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Congresul AMVAC/RoSAVA 2012

Devenit deja o tradiție, Congresul anual AMVAC/RoSAVA a ajuns anul acesta la cea de-a VII-a ediție, reunind peste 1.200 de participanți atât din România, cât și din diferite colțuri ale lumii.
Evenimentul se va desfășura pe parcursul a trei zile, la Sinaia, în perioada 8-10 noiembrie 2012, în cadrul Centrului Internațional de Conferințe "Casino Sinaia".
Prima zi a manifestării științifice va debuta cu "WSAVA Day", dedicată Medicinei feline, sub directa coordonare a Dr. Margie Scherk,) Vancouver, Canada, fondatoare a "Cats Only Veterinary Clinic", în Vancouver, în 1986.
Dr. Margie Scherk a absolvit Universitatea din Guelph în 1982, iar în 1995 a devenit cunoscută în specialitatea practică felină, fiind certificată de Consiliul American al Medicilor Veterinari (ABVP). Printre cele mai importante realizări ale sale se numără utilizarea plasturelui transdermic cu fentanil în atenuarea durerii la animale de companie. În calitate de co-autor a contribuit la realizarea de lucrări de specialitate, a scris un capitol pentru două ediții ale Manualului de Medicină Internă Veterinară, numeroase capitole în „Little's The Cat" etc.
În paralel cu "WSAVA Day", se vor desfășura și un Workshop dedicat managementului susținut de Dr. Cristi Mătură, în cadrul caruia vor fi abordate strategia și planificarea activităților în cabinetul veterinar, și un Workshop destinat Cardiologiei, susținut de renumitul doctor Jean-François Rousselot, membru al Academiei Veterinare din Franța.
Cea de-a doua zi a manifestării va dezbate cinci teme importante pentru domeniul veterinar: Dermatologie - susținută de renumitul profesor dr. Danny Scott, diplomat în American College of Veterinary Dermatology; Management - temă susținută de Dr. Cristi Mătură; Urgențe - ale cărui lucrări vor fi prezidate de Dr. Mario Codreanu și Dr. Norin Chai (Franța); Endocrinologie - prezentată și susținută de prof. dr. Viorel Andronie, președintele Colegiului Medicilor Veterinari din România, Chirurgie Reconstructivă - al cărui lector este Prof. dr. Dupré Gilles de la University of Vienna, Austria.
Ultima a zi Congresului va continua seria dezbaterilor din zilele precedente, cu teme noi precum Neurologie, susținută de Dr. Rick Lecouteur, Imagistică, ai cărui lectori vor fi Dr. Florin Grosu și Dr. Peter van Dongen (Marea Britanie), Ortopedie - Dr. Johan van Tilburg, Imunologie - Dr. Dragoș Cobzaru, Oftalmologie - Dr. Iuliana Ionascu și Dr. Pip Boydel (Marea Britanie) și Oncologie - Prof. dr. Michael J. Day (University of Bristol).
Vă așteptăm la un eveniment științific de amploare, cu participanți și lectori de renume, ce vor susține workshopuri interesante și vor prezenta lucrări științifice de actualitate, noutăți și tehnici moderne utile în practica curentă a fiecăruia.
Dr. Nicolae Valentin, Redactor-șef „Practica veterinară.ro"

Dr. Valentin Nicolae
Redactor-șef

sumar

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Pericardiocenteza la câine și pisică

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Dr. Delia Bagiu
Spitalul Veterinar „Dr. Șerdean", Timișoara

Pericardiocenteza este o procedură relativ simplă care ameliorează rapid simptomele tamponadei cardiace secundare efuziunii pericardice idiopatice sau neoplaziilor cardiace. Ultrasonografia este o metodă sigură și neinvazivă folosită în scopul de a diagnostica și trata efuziunea pericardică.

Dezvoltarea tehnologică, apariția unor noi generații de aparate de ultrasonografie și totodată îmbunătățirea calității imaginii au dus la crearea unor noi posibilități de diagnostic și tratament al efuziunii pericardice la animalele domestice. Este imperativ să se aibă în vedere că ultrasonografia este menită să mărească, dar nu să înlocuiască informațiile obținute prin examenul fizic și radiografia toracică.

Examenul radiologic în efuziunea pericardică

Constatările radiologice, la animalele cu efuziune pericardică, includ o siluetă cardiacă globuloasă, efuziune pleurală și mărirea în volum a venei cave caudale. În cazul metastazelor, se pot observa modificări nodulare sau interstițiale la nivelul parenchimului pulmonar. În unele cazuri, atunci când o cantitate mare de efuziune pericardică este absentă, poate exista o extinderea regională a siluetei cardiace asociate cu prezența unei tumori în atriul drept sau la baza cordului. Constatările radiografice pot fi normale sau doar ușor anormale în unele cazuri de efuziune pericardică. Hemoragia acută conduce la tamponada cardiacă, cu acumulări relativ mici de lichid, prin urmare examinarea fizică și ecografică la acești pacienți rămâne importantă. Dimensiunea siluetei cardiace depinde de stadiul de cronicizare a bolii. Astfel, efuziunea pericardică în care lichidul se acumulează pe o perioadă mai lungă de timp (efuziune pericardică cronică) produce în general o siluetă cardiacă mai mare decât în cazul efuziunii pericardice acute care se acumulează rapid. La câinii la care efuziunea pericardică se dezvoltă secundar unei endocardite sau

unei cardiomiopatii dilatative, silueta cardiacă poate indica cardiomegalie generalizată, nefiind asociată obligatoriu cu efuziune pericardică.

Electrocardiograma

Anomalii electrocardiografice sunt adesea prezente la câinii cu efuziune pericardică, cele mai frecvente fiind tahicardia sinusală și prezența complexelor QRS subvoltate. Alternanța electrică este constatată mai rar, dar este patognomonică pentru efuziunea pericardică. De asemenea, pot fi observate diverse aritmii ca urmare a dezvoltării unor tumori miocardice.

Efuziunea pericardică idiopatică la câine

Efuziunea pericardică idiopatică este o afecțiune mai puțin frecventă, care afectează, de obicei, masculii din rasele mari sau gigant. Cele mai multe cazuri prezintă un istoric sugestiv pentru insuficiența cardiacă cronică dreaptă (ascită, letargie, pierdere în greutate), deși ele pot dezvolta și tamponada cardiacă acută. Cea mai frecventă anomalie întâlnită la examenul clinic este prezența zgomotelor cardiace înăbușite; ocazional, poate fi întâlnit și puls slab. Prezența pe electrocardiogramă (ECG) a unui complex QRS diminuat este frecventă, în timp ce alternanța electrică se întâlnește foarte rar. În cazul acestor pacienți, motivul dezvoltării efuziunii pericardice rămâne necunoscut, chiar și în ciuda unor încercări mai amănunțite de diagnosticare.

Tumorile cardiace și pericardice

La animalele mici, neoplaziile cardiace și pericardice sunt mai puțin frecvente. În ciuda incidenței scăzute a acestora, aceste tumori sunt importante, din cauza efectelor lor potențiale asupra sistemului cardiovascular. Diagnosticul tardiv și invazivitatea acestor tumori fac tratamentul dificil.

La câine, hemangiosarcomul este neoplazia cardiacă cel mai frecvent întâlnită, cu o incidență variind între 40,5 și 69%. Chemodectomul este al doilea tip de tumoare cardiacă întâlnit, cu o incidență între 5 și 17,3%. Limfosarcomul și carcinomul tiroidian sunt următoarele tipuri, ca frecvență, de neoplazii cardiace identificate.

La pisici, diagnosticul histologic cel mai frecvent al neoplaziei cardiace este limfosarcomul. Potrivit unor studii, limfosarcomul cuprinde mai mult de 30% din toate neoplasmele cardiace la feline. Tumoarea implică de obicei miocardul, dar clinic se decelează efuziunea pericardică. Limfosarcomul de multe ori poate fi diagnosticat prin evaluarea citologică a efuziunii pericardice obținute prin pericardiocenteză. Restul de neoplasme cardiace raportate la feline sunt carcinoame diverse (19%), hemangiosarcomul (8,6%), tumori aortice (3,4%) și fibrosarcoame (3,4%).

Mezoteliomul este un neoplasm malign mai puțin frecvent la câine care afectează pericardul parietal și visceral, precum și suprafețele mezoteliale ale pleurei și peritoneului. Tumoarea poate afecta mai multe cavități ale corpului care duc la acumularea de lichid în cavitățile pericardică, pleurală și peritoneală. Mezoteliomul poate fi dificil de diagnosticat din cauza similitudinii dintre celulele neoplazice și celulele mezoteliale reactive observate la evaluarea citologică a efuziunilor. Atunci când la o ecografie de control se observă un pericard îngroșat, diagnosticul diferențial trebuie să se facă și față de mezoteliom. Pentru un diagnostic definitiv este necesară efectuarea unei biopsii. La pisică, mezoteliomul, deși a fost diagnosticat, este rar întâlnit.

Tumorile cardiace perturbă funcția normală a țesuturilor din care provin, ceea ce duce la alterarea funcției cardiovasculare. Efectele fiziologice ale unei tumori sunt influențate de dimensiunea și localizarea tumorii, precum și de prezența sau absența efuziunii pericardice (un număr relativ mic de tumori cardiace pot evolua fără efuziune pericardică). Semnele clinice pentru care animalul este adus la veterinar sunt adesea nespecifice, incluzând anorexie, letargie, intoleranță la exerciții, distensie abdominală, slăbiciune, colaps, dispnee și sincopă. De obicei, efuziunea pericardică secundară și concomitent tamponada cardiacă includ: puls slab, distensie venoasă jugulară, zgomote pulmonare și cardiace înăbușite, aritmii, hepatomegalie, spleanomegalie, dispnee, tahicardie și ascită.

Ecocardiografia în efuziune pericardică

Ecocardiografia este cea mai exactă metodă neinvazivă de diagnosticare a efuziunii pericardice și a tumorilor intrapericardice. În trecut, au fost folosite metode mai invazive de diagnosticare, cum ar fi pericardiografia cu contrast pozitiv, pneumopericardiografia și angiografia. Recent, troponinele cardiace au fost folosite pentru a ajuta la diagnosticarea cazurilor de efuziune pericardică la câine, deoarece câinii cu efuziune pericardică au o concentrație mult mai mare de troponine (cTnI) decât câinii sănătoși. Ultrasonografia normală permite vizualizarea directă, evaluarea tumorii și a efectului acesteia asupra funcției cardiace.

Examinarea atât pe partea stângă, cât și pe dreapta toracelui trebuie efectuată pentru a furniza clinicianului o imagine cât mai exactă a originii tumorii. Clasificarea histologică a tumorii poate fi de multe ori suspectată în funcție de localizarea acesteia în cord și/sau pericard. Prezența unei mase la nivelul atriului drept la câine indică cel mai probabil un hemangiosarcom, în timp ce o masă situată adiacent bazei cordului, între aorta și artera pulmonară, este cel mai probabil un chemodectom. În ceea ce privește tumorile intrapericardice, acestea pot fi identificate ecocardiografic în proporție de 17-69%. O serie de examene ecocardiografice poate fi necesară înainte de a identifica o masă la nivelul cordului. Efuziunea pericardică, în multe cazuri, permite vizualizarea mai bună a bazei de inimă și auriculelor, conducând astfel la o detectare mai facilă a maselor cardiace.

Efuziunea pericardică și pericardiocenteza

Efuziunea pericardică este un semn clinic secundar frecvent la animalele cu neoplazii cardiace. Ecocardiografic, efuziunea pericardică este văzută ca un spațiu anecogen sau hipoecogen între epicard și pericard. Efuziunea pericardică duce la o creștere a presiunii intrapericardice rezultând astfel diferite modificări hemodinamice. Tamponada cardiacă apare atunci când presiunea intrapericardică este egală sau depășește presiunea de umplere a ventriculului drept în timpul diastolei, ceea ce conduce la o scădere a debitului cardiac. Ca urmare, apar semnele clinice ale insuficienței cardiace drepte și ale șocului cardiogen. Presiunea intrapericardică depinde de volumul colecției, rata de acumulare a lichidului și caracteristicile fizice ale pericardului. Volumele mici de efuziune pericardică ce se acumulează rapid (efuziune pericardică acută) pot provoca creșteri mari ale presiunii intrapericardice, în timp ce volumele mari de efuziune pericardică ce se acumulează lent (efuziune pericardică cronică) pot duce la creșteri mici ale presiunii intrapericardice și sunt puțin semnificative din punct de vedere hemodinamic. O reducere a volumului pericardic poate fi observată fie în cazul unui neoplasm pericardic, fie în cazul inflamației cronice a pericardului. Colapsul atriului sau ventriculul drept poate fi observat ecocardiografic în timpul diastolei, sugerând tamponada cardiacă. Pericardiocenteza este metoda recomandată pentru a restabili presiunea intrapericardică normală și umplerea ventriculară.

Pericardiocenteza este folosită pentru a stabiliza animalele cu tamponadă cardiacă și pentru a obține probe de lichid în vederea confirmării diagnosticului. La unii pacienți poate fi necesară sedarea pentru a evita mișcările bruște neașteptate în timp ce acul este introdus. La pacienții aflați într-o stare critică, este recomandată anestezia locală a mușchilor intercostali și a pleurei parietale. Pericardiocenteza se face la nivelul hemitoracelui drept, cu câinele în poziție culcată lateral stânga. Abordarea unilaterală dreaptă este utilizată pentru a evita lezionarea arterei coronare stângi. Hemitoracele lateral drept este pregătit chirurgical între spațiile intercostale II și VIII. Ecocardiografia poate fi de ajutor în găsirea spațiului intercostal optim pentru pericardiocenteză, dar în cazul în care ecocardiografia nu este disponibilă, este recomandată efectuarea pericardiocentezei începând cu spațiile intercostale IV și V imediat dorsal de joncțiunea condrocostală. Mai multe tipuri de catetere pot fi utilizate pentru efectuarea acestei proceduri.

Acul cateterului este introdus prin piele în cavitatea toracică și este lent ghidat spre pericard. Atunci când efuziunea pleurală este prezentă, în cateter poate fi observat inițial un lichid limpede serosangvinolent. Acul trebuie să fie introdus în continuare până când lichidul de efuziune pericardică (în general, hemoragic) este observat în cateter. Apoi, o dată ce cateterul este poziționat în pericard, acul este scos din torace. Stiletul se scoate din cateter, iar seringa de capacitate mare, între 30 și 60 ml, este apoi atașată la cateter și se realizează aspirarea lichidului. Poziția cateterului trebuie ajustată în timpul procedurii pentru a elimina cât mai mult lichid din spațiul pericardic. În cazul în care cauza efuziunii nu

FOTO: FOTOLIA

este cunoscută, o probă de lichid ar trebui să fie trimisă spre analiză. Determinarea pH-ul lichidului pericardic s-a folosit anterior pentru a diferenția efuziunile neoplazice de cele non-neoplazice, dar s-a demonstrat că acest tip de analiză nu este relevant.

Complicațiile grave asociate pericardiocentezei sunt rare și includ: perforarea cordului, aritmii, leziuni ale arterei coronare sau ale tumorii, cauzând astfel hemoragie intrapericardică. Puncția cordului poate avea loc atunci când cateterul este avansat accidental în cord, acțiune care în mod obișnuit nu duce la complicații semnificative dacă cateterul este retras rapid și repoziționat. Atunci când există îndoieli dacă lichidul aspirat provine sau nu din pericard, o probă de lichid ar trebui să fie pusă deoparte și evaluată frecvent pentru a observa dacă se formează coaguli. Lichidul din pericard nu ar trebui să coaguleze, cu excepția cazului în care provine dintr-o hemoragie în curs de desfășurare sau de la o hemoragie foarte recentă. O mostră de lichid poate fi centrifugată și examinată pentru a stabili dacă are caracteristicile efuziunii pericardice și pentru calcularea hematocritului. Supernatantul din efuziunea pericardică este de foarte multe ori xantocromic, iar hematocritul este adesea mult mai mic comparativ cu cel din sângele periferic. Monitorizarea electrocardiografică (ECG) ar trebui să fie efectuată în timpul pericardiocentezei. Contactul cateterului sau acului cu cordul poate induce ectopie supraventriculară sau ventriculară și poate necesita retragerea cateterului și/sau tratamentului antiaritmic.

La pisică, se recomandată efectuarea pericardiocentezei ecoghidate atât pentru reducerea marjei de eroare, cât și datorită faptului că volumul efuziunii pericardice este redus. De asemenea, pisicile au nevoie, aproape întotdeauna, de un anumit nivel de sedare. ■

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Effective management of the diabetic cat

Margie Scherk, DVM, Dip ABVP (feline practice)

catsINK, Vancouver, B.C., Canada

Abstract

Diabetes mellitus is one of the two most common endocrine disorders in cats. While we tend to think of diabetes as a disease entity, we should remember that it really is a heterogeneous group of disorders in which insulin production is reduced or in which tissue cells are resistant to the effects of insulin, resulting in impaired glucose homeostasis. From a clinical perspective, regardless of the cause, diabetes mellitus (DM) can be challenging to diagnose and treat in the cat because of their stress-induced hyperglycemia.

Keywords: *diabetes mellitus, insulin, stress-induced hyperglycemia*

Pathophysiology review

Insulin is secreted after a meal, to facilitate utilization and storage of glucose, fat and amino acids in three primary tissues: liver, muscle and fat. A mild insulin deficiency results in decreased transfer of ingested nutrients into tissues causing mild to moderate hyperglycemia. Severe insulin deficiency not only hampers tissue uptake of ingested fuels, but also results in marked compensatory glucose overproduction along with excessive mobilization of the body's protein and fat stores. Combined with glucagon excess (relative or absolute), this results in an increased delivery of fatty acids to the liver, their oxidation to ketone bodies (beta-hydroxybutyrate, acetoacetate, and acetone), and a clinical state of ketoacidosis. Because there is no insulin available to deliver the glucose into the cells, cells starve and polyphagia with concurrent weight loss occurs. Unabsorbed glucose (hyperglycemia) spills into the urine drawing water with it. This causes polyuria and compensatory polydypsia.

Classification And Differentiation Between Type 1 And Type 2 Diabetes

In human diabetes, Type 1 refers to a condition of insulin dependency seen in people who are generally lean, young and prone to ketogenesis. It is caused by immune-mediated beta cell depletion, causing an absolute insulin deficiency. Type 2 DM usually occurs in the older human, often obese but less prone to the development of ketoacidosis. The underlying problem is one of insulin receptor and post receptor defects, interfering with insulin uptake by tissues. This insulin resistance and associated hyperglycemia, causes the beta cells to produce more insulin, thus this state is one of a relative insulin deficiency. Type 2 may be controlled, at least initially, with weight loss, diet and oral hypoglycemic agents.

