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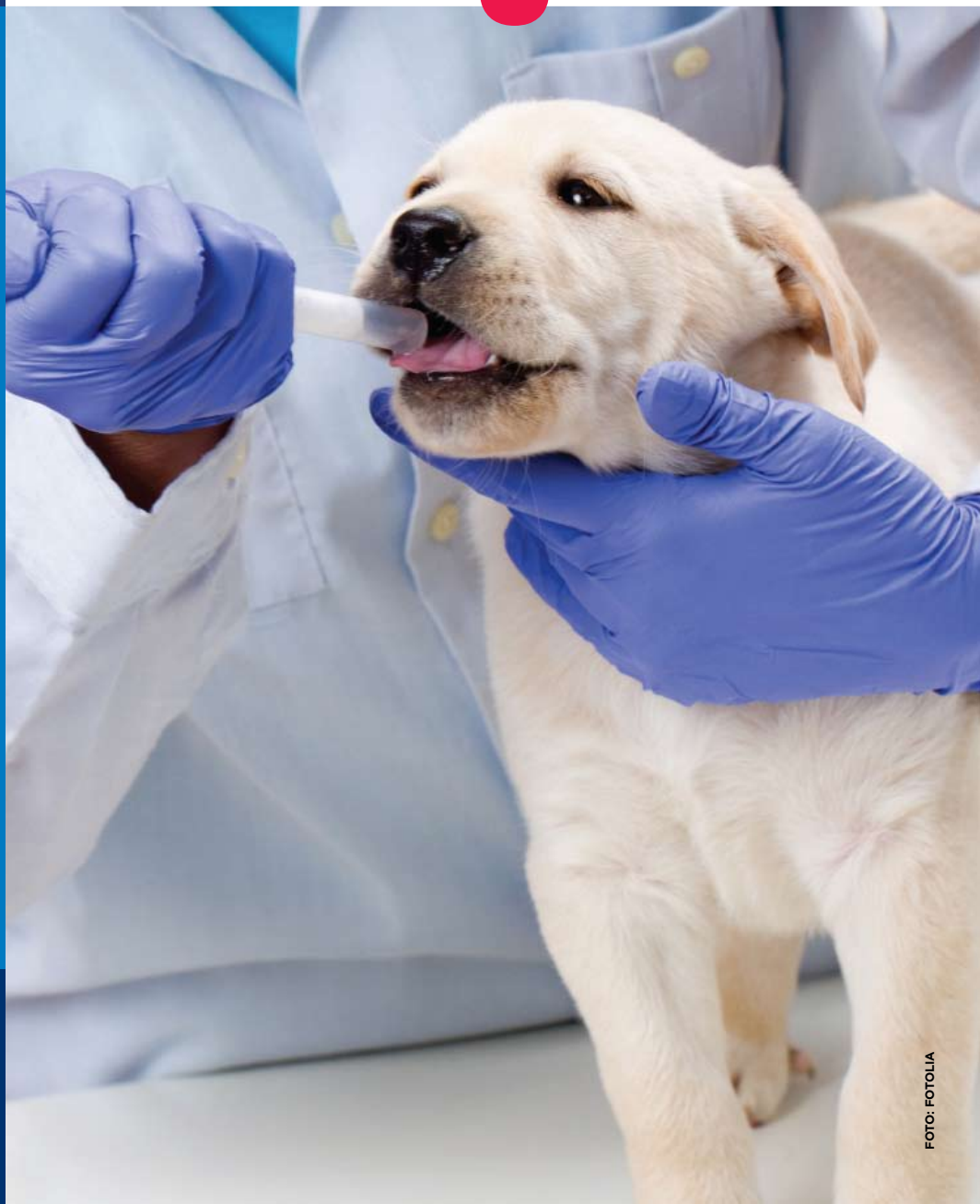
Practica Veterinară +ro

TEMA EDIȚIEI:

Gastroenterologie

Preț: 25 RON
Anul III • Nr. 7 (2/2012)



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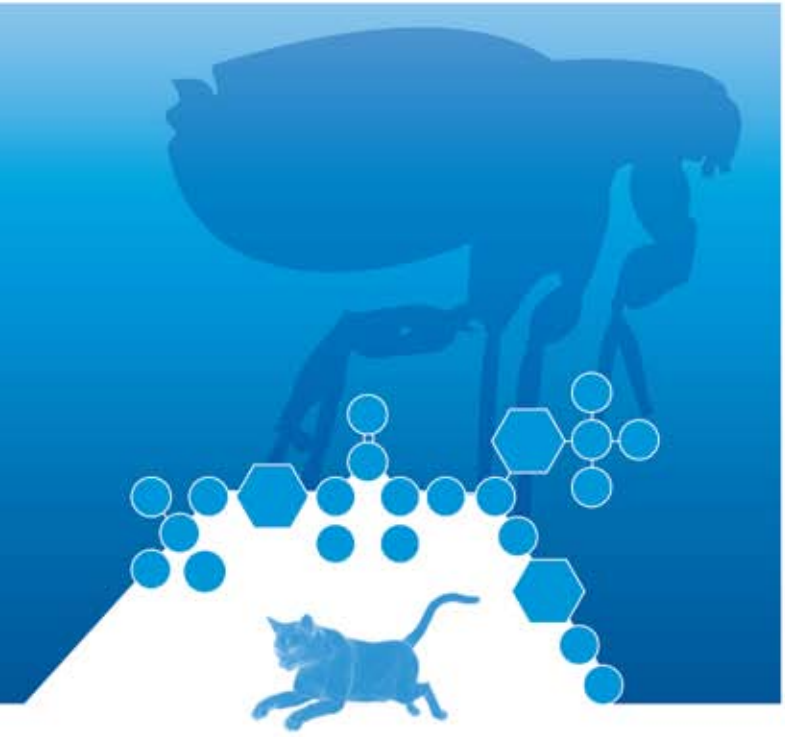
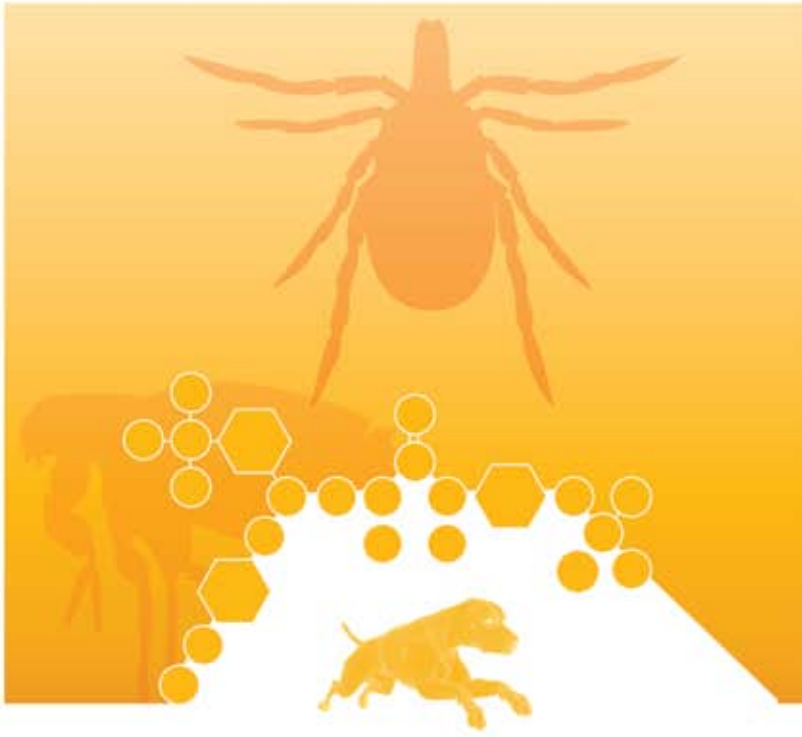
Soluții avansate pentru a combate eficient puricii şi căpuşele





