Persian (1) cats.

At initial presentation, blood glucose concentrations ranged from 15.3 to 30.8 mmol/litre (median 21.9 mmol/litre) and fructosamine concentrations from 383 to 1050 $\mu\text{mol/litre}$ (median 613.5 $\mu\text{mol/litre}$). Six weeks later, eight cats were considered to be well regulated. Clinical signs had resolved, blood glucose nadirs ranged from 3.0 to 7.9 mmol/litre (median 4.8 mmol/litre) and fructosamine concentrations were 266 to 394 $\mu\text{mol/litre}$ (median 356 $\mu\text{mol/litre}$). In 14 cats, glycaemic control was judged to be moderate to poor because of persistence of clinical signs, blood glucose nadir concentrations of between 9 and 26.9 mmol/litre (median 16.8 mmol/litre) and fructosamine concentrations between 410 and 1388 $\mu\text{mol/litre}$ (median 659 $\mu\text{mol/litre}$). There were significant differences between the fructosamine and blood glucose nadir concentrations in cats with good and those with moderate to poor glycaemic control ($P < 0.001$). In two of these 14 cats, concurrent hypercortisolism was diagnosed (see below); glycaemic control in these two cats was considered to be poor because of persistence of clinical signs, blood glucose nadir concentrations of 19.5 and 19.3 mmol/litre and fructosamine levels of 518 and 742 $\mu\text{mol/litre}$, respectively.

Low-dose dexamethasone suppression test

In 19 of the 22 diabetic cats, cortisol concentrations four and eight hours after dexamethasone administration were below the level of detection of the assay (5.5 nmol/litre); in one other cat, the cortisol concentration was 11.0 nmol/litre at four hours and below the level of detection at eight hours. The cortisol concentration four and eight hours after dexamethasone administration did not differ between the cats with good glycaemic control ($n = 8$) and those with moderate to poor control ($n = 12$). Baseline cortisol concentrations of the well-regulated diabetic cats were significantly higher ($P = 0.001$) than those of cats with moderate to poor control. Cortisol concentrations at baseline and four and eight hours after dexamethasone administration did not differ between the diabetic cats and the healthy cats (Fig 1).

In the remaining two of the 22 diabetic cats, the LDDS test was abnormal. One cat had baseline, four and eight hour cortisol concentrations of 132.4 nmol/litre, $<$ 5.5 nmol/litre and 135.2 nmol/litre, respectively. The cat developed diabetic ketoacidosis four months after diagnosis and was euthanased at the owner's request. Histological examination of tissue specimens revealed bilateral adrenal hyperplasia and a pituitary adenoma, which was positive for ACTH using immunohistochemistry. The other cat had base-

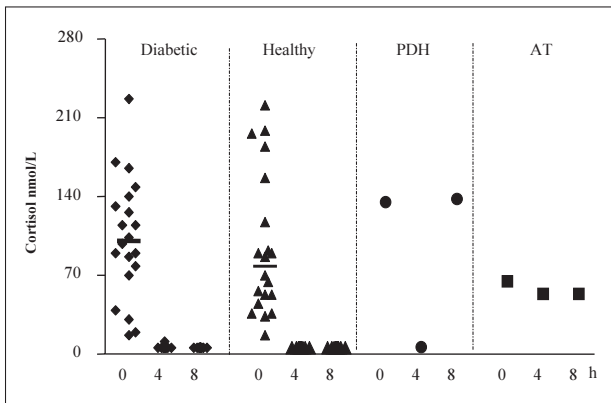


Figure 1: Serum cortisol concentrations 0, 4 and 8 hours after administration of dexamethasone in 20 diabetic cats, 20 healthy cats and two cats with concurrent pituitary-dependent hyperadrenocorticism (PDH) and adrenocortical tumour (AT), respectively. The data from the healthy cats are from a previous study (Zimmer et al JSAP, 2000).

line, four and eight hour cortisol concentrations of 63.5 nmol/litre, 52.4 nmol/litre and 52.4 nmol/litre, respectively. Unilateral adrenalectomy was performed, and histological examination of the tissue confirmed an adrenocortical adenoma in the left adrenal gland.

Ultrasonographic examination

Both adrenal glands could be visualised in all the diabetic cats. The glands appeared less echogenic than the surrounding tissue and were oblong and oval to bean-shaped (Figs 2a and 2b). The length of the right and left adrenal glands and the width of the left adrenal gland were not significantly different between diabetic and healthy cats. The right adrenal gland was thinner in the diabetic cats compared with those of the healthy cats ($P = 0.045$).