Generally, diabetes is a disorder of the older, often overweight cat, similar to the Type 2 human patient. Risk factors include body weight >7 kg, older age (>10 years), male gender, neutered. Henso showed that increased body condition score (BCS) in nondiabetic cats is associated with increased circulating concentrations of IAPP and insulin. Obese cats appear to have a defect in insulin secretion along with lower tissue sensitivity to insulin. Unlike human Type 2, however, by the time the diagnosis of diabetes is made, most cats are insulin dependent although not prone to ketogenesis. In addition to these differences, cats may also develop diabetes secondary to primary pancreatic disease, endocrinopathies (acromegaly or hyperadrenocorticism), or drug therapy (glucocorticoids and progestins). Inflammation is another recognized predisposing factor for susceptible individuals to develop diabetes. Franchini has shown at a molecular level that the inflammation induced by bacterial or viral infection can, via molecules recognized by toll gate receptors, damage endocrine pancreatic tissue.

Additionally, in cats pancreatic islet amyloid deposits are believed to interfere with insulin secretion, and that oral hypoglycemics (such as the secretagogue sulfonylureas) may actually increase islet amyloid polypeptide (IAPP) deposition. IAPP is co-secreted with insulin. Islet amyloidosis occurs in 90% of humans with Type 2 DM (O'Brien).

Thus, feline diabetes shares several similarities with the disease in humans. Impaired beta-cell function, decreased beta-cell mass, insulin resistance that is often related to obesity, and pancreatic amyloid deposition are among these common features (Zini, March 2010). Unlike in humans, DM does not predispose cats to hypertension.

Diagnosis

In the stressed patient, epinephrine release causes hyperglycemia and glucosuria. Therefore, even in cats with history and clinical findings of polyuria/polydypsia, polyphagia, weight loss, hyperglycemia and glucosuria, it is essential to differentiate between this stress response and diabetes. This can be done through verifying that the hyperglycemia and glucosuria are persistent over time. However, because stress recurs a better option is to request that a fructosamine level be run on the previously collected sample. Fructosamine measures the protein bound glucose levels over the preceding 10 - 20 days. It can be affected by protein metabolism as well, hence hyperthyroidism, with more rapid muscle turnover, may result in artificially lower fructosamine values.

Urine ketone measurement is routinely performed in cats with diabetes mellitus to identify impending or established ketoacidosis. The urinary ketone dipstick test has a low sensitivity as it quantifies the less abundant ketone acetoacetate. Beta-hydroxybutyrate (beta-OHB)

is the predominant serum ketone. Determination of plasma beta-OHB concentration was shown to be a useful method to distinguish between diabetic and non-diabetic sick cats (Zeugswetter).

Therapy and management of the diabetic cat

Good glycemic control soon after diagnosis is associated with increased probability of remission and should be the goal of insulin therapy (Roomp, Marshall). In a study published in 2010, clinical remission of diabetes was evaluated. Ninety cats with newly diagnosed diabetes were followed until death or remission. Remission was defined as normoglycemia without insulin for >4 weeks. Likelihood of remission was found to be greater in older cats and in cats with higher body weight. Remission was less likely in cats with increased serum cholesterol and was of shorter duration when serum glucose was higher, i.e., less well regulated (Zini, Nov 2010).

Insulin choice: There are many types of insulin available: they are derived from several sources and have several durations of action. In the United States, the FDA has eliminated any animal sourced insulin from the market. Insulins are currently produced from human recombinant technology. Beef-pork and beef sourced insulins may be better suited to cats because of closer structural similarity to feline insulin.

Speed of onset and the duration of the insulin:

1. Regular (fast) - rapid onset of action (0.5h), max. effect (1-5h), end effect (8h)
2. NPH (intermediate) - onset of action (1.5h), max. effect (4-12h), end effect (24h)
3. Lente - onset of action (2.5h), max. effect (7-15h), end effect (24h)
4. Semilente - onset of action (1.5h), max. effect (5-10h), end effect (16h)
5. Combination: 70% NPH: 30% regular - onset of action (0.5h), max. effect (4-8h), end effect (24h)
6. Ultralente (long acting) - onset of action (4h), max. effect (10-30h), end effect (36h)
7. Synthetic insulin analogs: glargine and detemir (ultra-long acting) once a day in humans

These values are for comparison only and reflect human metabolism. **Insulin responses vary with the individual. Every cat is different and will respond differently to the insulin they take in the management of diabetes.**

Protamine zinc insulin (PZI) is a long-acting, beef-pork insulin that was considered by many to be the insulin of choice for cats because of its molecular similarity to feline insulin. Since November 2009, PZI-R, a human recombinant DNA insulin, has come on the veterinary market as ProZinc™.

Caninsulin™, an intermediate acting porcine insulin, has been available for over 15 years. Its peak activity is ~3 hours and duration of 6-10 hours. In the United States, this product is known as Vetsulin™.

Glargine (Lantus™) is a long-acting human recombinant DNA insulin analog that forms microprecipitates at the site of injection from which small amounts of insulin glargine are slowly released. Thus the glucose nadir occurs later than with PZI-R or a lente/ultralente insulin. Insulin detemir (Levemir™) is similar to, but may be more predictable in cats than glargine.

It is critical to know the **concentration** of the insulin you are using and to match the syringes to that strength. For correct dosing, insulin should be administered using syringes specifically calibrated for the strength of insulin used. For example, most insulins are 100 Units/ml (U100) and micro-fine or ultra-fine U100 syringes should be used with them. With U-100 insulin, when only small amounts of insulin are needed, using a 3/10cc or 5/10cc U-100 allows even the tiniest dose to be measured accurately.

Caninsulin™/ Vetsulin™ and ProZinc™ are U40 insulins, and U40 syringes must be used to dose appropriately. Use of U100 syringes for a U40 insulin risks miscommunication and tragic consequences.

While there are guidelines in choosing the starting dose of insulin for a patient, the maximum dose for that patient will be the dose that he/she needs to resolve his/her clinical signs of excessive urination and drinking, lethargy and weakness. The majority of cats require twice daily injections, regardless of the type of insulin selected.

Client counselling

Once the cat has been determined to be diabetic, client counselling is very important. Initially, most clients are intimidated at the thought of administering insulin injections. Booking a discharge or demonstration appointment with the nurse-technologist works well, as nurses are often more patient than veterinarians are at explaining and guiding the learning client.

At this appointment, review the pertinent facts about insulin storage (refrigerator), handling (gently), resuspension (gently), drawing up into the syringe, administration (upon exhalation of client, walk through the door of the tent, OR pull the tent over the needle, think canvas, practise on a cat using saline), single use only of insulin syringes for sterility and sharpness sake.

Show the client how to keep a diary, recording date, time of insulin administration, dose administered, activity level, BM, amount urinated (and size of clumps of clumping litter), amount eaten, and amount drunk (by difference, measure amount left in bowl the next morning).

Counsel on diet to be fed, as determined by the veterinarian. Lower carbohydrate, higher protein diets may be more effective for glycemic control. Controversy remains and there is no scientific consensus on carbohydrates: to date there is no clear evidence that they either cause or are contraindicated in the treatment of feline diabetes. The native diet for a cat (bird or mouse) is high protein, moderate fat, low carbohydrate, it is reasonable to feed this macronutrient profile for any cat. Cats should have free access to food all the time, rather than feeding twice daily.

Some cats refuse to eat the diets we recommend. For those patients and for clients unwilling/unable to offer those diets, here is a website which lists the protein and carbohydrate proportions of grocery store brands: http://www.sugarcats.net/sites/jmpeerson/. Other helpful

websites for clients to use for information, support and encouragement (including teaching techniques) follow: www.petdiabetes.com, www.felinediabetes.com, www.sugarcats.com and www.cat-dog-diabetes.com/cats-diabetes-mellitus.asp

Monitoring urine parameters at home is justified for:

- Cats with transient diabetes- to identify when/if glucosuria recurs
- Cats on oral hypoglycemics to determine if glucosuria resolves
- Cats previously or currently ketoacidotic - to monitor for ketones.

A really good chapter to use as a client handout may be found in Vet Clinics of North America: May 1995, pp 753-759, entitled: Home management of cats and dogs with diabetes mellitus: Common questions asked by veterinarians and clients, by Drs. Arnie Plotnick and Deb Greco.

Follow-Up Care And Monitoring

At the discharge time, book an appointment for a blood glucose curve and re-evaluation for 14 days later. Let the client know that you will call daily for the first 3-4 days, to be supportive and available for questions, to find out how the kitty is doing, and to ascertain that they are observing the parameters you need diarized for evaluation. Let them know that it is unlikely that the initial dose will be the perfect one, and that, as they approach the "right" dose for this cat, there will initially be a marked reduction in urine output and drinking, however, after 3-4 days, these amounts will increase again as the cat's glucose homeostasis re-equilibrates.

The timeline for care that the author uses is:

- Diagnose diabetes mellitus; start insulin, diet and diary;
- 10-14 days later: in-clinic BG curve, adjust dose, teach ear prick BGs, add BID BG monitoring to diary for practice;
- Another 10-14 days later: in-clinic BG curve, fructosamine, adjust dose;
- Subsequent BG curves are performed at home, follow-up by email, phone or fax to adjust dose;
- Recheck kitty q4-6 months (exam, fructosamine, U/A) as long as he/she is stable.

At the **blood glucose (BG) curve** appointment, hospitalize the cat with food and water, after weighing him/her and ascertaining what time the insulin was administered and what dose the client gave. Measure BG immediately, to get a starting level. Using a 25G needle works well, as a mere drop or two of blood are needed for the portable glucometers. Plot the values on a graph for easier interpretation. Submit a serum fructosamine as well to determine how the average glycemic control has been over the past 10-20 days.

Continue measuring the BG every 1-1.5 hours over a 12 hours period. Ear sampling and a calm, reassuring manner will help to minimize the stress (and its associated BG elevations) somewhat. Nevertheless, the readings generally will be higher than what is occurring at home, therefore it is imperative to read the client's diary and take the clinical signs into consideration when adjusting the insulin dose. Once the blood glucose goes up for two consecutive measurements, the curve can be stopped (Note this does NOT apply in the case of a cat in diabetic ketoacidosis).

Use of the marginal ear vein is an accurate and easy technique for the measurement of BG. It is a useful technique in the clinic and, if the concept is introduced to clients with confidence and compassion, many are willing to perform curves at home. In general, these curves are more accurate as the cat's stress level is lower. Additionally, it is valuable for clients to be able to measure a spot glucose if their cat "doesn't look right" before deciding to give insulin or not.

The goals of performing a BG curve are to determine:

1. whether the insulin is being absorbed
2. the glucose nadir (value and time to reach it)
3. the duration of insulin effect
4. the duration of insulin effect
5. and to assess the fluctuations of glucose levels in this individual patient!

When using glargine, the protocol for regulation and curving is somewhat different. The following recommendations come from Dr. Jacquie Rand:

- Measure glucoses every two hours for a minimum of 12 hours daily for the first three days. This is in order to determine whether hypoglycemia is occurring as well as to assess how long the insulin is lasting in the individual. After this initial three day period, dose adjustments are based on the pre-insulin BG (vs. nadir as with other types of insulin).
- If at a 7 day hospital recheck, the pre-insulin BG concentration is > 290 mg/dl (16 mmol/L), increase the dose by 1 U/cat. A 12 hours curve should be done on the following day to make sure that hypoglycemia is not occurring at this increased dose.
- Do not change the dose if the pre-insulin BG concentration is 220-290 mg/dl (12-16 mmol/L).
- The dose should be decreased by 0.5-1 U/cat if the pre-insulin BG concentration is <180 mg/dL (10 mmol/L). I f biochemical hypoglycemia is present, the dose should be decreased by 1 U/cat. If clinical signs of hypoglycemia are present, the glargine dose should be decreased by 50%.

If a BG drops below normal range (<80mg/dl or <4.4 mmol/l), the staff person should notify the veterinarian after offering the cat some palatable food, as he/she may wish to administer dextrose intravenously to avoid a **hypoglycemic crisis**. Signs of hypoglycemia include weakness, lethargy, trembling, head tilt, ataxia, coma and death. If a hypoglycemic cat is offered food and doesn't eat right away, or if signs are severe, then corn syrup should be rubbed on the oral buccal mucosa while preparing to administer an intravenous dose of 50% dextrose.

The "**Somogyi effect**" is rebound hypoglycemia-induced hyperglycemia. If the cat's BG drops too low, the body reacts by releasing catecholamines (epinephrine), glucagon, glucocorticoids and growth hormone. This causes a rapid release of glucose into the serum causing this rebound to occur. It is important to not be tempted to increase the insulin dose in these individuals, as this

would accentuate the problem and eventually cause a hypoglycemic crisis. "Spot checks" of BG levels should be avoided as they can be misleading and can mask a rebound effect, and be misinterpreted as needing more insulin.

Over the next month or two, by performing blood glucose curves, measuring serum fructosamine and reassessing the cat clinically and historically (diary) every 2 weeks, the insulin dose suitable for this patient will be determined. Thereafter, it is advisable to see the stable diabetic cat every 4 - 6 months for a fructosamine. Consider, also, on these rechecks, to collect a sterile urine sample for urinalysis, as diabetic cats are more prone to bacterial urinary tract infections than non-diabetic individuals. If a diabetic patient becomes ill, then a glucose curve should be run as well as any other tests appropriate to their condition.

Update on glucometers

In a study comparing AlphaTRAK, Ascensia ELITE and reference hexokinase methods for determining serum glucose, the AlphaTRAK meter results did not differ from the reference method, however results from the Ascensia ELITE were significantly lower. The superior performance of the AlphaTRAK meter supports its use to monitor blood glucose levels in cats (Zini, 2009).

In a UK study (Dobromylskyj), six portable blood glucose monitors (PBGM) were compared to the reference method. Percentage of acceptable readings (unacceptable readings would result in a inappropriate clinical decision)

Meter 1: Accu-Chek Active (Roche): 95.3% (81 samples)
Meter 2: Ascensia Breeze (Bayer): 81.2% (69 samples)
Meter 3: Accu-Chek Compact (Roche): 96.5% (82 samples)
Meter 4: One-Touch Ultra (LifeScan): 85.9% (73 samples)
Meter 5: Supreme Plus (Hypoguard): 95.3% (81 samples)
Meter 6: Freestyle (TheraSense): 92.7% (77 samples)

Percentage of readings in zone where PBGM indicates opposite of reference, i.e., PBGM says hypoglycemic when reference says hyperglycemic (for instance, PBGM = 3.5 mmol/l; reference = 10 mmol/l) or PBGM says hyperglycemic when reference says hypoglycemic (for instance, PBGM = 14.5 mmol/l; reference = 2 mmol/l)

Accu-Chek Active (Roche): 2.4%
Ascensia Breeze (Bayer): 3.5%
Accu-Chek Compact (Roche): 2.4%
One-Touch Ultra (LifeScan): 3.5%
Supreme Plus (Hypoguard): 1.2%
Freestyle (TheraSense): 2.4%

Meter 3 had the smallest mean differences overall, together with the highest percentage of clinically acceptable readings.

Oral hypoglycemic agents

Sulfonylureas

The primary action of sulfonylureas (e.g. glipizide, glyburide) is to increase insulin release. Long-term success rate is estimated to be approximately 35%, but which cats will respond cannot be predicted. The ideal patient for treatment with glipizide is a stable, non-ketotic diabetic cat of optimum to obese body weight that has mild clinical signs with no complicating diseases. Patients that are emaciated,

dehydrated, debilitated have recently lost >10% of their body weight or have concomitant disease are not good candidates. Glipizide can be tried in any cat whose owners refuse to give injections.

Adverse effects are minimal. Vomiting is most common (approximately 15%). Increased liver enzymes and icterus develop within 4 weeks of initiating therapy in approximately 10%. Hypoglycemia occurs in approximately 12-15% of responder cats; usually these cats are transient diabetics. Most cats that respond without continued adverse effects can be treated with glipizide for life, but glipizide loses effectiveness in at least 5-10%. The period from initiation of therapy until failure is unpredictable, ranging from weeks to >3 years.

Glipizide treatment should be instituted at a dosage of 2.5 mg/cat PO BID with food, and the cat examined after 1 and 2 weeks. A history, complete physical examination, body weight, blood glucose concentration and urine glucose/ketones should be evaluated. If no problems occurred during the first 2 weeks, the dosage should be increased (5 mg/cat BID). If ketonuria is found, the medication should be discontinued and insulin therapy initiated. If vomiting or icterus is present, the drug should be discontinued until the problem resolves. Most cats will tolerate the medication if started at a lower dosage and gradually increased. If hepatic enzyme elevation or icterus occurred with the first administration, liver enzymes and serum bilirubin concentration should be checked periodically after re-initiation. If problems recur, the drug administration should be stopped and the cat placed on insulin.

Once a dosage of 5 mg BID has been given for 2 weeks, the previously mentioned parameters and a 10-12 hours glucose curve should be checked every 4 weeks. Response to therapy is evidenced by resolution of clinical signs, blood glucose concentrations during the curve <11-17 mmol/L and lack of glycosuria. Time until response varies, so therapy at the full dosage should continue for 12 weeks unless a contraindication develops.

If no response is seen after 12 weeks, glipizide administration should be stopped and insulin therapy instituted. If clinical signs and glycosuria resolve and blood glucoses are <200 mg/dl, glipizide therapy should be stopped and the serum glucose concentration re-evaluated in 1 week. If hyperglycemia is present then, glipizide should be reinitiated. If normoglycemia is present, no medication is warranted. Glipizide can be used again, however, at any time if hyperglycemia recurs. The patient should be rechecked every 3 months to ensure ongoing control.

Cats that have resolution of clinical signs according to the owner, stable body weight and normal physical examinations but serial blood glucoses > 17 mmol/L present a clinical dilemma. Either the clinical signs have not truly resolved or the hyperglycemia is due to stress. Cats such as this should ideally be monitored by serum glycated hemoglobin (GHb) or fructosamine concentrations to determine overall glycemic control. If these tests are not available, urine glucose can be monitored at home when the cat is not stressed. If glycosuria is absent or GHb or fructosamine concentrations are normal, glipizide therapy can proceed. If glycosuria is present or glycated protein levels are elevated, insulin should be used instead.

Transition metals

Transition metals are insulin-mimetic. Low doses (0.2 mg/kg/day) of vanadium decrease blood glucose and alleviate clinical signs in cats with early type II DM (D.S. Greco, personal observation). Unfortunately this is not a population we are likely to see often. In cats treated with vanadium, mild gastrointestinal signs may occur and one cat developed reversible renal failure. Large clinical studies on the effect of vanadium or chromium in diabetic cats are lacking.

Biguanides

The biguanides (e.g. metformin) inhibit hepatic glucose release and improve peripheral insulin sensitivity. Doses of 25-50 mg/cat BID should attain plasma concentrations used for treating human DM, but results in diabetic cats are not promising. In the single published study, 6 cats (5 newly-diagnosed, 1 insulin-treated) received metformin at a gradually increasing dosage. One cat was found dead after 2 weeks and no response was seen after 6-7 weeks in 4 cats. In 1 cat, glycemic control improved after 7 weeks (dose = 50 mg daily) and metformin was used successfully for 5 months. Side effects noted in healthy cats include inappetence, weight loss and vomiting.

Alpha-glucosidase inhibitors

Alpha-glucosidase inhibitors (e.g. acarbose) impair intestinal glucose absorption by decreasing fiber digestion and hence glucose production from food sources. In 5 dogs, a combination of acarbose and insulin provided better glycemic control over insulin alone. However, the final conclusion was that, due to expense and adverse effects, acarbose is primarily indicated for poorly controlled diabetic dogs for which the cause for the poor control cannot be identified. Acarbose may be administered at a dosage of 12.5-25 mg/cat BID with meals. Side effects include flatulence, semi-formed stools or diarrhea.