Eficacitate cu cea mai lungă durată aprobată pentru un produs antipurici pentru pisici

6 weeks

O singură doză de metaflumizonă determină reducerea în proporție de 99,4% a puricilor și a ouălor. Efectul se menține până la 45 de zile

Acțiune rapidă: 99,4% din purici sunt omorâți în decurs de 24h

100% mortalitate împotriva căpuşelor în diferite stadii de viață

Efect de desprindere unic: efectul apare în doar 2 ore de la aplicare şi acționează asupra a 90% din căpuşe.
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Practica Veterinară +ro

Revistă de educație medicală continuă

editorial

SCIVAC - Italian Companion Animal Veterinary Association

SCIVAC (Asociația italiană pentru animale de companie) este o organizație profesională italiană cu sediul în Cremona. Obiectivul general al SCIVAC este de a îmbunătăți îngrijirea veterinară a animalelor de companie, specializarea medicilor veterinari, prin colaborarea cu cei mai mai buni specialiști, tehnologii avansate într-o locație de excepție - Palazzo Trecchio - din Cremona, unde se organizează cursuri, workshop-uri și seminarii la un înalt nivel științific și profesional.

Palazzo Trecchi este o clădire istorică situată în centrul orașului Cremona, fiind una dintre cele mai importante clădiri din perioada Renașterii în Cremona, care a găzduit, prin urmare, regi, împărați, episcopi, distinși bărbați și lideri, printre care Louis XII al Franței, Charles V al Spaniei și Giuseppe Garibaldi. În jurul anului 1840, arhitectul Brilli a renovat palatul în stil neogotic la comanda marchizului Manfredo Alessandro Trecchi. Fațada este acum decorată cu elemente gotice și maure, cum ar fi friza orizontală mare și crenelurile încoronării. În curte arcadele sunt susținute de optsprezece coloane de marmură roșie, Verona. Astăzi, palatul găzduiește întâlniri, împreună cu evenimente publice și private. Acesta este sediul Asociației SCIVAC.

Organizarea de evenimente și cursuri practice dedicate educației veterinare are certificat ISO9001;2008 din 18 februarie 2004, iar lectorii sunt diplomați ai Colegiilor europene sau americane.

Cooperarea dintre companii de top din industria de sănătate animală asigură un standard ridicat al echipamentelor utilizate în timpul cursurilor.

În fiecare an, SCIVAC organizează Congresul anual la Rimini, o locație de excepție, cu un program interesant și ospitalitate italiană. Din acest an, în urma colaborării dintre SCIVAC și AMVAC, membrii AMVAC au reducere la Congresul de la Rimini și la workshop-urile organizate în timpul anului. De asemenea, mai sunt multe proiecte pe cale de realizare între cele două organizații profesionale.

Mai multe detalii puteți găsi pe site-ul www.palazzotrecchi.it.

Dr. Valentin Nicolae
Redactor-șef

Hipertiroidismul la pisică

Hipertiroidismul la pisică este cea mai frecventă endocrinopatie diagnosticată la pisici. Recent, medicii veterinari au descoperit că 10% dintre pisicile cu vârsta de peste 9 ani suferă de hipertiroidism. Se pare că prevalența hipertiroidismului crește o dată cu înaintarea în vârstă.

Semne frecvente ale hipertiroidismului la pisică

Scădere în greutate, poliurie, polifagie, polidipsie, hiperactivitate, tahicardie. În unele cazuri pot apărea: vomă, diaree, anorexie, hipertensiune arterială, alopecie, vocalizare crescută, intoleranță la căldură.

Patogeneza

Unele studii arată că hipertiroidismul la pisică apare ca urmare a hiperplaziei nodulare benigne însoțite și de hipersecreția hormonilor tiroidieni: tiroxina (T4) și triiodotiroxina (T3). Inhibarea secreției de TSH (hormonal de stimulare tiroidiană) determină o atrofie a glandei tiroide.

Există cazuri rare când boala apare ca urmare a carcinomului malign tiroidian.

Glanda tiroidă utilizează iodul pentru producerea hormonilor care ajută la reglarea metabolismului, temperaturii corpului, tensiunii arteriale, bătăilor inimii, funcțiilor gastrointestinale.

Hipersecreția de hormoni tiroidieni este determinată și de consumul crescut de iod din diferite surse de nutriție: recompense, hrana altor animale, resturi de mâncare etc. Absorbția crescută de iod determină creșterea în volum a glandei tiroide, producând în acest mod cantități excesive de hormoni tiroidieni care induc boala multisistemică.

Factorii de risc: utilizarea litierei, dormitul pe covor, expunerea la produși din ignifugare polibromurați, consumul de izoflavonoide din soia, consumul de Bisfenol-A din hrană, preferința pentru hrana la conservă, expunerea la insecticide, lipsa de medicamente antiparazitare, hrănirea cu hrană pentru puiuți, frecvența crescută a curățării covorului, expunerea la gazul de la șeminee ani la rând, vârsta înaintată.

Diagnosticul

Diagnosticul este dat prin:

- prezența unuia sau mai multor semne tipice;
- concentrație serică crescută de tiroxină (T4).