In the cat with pituitary-dependent hypercortisolism, the adrenal glands appeared bilaterally symmetric in size and shape, and their size was within the range determined for the healthy and diabetic cats. In the cat

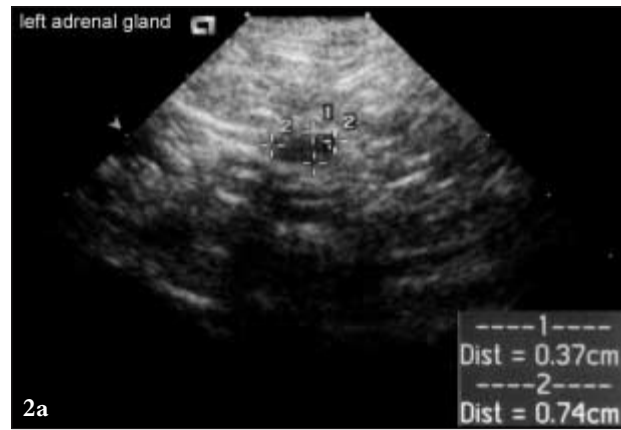


Figure 2 a and b: Ultrasonogram of the left and right adrenal glands of a diabetic cat. The adrenal glands are oval to bean-shaped and hypoechoic compared with the surrounding tissue. The left adrenal gland measures 0.78 cm in length and 0.37 in width, the length of the right adrenal gland is 0.9 cm, the width 0.37 cm.

with an adrenocortical adenoma, the left and right adrenal glands differed markedly in size. The length and width of the non-tumour bearing adrenal gland were at the lower end of the normal range, whereas the length of the tumour-bearing gland was at the upper end of the normal range and the width was slightly greater than normal. The measurements of the length and width of the adrenal glands are given in Table 1.

Table 1: Ultrasonographic measurements of the length and width of the adrenal gland in 20 diabetic cats, 20 healthy cats and two cats with concurrent pituitary-dependent hyperadrenocorticism (PDH) and an adrenocortical tumour (AT), respectively. The data from the healthy cats are from a previous study (Zimmer et al JSAP, 2000).

Cats	Length right adrenal (cm)	Width right adrenal (cm)	Length left adrenal (cm)	Width left adrenal (cm)
DM (n = 20) range (median)	0.46–1.32 (1.01)	0.26–0.48 (0.36)	0.71–1.13 (0.98)	0.24–0.46 (0.35)
Healthy (n = 20) range (median)*	0.67–1.37 (0.98)	0.29–0.45 (0.39)	0.45–1.33 (0.89)	0.30–0.53 (0.39)
DM + AT n = 1	0.66	0.32	1.15	0.5
DM + PDH n = 1	1.1	0.29	0.92	0.27

Discussion

In the present study, 20 of 22 diabetic cats had complete suppression of cortisol concentrations four and eight hours after administration of dexamethasone (0.1 mg/kg). The study was done under standardised clinical conditions by excluding cats with obvious concurrent disease, using a standardised treatment protocol and performing the LDDS test six weeks after initiating therapy in all cats. To our knowledge, there are no studies comparable with the present one. In the only other study in which dexamethasone testing was undertaken in diabetic cats (Zerbe et al., 1987), the conditions varied; for example, the time between the diagnosis of diabetes mellitus and the LDDS test ranged from two to 358 days, and cats with serious illness such as diabetic ketoacidosis were included in the study. Another difference was the test protocol. Zerbe et al. (1987) assessed adrenal function by measuring cortisol levels after a combined dexamethasone suppression-ACTH stimulation test. Diabetic cats had more variation in cortisol concentrations before and after administration of dexamethasone compared with healthy or ill non-diabetic cats. In contrast, in our study, suppression of the cortisol concentration below the level of detection of the assay occurred in all 20 cats without concurrent hypercortisolism (with the exception of one cat with a cortisol concentration of 11.0 nmol/litre at four hours). Our results were in agreement with those of 20 healthy cats, evaluated previously using the same test protocol (Zimmer et al., 2000). The disparity in the results of our study and the one by Zerbe and others is believed to be due mainly to the different study conditions.

Various studies have demonstrated hyperactivity of the hypothalamus-pituitary-adrenal gland axis in human diabetic patients, which may lead to abnormal results of the dexamethasone suppression test (Hudson et al., 1984; Roy et al., 1990; Roy et al., 1991). Poor glycaemic control, hypoglycaemic episodes, obesity, recent weight loss, depression and stress of hospitalisation are discussed as possible confounding factors (Roy et al., 1990). The relevance of the hyperactivity in humans is twofold: firstly, it may complicate the diagnosis of concurrent hypercortisolism and secondly, it may worsen regulation and the vascular complications of diabetes (Roy et al., 1991). Interestingly, in cats, the quality of glycaemic control did not affect the results of the LDDS test; in cats with good glycaemic control as well as those with moderate to poor control, there was complete suppression of cortisol release/production. The fact that well controlled diabetic cats had higher basal cortisol concentrations than moderately to poorly controlled cats is difficult to explain. It has been shown that baseline cortisol concentrations can vary considerably in healthy cats,