Thiazolidinediones

Thiazolidinediones increase target tissue sensitivity to insulin by binding to a novel receptor called the peroxisome proliferator-activated receptor- y (PPAR-y); they have received little attention for use in diabetic cats. Recent work suggests that darglitazone has beneficial effects in obese non-diabetic cats to decrease insulin secretion and glucose concentrations in a glucose tolerance test, but no work has been done in diabetic cats.

Acromegaly

Acromegaly has been studied in the last several years with an increased level of interest as it has been discovered that 1/4-1/3 of cats with diabetes may have unrecognized acromegaly. This condition is usually caused by an adenoma in the pars distalis of the anterior pituitary gland that secretes excessive growth hormone (GH). Less commonly, pituitary hyperplasia is suspected to result in acromegaly. Insulin-like growth factor 1 (IGF-1) is produced in the liver in response to the GH. GH has catabolic and diabetogenic effects, while IGF-1 has anabolic effects.

The characteristic signs of acromegaly are insulin resistance, believed to be caused by a GH-induced post-receptor

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Vetinnov

Experienţă practică: Înfiinţată în Octombrie 2003, Vetinnov este o companie condusă de un medic veterinar cu 17 ani experienţă în practica medicală şi chirurgicală în clinici veterinare.

Inovaţie în grupa produselor veterinare: Primul produs patentat, EasyPill® (drajeu pentru administrarea orală de tablete, comprimate) a beneficiat de sprijiin total din partea Agenţiei Franceze Pentru Invenţii şi Inovaţii OSEO. O echipă de ingineri, tehnicieni şi medici veterinari au lucrat la dezvoltarea acestor produse. Dupa 6 ani, s-a ajuns la o întreagă gamă de produse nutriţionale suplimentare foarte palatabile. Vetinnov comercializează produsele EasyPill® şi Vet Tasty-Treat® către cele mai importante companii farmaceutice din peste 20 de ţări.

defect in the tissues. Most are middle-aged to older, neutered male mixed breed cats. Physical changes consisting of prognathism and a broad face, large thickened limbs with clubbed paws and organomegaly may be subtle. Upper respiratory stridor associated with structural changes may be seen. Organomegaly is common as hypertrophic cardiomyopathy and renomegaly. In addition, arthropathies occur and, in some cases, there may be neurological signs from intracranial tumour expansion.

Classic signs of diabetes: PU/PD with polyphagia are present despite increasing doses of insulin. Uncharacteristic of diabetes, however, is concurrent weight gain. There are two populations of acromegalic cats: those who have been diabetic for some time and then deteriorate while the second group consists of those cats who appear to be acromegalic from the beginning of their diabetes.

Other differentials for an insulin resistant or uncontrolled diabetic include treatment failure of compliance or comprehension, inappropriate insulin handling, resistance associated with concurrent, uncontrolled inflammatory or infectious conditions, hyperprogesteronemia or hyperadrenocorticism.

Insulin growth factor-1 is the screening test with confirmation of diagnosis by imaging the pituitary gland. If possible, GH measurements should be measured. No single antemortem test is 100% reliable as there may be false positives and negatives. Because GH is secreted in a pulsatile fashion, there may be false negatives, i.e., normal GH values in an acroegalic cat. IGF-1 is secreted continuously and is, therefore, theoretically more reliable. Contrast enhanced CT or MRI studies are used for diagnosis as well as for treatment planning, should radiation or stereotactic radiosurgery be a consideration.

There are several therapeutic options. Conservative treatment with high doses of insulin as needed may be used, however the risk is that iatrogenic hypoglycemia may occur if the insulin dose is too high for the GH surge at the time. Thus, should this form of treatment be the one chosen, the client and veterinary team needs to work closely together to ensure that the client is able to assess blood glucose levels and trends.

Medical therapeutic options for people are of three kinds:

1. Somatotropin analogues control GH and IGF-1 secretion in about 50% of humans. Octreotride was effective in treating a small number of cats but did not result in normalization of GH after a single IV injection in one study.

2. Pegvisomant is a GH-receptor antagonist that is used in humans but does not appear to be effective in cats.

3. 70% of humans respond to dopamine antagonists such as bromocriptine and L-deprenyl (Selegiline). These have not been properly evaluated in cats.

Currently the best treatment option is radiation therapy: by reducing the bulk and function of the pituitary mass, neurological signs associated with mass as well as insulin resistance improve. Adjustment of insulin doses is not straightforward as resolution of insulin resistance can occur immediately or months after radiotherapy. Hepatic IGF-1 hyperproduction does not always resolve, so while diabetic management may become significantly easier or diabetes may resolve, the anabolic effects (polyphagia, bone growth, organomegaly, etc.) may still cause problems.

Stereotactic radiosurgery using a gamma knife to reduce the tumour mass is being investigated at Colorado State University. Another technique, transsphenoidal cryohypophysectomy, has been attempted in two cats with favourable long-term results in one cat.

Because there are chronic, ongoing changes associated with the effects of the IGF-1, namely possible arthropathy, HCM, renal insufficiency and hypertension, these, along with quality of life must be addressed regardless of form of therapy. ■

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ROYAL CANIN

Approach to feline iris melanoma

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Abstract

Feline iris melanoma is a common primary intra-ocular tumor with a high potential for metastasis. Ocular signs vary from focal to diffuse hyperpigmentation of the anterior iris, to involvement of the sclera and choroid. The evolution of initial clinical signs may take from months to years until metastasis occur. The approach to a case is awkward with early signs as hyperpigmentation can indicate iris freckles or the early stage of iris melanoma. The diagnostic and treatment approach is controversial, as some studies showed that early enucleation does not improve the prognosis significantly, and may induce early metastasis. Unless there are ocular complications such as uveitis or glaucoma, it may be preferable to choose a conservative approach and monitor the tumor's progression.

Keywords: *iris, melanoma, interferons, enucleation*

Introduction

Melanomas are the most common primary intraocular tumors of cats[13,14,15,18,29]. Ocular melanomas in cats are locally invasive and an early diagnosis is desirable to confirm clinical diagnosis, to establish the effective treatment and to determine the prognosis, as they have a high potential for metastasis[3,14,18,24,27,29,30]. In the cat, ocular melanomas are more common than oral and dermal ones, and ocular and oral ones are more malignant that dermal ones, with higher rates of mortality and metastasis[13,21,24]. Feline melanoma in the cat tends to involve the anterior uvea, iris and ciliary body[12,14,27,28,29], generally affecting cats more than 10 years of age with no breed or sex predisposition[8,9,15,23,24,29].

Metastatic potential

The site of the tumor, the thickness of the primary tumor or depth of invasion, scleral venous plexus involvement and the mitotic index are used histologically to predict biologic behavior and as prognostic indicators[14,29,30]. Histologically, feline anterior uveal melanoma is composed of melanocytes classified as spindle, plump spindle, and pleomorphic, and the iris appears to be the most common tissue of origin[14,24,28,30].

Metastatic disease was confirmed in 63% of 16 cats with intraocular melanoma reviewed in one series[15]. Feline intraocular melanoma is considered to have a greater metastatic potential than in dogs, the regional lymph nodes, liver and lungs being the main sites for metastasis[3,14,23,28,30]. One report described long bone metastasis that appeared 5 months post-exenteration for a large primary uveal melanoma in a cat[24].

Some studies have reported a slow growth rate of the tumour with a long period between detection and occurrence of clinical signs of metastasis up to 2 years following initial presentation[3,9,29,30]. That metastasis can occur in early stages, suggesting the benefit of early enucleation[15,16,29].

Clinical signs

Focal or diffuse iris hyperpigmentation (Figure 3) is frequently seen without an obvious mass lesion[19,22,23,26,29,30]. Focal areas of hyperpigmentation may persist for the life of the cat or may expand with no effect on vision or health (Figure 1, 1a, 1b)[16].

In some cats, over many months or years, the pigmented cells may infiltrate the iris stroma and the morphology of the cells can change with no clinical signs[10]. Histologically, the onset of melanoma is marked by the small angular cells turning into rounded cells with a round nucleus and a prominent nucleolus[10]. The hyperpigmented area may develop irregular iris masses (Figure 2) leading to anisocoria, dyscoria, infiltration of iridocorneal angle with tumor's cells and secondary glaucoma, corneal edema, hyphema and anterior uveitis[15,16,19,22,24,26,30].

Atypical melanoma appears to originate multifocally, from any part of the uvea other than the anterior iris and progresses more rapidly[14].

Differential diagnosis should include: iris freckles or nevi, melanosis, pigmented uveal cysts, iridal discoloration due to inflammation, melanosis secondary to chronic inflammation and other uveal neoplasia[14,22,25].

Diagnostic and therapeutic approach

A complete ophthalmic examination should be performed to assess the location and extent of lesion's involvement. Depending on the ocular signs presented, one might perform gonioscopy to examine the involvement of the iridocorneal angle, ultrasound and ultrabiomicroscopy to evaluate thickening of the iris root and cilliary cleft[23]. Ocular ultrasound

Figure 1. Russian Blue male cat, in 2005, eyes with no iris changes

examination will help to diagnose cysts that do not transilluminate because of excessive pigmentation (Figure 4)[30]. Ultrasound biomicroscopy uses high-frequency (40-MHz to 100-MHz)[5,15] waves to image the anterior segment of the eye and is useful to define the tumor shape and the extent of local invasion (Figure 5)[5].

Fine-needle aspiration biopsy. The prognostic and diagnostic value of fine-needle aspirates of the iridal surface is unclear but worthy of further study[23,24]. This technique has 90% accuracy, but may have several complications: hemorrhage, reflex uveitis and a low risk of tumor seeding in man[6]. Additionally, the risk of obtaining inaccurate biopsy samples is to be considered[23]. This technique had been performed in cats with variable results (Boydell, P. - Unpublished data).

In human medicine, a cannula aspirating technique has been developed for obtaining accurate diagnosis in 100% of the iris melanoma's cases, that provide sufficiently large sample size[17].

Figure 1a. Eyes in 2011, areas of iris hyperpigmentation, pupil size normal

Figure 1b. Eyes in 2012, progression of multiple hyperpigmented areas, consistent with iris melanosis, iris freckles or iris melanoma as differential diagnosis; no thickening of the iris (what would be the appropriate approach?)

A systemic examination should also be performed to evaluate to check for metastatic disease. This may include complete blood count and serum chemis-

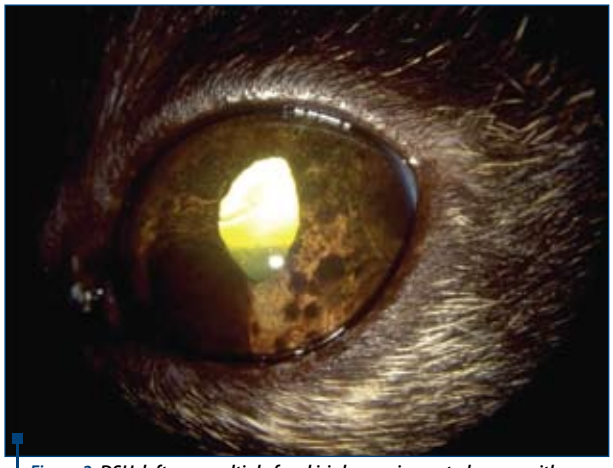

Figure 2. DSH, left eye, multiple focal iris hyperpigmented areas, with thickening of the iris ventro-nasally and misshapen of the pupil, likely to be iris melanoma

Figure 3. DSH, focal iris pigmentation in right eye

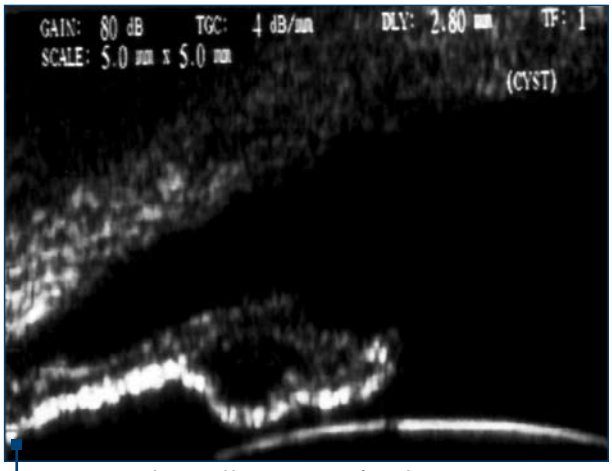

Figure 4. DSH, ultrasound biomicroscopy of uveal cyst

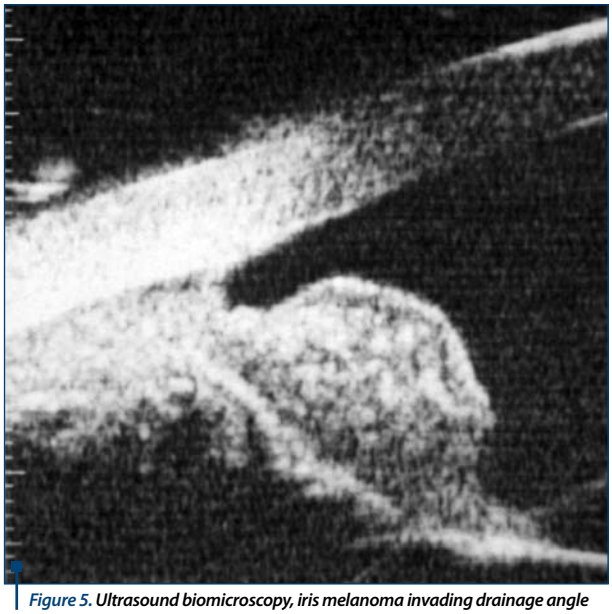

Figure 5. Ultrasound biomicroscopy, iris melanoma invading drainage angle

try panel, thoracic/abdominal radiography/ultrasonography. As many case-report studies revealed no evidence of metastasis and unremarkable blood changes at the initial presentation[23], the value of further investigations may be questionable.

Controversy exists regarding the best approach for feline iris melanoma[18]. The value of early enucleation in preventing metastasis has not yet been demonstrated[16,18,30]. Difficulty is that many cats may have only pigmentary changes of the anterior iris and no other intraocular signs, making the decision to enucleate difficult[30] as not all iris hyperpigmentation lesions are melanomas or malignant. Nevertheless, enucleation had been chosen in cats presenting only hyperpigmentation changes (iris freckles)[11,16,21,23] - Boydell, P. - Unpublished data.

Several therapeutic options in the management of uveal melanoma have evolved. In man, the choroidal melanomas are the most malignant of the intraocular melanomas, whereas in dogs they are unusual. For many years, it was believed that urgent and radical treatment of uveal melanoma was life-saving. Then, in 1978, Zimmerman and colleagues hypothesised that enucleation actually caused early metastatic death, either by disseminating tumour cells or by interfering with immunological or other defence mechanisms[31], performed on mice showed that enucleation of a melanoma-containing eye promotes intravascular showering of melanoma emboli that might develop into metastases[20].

Studies suggested that when enucleation is performed in early stages when only the iris stroma is involved, the survival time is the same as the control group with enucleation due to other ocular diseases[16]. However, the prognosis is poorer when enucleation is performed after cilliary body involvement[16,30]. Even with malignant transformation, cats with tumors confined histologically to the iris had good survival times, whereas cats with extensive ocular involvement or secondary glaucoma had shorter survival times[15]. The treatment of choice may depend on the tumor growth rate, the age of the patient and it may be appropriate only to observe the tumor[23,30].

If enucleation is not performed, frequent reevaluation and conservative treatment are recommended, with enucleation advised if progression such as increased iridal pigment changes, uveitis or glaucoma is observed[23]. The globe should always be submitted for histopathologic examination to confirm the neoplastic disease and to assess the prognostic histologic factors[2].

One study reported virally induced uveal melanoma, following the injection of an RNA tumor virus C-type Oncornavirus or retrovirus (FeLV-FeSV) into the anterior chamber of newborn kittens[1]. Immunohistochemical research on 10 cats with diffuse iris melanoma failed to detect feline leukemia virus/feline sarcoma virus, and reported that these are unlikely to play a role in the tumorigenesis of feline diffuse iris melanomas[8].

Diode laser photocoagulation

One study on 23 dogs reported non-invasive laser photocoagulation as a safe and effective method of treatment for isolated, pigmented iris masses in dogs, but with minor complications including dyscoria, iris hyperpigmentation and corneal edema[7]. The lesions exhibited no enlargements during the period of 6 months - 4.5 years, no glaucoma or cataract were reported, but five cases required more than one session, three eyes received two treatments and two cases received three treatments[7].

Diode laser photocoagulation was performed in 8 cats with suspected or confirmed iris melanoma but long-term results are not yet available (Boydell, P. - Unpublished data).

Interferons

Recently, interferons have proved useful in the treatment of human high-risk melanoma and have reduced the frequency of clinical recurrence[2], in humans, chemoimmunotherapy with a chemotherapy regimen including interferon alfa had been of benefit in approximately 20% of patients and contributed to prolonged survival[2,25]. The dose alfa-interferons have been administered in cats with suspected iris melanoma with no evidence of reported good long-term results but worthy of further studies (Boydell, P. - Unpublished data). In March 2011, the U.S. Federal Drug Administration approved administration of interferon-alfa for cutaneous melanoma in humans.

Summary

As diagnostic and treatment possibilities may be associated with the high risk of metastasis and ocular complications, enucleation is a controversial option. In case of focal anterior iris hyperpigmentation, trying to determine the nature of the lesion by fine-needle aspiration biopsy could lead to intraocular complications and early metastasis. The less invasive investigative procedures are the ultrasound scanning, ultrabiomicroscopy, tonometry and gonioscopy. Depending on the lesion's size and invasiveness in the absence of tumor-associated complications such as glaucoma and uveitis, it might be safer only to monitor its progression.

Conclusion

The decision making process in such cases is not easy. There are pros and cons to each approach option and the clinician must ensure that the client has sufficient information to allow an informed decision of how to proceed. This takes considerable time and effort and requires a degree of understanding on the client's part. Where there is uncertainty, the safest option may be to enucleate an eye where melanoma is suspected rather than biopsy to confirm but then one must be prepared to explain why there is no evidence of tumour on histologic examination of the excised globe. Enucleation of an eye where the appearance is strongly suggestive of melanoma may reduce the odds of metastasis compared to those odds if the eye were to be left in situ but it may take several years before secondaries become apparent. What would one advise if there was a possible early melanoma in the remaining eye of a one-eyed cat?

The authors' general recommendation is that administration of low dose interferon alpha, which has no reported side effects but is not licensed in any form for use in the cat, may be initiated for any abnormal iris pigmentation, and that the eye be enucleated if there is progression to thickening of the pigmented area and growth towards the iris base. Aspiration biopsy would be offered but not recommended. The owner must make the ultimate decision and, in light of the available evidence this should be supported by all clinicians involved. ■

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Fibrosarcom penian, hemangiosarcom splenic şi subcutanat la câine

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Prezentăm un caz clinic de fibrosarcom penian la un câine în vârstă de 8 ani, castrat, ce prezenta sângerare la urinare. Se atrage atenţia asupra importanţei integrării informaţiilor paraclinice în contextul clinic. Integrarea tuturor datelor anamnetice, clinice şi paraclinice duce la decelarea altor procese patologice, în afară de cele evidente cu care animalul se prezintă la medic.