Evaluare inițială:

- Istoric și examen clinic;
- Se examinează tot ce este aparent sănătos la pisicile cu vârsta de peste 7 ani;
- Evaluarea semnelor clinice.

Evaluare standard:

- Hemoleucograma completă;
- Profil biochimic, inclusiv dozarea hormonului T4 (tiroxină) și teste ale funcțiilor renale.

Evaluare ulterioară (figura 1)

Tratamentul

Înainte de a se alege metoda terapeutică, este recomandat ca pisica bolnavă să fie evaluată pentru diagnosticul diferențial cu alte boli, în special cele renale și cardiace.

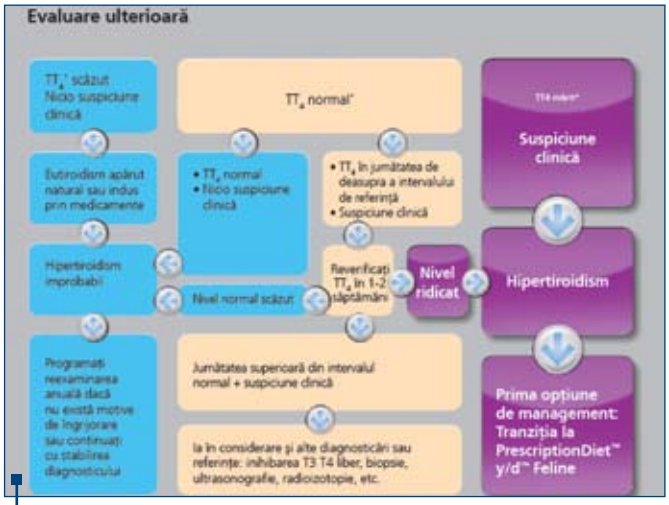

Figura 1

Hipertiroidismul poate fi tratat medical (terapia medicamentoasă), chirurgical (tiroidectomie), cu izotopi radioactivi (I 131), dar și nutrițional prin hrănirea cu **dieta Hill's Feline y/d** (hrană cu cantitatea de iod restricționată).

În cazul în care medicul veterinar optează pentru tratamentul nutrițional cu dieta Hill's y/d Feline, se recomandă obligatoriu întreruperea tratamentului cu medicamente înainte de a se începe hrănirea cu y/d Feline. Avantajul tratamentului nutrițional este că elimină efectele secundare determinate de terapia medicamentoasă: tulburările gastrointestinale și modificările hematologice.

Pe parcursul tratamentului nutrițional se vor efectua:

Reexaminări inițiale la 4,8 și 12 săptămâni după ce ia sfârșit perioada de tranziție urmărindu-se: istoricul bolii, examenul clinic (inclusiv SCF), analiza biochimică (uree, creatinină, T4), analiza urinei.

Reexaminări pe termen lung, la fiecare 6 luni: istoricul bolii, examenul clinic (inclusiv SCF), analiza biochimică (uree, creatinină, T4), analiza urinei. ■

Bibliografie

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2. Kraft W, Buchler F. Hipertiroidismul: Incidența la pisici. Tierraztl Prax Ausg K Kleintiere Heimtiere 1999;27:386-8.
3. Symes H, Winter 2008/2009. Hipertiroidismul la pisici. Endocrinologul. Nr. 90, pp 10-12.

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Nutriție dovedită clinic pentru controlul pisicilor cu hipertiroidism

Îmbunătățește sănătatea tiroidei în 3 săptămâni atunci când este administrată ca singura sursă de nutriție[4]

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Nutriție completă de zi cu zi pe gustul pisicilor

✓ Dietă disponibilă atât în formulă uscată cât și formulă umedă pentru un proces de fidelizare ușor

Beneficii suplimentare pentru clienți	Caracteristici
Ajută la susținerea sănătății renale	Fosfor controlat și cantitate scăzută de sodiu
Susține sănătatea tractului urinar	Magneziu controlat și pH urinar (6.4-6.6)
Susține sănătatea dermatologică și strălucirea blănii	Niveluri ridicate de acizi grași Omega-3 și Omega-6

Fidelizarea este importantă

Iodul din alte surse de nutriție - recompense, hrana altor animale, resturi de mâncare etc. – poate compromite eficiența nutriției cu cantitate restricționată de iod. Este foarte important ca hrana y/d™ să fie administrată ca singura sursă de nutriție.

[4] Yu S, et al. Nivelul controlat de iod normalizează nivelul total de serum de tiroxină la pisicile cu hipertiroidism apărut în mod natural, J Vet intern Med 2011; 25:683-684.

sumar

Anul III • Nr. 7 (2/2012)

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Electromagnetica Business Park
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axele A-D, Sector 5, București
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Specii ţintă: Câini (toate tabletele aromate) şi pisici (numai tabletele de 15 mg).

ndicaţii: tratamentul infecţiilor bacteriene ale tractului digestiv, respirator şi urogenital, în infecţii ale pielii, în infecţii secundare ale rănilor şi în otite externe, cazuri în care experienţa clinică indică enrofloxacina ca medicament de elecţie.

Contraindicaţii: Cartilajul articular poate fi afectat în perioada de creştere rapidă, de aceea nu administraţi produsul la câinii cu vârstă mai mică de 1 an sau în mod excepţional la câinii din rasele de talie mare cu o perioadă de creştere de până la 18 luni. Nu se recomandă administrarea la pisici cu vârstă mai mică de 8 săptămâni. Nu administraţi la câini/pisici cu alergii la substanţa activă sau la oricare din excipienţi. Nu administraţi la câinii cu aplopexie deoarece enrofloxacina poate cauza stimularea sistemului nervos central.

Înainte de prescriere citiţi Sumarul Caracteristicilor Produsului.

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Inovaţia şi cunoştinţele noastre sunt dedicate sănătăţii. De aceea folosim determinarea, tenacitatea şi experienţa noastră, într-un singur scop: dezvoltarea unor produse eficiente şi sigure, de cea mai înaltă calitate.