and can be much higher than the concentrations determined in the present study (Schoeman et al., 2000). We thus assume that the difference observed in our study was due to variations that are not clinically relevant and may not be obvious if a larger group of cats would have been examined. Based on our results, using the LDDS test for the diagnostic work-up of diabetic cats with poor glycaemic control appears justified. It has been debated that in such cases, poor glycaemic control may lead to false positive results. Most clinicians therefore recommend pursuing good glycaemic control before testing for endocrine disorders, which, unfortunately, is rarely achievable in cats with hypercortisolism.

However, there are two limitations of our study that should be considered: the duration of diabetes mellitus and the dose of dexamethasone used. It was suggested that the dysregulation of the hypothalamus-pituitary-adrenal gland axis in human diabetic patients is a function of the duration of the disease; the longer the duration, the more obvious the dysregulation (Roy et al., 1991). All the cats in our study underwent an LDDS test at six weeks after the diagnosis of diabetes, which represents a relatively early stage of the disease. Therefore, we do not know whether the risk of abnormal results of the LDDS test increases with time. Nevertheless, it appears reasonable to recommend work-up for hypercortisolism relatively early in the course of diabetes mellitus.

Many endocrinologists currently recommend carrying out the LDDS test in cats using 0.1 mg/kg of dexamethasone, which is ten-fold higher than the dose used in dogs (Peterson and Graves, 1988; Myers and Bruyette, 1994; Feldman and Nelson, 2004; Gunn-Moore, 2005). The rationale behind this is that in some healthy cats, the administration of 0.01 mg/kg of dexamethasone (i.e., the dose used in dogs) failed to suppress the plasma cortisol concentration, and thus led to false-positive test results (Smith and Feldman, 1987; Peterson and Graves, 1988). In dogs, it has been demonstrated that resistance to dexamethasone is not an "all or nothing" phenomenon, but may be compared with a "sliding scale" (Kooistra et al., 1997). In cats, the number of cases with hypercortisolism is currently too low to draw a similar conclusion. It was shown, however, that a normal dexamethasone test result may become abnormal within six months in a cat with hypercortisolism (Meij et al., 2001). It was also reported that in a cat with hypercortisolism, cortisol suppression was not achieved with a dexamethasone dose of 0.01 mg/kg, but was achieved with a dose of 0.1 mg/kg (Nelson et al., 1988). It is therefore possible that in cases of mild hypercortisolism, false negative test results occur, particularly when a dexamethasone dose of 0.1 mg/kg is used.

Two of the 22 diabetic cats had hypercortisolism. Interestingly, in both cases, the diagnosis was made only

after receiving the results of the LDDS test and was supported by the ultrasonographic finding of an adrenal mass in one of the two cats. The clinical signs of these two cats did not differ in severity from other poorly regulated diabetic cats in our study. In addition, more typical signs of hypercortisolism such as thin skin, skin tears and a pot-bellied appearance were absent. In human medicine, it was shown that the prevalence of pre-clinical hypercortisolism in patients with poorly controlled diabetes appears to be considerably higher than previously believed (Leibowitz et al., 1996). Whether this also occurs in diabetic cats requires further investigation, using screening-tests with a high sensitivity.

Ultrasonography revealed that the size of the adrenal glands of the 20 diabetic cats that did not have hypercortisolism did not differ from that of 20 healthy cats (Zimmer et al., 2000). It can therefore be concluded that diabetes mellitus, at least in the short term, does not lead to a measurable increase in adrenal gland size in cats. Whether the results would differ in cats with long-term diabetes requires further study. In dogs, adrenal ultrasonography is a valuable tool to differentiate individuals with pituitary-dependent hypercortisolism and those with adrenocortical tumours. However, a diagnosis of hypercortisolism should not be based on the results of ultrasonography alone (Feldman and Nelson, 2004). Until now, there has been considerably less information on the value of

adrenal ultrasonography in cats compared with dogs. Of 41 cats with hypercortisolism, pituitary-dependent disease and adrenocortical tumours were correctly diagnosed in 34 (83%; Feldman and Nelson, 2004). In the present study, the size of the adrenal glands in the cats with hypercortisolism did not differ from those without hypercortisolism. In the cat with pituitary-dependent hypercortisolism, the adrenal glands appeared unremarkable, and hypercortisolism would not have been suspected based on the ultrasonographic findings alone. In the cat with an adrenocortical tumour, there was a distinct difference in the size of the two adrenal glands. Hypercortisolism due to an adrenocortical tumour was further suspected because of abnormal LDDS test results.