Pacient: Bibi
Semnalmente: câine metis, castrat, vârsta >8 ani.
Motivul prezentării la medic: sângerare la urinare.

Anamneză

Hematurie la începutul urinării şi independent de aceasta, strangurie absentă, polakiurie absentă, disurie absentă; nu s-a putut preciza existenţa vreunei traume.

Restul anamnezei (consumul de apă, scaunul etc.) aparent normal.

Trebuie precizat că animalul era fără stăpân, iar datele anamnetice au fost limitate.

Examenul clinic

Greutate corporală: 20 kg - moderat subponderal, temperatura corporală: 39,3°C, alert, stare generală bună, tahipnee, tahicardie (frecvenţă cardiacă: 172 de bătăi/minut) - cel mai probabil din cauza stresului, tahisfigmie - amplitudine normală şi sincronizare cu bătăile cardiace, ascultaţia cardiacă şi respiratorie normală, mucoase aparente roz-pal şi umede, timp de reumplere capilară normal, hidratare normală, limfonoduli explorabili normali. La palparea abdominală s-a constatat splenomegalie moderată, fără reacţie de apărare. Tuseul rectal a evidenţiat prostata involuată. La nivelul ostiumului prepuţial era prezentă o cantitate mică de secreţie hemoragică; segmentul anterior al glandului cu aspect normal. S-a identificat o formaţiune de consistenţă dură-elastică situată pe zona de proiecţie a corpului penian şi a bulbului glandular, nedureroasă, neaderentă la tegumentul prepuţial sau la peretele abdominal, având dimensiuni aproximative de 8,5/5 cm; prolabarea penisului a fost imposibil de realizat, date fiind dimensiunile mari ale formaţiunii. De asemenea, s-a observat o formaţiune de consistenţă elastică, nedureroasă, neaderentă la piele şi aderentă la ţesuturile profunde, localizată în zona dorsolaterală lombară stângă, dimensiuni: 8/4 cm.

Diagnostic diferenţial

- **Hematurie** - originea poate fi renală, ureterală, vezicală, uretrală (calculi, inflamaţii, infecţii, neoplazii, traume), prostatică (prostatite, hiperplazia benignă de prostată, neoplazii), peniană (balanite, fracturi de os penian, tumori, inclusiv tumora veneriană Sticker) sau prepuţială (postite, tumori, traume);
- **Splenomegalie** - congestie, hiperplazie, hematopoieză extramedulară, inflamaţii, infecţii cronice, torsiune splenică;
- **Formaţiunea de pe zona de proiecţie a penisului** - abces, hematom, nodul sau tumoră;
- **Formaţiunea subcutanată** - abces, hematom, nodul sau tumoră.

Examene paraclinice

Examenul hematologic

- plasma clară, incoloră

	Valori de referinţă
Hct 24,8%	37-54%
Eritrocite 3,18x10^6/µl	5,4-7,8x10^6/µl
Hemoglobină 8,2g/dl	13-19 g/dl
VEM 78 fl	64-74 fl
HEM 25,8 pg	22-27 pg
CHEM 33 g/dl	34-36 g/dl
Reticulocite 98‰	0-10‰
# 311640/µl	0-60000/µl
Metarubricite 15%	<5%

Morfologie eritrocitară: policromazie, hipocromie, macrocitoză, anizocitoză, codocite frecvente, keratocite, schistocite, metarubricitoză, corpi Howell-Jolly frecvenți.

	Valori de referință
Leucocite 21,3x10^3/µl	6-17x10^3/µl

Având în vedere numărul mare al metarubricitelor (15%) și că aparatul de hematologie le numără ca leucocite (metarubricitul fiind o celulă nucleată), a fost nevoie de corecția numărului absolut al leucocitelor (aceasta se aplică la peste 5 metarubricite identificate la 100 de elemente figurate ale liniei albe).

Număr leucocite corectat: 21,3x100/(100+15)=18,5x10^3/µl

	Valori de referință
Neutrofile segmentate # 15170 (82%)	3000-11500/µl (60-77%)
Neutrofile nesegmentate # 0	0-300/µl (0-3%)
Eozinofile # 0	100-1250/µl (2-10%)
Bazofile # 0	rare (rare)
Limfocite # 740 (4%)	1000-4800/µl (12-30%)
Monocite # 2590 (14%)	150-1350/µl (3-10%)

Morfologie leucocitară normală.

	Valori de referință
Trombocite 517x10^3/µl	160-430x10^3/µl

Morfologie trombocitară: macrotrombocite prezente.

În urma examenului hematologic se poate observa prezența unei anemii macrocitare hipocrome regenerative moderate. Macrocitoza, policromazia, anizocitoza, codocitele și corpii Howell-Jolly frecvenți sunt elemente sugestive ale regenerării, fapt confirmat prin numărarea reticulocitelor. Metarubricitoza poate fi și ea un indicator al regenerării medulare, însă prezența keratocitelor și a schistocitelor a ridicat suspiciunea existenței unui proces patologic la nivelul patului capilar splenic sau hepatic. Gradul înalt regenerativ și proteina totală normală (vezi examenul biochimic) sunt indicatori ai unei anemii hemolitice, dar și ai unei anemii posthemoragice cronice. De aici rezultă că, în afară de componenta hemoragică a anemiei, există și posibilitatea prezenței unei componente hemolitice extravasculare (plasma clară, incoloră), însă de magnitudine redusă, bilirubina totală fiind normală (vezi examenul biochimic).

Leucocitoza cu neutrofilie matură, limfopenie, eozinopenie și monocitoza s-a încadrat în leucograma de stres.

Trombocitoza cu macrotrombocite indică o trombopoieză activă consecutivă hemoragiei cronice.

Examenul biochimic sangvin

Parametri	Valori de referință
ALT 26 U/L	10-100 U/L
PT 6,9 g/dl	5,2-8,2 g/dl
Alb 2,8 g/dl	2,2-3,9 g/dl
Bil-T 0,2 mg/dl	0-0,87 mg/dl
Chol 201 mg/dl	110-320 mg/dl
ALP 1253 UI/L	23-212 U/L
Glu 103 mg/dl	77-150 mg/dl
BUN 18 mg/dl	7-30 mg/dl
Cre 1 mg/dl	0,3-1,16 mg/dl
Ca 9,4 mg/dl	7,92-12 mg/dl
P 5,03 mg/dl	2,5-6,8 mg/dl
Amy 1113 U/L	500-1500 U/L
AST 64 U/L	10-50 U/L
CK 612 U/L	50-440 U/L
LDH 355 U/L	40-120 U/L
Fe	-
Echilibrul acidobazic şi electroliții	-

La examenul biochimic sangvin s-a constatat o creștere semnificativă a ALP, ce poate fi un element indicator al prezenței tumorale. Creșterea enzimelor tisulare CK, LDH și AST se explică cel puțin prin prezența unei distrucții de țesut la nivelul formațiunilor anterior descrise. Trebuie precizat că creșterea AST în lipsa creșterii ALT nu semnifică leziune hepatocitară.

Sideremia nu s-a efectuat întrucât nu a existat posibilitatea determinării fierului seric, iar echilibrul acidobazic și electroliții - din considerente financiare.

Examenul de urină

Examenul biochimic urinar

Sursa de urină	sondaj uretral
Culoare	galbenă
Turbiditate	absentă
Greutate specifică (refractometru)	1,048
pH	6,8
Proteină	++ (100mg/dl)
Glucoză	negativ
Corpi cetonici	negativ
Bilirubină	+ (0,5mg/dl)
Urobilinogen	normal
Sânge	+ (0,1mg/dl)
Leucocite	250/µl!

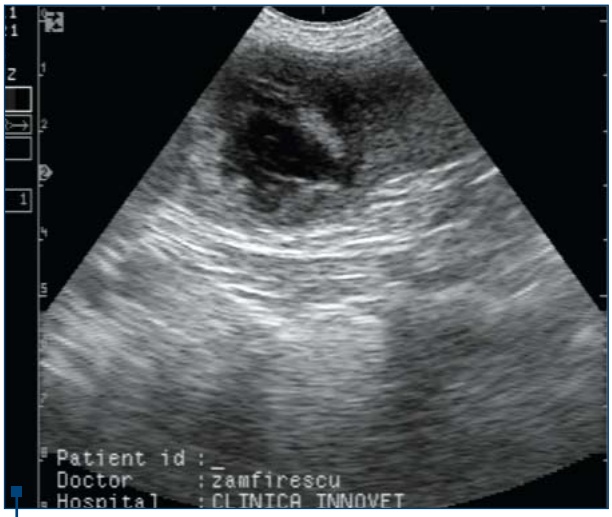

Figura 1

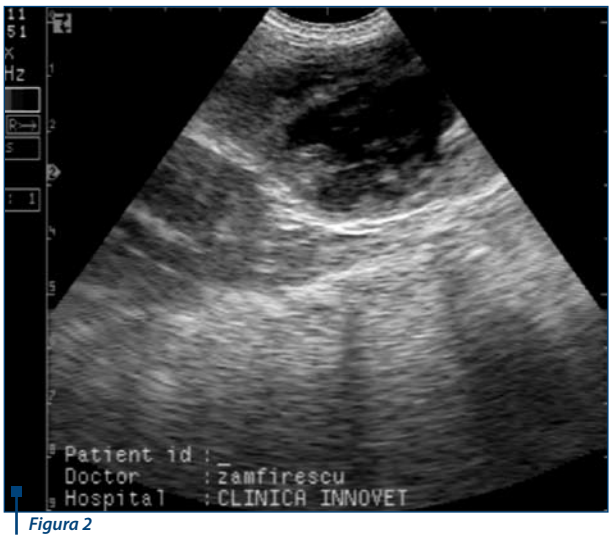

Figura 2

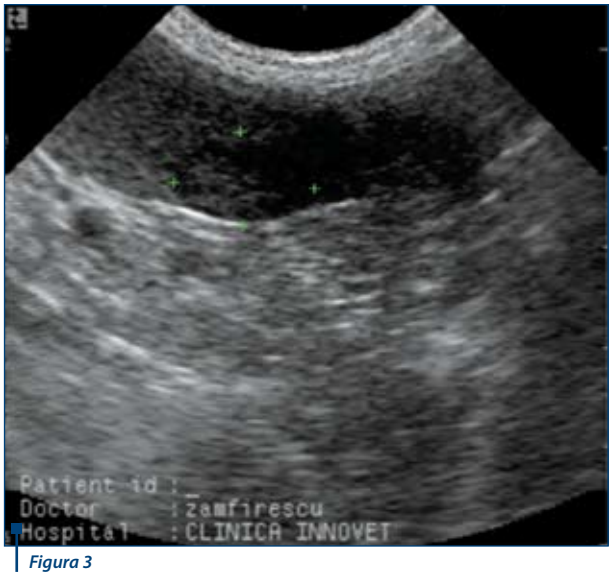

Figura 3

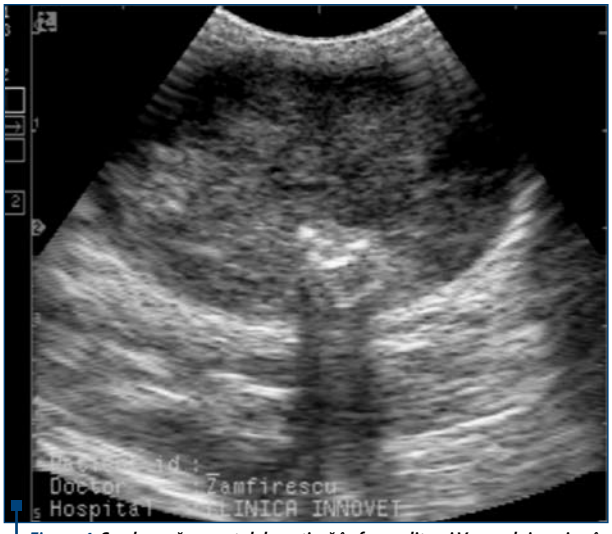

Figura 4. *Se observă aspectul de gutieră în forma literei V a osului penian în secțiune transversală și umbra acustică generată de acesta*

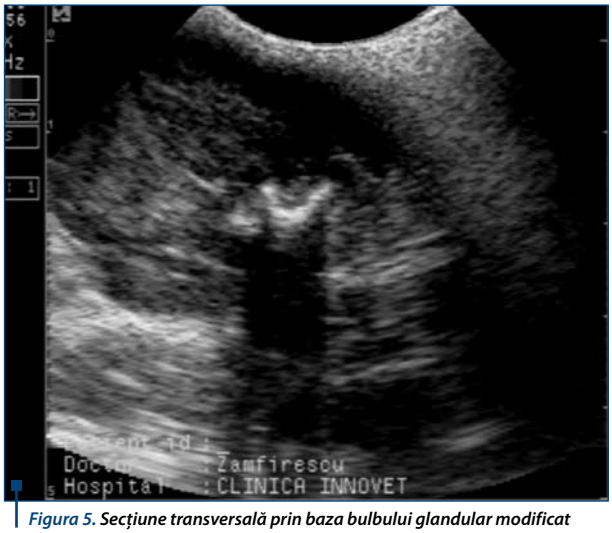

Figura 5. *Secțiune transversală prin baza bulbului glandular modificat*

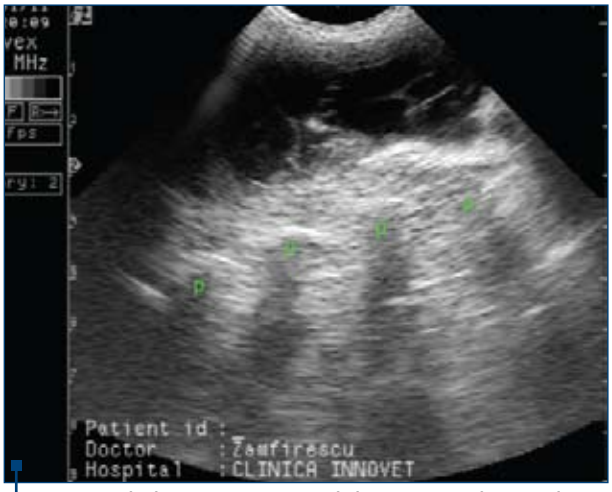

Figura 6. *Umbrele acustice cu aspect radial sunt generate de procesele spinoase (p) ale vertebrelor lombare*

■ **Sedimentul urinar**

Leucocite	0-2/hpf (normal 0-5/hpf)
Eritrocite	până la 10/hpf (normal 0-5/hpf)
Celule epiteliale	rare

hpf=high power field (x40).

Examenul de urină a evidențiat proteinurie moderată și hematurie microscopică. Având în vedere datele anamnetice, hematuria provenea cel mai probabil de la nivel penian, dar cateterismul sau o sursă de la nivelul tractului urinar superior putând fi incriminate, lucru ce trebuia confirmat sau infirmat. Am simțit nevoia să subliniez aici două elemente importante: unul legat de hematuria microscopică indusă de cateterismul uretral - niciodată nu se va evalua o probă de urină obținută în urma sondajului uretral (sau cistocenteza, după caz) pentru hematuria microscopică; celălalt legat de piuria biochimică afișată de aparatele de biochimie urinară care folosesc reacția diesterazică pentru identificarea leucocitelor, ceea ce duce foarte des la identificarea eronată a acestora în urină (în special la feline) - singura metodă de cuantificare a leucocitelor în urină rămâne sedimentul urinar.

Imagistic

Examenul ecografic:

■ Abdominal, s-au identificat:

✓ o formațiune ce deformează conturul splenic, aproximativ 4 cmØ, localizată în corpul splenic, delimitată de o „cămașă" cu ecogenitate crescută și contur interior anfractuos, centru anecogen ce generează distal o reducere a atenuării ultrasunetelor (figurile 1 și 2);

✓ un nodul intraparenchimatos ce realizează o discretă deformare a conturului splenic, contur oval în secțiune longitudinală având aproximativ 1,4/0,9 cm, situat în corpul splenic, izoecogen cu parenchimul splenic fiind delimitat de acesta de un contur hipoecogen; nu se constată fenomene de atenuare sau întărire posterioară (figura 3);

✓ restul parenchimului splenic și vascularizația - aparent normale ultrasonografic;

✓ nu s-au evidențiat alte elemente patologice la ecografia abdominală.

■ Penian - s-a constatat transformarea unui segment al corpului penian și bulbului glandular într-o formațiune cu contur neted, mediu-hipoecogenă neomogenă, ecotextura medie neomogenă, formațiune care în porțiunea cu grosime maximă avea aproximativ 4,47/3,53 cm abordare transversală (figurile 4 și 5).

■ Formațiunea cutanată - contur neted, 7/4 cm pe secțiune longitudinală, aspect multilacunar, fenomen de întărire posterioară evidentă (figura 6).

Având în vedere că la examenul ecografic s-a depistat o formațiune cavitară splenică, diagnosticul diferențial trebuia făcut între hiperplazia nodulară/hematom, abces sau tumoră. Întrucât starea generală a animalului era bună, nu era febril şi la examenul hematologic magnitudinea leucocitozei nu era mare şi nu s-au identificat neutrofile imature cu sau fără modificări toxice, era foarte puțin probabil să fie vorba de un abces splenic, astfel că în diagnosticul diferențial au rămas hiperplazia nodulară/hematomul şi tumora splenică.

Examenul radiologic toracic, în vederea depistării unor eventuale metastaze, nu s-a efectuat din rațiuni financiare.

Examenul ecocardiografic - pentru identificarea unor posibile tumori, fiind cunoscut faptul că aproximativ 25% din hemangiosarcoamele splenice metastazează în atriul drept - nu s-a efectuat tot din motive financiare.

Intervențional

Puncția ecoghidată cu ac fin prin tehnica mâinii libere:

- din formațiunea peniană: examen citologic → fibrosarcom.
- din formațiunea cutanată: examen citologic → neconcludent.

Tratament

Fiind vorba de o leziune cavitară splenică, s-a optat pentru splenectomie totală (figura 7). În cazul formațiunii peniene s-a recurs la amputarea penisului, cu efectuarea uretrostomei scrotale (figurile 8, 9, 10, 11) şi în cazul formațiunii cutanate la ablația acesteia.

Chimioterapia a fost refuzată de proprietar.

Diagnostic histopatologic

Hemangiosarcom splenic şi muscular, fibrosarcom penian.

Monitorizare

Şase luni mai târziu, câinele era normal din punct de vedere clinic (orice investigații ulterioare au fost respinse de proprietar).