Congresul Rimini 2012

SCIENTIFIC PROGRAMME

RIMINI June 8th-10th 2012

scivac

73rd INTERNATIONAL SCIVAC CONGRESS
June 8th-10th 2012, RIMINI

	Sala Anfiteatro 1	Sala Anfiteatro 2	Sala dell'Arengo	Sala del Castello 1	Sala del Castello 2	Sala dell'Arco	Sala della Marina	Sala del Parco	FREE SESSION Sala del Tempio 1	FREE SESSION Sala del Tempio 2	ADVANCED SESSION Sala del Ponte	ADVANCED SESSION Sala del Lavatoio	Workshop Sala del Borgo	Workshop Sala del Porto	Workshop Sala dell'Orologio	Workshop Sala del Faro
FRIDAY JUNE 8th MORNING	Respiratory Disease Lynelle Johnson (USA)	Emergency and critical care Nuno Felix (P)	Surgery Jilles Dupré (A)	Oncology	Exotic Animals Angela Lennox (USA)	Reproduction	Physiotherapy Alessandro Piras (IRL)		To be defined	To be defined						
FRIDAY JUNE 8th AFTERNOON	Respiratory Disease Lynelle Johnson (USA)	Emergency and critical care Nuno Felix (P)	Surgery Jilles Dupré (A)	Oncology Miriam Kleiter (AU)	Exotic Animals Angela Lennox (USA)	Reproduction	Physiotherapy	Cardiology	To be defined	To be defined	Orthopedic	Behaviour	Endoscopy Storz			
SATURDAY JUNE 9th MORNING	Gastro-enterology Robert Washabau (USA)	Anesthesia Peter Kronen (CH)	Orthopedic Steven Budsberg (USA)	Cardiology Virginia Luis-Fuentes (USA)	Feline Medicine Margie Scherk (CND)	Practice management	Behaviour	Internal Medicine	To be defined	To be defined	Imaging	Emergency and critical care	Surgery mini-invasiva Gilles Dupré (A)	Cytology Zeiss	Reproduction	
SATURDAY JUNE 9th AFTERNOON	Gastro-enterology Robert Washabau (USA)	Anesthesia Peter Kronen (CH)	Orthopedic Steven Budsberg (USA)	Cardiology Virginia Luis-Fuentes (USA)	Feline Medicine Margie Scherk (CND)	ANMVI	Leishmaniasis	Neurology	To be defined	To be defined	Dermatology	Oncology	Surgery Storz			
SUNDAY JUNE 10th MORNING	Urology Joseph Bartges (USA)	Dermatology Citology	Imaging Gabriela Seiler (USA)	Neurology	Dentistry Jennifer Rawlinson (USA)	ANMVI	Opthalmology David Maggs (USA)	Technicians	To be defined	To be defined	Gastro-enterology	Feline Medicine			Bibliography Research	
SUNDAY JUNE 10th AFTERNOON	Urology Joseph Bartges (USA)	Dermatology Citology	Imaging Gabriela Seiler (USA)	Neurology	Dentistry Jennifer Rawlinson (USA)	ANMVI	Opthalmology David Maggs (USA)	Technicians	To be defined	To be defined						

CONTINUING EDUCATION · ADVANCED LEVEL · ADVACED SESSION · WORKSHOP · TECHNICIANS · FREE SESSIONS

SCIENTIFIC PROGRAMME

RIMINI June 8th-10th 2012 — scivac

SCIENTIFIC PROGRAMME - FRIDAY, JUNE 8th 2012

	SALA ANFITEATRO 1	SALA ANFITEATRO 2	SALA DELL'ARENGO	SALA DEL CASTELLO 1	SALA DEL CASTELLO 2	SALA DELL'ARCO	SALA DELLA MARINA
	INTERNAL MEDICINE by Lynelle Johnson — ROYAL CANIN	EMERGENCY AND CRITICAL CARE — ROYAL CANIN	SURGERY by Gilles Duprè — Hills	ONCOLOGIA (only italian) — Hills	EXOTIC ANIMALS by Angela Lennox	RIPRODUZIONE (only italian) — ROYAL CANIN	PHYSIOTHERAPY — ROYAL CANIN
	Chairperson: Federico Fracassi	*Chairperson: Marco Bertoli*	*Chairperson: Emanuela Morello*	*Chairperson: Paolo Buracco*	*Chairperson: Vittorio Capello*	*Chairperson: Mauro Ronchese*	*Chairperson: Francesca Cazzola*
10.00	Rhinitis in dogs and cats **Lynelle Johnson (USA)**	Drugs and Emergency: drugs that should not be lacking in emergency room and when to use them **Nuno Felix (P)**	Brachycephalic airway syndrome **Gilles Duprè (A)**	Fattori prognostici: mettiamo un po' di ordine **Laura Marconato (I)**	Sedation, anesthesia and monitoring, and analgesia of exotic companion mammals - Part 1 **Angela Lennox (USA)**	Il neonato sano: cosa fare e soprattutto cosa non fare **M. Carmela Pisu (I)**	Tenomuscular diseases: team's job or surgical therapy only? **Alessandro Piras (UK)**
10.40	Medical treatment of tracheal collapse **Lynelle Johnson (USA)**	Fluid therapy, restoring an effective circulation, historical analysis and state of the art **Nuno Felix (P)**	Diagnosis and surgical treatment of laryngeal paralysis **Gilles Duprè (A)**	Aspetto citologico, grado istologico, margini chirurgici, immunoistochimica: in aiuto al clinico **Giuliano Bettini (I)**	Sedation, anesthesia and monitoring, and analgesia of exotic companion mammals - Part 2 **Angela Lennox (USA)**	Il neonato ortano **Manuela Farabollini (I)**	Clinical and diagnostic approach for the correct diagnosis of tenomuscular diseases **Alessandro Piras (UK)**
11.20	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION
12.00	Living with a breath of air: common and less common clinical signs of different larynx diseases **Davide de Lorenzi (I)**	Vital electrolyte imbalances, diagnosis and therapy **Paolo Gaglio (I)**	Tracheotomy, permanent tracheostomy: Indications, surgical technique, post surgical cures **Gilles Duprè (A)**	Mastocitoma: quando è o non è chirurgico? **Paolo Buracco (I)**	Critical care of exotic companion mammals **Angela Lennox (USA)**	Farmacologia, farmacocinetica e farmacodinamica nel neonato **Ivano Eberini (I)**	Supraspinatus tendonitis and contraction of the iliopsoas muscle: is physiotherapy the right answer? **Lisa Piras (I)** **Daniele Corlazzoli (I)**
12.40	LUNCH BREAK AND SCIVAC EXHIBITION						
	Chairperson: Federico Fracassi	*Chairperson: Paolo Gaglio*	*Chairperson: Guido Pisani*	*Chairperson: Giuliano Bettini*	*Chairperson: Vittorio Capello*	*Chairperson: M. Carmela Pisu*	*Chairperson: Andrea Martinoli*
14.00	Chronic bronchitis in dogs **Lynelle Johnson (USA)**	Acid base and fluid therapy **Marco Bertoli (I)**	Sialoceles and salivary glands surgery **Gilles Duprè (A)**	Il mastocitoma multiplo: chirurgia o chemioterapia? **Damiano Stefanello (I)**	Critical care of the avian patient **Angela Lennox (USA)**	Il neonato patologico: principali patologie neonatali **M. Cristina Veronesi (I)**	Muscle/peripheral nerves: how to recognize and treat a non-spinal disease **Daniele Corlazzoli (I)**
14.40	Bacterial pneumonia **Lynelle Johnson (USA)**	COFFEE BREAK AND SCIVAC EXHIBITION	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS	Il neonato patologico: diagnosi **M. Cristina Veronesi (I)**	The athletic dog and the physiopathology of exercise **Alessandro Piras (UK)**
15.20	COFFEE BREAK AND SCIVAC EXHIBITION	Fluid in SIRS and human serum albumin **Fabio Viganò (I)**	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION
16.00	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS	Reconstructive surgery of the head: flaps **Gilles Duprè (A)**	Terapia antiblastiche nel mastocitoma: vecchie attuali e nuove **Laura Marconato (I)**	Critical care of the avian patient **Angela Lennox (USA)**	SHORT COMMUNICATIONS	Physiotherapy: what are the possibilities, modality and goals of treatment? **Ludovica Dragone (I)**
16.40	Felines asthma **Lynelle Johnson (USA)**	Enteral and parenteral nutrition **Nuno Felix (P)**	Surgery of the external and middle ear **Gilles Duprè (A)**	La radioterapia: usiamola prima che sia troppo tardi! **Miriam Kleiter (AU)**	Sedation, anesthesia and monitoring, and analgesia of the reptile patient **Angela Lennox (USA)**	Il neonato patologico: terapie **Alessandro Rota (I)**	Trigger points and pnf: new frontiers of physiotherapy **Ludovica Dragone (I)**