In conclusion, complete suppression of the cortisol concentration occurred four and eight hours after the LDDS test using 0.1 mg/kg of dexamethasone in 20 of 22 cats with diabetes mellitus. The results of the LDDS test did not differ between diabetic cats with good glycemic control and those with moderate to poor control. In two of 22 diabetic cats, the LDDS test was abnormal, and hypercortisolism was confirmed by histological examination of tissue samples. Based on our results, we conclude that the LDDS test can be used to diagnose concurrent hypercortisolism in cats with diabetes mellitus. Diabetes mellitus does not lead to an increase in adrenal gland size as determined by ultrasonography.

Etude d'un test de suppression à la dexaméthasone faiblement dosée et mesure échographique des surrénales chez des chats atteints de diabète sucré

Les buts du présent travail étaient d'examiner le résultat d'un test de suppression à la dexaméthasone faiblement dosée ainsi que la taille des surrénales mesurée par échographie chez des chats atteints de diabète sucré. 22 chats ont été inclus dans l'étude. Chez 19 d'entre eux, le taux de cortisol, 4 et 8 heures après l'application de dexaméthasone (0.1 mg/kg) se trouvait en dessous des limites de sensibilité du test (< 5.5 nmol/l). Chez un chat, le taux de cortisol après 4 heures atteignait 11 nmol/l et passait en-dessous du seuil de détection après 8 heures. Ces résultats concordent avec ceux obtenus dans une étude précédente sur des chats en bonne santé. On ne constatait pas de différences quant au taux de cortisol après 4, respectivement 8 heures entre les chats présentant un bon (n=8) et un moyen à mauvais (n=12) contrôle du métabolisme. Les surrénales des chats atteints de diabète sucré n'étaient pas plus grosses que celles des chats sains. Les résultats de tests de suppressions étaient anor-

Esame di un test di soppressione al desametazone, dosato debolmente, e di misura ultrasonografica dei surreni nei gatti affetti da diabete mellito

Scopo di questa ricerca era di esaminare l'esito del test al desametazone (test LDDS) dosato debolmente e la grandezza rilevata dei surreni nei gatti affetti da diabete mellito. Nello studio sono stati esaminati 22 gatti. In 19 gatti la concentrazione di cortisolo nelle 4 a 8 ore dopo l'applicazione di desametazone (0.1 mg/kg) restava sotto la misura limite del test (< 5.5 nmol/l). Per un altro gatto la concentrazione di cortisolo era di 11.0 nmol/l dopo 4 ore e si situava dopo 8 ore sotto la misura limite. Questi risultati concordano con quelli raggiunti precedentemente da un altro studio effettuato su gatti sani. Gatti con un controllo del metabolismo buono (n=8) e moderatamente cattivo (n=12) non mostravano differenza riguardo alla concentrazione di cortisolo dopo 4 e dopo 8 ore. I surreni dei gatti con diabete mellito non erano più grandi di quelli dei gatti sani. I risultati del test LDDS erano anormali in 2 gatti con diabete mellito. Uno dei due gatti aveva un adenoma dell'ade-

maux chez 2 chats atteints de diabète sucré. L'un d'entre eux présentait un adénome du lobe antérieur de l'hypophyse, l'examen échographique révélant des surrénales de taille normale. Chez l'autre chat, les surrénales étaient de taille différente à l'échographie et l'examen histologique révélait un adénome du cortex surrénal dans la plus grosse d'entre elle.

En résumé, on constate que le test de suppression à la dexaméthasone faiblement dosée (0.1 mg/kg) donne des résultats normaux chez les chats atteints de diabète sucré quelle que soit la qualité de la stabilisation de leur métabolisme. On constate en outre que le diabète sucré ne conduit pas, chez le chat, à un grossissement mesurable des surrénales. Il s'agit maintenant, sur la base d'autres études, de déterminer si la dose de dexaméthasone utilisée est adaptée au diagnostic des formes légères d'hypercorticisme.

noipofisi, ma dall'analisi ultrasonografica i surreni risultavano di grandezza normale. Negli altri gatti entrambi i surreni erano, con l'ultrasonografia, di differenti grandezze e le analisi istologiche provavano la presenza di un adenoma adrenocorticale della grandezza di entrambi i surreni. Riassumendo si può affermare che il test LDDS con 0.1 mg/kg di desametasone nei gatti con diabete mellito risulta normale, indipendentemente dalla qualità del metabolismo. Inoltre si è costatato che il diabete mellito nei gatti non porta necessariamente ad un ingrossamento dei surreni. Studi futuri dovrebbero identificare se la dose di desametasone utilizzate è adatta anche per diagnosticare delle forme leggere di ipercorticisme.

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