Concluzii

În concluzie, se poate spune că integrarea tuturor datelor anamnetice, clinice şi paraclinice duc la decelarea altor procese patologice, în afară de cele evidente cu care animalul se prezintă la medic. Încă o dată se atrage atenția asupra importanței integrării informațiilor paraclinice în contextul clinic. ■

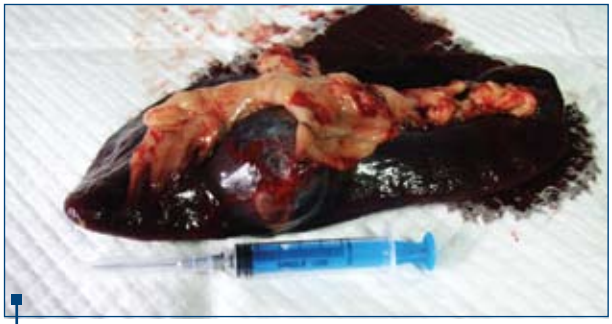

Figura 7

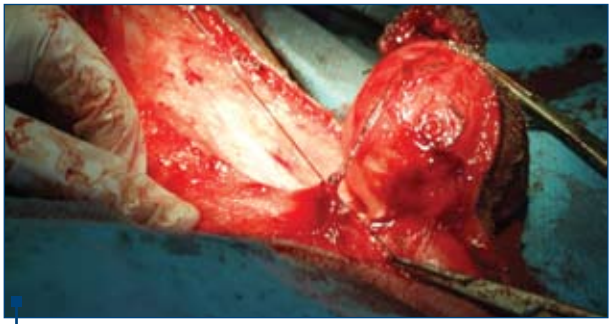

Figura 9

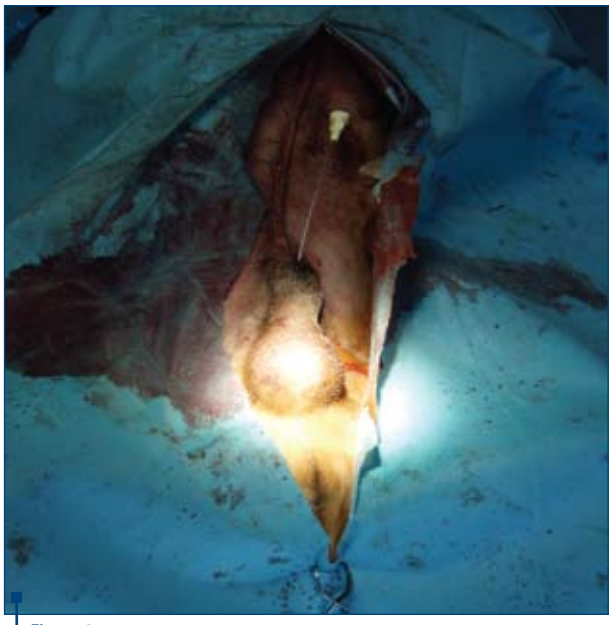

Figura 8

Figura 10

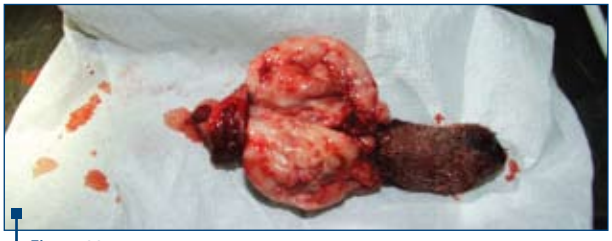

Figura 11

AMVAC
Asociația Medicilor Veterinari pentru Animale de Companie
RoSAVA
www.amvac.ro

CONGRES AMVAC

8-10 noiembrie 2012, Sinaia, România

Publicație acreditată de Colegiul Medicilor Veterinari din România

REVISTA ASOCIAȚIEI MEDICILOR VETERINARI PENTRU ANIMALE DE COMPANIE

Practica Veterinară +ro

Preț: 25 RON
Anul III • Nr. 8 (3/2012)

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A CONGRESULUI NAȚIONAL
ORGANIZAT DE CĂTRE
ASOCIAȚIA MEDICILOR VETERINARI PENTRU ANIMALE DE COMPANIE

SINAIA
8-10 NOIEMBRIE 2012

CENTRUL INTERNAȚIONAL DE CONFERINȚE CASINO SINAIA

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Congresul AMVAC 2012

AMVAC
Asociația Medicilor Veterinari pentru Animale de Companie
RoSAVA

CONGRES AMVAC/RoSAVA -2012
JOI 8 NOI 2012
PROGRAM STIINTIFIC

	Program		Program		Program
	SALA TEATRU 400 loc.		HOTEL PALACE SALA GEORGE ENESCU 120 loc		SALA CAROL I
	WSAVA DAY		CARDIOLOGIE WORKSHOP		
	IWSAVA Global Veterinary Development	08.00 08.45	1.Lectura EKG / Reading the EKG I 1 1/2 ore		
	INSCRIERE CONGRES RoSAVA	08.45 09.00	PAUZA DE CAFEA		
		09.00 09.45	1.Lectura EKG / Reading the EKG I 1 1/2 ore		
			JEAN FRANCOIS ROUSSELOT		
		09.45 10.00	PAUZA DE CAFEA		
	MEDICINA FELINA		CARDIOLOGIE WORKSHOP		MANAGEMENT
13.00 13.45	Therapeutic Implications of Renal Insufficiency: new thoughts / Implicatiile terapeutice ale insuficientei renale, noutati	10.00 10.45	Lectura imaginii eco-cardiografice / Reading the Cardiac Ultrasound I 1 1/2 ore		Program Workshop Management AMVAC 2012
	MARGIE SCHERK		JEAN FRANCOIS ROUSSELOT		
14.00 14.45	Untangling the complexities of the FLUTD complex / Descifrand complexitatile FLUTD	11.oo 11.45	Lectura imaginii eco-cardiografice / Reading the Cardiac Ultrasound I 1 1/2 ore		Strategia si planificarea activitatii unui cabinet veterinar
	MARGIE SCHERK		JEAN FRANCOIS ROUSSELOT		
14.45 15.15	COFFEE BREAK / PAUZA DE CAFEA		CARDIOLOGIE WORKSHOP		
	MEDICINA FELINA				SALA CAROL I
15.15 16.00	Dealing with that darned diabetic cat / Tratand pisica diabetica -I-	12.00 12.45	Diagnosticul si tratamentul fibrilatiei atriale,extrasistolele si tahicardia ventriculara / The Diagnostic and Treatment of Atrial Fibrilation, Extrasistoles and Ventricular Tachycardia 45min	15.10 - 16.00	Repere, cifre, procente, idei.... Indicatori de profitabilitate, preturi, cheltuieli
	MARGIE SCHERK		JEAN FRANCOIS ROUSSELOT		CRISTI MATURA
16.15 17.00	Dealing with that darned diabetic cat / Tratand pisica diabetica-II-	13.00 13.45	Diagnostic et traitement de l' hypertension artérielle/Diagnosticul si tratamentul in hipertensiunea arteriala.45min	16.10 - 17.00	Strategia in libera practica veterinara Planul strategic al unui cabinet veterinary Pregatirea si dezvoltarea unui plan strategic Modele de evaluare a unui cabinet veterinary
	MARGIE SCHERK		JEAN FRANCOIS ROUSSELOT		CRISTI MATURA
17.00 17.30	COFFEE BREAK / PAUZA DE CAFEA			17.00 17.10	COFFEE BREAK / PAUZA DE CAFEA
	MEDICINA FELINA				MANAGEMENT
17.30 18.15	Mellow Yellow: Winning with Hepatic Lipidosis / Castigand in lupta cu lipidoza hepatica			17.10 - 18.00	Alegerea unei strategii competitive Implementarea planului strategic Dezvoltarea si mentinerea unui brand de succes
	MARGIE SCHERK				Masa rotunda Lider sau manager intr-un cabinet veterinar ?
18.30 19.15	Triaditis A Troublesome Threesome / Triadita ("colangită, pancreatită IBD"): O treime problematica			18.10- 19.30	Managementul schimbarii - incep sa se schimbe regulile pietei ? Are cabinetul veterinar nevoie de manager ? Dar de lider ? Liderul si... liderul de succes. Rolul si functiile unui lider. Alegerea oamenilor potriviti si importanta feedback-ului.
	MARGIE SCHERK				CRISTI MATURA
	Medium lecture		Advanced Lecture		I - Interactiv
	WORKSHOP		WORKSHOP		WORKSHOP
	SIMULTANEOUS TRANSLATION ENGLISH-ROMANIAN		- Lectii tinute in limba Engleza		- Lectii tinute in limba Romana
			- Lectii tinute in limba Romana		- Lectii tinute in limba Romana

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VINERI 9 NOI 2012

PROGRAM STIINTIFIC

	Program	Program	Program	Program
	SALA TEATRU 400 loc.	SALA FERDINAND 200 loc	SALA N.GRIGORESCU 120 loc	HOTEL PALACE SALA GEORGE ENESCU 120 loc
	DERMATOLOGIE	MANAGEMENT	URGENTE	ENDOCRINOLOGIE
08.30 09.15	Economical considerations in the treatment of Canine Atopic Dermatitis/Considerente economice, în tratamentul dermatitei atopice canine	Managementul Clinicii veterinare-CAND, CE si CUM comunicam ! / Management of the Veterinary Practice - WHERE,WHY and How Do We Communicate !	Edemul pulmonar-abordare clinica si terapeutica diferentiata / Pulmonary Edema - Clinical Approach and Differntiated Therapy	Pancreatitele / Pancreatitis
	ALBERTO MARTIN CORDERO	CRISTI MATURA	MARIUS CODREANU	ANDRONIE VIOREL
09.30 10.15	2.Atopic Dermatitis: Short, middle and long term management / Dermatita atopica: Managementul de de durata scurta, medie si lunga	Management clinicii veterinare - Ce ar trebui să ştim când ne hotărâm să deschidem o clinică veterinară/Management of the Veterinary Practice - What we should know when we decide to open a veterinary clinic !	Anesthesia in Birds/Anestezia la pasari	Chirurgia aparatului genital la carnivore: simplu sau complicat / Surgery of Reproductive Organs in Carnivores : Simple or Complicated
	ALBERTO MARTIN CORDERO	CRISTI MATURA	NORIN CHAI	ALEXANDRU DIACONESCU
10.15 10.45	PAUZA DE CAFEA & EXPOZITIE	PAUZA DE CAFEA & EXPOZITIE	PAUZA DE CAFEA & EXPOZITIE	PAUZA DE CAFEA & EXPOZITIE
	DERMATOLOGIE	WORKSHOP MEDICINA FELINA	EXOTICE	CHIRURGIE RECONSTRUCTIVA
10.45 11.30	3.Practical approach to ear disease / Abordarea practica fata de afectiunile urechii	Feline friendly practice: get CATtitude: (interactive hours, with videos) (includes a lot of practice tips) / O clinica pentru feline cu atitudine (ore interactive, cu filme)(include multe sfaturi)	Surgery and Endoscopy in Birds / Chirurgia si endoscopia la pasari	Chirurgie des voies respiratoires; Paralysie Laryngée: Du diagnostic au traitement/Chirurgia cailor respiratorii;paralizia laringelui
	ALBERTO MARTIN CORDERO	MARGIE SCHERK	NORIN CHAI	GILLES DUPRE
11.45 12.30	Deschiderea oficiala a Congresului Amvac 2012 / Official Opening Ceremony of the 2012 RoSAVA Congress	Feline friendly practice: get CATtitude: (interactive hours, with videos) (includes a lot of practice tips) / O clinica pentru feline cu atitudine (ore interactive, cu filme)(include multe sfaturi)	Bacterial diseases in Exotics / Boli bacteriene la exotice	Chirurgie des voies respiratoires; Brachycéphales: Ce n'est pas seulement ce que vous croyez! / Chirurgia cailor respiratorii : Brahicefalicii : Nu sunt ceea ce credeti
		MARGIE SCHERK	NORIN CHAI	GILLES DUPRE
12.30 14.30	PRINZ & EXPOZITIE	PRINZ & EXPOZITIE	PRINZ & EXPOZITIE	PRINZ & EXPOZITIE
	DERMATOLOGIE	NEUROLOGIE	IMUNOLOGIE	CHIRURGIE RECONSTRUCTIVA
14.30 15.15	Clinical Approach to Allergic Skin Disease in Dogs and Cats / Abordarea clinica a bolilor alergice de piele la caini si pisici	Neurological examination/Examinarea neurologica	Clinical uses of colostrum derivated product in pets health:benefits/Beneficii ale utilizarii derivatelor de colostru pentru sanatatea animalelor de companie	Chirurgie mini-invasive (laparoscopie-thoracoscopie): Le futur est déjà là / Chirurgie minim-invasiva (laparascopie-toracospopie) - Viitorul este deja aici
	DANNY SCOTT	RICK LECOUTEUR,	GABRIELE BRECHIA	GILLES DUPRE
15.30 16.15	Differential Diagnosis for Allergic Reaction Patterns in the Skin of Cats / Diagnostic diferential pentru diverse moduri de reactie alergica la nivelul pielii la pisici	The Diagnostic Plan for Neurological Patients / Planul de diagnostic pentru pacientii neurologici	Solutii complexe in tratamentul afectiunilor tractului digestiv / Complex solutions in the Treatment of Gastrointestinal Diseases	Chirurgie oncologique: Les clés du succès/Chirurgia oncologica:cheia succesului
	DANNY SCOTT	RICK LECOUTEUR,	ALEXANDRU STALARU	GILLES DUPRE
16.15 16.45	PAUZA CAFEA & EXPOZITIE	PAUZA CAFEA & EXPOZITIE	PAUZA CAFEA & EXPOZITIE	de medicam
	DERMATOLOGIE	ORTOPEDIE	HEMATOLOGIE	MANAGEMENT
16.45 17.30	"Allergy Testing" in Dogs and Cats / Teste alergice la caini si pisici	ABCDE - how to score hips and elbows in 21st century? / Evaluarea bazinului si a coatelor in secolul XXI	Integrarea analizelor medicale in diagnosticul modern al afectiunilor medicale / Integrating Medical Analysis in Modern Diagnostic of Medical Conditions	Opinion poll relating to veterinary practice selection by pet owners/Sondaj referitor la criteriile de selectie ale cabinetelor veterinare de către proprietarii de animale de companie
	DANNY SCOTT	DENIS NOVAK	CRISTIAN POPOVICI	MIHAI CERNEA
17.45 18.30	Diagnostic si tratament in hipertiroidismul la pisică - Cazuri clinice / The Diagnostic and Treatment of Hyperthyroidism in cat - Clinical Cases	Patologii feline - Fibrosarcomul felin si Carcinomul scvamocelular - diagnostic si protocol terapeutic / Feline Phatology - Feline Fibrosarcoma and Squamocelular Carcinoma		The Utility of Molecular Biology Tests in Small Animals Pathology?/Utilitatea testelor de biologie moleculara in patologia animalelor de companie?
	SERBAN URSACHI	CRINGANU DAN		TURCITU MIHAI
	INCHIDEREA EXPOZITIEI	INCHIDEREA EXPOZITIEI	INCHIDEREA EXPOZITIEI	INCHIDEREA EXPOZITIEI
	North American Veterinary Conferance-NAVC	Advanced Lecture	M - Masterclass	Medium lecture
	PLATINUM	GOLD	SILVER	BRONZ
	lectures given in English	- Lectii tinute in limba Engleza	lectures given in English	lectures given in English
	lectures given in Romanian	- Lectii tinute in limba Romana	lectures given in Romanian	lectures given in Romanian

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CONGRES AMVAC/RoSAVA -2012

SÂMBĂTĂ 10 NOI 2012

PROGRAM STIINTIFIC

	Program	Program	Program	Program	
	SALA TEATRU 400 loc.	SALA FERDINAND 200 loc	SALA N.GRIGORESCU 120 loc	HOTEL PALACE SALA GEORGE ENESCU 120 loc	
	DERMATOLOGIE	DERMATOLOGIE	IMUNOLOGIE	ONCOLOGIE	
08.30 09.15	Clinical approach to allergic skin disease/Abordarea clinica a bolilor alergice	Demodecia / Demodicosis	Managementul bolilor infectioase transmise de ectoparaziti la animalele de companie / Management of Ectoparasite-Transmitted Infectious Diseases in Small Animals	Criterii de diagnostic pozitiv si diferential intre leucemii si reactiile leucemoide / Positive and Differencial Diagnostic Criteria Between Leukemia and Leukemoid reactions	08.30 09.15
	ALBERTO MARTIN CORDERO	ROBERT POPA	DRAGOS COBZARIU	EMILIA SALINT	
09.30 10.15	Medical Management of Allergic Dogs and Cats / Managementul medical in alergiile la caini si pisici	Hipertiroidismul la pisica / Hyperthyroidism in Cats	Managementul fracturilor / Fracture Management	Oncologia comparata,un nou concept,o noua varianta de practica medicala / Compared Oncology , a New Concept, A New Option of Medical Practice	09.30 10.15
	DANNY SCOTT	CRISTINA FERNOAGA	JOHAN VAN TILBURG	NICOLAE MANOLESCU	
10.15 10.45	PAUZA DE CAFEA & EXPOZITIE	PAUZA DE CAFEA & EXPOZITIE	PAUZA DE CAFEA & EXPOZITIE	PAUZA DE CAFEA & EXPOZITIE	10.15 10.45
	DERMATOLOGIE	IMAGISTICA	ORTOPEDIE	ONCOLOGIE	
10.45 11.30	Pursuing Anecdotes in Canine Dermatology / Cautand Anecdote in Alergia Canina	Imagistica prin Rezonanta Magnetica / Imagistics through Magnetic Resonance	Ligamentul incrucisat abordari practice / The Cruciate Ligament - Practical Approach	Mastocitomul / The Mastocytoma	10.45 11.30
	DANNY SCOTT	FLORIN GROSU	JOHAN VAN TILBURG	MILITARU MANUELA	
11.45 12.30	Recent Studies in Feline Dermatology / Studii recente de dermatologie felina	Mielografia, CT, IRM, cand si de ce? / Myelography, Ct MRI, When and Why?	Scheme de tratament in oftalmologie / Treatment Protocols in Ophtalmology	An Overview of Immune-Mediated Disease I / O privire de ansamblu asupra bolilor imuno-mediate I	11.45 12.30
	DANNY SCOTT	FLORIN GROSU	IULIANA IONASCU	MICHAEL DAY	
12.30 14.30	PRINZ & EXPOZITIE	PRINZ & EXPOZITIE	PRINZ & EXPOZITIE	PRINZ & EXPOZITIE	12.30 14.30
	NEUROLOGIE	IMAGISTICA	OFTALMOLOGIE	IMUNOLOGIE	
14.30 15.15	Localization of Nervous System Problems / Localizarea problemelor sistemului nervos	Radiography: tips and hints for taking good radiographs / Radiografia : Sfaturi si trucuri pentru radiografii bune	Testul cu fluoresceina sau testul Schirmer? / The Fluorescein or the Schirmer Test	An Overview of Immune-Mediated Disease II / O privire de ansamblu asupra bolilor imuno-mediate II	14.30 15.15
	RICK LECOUTEUR,	PETER VAN DONGEN	IULIANA IONASCU	MICHAEL DAY	
15.30 16.15	Neuromuscular Disorders of Dogs and Cats / Afectiuni neuromusculare la caini si pisici	Radiology: basic film reading / Radiologia: Interpretarea de baza a radiografiilor	Ophthalmic Emergencies I / Urgentele oftalmice I	An update on Vaccination I / Noutati in vaccinare I	15.30 16.15
	RICK LECOUTEUR,	PETER VAN DONGEN	PIP BOYDEL	MICHAEL DAY	
16.15 16.45	PAUZA CAFEA & EXPOZITIE	PAUZA CAFEA & EXPOZITIE	PAUZA CAFEA & EXPOZITIE	PAUZA CAFEA & EXPOZITIE	16.15 16.45
	NEUROLOGIE	IMAGISTICA	OFTALMOLOGIE	IMUNOLOGIE	
16.45 17.30	Localization of Spinal Cord Disorders / Localizarea afectiunilor coloanei vertebrale	Radiology of the thorax and abdomen / Radiologia toracelui si abdomenului	Ophthalmic Emergencies II / Urgentele oftalmice II	An update on Vaccination II / Noutati in vaccinare II	16.45 17.30
	RICK LECOUTEUR,	PETER VAN DONGEN	PIP BOYDEL	MICHAEL DAY	
17.45 18.30	Seizures & Epilepsy / Crizele si epilepsia	Radiology of the appendicular skeleton / Radiologia extremitatilor	Intracranial Emergencies / Urgentele intracraniene	Sterilitatea la caine / Sterility in Dogs	17.45 18.30
	RICK LECOUTEUR,	PETER VAN DONGEN	PIP BOYDEL	IANCU MORAR	
	INCHIDEREA EXPOZITIEI	INCHIDEREA EXPOZITIEI	INCHIDEREA EXPOZITIEI	INCHIDEREA EXPOZITIEI	
	Interactiv	North American Veterinary Conferance-NAVC	Prezentare Produs	Patologie EQUINE	
	PLATINUM	GOLD	SILVER	BRONZ	
	lectures given in English	lectures given in English	lectures given in English	lectures given in English	
	lectures given in Romanian	lectures given in Romanian	lectures given in Romanian	lectures given in Romanian	

Word before

Dear Colleagues and Friends,

It is the pleasure of the EEVC to welcome you all to our Professional Training course and The 7 meeting of the Eastern European Veterinary Conferance in the exquisitely beautiful city Sinaia in Romania.