CONTINUING EDUCATION — ADVANCED LEVEL — SHORT COMMUNICATIONS — SIMULTANEOUS TRANSLATION ITALIAN → ENGLISH

scivac RIMINI June 8th-10th 2012

SCIENTIFIC PROGRAMME

SCIENTIFIC PROGRAMME - SATURDAY, JUNE 9th 2012

	SALA ANFITEATRO 1	SALA ANFITEATRO 2	SALA DELL'ARENGO	SALA DEL CASTELLO 1	SALA DEL CASTELLO 2	SALA DELL'ARCO	SALA DELLA MARINA
	GASTROENTEROLOGY by Robert Washabau	ANAESTHESIA	ORTHOPAEDICS by Steve Budsberg ROYAL CANIN	CARDIOLOGY ROYAL CANIN	FELINE MEDICINE by Margie Scherk ROYAL CANIN	PRACTICE MANAGEMENT (only italian)	MEDICINA COMPORTAMENTALE (only italian) ROYAL CANIN
	Chairperson: Ugo Lotti	*Chairperson: Federico Corletto*	*Chairperson: Filippo Maria Martini*	*Chairperson: Marco Poggi*	*Chairperson: Magda Gerou-Ferriani*	*Chairperson:*	*Chairperson: Daniele Meriano*
9.00	Diagnosis and management of swallowing disorders **Robert Washabau (USA)**	Anaesthesia guidelines: where are we? **Peter Kronen (CH)**	Minimally invasive fracture repair: good idea or disaster in the making? **Steve Budsberg (USA)**	Therapeutic goals in cardiac disease **Virginia Luis Fuentes (UK)**	Feeding cats with different nutritional needs: a dilemma in the multicat household - Part 1 **Margie Scherk (CND)**	Liberalizzazioni: vantaggi per le strutture gestite dal management **Massimo Serreri (I)**	Lo sviluppo mentale del cucciolo: neurobiologia, scienze cognitive e medicina comportamentale a confronto **Sabrina Giussani (I)**
9.40	Difficult vomiting disorders: pathogenesis, diagnosis and therapy **Robert Washabau (USA)**	Anaesthesia service: is it just an expensive waste of time? **Guido Pisani (I)**	Pet Obesity - Orthopedic problems **Steve Budsberg (USA)**	Treatment of emergencies in cardiology **Cecilia Quintavalla (I)**	Feeding cats with different nutritional needs: a dilemma in the multicat household - Part 2 **Margie Scherk (CND)**	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS
10.20	COFFEE BREAK AND SCIVAC EXHIBITION	SHORT COMMUNICATIONS	COFFEE BREAK AND SCIVAC EXHIBITION	Management of canine chronic mitral valve disease **Virginia Luis Fuentes (UK)**	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION
11.00	Feline, constipation, obstipation, megacolon: breaking the circle **Robert Washabau (USA)**	COFFEE BREAK AND SCIVAC EXHIBITION	Medical management of canine osteoarthritis: what is the evidence for the products we use? **Steve Budsberg (USA)**	COFFEE BREAK AND SCIVAC EXHIBITION	Getting calories in: feeding the inappetent or anorectic cat **Margie Scherk (CND)**	La pubblicità: indagine su forme e costi nel 2012 **Bartolomeo Borgarello (I)**	L'ABC della relazione di attaccamento **Marzia Possenti (I)**
11.40	SHORT COMMUNICATIONS	Anaesthesia service: could it be a simple way to improve patient management? **Paolo Franci (I)**	SHORT COMMUNICATIONS	Management of canine dilated cardiomyopathy **Virginia Luis Fuentes (UK)**	Managing feline constipation: relieving a hard problem **Margie Scherk (CND)**	Cosa si aspetta la clientela dal veterinario: indagine ANMVI **Marco Viotti (I)**	Strumenti cognitivo-relazionali per l'educazione del cucciolo **Maria Chiara Catalani (I)**
12.20	LUNCH BREAK AND SCIVAC EXHIBITION						
	Chairperson: Ugo Lotti	*Chairperson: Paolo Franci*	*Chairperson: Michele d'Amato*	*Chairperson: Cecilia Quintavalla*	*Chairperson: Saverio Paltrinieri*	RESPONSABILITÀ CIVILE A.N.M.V.I. con Daria Scarciglia *Chairperson: Carlo Pizzirani*	LEISHMANIOSI CANINA (only italian) *Chairperson: George Lubas*
14.00	Feline hepatobiliary disease what's new in diagnosis and therapy? **Robert Washabau (USA)**	How expensive should a good anaesthetic be? **Peter Kronen (CH)**	Lameness exam: what am I missing? **Steve Budsberg (USA)**	Management of atrial fibrillation **Manuela Perego (I)**	Bronchopulmonary disease in cats-and acute respiratory distress **Margie Scherk (CND)**	Che cos'è la responsabilità civile professionale? - **Daria Scarciglia (I)**	Decorso, prognosi e monitoraggio del cane affetto da leishmaniosi **Alessandra Fondati (I)**
14.40	Canine hepatic disease: from diagnosis to therapy **Robert Washabau (USA)**	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS	Prevenire la responsabilità: il consenso informato, la scienza e la coscienza **Daria Scarciglia (I)**	PAUSA ED ESPOSIZIONE COMMERCIALE
15.20	COFFEE BREAK AND SCIVAC EXHIBITION	How to choose your anaesthetic equipment wisely according to your budget and case load **Peter Kronen (CH)**	Technical errors - one subset of fracture failures **Steve Budsberg (USA)**	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	Ci scrive l'avvocato del cliente. Ho la polizza, ma ho ragione: rinuncio a difendermi? **Daria Scarciglia (I)**	COMUNICAZIONI BREVI
16.00	Canine inflammatory bowel disease: the eight (8) components of therapy **Robert Washabau (USA)**	Finding the right place for anaesthesia in the budget of your clinic **Marco Viotti (I)**	Feline osteoarthritis - what we know and don't know! **Steve Budsberg (USA)**	Management of heart failure in cats **Virginia Luis Fuentes (UK)**	Snotting and snuffling: the cat with chronic upper respiratory disease **Margie Scherk (CND)**	Quali soluzioni assicurative? **Francesco Amodeo (I)**	Patogenesi e gestione della proteinuria nel cane affetto da leishmaniosi **Andrea Zatelli (I) - Eric Zini (I)**

CONTINUING EDUCATION ADVANCED LEVEL SHORT COMMUNICATIONS SIMULTANEOUS TRANSLATION ITALIAN → ENGLISH

SCIENTIFIC PROGRAMME

RIMINI June 8th-10th 2012 — scivac

SCIENTIFIC PROGRAMME - SUNDAY, JUNE 10th 2012

	SALA ANFITEATRO 1	SALA ANFITEATRO 2	SALA DELL'ARENGO	SALA DEL CASTELLO 1	SALA DEL CASTELLO 2	SALA DELL'ARCO	SALA DELLA MARINA	SALA DEL PARCO
	NEPHROLOGY AND UROLOGY	CITOLOGIA DERMATOLOGICA (only Italian)	IMAGING - by G. Seiler AGFA Meyer	NEUROLOGIA (only Italian)	DENTISTRY by Jenniler Rawlinson	SICUREZZA SUL LAVORO con Carlo Pizzirani	OPHTALMOLOGY by David Maggs	ATAV TECHNICIANS
	Chairperson: P. Scarpa e W. Bertazzolo	*Chairperson: Federico Leone*	*Chairperson: Giliola Spattini*	*Chairperson: Daniele Corlazzoli*	*Chairperson: Mirko Radice*	*Chairperson: Giorgio Neri*	*Chairperson: Alberto Crotti*	*Chairperson: Irene Bendoni*
9.00	Urolithiasis - Struvite, urate, cystine: Medical management of struvite, urate, and cystine uroliths including medical dissolution and prevention **Joseph Bartges (USA)**	Come ottimizzare il prelievo citologico in base al tipo di lesione: tecniche di prelievo, allestimento dei vetrini ed indicazioni per l'esame citologico **Francesco Albanese (I)**	Diagnostic imaging of the lymphatic system **Gabriela Seiler (USA)**	Neurolocalizzazione al midollo spinale: quando le cose si complicano **Gualtiero Gandini (I)**	Performing a complete oral examination **Jenniler Rawlinson (USA)**	La valutazione dei rischi **Carlo Pizzirani (I)**	How to do a great eye exam? **David Maggs (USA)**	Practice Management **Relatore da definire**
9.40	COFFEE BREAK AND SCIVAC EXHIBITION	La lesioni elementari in dermatologia: la clinica ce le fa riconoscere, la citologia ce le fa interpretare! **Francesco Albanese (I)**	Imaging of disorders of the lymphatic system in the dog **Gabriela Seiler (USA)**	Mielografia, CT e RMnella diagnosi delle patologia spinali: l'una vale l'altra? **Cristian Falzone (I)**	COFFEE BREAK AND SCIVAC EXHIBITION	L'impiantistica a norma di legge **Carlo Pizzirani (I)**	The 7 colors of corneal pathology **David Maggs (USA)**	Practice Management **Relatore da definire**
10.20	Urolithiasis - UTI: Management of UTI including complicated and resistant UTI **Joseph Bartges (USA)**	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION	Intraoral radiography **Jenniler Rawlinson (USA)**	PAUSA ED ESPOSIZIONE COMMERCIALE	COFFEE BREAK AND SCIVAC EXHIBITION	COFFEE BREAK AND SCIVAC EXHIBITION
11.00	The laboratory in urolithiasis: how, when and why **Paola Scarpa (I)**	Neoplasie cutanee: quando la clinica supporta l'esame citologico! **Francesco Albanese (I)**	Lymphoma or IBD? Diagnostic imaging of small intestinal disease **Gabriela Seiler (USA)**	Neoplasie spinali: dalla diagnosi al trattamento chirurgico **Massimo Baroni (I)**	Oral pathology recognition and treatment recommendations **Jenniler Rawlinson (USA)**	Dipendenti, collaboratori e tirocinanti: i doveri del datore di lavoro **Carlo Pizzirani (I)**	Ocular pharmacology (which drug when?) **David Maggs (USA)**	Il ruolo del tecnico veterinario nel settore dell'anestesia **Irene Bendoni (I)**
11.40	Dysuria: when the imaging is essential **Giliola Spattini (I)**	Lesioni follicolari: quello che la citologia non dice **Chiara Bracheiente (I)**	Advances in diagnostic imaging of splenic disorders **Gabriela Seiler (USA)**	Neoplasie spinali: Radioterapia quando e perché **Simona Cancedda (I)**	Feline oral pathology recognition and treatment recommendations **Jenniler Rawlinson (USA)**	Sistri: facciamo chiarezza **Giorgio Neri (I)**	Cherry eye surgeries in dogs **David Maggs (USA)**	Preparazione e assistenza del cane e del gatto nelle varie fasi del periodo perioperatorio **Paolo Franci (I)**
12.20	LUNCH BREAK AND SCIVAC EXHIBITION							
	Chairperson: Paola Scarpa	*Chairperson: Enrico Bottero*	*Chairperson: Federica Rossi*	*Chairperson: Stefania Gianni*	*Chairperson: Mirko Radice*	*Chairperson: Giorgio Neri*	*Chairperson: Alberto Crotti*	*Chairperson: Irene Bendoni*
13.40	Urolithiasis - Calcium oxalate: Management of calcium oxalate uroliths by invasive and minimally invasive (laser lithotripsy and cystoscopic-assisted cystotomy) and prevention **Joseph Bartges (USA)**	Cellule fusate: il confine tra la realtà e l'inganno **Mario Caniatti (I)**	Diagnostic imaging workup of patients with pleural effusion - imaging of the thoracic duct **Gabriela Seiler (USA)**	L'approccio diagnostico moderno alle ernie discali **Nicola Gasparinetti (I)**	Practical therapies for periodontal disease **Jenniler Rawlinson (USA)**	Mezzi e sistemi per la prevenzione degli incendi **Carlo Pizzirani (I)**	Feline keratoconjunctivitis – clinical signs, the latest on diagnosing and treating Herpesvirus (first part) **David Maggs (USA)**	Preparazione e assistenza degli animali esotici e da reddito nelle varie fasi del periodo perioperatorio **Elisa Bortolami (I)**
14.20	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS	SHORT COMMUNICATIONS	Legislazione e obblighi documentali **Carlo Pizzirani (I)**	Feline keratoconjunctivitis – clinical signs, the latest on diagnosing and treating Herpesvirus (second part) **David Maggs (USA)**	Riconoscere e rispondere in modo adeguato alle emergenze anestesiologiche **Paolo Franci (I)**
15.00	Urolithiasis – Nephrolithiasis: Medical and surgical management of nephroureterolithiasis **Joseph Bartges (USA)**	Neoplasie rotondocellulari: una diagnosi sempre facile? Obiettivi e limiti della citologia **Mario Caniatti (I)**	Challenging imaging cases of pleural effusion **Gabriela Seiler (USA)**	Ernie discali: per ogni singolo caso l'idonea terapia **Daniele Corlazzoli (I)**	The systemic impact of periodontal disease **Jenniler Rawlinson (USA)**	Casi particolari: a domanda risposta **Carlo Pizzirani (I)**	Feline keratoconjunctivitis – clinical signs, the latest on diagnosing and treating Chlamydia, and Mycoplasma **David Maggs (USA)**	Utilizzo, manutenzione e disposizione delle attrezzature anestesiologiche **Elisa Bortolami (I)**
15.40	CONGRESS CLOSES							