The presentations from the first day are supported by MARGIE SCHERK in the framework WSAVA. Following 2 days for the congress include presentations supported by outstanding figures of veterinary medicine in the country and abroad, 3 workshops of Orthopedics, Cardiology and one the management of the feline medicine.

From the Comitee of organization of AMVAC/RoSAVA, we address all colleagues and friends (specialists and residents) the invitation to participate at this scientific and educational event.

We are waiting for you fondly!

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DVM Clinica Di-Vet Bucharest

Thursday Nov 08th 2012 Preconference Program

WSAVA - Conference Registration

08:30 - 10:15 - RoSAVA entry congress
10:15 - 10:45 - Coffee brake
10:45 - 11:30 - Speaker: MARGIE SCHERK (CDN)
Therapeutic Implications of Renal Insufficiency; new thoughts
11:45 - 12:30 - Speaker: MARGIE SCHERK (CDN)
Untangling the complexities of the FLUTD complex
12:30 - 14:30 - Lunch break
14:30 - 15:15 - Speaker: MARGIE SCHERK (CDN)
Dealing with that darned diabetic cat I
15:30 - 16:15 - Speaker: MARGIE SCHERK (CDN)
Dealing with that darned diabetic cat II
16:15 - 16:45 - Coffee break
16:45 - 17:30 - Speaker: MARGIE SCHERK (CDN)
Mellow Yellow: Winning with Hepatic Lipidosis
17:45 - 18.30 - Speaker: MARGIE SCHERK
Triaditis: A Troublesome Threesome

Friday November 09th 2012 Scientific Program

HALL THEATRE

08:30 - 09:15 - Speaker: ALBERTO CORERRO (Mexic)
Cheap and expensive ways to treat Atopic Dermatitis
09:30 - 10:15 - Speaker: TURCITU MIHAI (Bucharest)
The Utility of Molecular Biology Tests in Small Animals Pathology?
10:15 - 10:45 - Coffee break and EEVC exhibition
10:45 - 11:30 - Speaker: ALBERTO CORERRO (Mexic)
Atopic Dermatitis: Short, middle and long term management
11:45 - 12:30 - Speaker: ALBERTO CORERRO (Mexic)
Practical approach to ear disease
12:30 - 14:30 - Lunch break and EEVC exhibition
14:30 - 15:15 - Speaker: DANNY SCOTT (USA)
Clinical Approach to Allergic Skin Disease in Dogs and Cats
15:30 - 16:15 - Speaker:DANNY SCOTT (USA)
Differential Diagnosis for Allergic Reaction Patterns in the Skin of Cats
16:15 - 16:45 - Coffee break and EEVC exhibition
16:45 - 17:30 - Speaker:DANNY SCOTT (USA)
"Allergy Testing" in Dogs and Cats
17:45 - 18:30 - GALA VET MEDICA

Friday November 09th 2012 Scientific Program

FERDINAND HALL

08:30 - 09:15 - Speaker: CRISTI MĂTURĂ (Bucharest)
Management of the Veterinary Practice - What we should know when we decide to open a veterinary clinic!
09:30 - 10:15 - Speaker: CRISTI MĂTURĂ (București)
Management of the Veterinary Practice - WHERE,WHY and How Do We Communicate !
10:15 - 10:45 - Coffee break and EEVC exhibition
10:45 - 11:30 - Speaker: MARGIE SCHERK (Canada)
Feline friendly practice: get CATtitude: (2-3 interactive hours, with videos) (includes a lot of practice tips) I
11:45 - 12:30 - Speaker: MARGIE SCHERK (Canada)
Feline friendly practice: get CATtitude: (2-3 interactive hours, with videos) (includes a lot of practice tips)II
12:30 - 14:30 - Lunch break and EEVC exhibition
14:30 - 15:15 - Speaker: RICK LECOUTEUR (USA)
Neurological examination
15:30 - 16:15 - Speaker: RICK LECOUTEUR (USA)
The Diagnostic Plan for Neurological Patients**1**
6:15 - 16:45 - Coffee break and EEVC exhibition
16:45 - 17:30 - Speaker: DENIS NOVAK (Serbia)
ABCDE - how to score hips and elbows in 21st century?
17:45 - 18:30 - VET MEDICA GALA

Friday November 09th 2012 Scientific Program

HALL N. GRIGORESCU

08:30 - 09:15 - Speaker: NORIN CHAI (France)
Anesthesia in Birds
09:30 - 10:15 - Speaker: MARIO CODREANU (Bucharest)
Pulmonary Edema - Clinical Approach and Differntiated Therapy
10:15 - 10:45 - Coffee break and EEVC exhibition
10:45 - 11:30 - Speaker: NORIN CHAI (France)
Surgery and Endoscopy in Birds
11:45 - 12:30 - Speaker: NORIN CHAI (Franța)
Bacterial diseases in Exotics
12:30 - 14:30 - Lunch break and EEVC exhibition
14:30 - 15:15 - Speaker: GABRIELE BRECHIA
Clinical uses of colostrum derivated product in pets health: benefits
15:30 - 16:15 - Speaker: ALEXANDRU VITALARU
Complex solutions in the Treatment of Gastrointestinal Diseases
16:15 - 16:45 - Coffee break and EEVC exhibition
16:45 - 17:30 - Speaker: Cristian Popovici (CLUJ)
Integrating Medical Analysis in Modern Diagnostic of Medical Conditions
17:45 - 18:30 - VET MEDICA GALA

Friday November 09th 2012 Scientific Program

HALL G. ENESCU - PALACE HOTEL

08:30 - 09:15 - Speaker: ALEXANDRU DIACONESCU
Surgery of Reproductive Organs in Carnivores : Simple or Complicated
09:30 - 10:15 - Speaker: VIOREL ANDRONIE (Bucharest)
Pancreatitis
10:15 - 10:45 - Coffee break and EEVC exhibition
10:45 - 11:30 - Speaker: GILLES DUPRE (Austria)
Chirurgie des voies respiratoires; Paralysie Laryngée: Du diagnostic au traitement
11:45 - 12:30 - Speaker: GILLES DUPRE (Austria)
Chirurgie des voies respiratoires; Brachycéphales: Ce n'est pas seulement ce que vous croyez!
12:30 - 14:30 - Lunch break and EEVC exhibition
14:30 - 15:15 - Speaker: GILLES DUPRE (Austria)
Chirurgie mini-invasive (laparoscopie-thoracoscopie): Le futur est déjà là
15:30 - 16:15 - Speaker: GILLES DUPRE (Austria)
Chirurgie oncologique: Les clés du succès
16:15 - 16:45 - Coffee break and EEVC exhibition
16:45 - 17:30 - Speaker: MIHAI CERNEA(CLUJ)
Opinion poll relating to veterinary practice selection by pet owners
17:45 - 18:30 - VET MEDICA GALA

Saturday November 10th 2012 Scientific Program

HALL THEATRE

08:30 - 09:15 - Speaker: DANNY SCOTT (USA)
Medical Management of Allergic Dogs and Cats
09:30 - 10:15 - Speaker: ALBERTO CORERRO(Mexic)
Flushing and cleaning techniques of the ear canal
10:15 - 10:45 - Coffee break and EEVC exhibition
10:45 - 11:30 - Speaker: DANNY SCOTT (USA)
Pursuing Anecdotes in Canine Dermatology
11:45 - 12:30 - Speaker: DANNY SCOTT (USA)
Recent Studies in Feline Dermatology
12:30 - 14:30 - Lunch break and EEVC exhibition
14:30 - 15:15 - Speaker: RICK LECOUTEUR (USA)
Localization of Nervous System Problems
15:30 - 16:15 - Speaker: RICK LECOUTEUR (USA)
Neuromuscular Disorders of Dogs and Cats
16:15 - 16:45 - Coffee break and EEVC exhibition
16:45 - 17:30 - Speaker: RICK LECOUTEUR (USA)
Localization of Spinal Cord Disorders
17:45 - 18:30 - Speaker: RICK LECOUTEUR (USA)
Seizures & Epilepsy

Saturday November 10th 2012 Scientific Program

HALL FERDINAND

08:30 - 09:15 - Speaker: CRISTINA FERNOAGĂ
The cat Hhyperthyroidism
09:30 - 10:15 - Speaker: ROBERT POPA
Demodicosis
10:15 - 10:45 - Coffee break and EEVC exhibition
10:45 - 11:30 - Speaker: FLORIN GROSU (Bucharest)
Imagistics through Magnetic Resonance
11:45 - 12:30 - Speaker: FLORIN GROSU (Bucharest)
Myelography, Ct MRI, When and Why?
12:30 - 14:30 - Lunch break and EEVC exhibition
14:30 - 15:15 - Speaker: PETER VAN DONGEN (UK)
Radiography: tips and hints for taking good radiographs
15:30 - 16:15 - Speaker: PETER VAN DONGEN (UK)
Radiology: basic film reading
16:15 - 16:45 - Coffee break and EEVC exhibition
16:45 - 17:30 - Speaker: PETER VAN DONGEN (UK)
Radiology of the thorax and abdomen
17:45 - 18:30 - Speaker: PETER VAN DONGEN (UK)
Radiology of the appendicular skeleton

Saturday November 10th 2012 Scientific Program

HALL N. GRIGORESCU

08:30 - 09:15 - Speaker: JOHAN VAN TILBOURG
Fracture Management
09:30 - 10:15 - Speaker: DRAGOȘ COBZARIU
Management of Ectoparasite-Transmitted Infectious Diseases in Small Animals
10:15 - 10:45 - Coffee break and EEVC exhibition
10:45 - 11:30 - Speaker: JOHAN VAN TILBOURG
The Cruciate Ligament - Practical Approach
11:45 - 12:30 - Speaker: IULIANA IONAȘCU (Bucharest)
Treatment Protocols in Ophtalmology
12:30 - 14:30 - Lunch break and EEVC exhibition
14:30 - 15:15 - Speaker: IULIANA IONAȘCU
The Fluoresceine or the Schirmer Test
15:30 - 16:15 - Speaker: PIP BOYDEL (Marea Britanie)
Ophthalmic Emergencies I
16:15 - 16:45 - Coffee break and EEVC exhibition
16:45 - 17:30 - Speaker: PIP BOYDEL (Marea Britanie)
Ophthalmic Emergencies II
17:45 - 18:30 - Speaker: PIP BOYDEL (Marea Britanie)
Intracranial Emergencies

Saturday November 10th 2012 Scientific Program

HALL G. ENESCU - PALACE HOTEL

08:30 - 09:15 - Speaker: NICOLAE MANOLESCU
Compared Oncology , a New Concept, A New Option of Medical Practice
09:30 - 10:15 - Speaker: BALINT EMILIA (Bucharest)
Positive and Differencial Diagnostic Criteria Between Leukemia and Leukemoid reactions
10:15 - 10:45 - Coffee break and EEVC exhibition
10:45 - 11:30 - Speaker: MILITARU MANUELA
The Mastocytoma
11:45 - 12:30 - Speaker: MICHAEL DAY (UK)
An Overview of Immune-Mediated Disease I
12:30 - 14:30 - Lunch break and EEVC exhibition
14:30 - 15:15 - Speaker: MICHAEL DAY (UK)
An Overview of Immune-Mediated Disease I
15:30 - 16:15 - Speaker: MICHAEL DAY (UK)
An update on Vaccination I
16:15 - 16:45 - Coffee break and EEVC exhibition
16:45 - 17:30 - Speaker: MICHAEL DAY (UK)
An update on Vaccination II
17:45 - 18:30 - Speaker: IANCU MORAR(CLUJ)
Sterility in Dogs

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Clinical Approach to Allergic Skin Diseases in Dogs and Cats

Danny W. Scott, DVM

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Allergic (hypersensitivity) skin diseases are common in dogs and cats (Table 1)[1]. The most common of these are atopic dermatitis, flea-bite hypersensitivity, and food allergy. This lecture will focus on these "big 3".

The keys to recognizing allergic skin reactions in dogs and cats are:

1. Knowing the allergic reaction patterns of the skin (which are somewhat different for the two species).
2. A thorough history.
3. A thorough physical examination.
4. Diagnosing and treating concurrent diseases (bacterial infection, yeast infection, ectoparasite infestation).

Allergic cutaneous reaction patterns in dog

The most common reaction pattern in dogs is symmetrical, initially nonlesional pruritus. Dogs with atopic dermatitis and food allergy often have subtle (focal erythema; small, nonspreading erythematous papules) or no obvious primary skin lesions. However, licking/chewing/scratching will eventually lead to self-trauma and secondary bacterial/yeast infections. One dog may have regional symmetry (e.g., front paws; both ears; both axillae), and another may have multicentric symmetry (combinations of paws, ears, face, axillae, groin, rump).

Less common reaction patterns include symmetrical, pruritic papulocrustous dermatitis of rump, hind limbs, and ventral abdomen (flea-bite hypersensitivity) and urticaria (reaction to drugs, insects, and food).

Table 1 Allergic Skin Diseases in Dogs and Cats

Atopic dermatitis (atopy)
Flea-bite hypersensitivity (flea allergy dermatitis)
Food allergy (food hypersensitivity)
Allergic contact dermatitis (contact hypersensitivity)
Adverse cutaneous drug reaction (drug allergy)
Intestinal parasite hypersensitivity
Hormonal hypersensitivity*
Mosquito-bite hypersensitivity†
Dirofilariasis‡

*Dog only †Cat only ‡Dog only

Allergic cutaneous reaction patterns in cats

The common reaction patterns in cats are: (1) miliary dermatitis, (2) eosinophilic granuloma complex, (3) symmetrical self-induced hypotrichosis, and (4) symmetrical, initially nonlesional pruritus. These reaction patterns will be covered in detail in a subsequent lecture. Cats very rarely develop urticaria.

History

A thorough history of when and on what body site(s) the reaction pattern began, how it has progressed, and if the condition has seasonal, episodic, or locational (e.g., indoors versus outdoors) exacerbations is very important. Is the animal receiving drugs? If appropriate, should fecal flotations/heartworm tests be performed? If glucocorticoids have been given, what was the specific product/specific dosage/response? Food allergy and adverse cutaneous drug reactions may respond poorly to glucocorticoids (as do bacterial and yeast infections, and scabies).

Physical examination and concurrent dermatoses

Deviations from the above-described reaction patterns could indicate concurrent dermatoses. For instance, symmetrically pruritic dogs that have spreading annular lesions (papules, pustules, crusts, alopecia, collarettes) and/or spreading areas of erythema and waxy/greasy surface exudate probably have bacterial or yeast infections, respectively. These conditions must be diagnosed (e.g., cytology) and eliminated with treatment so as to see what the actual allergy looks like. Another example: the miliary dermatitis reaction pattern in cats has a lengthy differential diagnosis which will be discussed in a subsequent lecture.

Once the clinician has corroborated the existence of an allergic reaction pattern, specific testing procedures (e.g., elimination diet; drug withdrawal; fecal flotation/heartworm test; serological or cutaneous Aallergy testing@) can be implemented. In addition, the clinician can now implement medical management (to be covered in a subsequent lecture) and be able to better interpret clinical responses. ■

References

1. Scott DW, et al: Muller & Kirk's Small Animal Dermatology, 6th ed., Saunders-Elsevier, Philadelphia, 2001.

Differential Diagnosis for Allergic Reaction Patterns in the Skin of Cats

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Cornell University

The most common cutaneous allergies in cats are atopic dermatitis, flea-bite hypersensitivity, and food allergy[1]. The four classic cutaneous reaction patterns in cats are:

1. Papulocrustous dermatitis ("miliary dermatitis").
2. Eosinophilic granuloma complex.
3. Symmetrical, initially lesionless pruritus.
4. Symmetrical self-induced hair loss.

It is very important to remember that these reaction patterns: (a) do not indicate a specific allergy, (b) can be seen in various combinations in the same cat, and (c) can be produced by nonallergic diseases (Tables 1, 2, 3, and 4). ■

Table 1 Differential Diagnosis for Symmetrical Initially Lesionless Pruritus

Atopic dermatitis Food allergy	Otodectic mange Adverse cutaneous drug reaction

Table 2 Differential Diagnosis for Eosinophilic Granuloma Complex

Atopic dermatitis Food allergy Flea-bite hypersensitivity Mosquito-bite hypersensitivity	Staphylococcal infection Adverse cutaneous drug reaction Idiopathic

Table 3 Differential Diagnosis for Symmetrical Self-Induced Hair Loss

Atopic dermatitis Food allergy Adverse cutaneous drug reaction	Ectoparasites (fleas, *Cheyletiella*, *Otodectes*, *Demodex gatoi*) Hyperthyroidism

Table 4 Differential Diagnosis for Papulocrustous Dermatitis ("Miliary Dermatitis")

Atopic dermatitis Food allergy Flea-bite hypersensitivity Adverse cutaneous drug reaction Hypereosinophilic syndrome	Ectoparasites (*Cheyletiella*, *Otodectes*, *Lynxacarus*, chiggers [trombiculidiasis], lice) Infections (staphylococcal, dermatoph yte)

References

1. Scott DW, et al: Muller & Kirk's Small Animal Dermatology, 6th ed., Saunders-Elsevier, Philadelphia, 2001.

"Allergy Testing" in Dogs and Cats

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Serological determination of allergen-specific IgE has been commercially available in the United States since 1985, and the number of companies offering these tests has increased dramatically. It is disappointing that there is no governmental oversight and quality control of these companies. The veterinary profession must hope that the companies are performing appropriately.