CONTINUING EDUCATION — ADVANCED LEVEL — SHORT COMMUNICATIONS — SIMULTANEOUS TRANSLATION ITALIAN → ENGLISH — TECHNICIANS

Feline trichomoniasis

Poching Pan
Northern Animal Clinic Hong Kong
Correspondence: Dr. Poching Pan Room G, 11/F, Paris Court, Sheung Shui Town Centre, Sheung Shui, N.T., Hong Kong

Abstract

Feline trichomoniasis is an emerging disease. In the past two decades, several investigators have recognized an association between feline large bowel diarrhea and trichomonad infection. Feline trichomoniasis is caused by Tritrichomonas foetus. The clinical signs of Tritrichomonas foetus in cats can be variable, ranging from asymptomatic infection to prolonged and intractable diarrhea which in some cases may wax and wane. The predominantly large bowel diarrhea typically presents with increased frequency of defecation, the passage of semi-formed to liquid often foul-smelling faeces, sometimes containing fresh blood and mucus. This is the disease now spread to worldwide. Case reports from UK, Norway, Greece, Spain, Australia, USA and Korea have been published for last decade.
Keywords: *feline trichomoniasis, bowel diarrhea, clinical sign*

Feline trichomoniasis is an emerging disease. In the past two decades, several investigators have recognized an association between feline large bowel diarrhea and trichomonad infection. Feline trichomoniasis is caused by *Tritrichomonas foetus*. The clinical signs of Tritrichomonas foetus in cats can be variable, ranging from asymptomatic infection to prolonged and intractable diarrhea which in some cases may wax and wane[1]. The predominantly large bowel diarrhea typically presents with increased frequency of defecation, the passage of semi-formed to liquid often foul-smelling faeces, sometimes containing fresh blood and mucus. This is the disease now spread to worldwide. Case reports from UK, Norway, Greece, Spain, Australia, USA and Korea have been published for last decade[2-7].

Tritrichomonas foetus is a single celled flagellated protozoan parasite that is known to be a pathogen of the bovine reproductive tract. However, feline trichomonads are indistinguishable from bovine *T. foetus* in morphology and sequence analysis of some rRNA genes[8]. Despite of it, there is only one research indicated isolates of *T. foetus* from cattle are infectious for the large intestine of cats[9]. Most of the researches highlight some important biological differences between the bovine and feline trichomonads, suggesting that spontaneous transmission between the two hosts is unlikely[8].

The parasite is maintained by fecal-oral transmission in cats. Most infected cats are purebred or shelter cats. Multiple cats environment is an important risk factor for feline trichomoniasis. One study shows *T. foetus* can be cultured from faeces containing high concentration of organisms after 24 hours[10]. Another study shows trophozoites of *T. foetus* survived exposure to distilled or tap water for 30 minutes, while they survived for at least 180 minutes in urine. Trophozoites survived for 30 minutes on dry cat food but survived for 120-180 minutes in canned cat food[9]. The result shows that the transmission of *T. foetus* in cats may occur via shared litter box, contaminated water and cat food than through exposure to contaminated cat litter. In the concern of public health, there are only three cases of human infection with *T. foetus*. However, all three people are with impaired immune systems[9].

The diagnoses methods for *T. foetus* in cats now include: 1) looking for motile trophozoites in fresh faecal smear with saline dilution 2) using the specific "InPouch™ TF" culture system or 3) by detection of *T. foetus* ribosomal DNA using PCR method. To looking for motile trophozoites in light microscopy requires fresh faecal samples. The experienced clinicians also help to increase the sensitivity and specificity of this method. *Pentatrichomonas hominis* and *Giardia spp.*, another two trophozoites, can be misdiagnosed as *T. foetus*: *T. foetus* and *Giardia spp.* are both pathogens in cats leading to chronic diarrhea. Although of similar in size (≈10–20μm in length), trichomonad trophozoites differ morphologically from those of *Giardia spp.*[8]. Trichomonad is presented by spindle shape and moved forwardly by undulating membrane. *Giardia spp.* is characterized as a pear shape and the typical two "eyes" as "clown face"; *T. foetus* and *P. hominis* are both trichomonads. *Tritrichomonas* exhibits 3 anterior flagella, however *Pentatrichomonas* is characterized by a predominance of 5 anterior flagella. Anterior flagella are difficult to enumerate in living motile specimens. Therefore, to distinguish tritrichomonads from pentatrichomanads, it requires specialized silver-staining techniques for adequate viewing[11]. Although the method of light microscopy method is convenient and inexpensive, the sensitivity is only about 13%.