This presentation will focus on the diagnosis of atopic dermatitis and food allergy in dogs and cats. As very few veterinary practitioners will perform intradermal testing, most of my comments will deal with serological testing. I will discuss atopic dermatitis and food allergy. It is essential to understand that "allergy tests" do NOT diagnose allergy. These tests indicate host reactivity to antigens, but do NOT document clinical disease.

Atopic dermtaitis

Atopic dermatitis is a genetically-predisposed inflammatory and pruritic allergic skin diseases with characteristic clinical features that is most commonly associated with IgE antibodies to environmental allergens[1]. Clinical signs may be seasonal (warm weather with pollens and molds; cold weather with house dust and danders), nonseasonal, nonseasonal with seasonal exacerbations, or progress from seasonal to nonseasonal. The majority of patients develop clinical signs between 6 months and 3 years of age. Familial involvement may be known. Certain dog breeds are predilected, and these vary from region to region. Clinical signs differ between dogs and cats.

Diagnosis is based on compatible history, physical examination, and exclusion of other diseases (food allergy, drug reaction, contact dermatitis, *Malassezia* dermatitis, bacterial folliculitis, ectoparasites etc.)[2]. Various diagnostic criteria[3-6] have been developed, with none being totally reliable nor internationally accepted.

Because most animals (normal, diseased, as well as atopic) will have positive serological tests, these cannot distinguish between normal and atopic individuals, and cannot be used for screening or diagnosis. Pitfalls inherent to allergy testing include:

1. **Timing.** Because some regions have distinct pollen and mold seasons, testing periods must be appropriately chosen.
2. **Drug Inhibition.** Glucocorticoids must be withdrawn for at least 3 weeks (pending the type of product used). I am also concerned about the long-term effects of cyclosporine.
3. **Parasitisms.** Ectoparasitisms and endoparasitisms can increase IgE levels and "false-positive" (clinically insignificant) reactions. It is advisable to diagnose and treat these, and do allergy testing 3 months later.
4. **Annual Vaccinations.** Annual vaccinations can increase IgE levels, and serological allergy testing should not be done until 8 weeks post-vaccines.
5. **Positive Reactions.** Even normal animals will have positive tests. Therefore, the veterinarian caring for the patient is the ONLY person (in concert with the owner) who can assess the significance of positive reactions.

Food allergy

The pathomechanism(s) of food allergy in dogs and cats is poorly understood. As serological tests only evaluate allergen-specific IgE and Type I hypersensitivity reactions, all other hypersensitivity reactions (e.g., Type III, Type IV) would not be detected. In addition, we know little about the exact allergen(s) involved, and the use of crude whole-food allergens is undoubtedly suboptimal. The cutaneous abnormalities of food allergy are indistinguishable from those of atopic dermatitis.

Diagnosis is based on compatible history, physical examination, and exclusion of other disease. We have been overwhelmed with novel protein and hypoallergenic (hydrolyzed) commercial foods. It is a shame that these are not carefully clinically assessed prior to marketing. History repeatedly shows that these diets fail to diagnose from 10 to 65% of food allergic dogs and cats[7,8]. The "gold standard" for the diagnosis of food allergy in dogs and cats remains the carefully chosen home-cooked diet. In addition, serological and intradermal testing are WORTHLESS for the diagnosis or exclusion of food allergy[7]. ■

References

1. Halliwell R: Revised nomenclature for veterinary allergy. Vet Immunol Immunopathol 114:207, 2006.
2. Olivry T, et al: The American College of Veterinary Dermatology Task Force on Canine Atopic Dermatitis. Vet Immunol Immunopathol 81:143, 2001.
3. Willemse TA: Atopic dermatitis: a review and reconsideration of diagnostic criteria. J Small Anim Pract 27:771, 1986.
4. Prélaud P, et al: Reevaluation of diagnostic criteria of canine atopic dermatitis. Rev Méd Vét 149:1057, 1998.
5. Favrot C, et al: A prospective study on the clinical features of chronic canine atopic dermatitis and its diagnosis. Vet Dermatol 20:23, 2009.
6. Terada Y, et al: Clinical comparison of human and canine atopic dermatitis using human diagnostic criteria (Japanese Dermatological Association 2009): proposal of provisional diagnostic criteria for canine atopic dermatitis. J Dermatol 38:1, 2011.
7. Scott DW, et al: Muller & Kirk's Small Animal Dermatology, 6th ed. W.B. Saunders, Philadelphia, Pennsylvania, 2001.
8. Olivry T, et al: A systematic review of the evidence of reduced allergenicity and clinical benefit of food hydrolysates in dogs with cutaneous adverse food reactions. Vet Dermatol 21:32, 2010.

Medical Management of Allergic Dogs and Cats

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Pruritus and the various aberrations of skin and hair coat that it provokes are, by far, the most common reasons for which dogs and cats are presented to veterinarians for dermatologic diagnosis[1].

Although many different dermatoses can be pruritic, and the differential diagnosis of pruritus is complicated, allergic (hypersensitive) skin diseases are certainly the most common causes of pruritus in dogs and cats.

Pruritus is defined as a sensation that elicits the desire to scratch[1]. The pathophysiology of pruritus is complicated and poorly understood for most diseases in most species. The literature is plethoric with information on various mediators and modulators of pruritus. However, the relative importance of these mediators and modulators in any given species, disease, or individual is rarely known.

In our practice, the most common reason for having difficulty in managing the allergic patient is failure to frequently reconsider the "threshold phenomenon" and the "summation of effects". Any "allergic" patient that is difficult to control or suddenly "comes out of control", needs to be reassessed for other problems (secondary bacterial pyoderma, secondary *Malassezia* dermatitis, flea infestation, dry skin, contact dermatitis etc.) before its allergy medicine is adjusted.

There are several categories of therapeutic agents, and many patients do better on combinations of these.

Systemic therapy

Glucocorticoids are, without a doubt, the most used and abused compounds in veterinary dermatology[1]. They are also the most consistently effective drugs in the management of allergic pruritus in dogs and cats, and can be used effectively and safely in many patients (Table 1). All glucocorticoids are not created equal. Thus, if a patient does not do well with one glucocorticoid, a different one may be more acceptable. Some cats and dogs do not appear to be able to convert prednisone to prednisolone; hence using the latter is more effective. The concept of "tolerable itchiness" must be stressed to the owners of dogs. Situations do arise wherein the use of glucocorticoids is undesirable or contraindicated. Examples would include: (1) objectionable acute or chronic side effects, (2) certain concomitant diseases (e.g., diabetes mellitus, pancreatitis, renal failure), (3) concurrent infections (bacterial, fungal, viral), (4) concurrent immunodeficiency states (e.g., FIV, FeLV), and (5) owners who are "cortisone" - or "steroid"-conscious. For these reasons, clinical and research interest in nonsteroidal antipruritic agents has "exploded" in the last several years.

Although nonsteroidal antipruritic agents are often useful in the management of allergic dogs and cats, they do not have an immediate antipruritic and anti-inflammatory effects. Hence, it is often necessary to give glucocorticoids along with the nonsteroidal agents for the first 3 to 7 days.

Table 1 Glucocorticoid Therapy in Dogs and Cats

Species	Drug	Dose*	Frequency*	Route
Dog	Prednisone/prednisolone‡	1 mg/kg	q24h	PO
	Methylprednisolone	0.8 mg/kg	q24h	PO
	Triamcinolone	0.2 mg/kg	q24h	PO
	Dexamethasone	0.1 mg/kg	q24h	PO
Cat	Methylprednisolone	20 mg/cat	†	SQ
	Prednisolone/prednisone‡	2 mg/kg	q24h	PO
	Triamcinolone	0.4 mg/kg	q24h	PO
	Dexamethasone	0.2 mg/kg	q24h	PO

**These doses and frequencies are for induction. For maintenance, the lowest alternate morning (dog) or evening (cat) dose achievable is indicated. In general, "safe" alternate-day doses of prednisone/prednisolone are <0.25 mg/kg (dog) or <0.5 mg/kg (cat). Triamcinolone and dexamethasone are not safe for alternate-day therapy in dogs, but may be acceptable in cats.*
†Can be given every 2 weeks for achieving remission (up to 4 times). Chronically, no more frequently than every 3 months.
‡Prednisolone is preferable.

Antihistamines

All "traditional" H_1-blockers have antihistaminic, anticholinergic, sedative, and local anesthetic effects[1,2]. They must be used with caution, if at all, in the presence of liver disease, glaucoma, urinary retention, gastrointestinal atony, seizures, pregnancy, and nursing bitches and queens. Responses are notoriously individualized and unpredictable. Thus, one often has to try several before the one that is "right" for the patient is found (Table 2). Each antihistamine should be tried for at least two weeks.

Concurrent antihistamine administration often allows reduced glucocorticoid doses. Antihistamines are often synergistic with Omega-3/-6 fatty acids and pentoxifylline.

Heterocyclic ("tricyclic") antidepressants

In addition to poorly-defined behavior-modifying properties, these agents are very potent H_1-blockers (Table 3)[1]. In addition to classic antihistamine side effects, these agents can also cause cardiac arrhythmias, lower seizure thresholds, and potentiate side effects of monoamine oxidase inhibitors (amitraz). Cardiac side effects have not been produced in dogs with normal cardiac function. Like antihistamines, heterocyclic antidepressants often act synergistically with glucocorticoids and Omega-3/-6 fatty acids.

Omega-3/Omega-6 fatty acids

Fatty acid supplements containing Omega-3/Omega-6 fatty acids are potent modulators of prostaglandin and leukotriene synthesis[1]. They rarely cause side effects. Numerous clinical trials have shown that these agents are useful in many allergic dogs and cats, and may also act synergistically with glucocorticoids and antihistamines. The literature is very confusing as concerns the "correct" dosage, ratio, and type of omega fatty acid to be used. Most of this is directly attributable to a failure to consider the patient's base diet. These products should be given for at least 3 weeks.

A commercial lamb and rice dog food with an Omega-6:Omega-3 fatty acid ratio of 5.5:1 was fed in a single-blinded, self-controlled clinical trial to 18 atopic dogs[3]. The pruritus in 8 of these dogs (44.4%) was controlled within 7 to 21 days, returned within 3 to 14 days after the diet was

Table 2 Antihistamine Therapy in Dogs and Cats†

Species	Drug	Dose	Frequency
Dog	Astemizole	0.25 mg/kg	q24h
	Brompheniramine†	0.5 mg/kg	q12h
	Azatadine	1 mg/dog	q24h
	*Chlorpheniramine†	0.4 mg/kg	q8h
	Cimetidine	6 mg/kg	q8h
	*Cetirizine†	1 mg/kg	q24h
	*Clemastine†	0.05-0.1 mg/kg	q12h
	Cyproheptadine	0.1-1 mg/kg	q12h
	*Diphenhydramine†	2 mg/kg	q8h
	Hydroxyzine†	2 mg/kg	q8h
	Loratadine	0.5 mg/kg	q24h
	Oxatomide	1-2 mg/kg	q12h
	Promethazine	1 mg/kg	q24h
	Terfenadine	5 mg/kg	q12h
	Trimeprazine	0.5 mg/kg	q12h
	Tripelennamine	1 mg/kg	q12h
Cat	*Cetirizine†	5 mg/cat	q12-24h
	*Chlorpheniramine†	2-4 mg/cat	q12h
	*Clemastine†	0.67 mg/cat	q12h
	*Cyproheptadine†	2 mg/cat	q12h
	Diphenhydramine	0.5 mg/kg	q12h
	Hydroxyzine	1-2 mg/kg	q12h
	Oxatomide	10-30 mg/cat	q12h

**Dr. Scott's favorite*
†Peer-reviewed publication(s) demonstrate efficacy

Table 3 Heterocyclic Antidepressant Therapy in Dogs and Cats†

Species	Drug	Dose	Frequency
Dog	*Amitriptyline†	1-2 mg/kg	q12h
	Clomipramine	1-3 mg/kg	q24h
	Doxepin	0.5-1 mg/kg	q12h
	Fluoxetine	1-2 mg/kg	q24h
	Imipramine	2-4 mg/kg	q24h
Cat	*Amitriptyline	5-10 mg/cat	q24h
	Buspirone	2.5 mg/cat	q12h
	Clomipramine	1.25-2.5 mg/cat	q24h
	Fluoxetine	1 mg/kg	q24h

**Dr. Scott's favorites*
†Peer-reviewed publication(s) demonstrate efficacy

withdrawn, and was again controlled when the diet was reinstated. Some of these dogs had failed to respond to recommended doses of commercial fatty acid supplements. The dog food supplied about 6 to 7 times the γ-linolenic acid (about 6 mg/kg) and eicosapentaenoic acid (about 9 mg/kg) as what is found in the commercial supplement. When the commercial dog foods being fed to these dogs were analyzed, tremendous variation in quantity, ratio, and types of omega-6/-3 fatty acids was found. In addition, it appears that atopic dogs have a partial deficiency in Δ6-desaturase and, in some cases, Δ5-desaturase activities. Hence, simply supplying these dogs with linoleic acid (omega-6) and α-linolenic acid (omega-3) may not be adequate. If the clinician really wants to know whether or not a dog will benefit from omega-6/-3 fatty acids, a commercial diet with controlled amounts and ratios is preferable.

Otherwise, selecting a commercial supplement without knowledge of the dog's base diet is fraught with misinterpretation and frustration.

Phosphodiesterase inhibitors

Phosphodiesterase inhibitors produce increased levels of intracellular cyclic AMP, which stabilizes cells and has anti-inflammatory effects[1]. Papaverine (150 to 300 mg/dog q12h PO) was not effective in pruritic dogs. Arofylline (1 mg/kg q12h PO) was effective in some dogs, but had a high incidence of gastrointestinal toxicity. Pentoxifylline (10 mg/kg q12h PO) produced significant reduction of pruritus in atopic dogs, but no dog was satisfactorily controlled[4]. Recent experience suggests that 25 mg/kg q12h is a more effective regimen. In 37 atopic dogs, pentoxifylline was effective alone in 19%, steroid-sparing in 10.5%, and synergistic with immunotherapy in 13.5%[5].

Pentoxifylline is synergistic with glucocorticoids, antihistamines, and omega fatty acids.

Pentoxifylline should be given with food for 4 weeks.

Leukotriene inhibitors

Zileuton is a 5-lipoxygenase inhibitor that is effective and safe in humans; but it was ineffective in atopic dogs (5 mg/kg q8h PO)[5]. In a double-blinded, placebo-controlled study with zafirlukast (0.5 to 1 mg/kg q12h PO) in atopic dogs, about 11% of the dogs had satisfactory control of their pruritus[6].

Cyclosporine

Cyclosporine is a potent inhibitor of T lymphocyte-dependent immune responses[1]. Its various effects include decreased IL-2, IL-3, IL-4, IL-5, TNF-α, and IFN-α production; inhibition of antigen presentation, eosinophil and mast cell production, histamine release from mast cells, neutrophil adherence, and growth and differentiation of B lymphocytes. The microemulsified forms are preferred (better absorption) over the original forms. Drugs that inhibit cytochrome P-450 enzymes (macrolides, azoles, tetracyclines, large doses of glucocorticoids) increase cyclosporine blood levels.

Recommended initial dosage is 5 mg/kg q24h PO for dogs and cats. Side effects are common (especially gastrointestinal) but usually mild. Cyclosporine was reported to be as effective as prednisone or methylprednisolone in atopic dogs (prednisone dose only half of what I use)[7]. Many dogs can eventually be controlled with 5 mg/kg q48h or even twice weekly. It appears that cyclosporine is more effective in early atopic dermatitis than it is in chronic disease. Recent studies have shown that cyclosporine therapy in dogs is associated

with disturbances in glucose metabolism[8] and frequent urinary tract infections[9].

Atopica® is not approved for use in dogs less than 6 months of age, dogs weighing less than 4 pounds, during pregnancy or lactation, or in malignant neoplasia. Patients on cyclosporine should probably receive only killed vaccines, and NOT be treated with ectoparasitic doses of avermectins.

Food does NOT reduce the clinical efficacy of cyclosporine in dogs. Cyclosporine is also effective for allergic pruritus, eosinophilic plaques, eosinophilic granulomas, and atopic dermatitis in cats[1,10]. Cats must be checked for FIV, FeLV, and *Toxoplasma* infection prior to therapy[11,12].

Miscellaneous

Agents found to be ineffective in pruritic dogs include aspirin, erythromycin, large doses of vitamin E, large doses of vitamin C, zinc methionine, doxycycline, the combination of tetracycline and niacinamide[1], and dextromethorphan[13].

Misoprostol is a prostaglandin E_1 analogue with anti-inflammatory properties, especially directed at the late-phase IgE reaction. Misoprostol (3 to 6 µg/kg q8h PO) produced a significant reduction in pruritus in some atopic dogs, but none of the dogs were satisfactorily controlled.

Gastrointestinal toxicity occurred in about one-third of the dogs[1].

Chinese herbal products[14] and commercial homeopathic remedies[15,16] have not been effective.

Topical therapy

Topicals can be useful adjuvants in the management of pruritus[1]. Moisturizing shampoos and rinses (colloidal oatmeal) reduce pruritus by rehydrating stratum corneum, re-establishing epidermal barrier funciton, and removing surface allergens, irritants, and microorganisms. Remember to use cool water. Added antipruritic effect can be attained with the local anesthetic pramoxine. A triamcinolone spray is useful for spot and regional therapy.

Tacrolimus is a topical macrolactam immunomodulator. Tacrolimus is a calcineurin inhibitor, thus inhibiting the activation and maturation of T lymphocytes and the activity of various cytokines (IL-2, IL-3, IL-4, IL-5, TNF-α). Unlike glucocorticoids, tacrolimus has no effect on collagen synthesis, thus avoiding the side effect of cutaneous atrophy. A 0.1% tacrolimus ointment was a useful spot treatment in several dogs with atopic dermatitis[17,18]. In humans, the most frequent side effect is burning at the site of application in up to 50% of patients. This burning is typically mild, transient, and decreased with continued application, but has not been recognized in dogs. In humans, ultraviolet light exposure is to be avoided (promotes photocarcinogenesis in the laboratory).

Tyrosine kinase inhibitors

Tyrosine kinase inhibitors (TKI) inhibit C-kit, Lyn, and Fyn, thus exerting antiproliferative effects on mast cells and inhibiting IgE-induced degranulation. A recent study suggested that masitinib (12.5 mg/kg/day) was useful for the reduction of pruritus in some atopic dogs[19]. However, there is concern over the frequency of proteinuria, hypoalbuminemia, and minimal change nephropathy in dogs and cats[19-21]. ■

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Pursuing Anecdotes in Canine Dermatology

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Residents in veterinary dermatology are often surprised to find out how much of what we say and do in our specialty is anecdotal. Let me share some recent anecdote hunts.

Localized demodicosis

Localized demodicosis was diagnosed in 46 dogs accounting for 0.6% of the canine dermatology cases and 0.1% of the canine hospital population over an 11-year period[1]. Seventy two percent of the dogs were less than 12 months old when examined. Rottweilers, collies, and German shepherds appeared to be over-represented. Lesions were most commonly seen on the periocular region, face, chin, and lip, but occurred in a variety of body sites. Lesions were asymptomatic and the dogs were otherwise healthy. No dog received miticidal therapy. Wherein follow-up was available (85% of the cases), all dogs spontaneously recovered and did not relapse.

Post-clipping hair follicle arrest

A retrospective study was conducted on 14 dogs with post-clipping hair follicle arrest over an 11-year period[2]. These dogs accounted for 0.2% of the canine dermatology cases. The dogs varied from 0.5 to 10 years of age when the close clipping occurred, with no apparent sex predilection. Close clipping occurred in all seasons of the year. Sled dogs, plush-coated dogs, golden retrievers, and American cocker spaniels appeared to be over-represented. All dogs regrew a normal hair coat after 7 to 30 months.

Schnauzer comedone syndrome

The Schnauzer comedo syndrome is a visually distinctive dermatosis of miniature schnauzers. The syndrome was diagnosed in 16 dogs, accounting for 0.2% of the canine dermatology cases and 0.04% of the canine hospital population over an 11-year period[3].

Interestingly, only two of the dogs were presented for only the syndrome, and 12 (75%) of the owners had not previously noticed the condition. Follow-up information was available for 10 (62%) of the dogs, and the syndrome was unchanged for 3 months to 9 years.

Idiopathic nasodigital hyperkeratosis

Idiopathic nasodigital hyperkeratosis is a visually distinctive disorder in dogs with a typical history and that are otherwise healthy[4]. The condition

FOTO: FOTOLIA

was diagnosed in 35 dogs, accounting for 0.4% of the canine dermatology cases and 0.1% of the canine hospital population over an 11-year period. English bulldogs, miniature poodles, miniature schnauzers, American cocker spaniels, and Doberman pinschers may be predisposed. Most dogs (71.4%) had only nasal involvement. The condition is usually asymptomatic, stable over time, and not reported to spontaneously resolve.

Fly-bite dermatitis

Fly-bite dermatitis was diagnosed in 35 dogs, accounting for 0.4% of the canine dermatology cases and 0.1% of the canine hospital population over an 11-year period[5]. Labrador retrievers appeared to be over-represented. Three different clinical presentations were recognized, and may be associated with the bites of *Simulium spp*. (black flies), *Chrysops spp*. (deer flies), or *Stomoxys calcitrans* (stable flies). The dermatoses occur during fly season in dogs that go outdoors. ■

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Recent Studies in Feline Dermatology

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Cetirizine for cats with allergic dermatitis

First generation (traditional H_1- antagonists) have antihistaminic, anticholinergic, and sedative effects. Second generation (nonsedating) H_1- antagonists cause minimal side effects.

Only four published clinical trials using antihistamines for the management of allergic pruritus in cats are available[1-4]. The antihistamines evaluated were all first-generation: chlorpheniramine, clemastine, cyproheptadine, and oxatomide.

Cetirizine is a second-generation antihistamine known to affect eosinophil function, and eosinophils are usually prominent in skin-biopsy specimens from allergic cats. A recent pharmacokinetic study of cetirizine in normal cats indicated that once-daily dosing (1 mg/kg) was appropriate, and side effects were not seen[5].

We have recently completed a clinical trial with cetirizine in 32 cats with allergic skin disease: 14 with atopic dermatitis, 16 with allergic dermatitis of undetermined cause (atopic dermatitis and/or food allergy)[2], with atopic dermatitis and food allergy. Cutaneous reaction patterns include self-induced alopecia, initially nonlesional pruritus, and eosinophilic granuloma complex[6]. In our study, 41% of the cats realized mild-to-moderate reduction in pruritus which was repeatable and sustainable. There was no association between cutaneous reaction pattern, age, or severity of pruritus and response to cetirizine.

Skin-biopsy findings in cats with allergic dermatitis

The significance of two histopathologic reaction patterns in cats with allergic dermatitis have been recently evaluated.

The prevalence of **infiltrative lymphocytic mural folliculitis** (ILMF) was evaluated in skin-biopsy specimens from 354 cats with various inflammatory dermatoses and from 33 cats with normal skin[7]. Although ILMF was present in 33/47 dermatoses studied, the prevalence of ILMF in allergic dermatoses was significantly greater than in nonallergic dermatoses. ILMF was not observed in normal skin.

The **depth of perivascular-to-interstitial eosinophilic inflammation** was evaluated in skin-biopsy specimens from cats with atopic dermatitis, food allergy, and flea-bite allergy[8]. Dermal inflammation was both superficial and deep in 93% of the cats. There was no difference in histopathological reaction pattern based on clinical diagnosis of clinical cutaneous reaction pattern.

Feline acne

Feline acne is an uncommon disorder of cats[9]. There is no apparent age, breed, or sex predilection. Owners are often not aware that their cat has acne. The etiopathogenesis of feline acne is unknown, and triggering factors such as stress, viral infections, allergies, lifestyle, and food/water bowl contact have not been corroborated.

About 58% of affected cats present with the asymptomatic comedone stage, and 42% present with secondary bacterial folliculitis/furunculosis. Cats with the asymptomatic comedonal stage can be observed or treated with various topicals. Cats with secondary bacterial folliculitis/furunculosis require topical and/or systemic antibiotic therapy. The comedonal stage persists for life.

Idiopathic eosinophilic granuloma in cats

In most instances, feline eosinophilic granulomas are associated with allergies, especially atopic dermatitis, food allergy, flea-bite allergy, and mosquito-bite allergy. Small numbers of cases have been attributed to ectoparasites (*Cheyletiella*, *Notoedres*, *Otodectes*), staphylococ al infection, foreign bodies (cactus tines, insect parts), allergic contact dermatitis, and idiopathy. A retrospective study was conducted on 55 cats with idiopathic eosinophilic granuloma[10].

Ninety-three percent of the cats had an age of onset of ≤4 years old. Lesions occurred most commonly on the lips, caudal thighs, and chin, and were usually asymptomatic. Papular-tonodular or linear lesions were seen in 70% and 30% of the cats, respectively. Seventy-eight percent of the cats received no treatment and - where follow-up information was available (67% of cases) - underwent spontaneous remissions with no relapses recorded. ■

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Bolile tractului urinar inferior la feline (BTUIF)

O actualizare științifică la tema Bolile Tractului Urinar Inferior la Feline (BTUIF)

Aproximativ 2-13% din totalul pisicilor care se prezintă în clinicile veterinare suferă de BTUIF. BTUIF însumează o serie de afecțiuni cu diverse etiologii și pentru care s-au identificat o serie de factori de risc, inclusiv consumul insuficient de apă, obezitatea și, mai recent, stresul. Indiferent de cauză, BTUIF sunt caracterizate de semne clinice comune neplăcute pentru pacienți și extrem de stresante pentru proprietari. Și mai îngrijorător este faptul că BTUIF obstructive necesită intervenție de urgență și pot pune în pericol viața pacientului. Cistita Idiopatică Felină (CIF), plăgile uretrale și urolitiaza sunt cele mai comune tipuri de BTUIF (55%, 21% și respectiv 21%). Majoritatea uroliților la pisici este reprezentată de complexe minerale de struviți sau oxalați de calciu a căror pondere a variat de-a lungul timpului. Semnele de BTUIF tind să reapară la pacienți, ceea ce subliniază importanța instituirii unor strategii de control al uroliltiazei atât cu struviți, cât și cu oxalați de calciu.

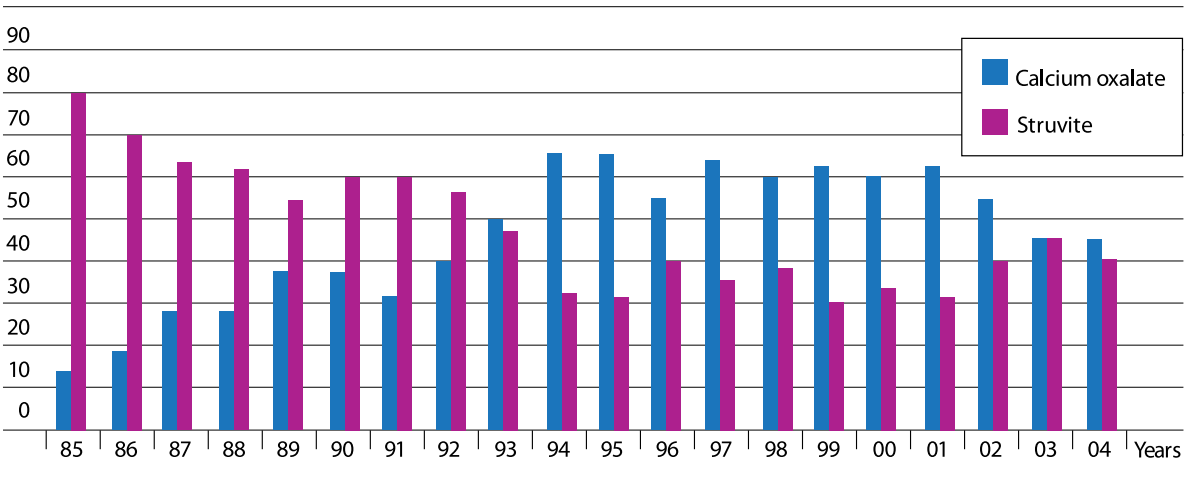

Figura 1. Evoluția procentuală a oxalaților de calciu și a struviților în urma analizei a 5.230 de cazuri[1] de urolitiază

Rata de Activitate a Produsului (APR), ultimul și cel mai puternic parametru pentru evaluarea eficacității dietelor urinare

Urolițiii se formează în medii în care cristalele precipită în soluții și agregă. Acest proces se desfășoară la nivel microscopic și implică o serie de factori care influențează, astfel că au fost foarte greu de anticipat situațiile în care urina a fost favorabilă sau nefavorabilă formării urolițiilor în condițiile în care pacientul consumă o anumită hrană. Din fericire, acum avem metode cantitative pentru a determina probabilitatea formării de cristale. Aceste metode sunt RSS (Super Saturație Relativă) și cea mai recentă și mult mai precisă metodă de determinare, Rata de Activitate a Produsului (APR), folosită de obicei în cercetarea umană, au fost folosite pentru a evalua eficacitatea dietelor urinare.

RSS este determinată prin măsurarea încărcării soluției cu minerale specifice, ca și pH-ul și volumul urinar. O valoare mare a RSS-ului (suprasaturație) înseamnă că echilibrul urinei este în favoarea precipitării atunci când dieta respectivă este dată în consum, în timp ce o valoare scăzută (nesaturată) înseamnă că echilibrul este în favoarea mineralelor care stau în soluție în loc să precipite sub formă de cristale. Valorile RSS rezultate sunt împărțite pe categorii în nesaturate, metastabile și suprasaturate.

Mai recent, Nestle Purina a fost prima companie de pet food care a folosit atât RSS, cât și APR, o altă metodă de măsurare, mult mai dinamică și sensibilă pentru determinarea riscului de formare a cristalelor și urolițiilor. Precum RSS, APR implică determinarea substanțelor dizolvate în urină și calcularea, dar APR este o metodă ce reflectă cu mai mare precizie ceea ce se întâmplă în situațiile *in vivo*. APR se determină prin analizarea mostrei de urină înainte și după incubarea cu cristal de struvit, oxalat de calciu sau alte tipuri de uroliți. Cu APR, cristalul care se dorește a fi analizat se adaugă în mostra de urină și se măsoară concentrația soluției înainte și după incubare. Concentrația soluției scade în mostra de urină care permite cristalului să crească în timpul incubării, în timp ce mineralele solubile cristalizează. Această mostră de urină are valoarea APR mai mare de 1, ceea ce indică creșterea riscului de formare a urolițiilor. În schimb, valorile APR mai mici de 1 indică faptul că acel cristal nu crește, ceea ce sugerează scăderea riscului de formare a urolițiilor[2]. Valorile APR mai mici de 1 indică de asemenea că urolițiii și cristalele din acea urină se dizolvă. Această metodă este mult mai complicată decât RSS și este rar folosită pentru evaluarea dietelor, oricum ea a fost utilizată pentru evaluarea noilor diete Purina Veterinary Diets Feline St/Ox și PRO PLAN CAT Sterilised.

Importanța volumului urinar și a greutății specifice a urinei

Creșterea aportului de apă este o strategie foarte recomandată pentru a ajuta la controlarea BTUIF și este susținută de numeroase publicații[3,4,5]. Creșterea aportului de apă duce la creșterea volumului urinar și la o urină mai diluată. Diluția urinei ajută la scăderea concentrației diferitelor componente urinare care duc la formarea urolițiilor, minimizând posibilitatea de formare a urolițiilor, diluarea mediatorilor inflamatori și un contact al substanțelor iritante cu mucoasa vezicală de mai scurtă durată.

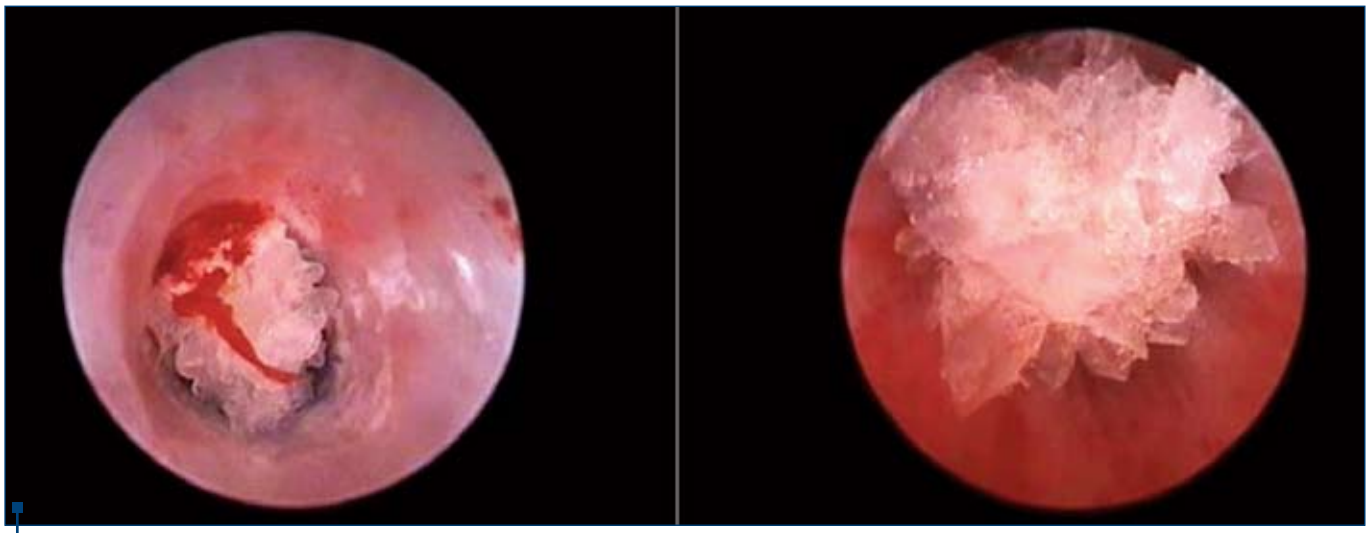

Figura 2. Dr. Chekroun/Nestlé-Purina. Imagine endo-uroscopică ilustrând uretroliți la pisica ce s-a prezentat în regim de urgență cu semene de BTUIF obstructivă

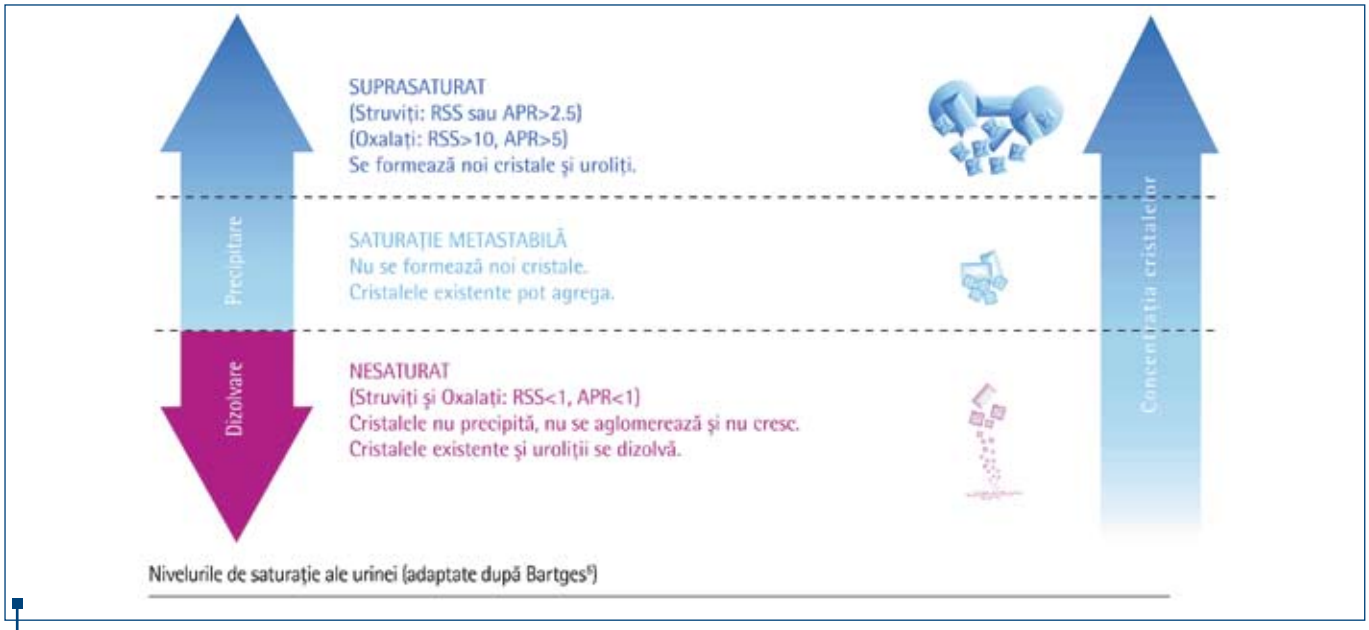

Figura 3

Importanța reducerii nivelului de grăsime

Deoarece supraponderalitatea și obezitatea sunt factori predispozanți pentru BTUIF, este important să menținem condiția fizică ideală la pisicile care prezintă acest risc.

Concluzie

O abordare integrată pentru asigurarea unui control pe termen lung de reducere a factorilor de risc asociați cu BTUIF este acum posibilă. Dezvoltat prin folosirea ultimelor cercetări nutriționale și a ultimelor tehnologii, Feline UR St/Ox™ încorporează strategii nutriționale complementare pentru controlul pe termen lung al urolitiazei cu struviți și oxalați de calciu, CIF, ca și dizolvarea rapidă a urolițiilor struviți. ■

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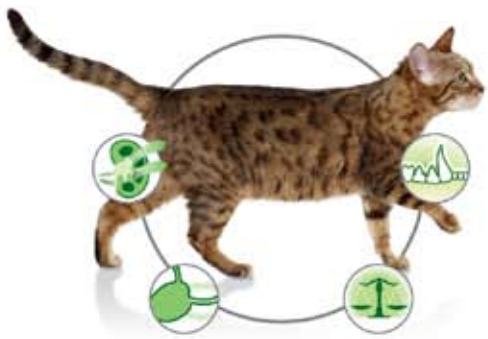

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