The specific "InPouch™ TF" culture system is first designed for detecting bovine trichomoniasis. The system can also be used in diagnoses of feline trichomoniasis. The culture system is easy-to-use and costs much less than PCR method (about 5 USD). The sensitivity is about 55%. In cattery or shelter environment, it may be a good diagnosed method to save money. To maximize the diagnostic utility of culture the cat feces should be inoculated into culture within a 6 hours period from voiding. The specimens should be kept and shipped at room temperature[10].

Definitive diagnosis is established via PCR testing for *T. foetus* DNA. Unlike other two methods, PCR testing can detect both live and dead trichomonads. PCR testing has the highest sensitivity, but may be cost-prohibitive for many cat owners. The cost of diagnostic is about 75 USD. The sensitivity can be as high as 95% in a good quality control lab. In Dr. Jody Gookin's study, she recommends the fecal samples should be as lima-bean size. The loose/diarrheic fresh sample is more possible to detect T. foetus than a formed old sample. The faecal sample is best collected by deep loop or saline flush without lubricant and kept unrefrigerated prior to testing. The patient should also stop any antibiotic treatment for at least 7 days before collecting the sample.

The only known effective treatment for *T. foetus* is ronidazole. Ronidazole is widely used for treatment for pigeons for *Trichomonas gallinae* infection. Oral absorption of ronidazole capsues in cats is rapid and complete. The previous recommended dosage was 30 mg/kg Q12H. In one study, there are cats developing neurological signs when taking ronidazole. The neurological signs include anorexia, hindlimb weakness and hyperesthetic etc. Cats recovered without additional treatment from days to weeks. It is believed the adverse effects of ronidazole reported in dogs may also occur in cats. The adverse effect of ronidazole in dogs includes seizures, opisthotonos, fine tremors, ataxia, hindquarter stiffness, dry mouth and gums, mild tachycardia and slow and shallow respiration[12]. Although neurological signs can occur at low dosages as well, the cats that received high dosage of ronidazole are likely to develop neurological signs. Therefore, ronidazole must be compounded to get a dose in a suitable size for cats. The mechanism of ronidazole neurotoxicity is unknown, formation of superoxide and semiquinone radicals from catecholamine-ronidazole oxidation - reduction reaction under aerobic condition is proposed. The new study shows the half-life of ronidazole was long and there was still drug remaining in the plasma 48 hours after a single dose. To decrease the risk of drug accumulation that may produce neurotoxicity and considering the long half-life of ronidazole in cat, once-daily administration is replaced by twice-daily administration. The recommended treatment of *T. foetus* in feline is now ronidazole 30mg/kg Q24H for 14 days[13].

Unfortunately, the treatment does not guarantee fully recovery in all infected cats. In Dr. Jody L. Gookin's study, improvement of fecal consistency can be immediate and dramatic. The faeces returned to normal after complete treatment. Nevertheless, relapse of *T. foetus* after ronidazole treatment can be as long as 20 weeks after treatment[14]. Moreover, in another study shows 88% of cats had resolution of their clinical signs within two years of onset of *T. foetus*-related diarrhea. Despite resolution of clinical signs, the owners of many cats reported subsequent infrequent and short-duration relapses of diarrhea[1]. This may due to *T. foetus* or possible developing of inflammatory bowel disease. Since ronidazole is not a licensed drug for cats, clinicians should discuss with owners about the pros and cons of the treatment. ■

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A practical approach to the chronic vomiting patient

Colin F. Burrows
BVetMed, PhD, Dip ACVIM, HonFRCVS
College of Veterinary Medicine, University of Florida

Abstract

Gastrointestinal disorders are a frequent complaint in small animal practice, ranking second only to skin disease as a reason for veterinary evaluation. These disorders have a variety of presenting signs which are ranked in an approximate order of descending importance and frequency in Table 1. Diarrhea and vomiting predominate, but it must be appreciated that like the others listed in Table 1, these are only signs of disease, and it is their varying manifestations and combinations that must be utilized to make a differential diagnosis.
Keywords: *gastrointestinal disorders, vomiting, diagnosis*

Most gastrointestinal disturbances are acute and reversible, requiring only supportive or symptomatic therapy to bring about a remission of signs. Specific diagnosis is often unnecessary, but may sometimes be extremely important, for example if the vomiting is due to infectious disease and other animals are at risk. The vomiting patient often presents a decisional challenge in this context in that it may be difficult to decide whether the animal needs an in-depth diagnostic evaluation or if symptomatic therapy alone may resolve the complaint. It should also be borne in mind that both acute and chronic vomiting result from a wide range of causes, many of which emanate from outside the gastrointestinal tract. This is an important clinical consideration, since treatment should always be directed at the underlying disorder. Nevertheless, symptomatic treatment may be essential in some patients while a diagnosis is pursued.

In contrast to acute disturbances, chronic gastrointestinal disorders are rarely self limiting, and, for treatment to be effective, it is usually essential to establish a specific diagnosis and prescribe appropriatetherapy. Most regurgitating patients, for example, fall into this category

The stomach: normal and abnormal function

The stomach has 3 important functions: 1) to act as an adjustable food reservoir, 2) to mix food with gastric secretions and begin the process of digestion, and 3) to gradually empty this mixture into the intestine for final digestion and absorption. In dogs and cats gastric disease disrupts these processes and usually causes vomiting.

Gastric Filling

The stomach acts as an adjustable food reservoir by its ability to increase in volume during a meal without an associated increase in intragastric pressure. This process, known as accommodation, is mediated through vagal inhibitory fibers. Receptive relaxation, another term associated with gastric filling, occurs at the time a bolus of food actually enters the stomach from the esophagus. In this case, vagal inhibitory fibers decrease lower esophageal sphincter pressure and fundic contractions to allow an individual bolus to enter without an increase in pressure (Figure 1). Failure to relax occurs in both inflammatory and neoplastic gastric disease and causes an increase in intragastric pressure with associated pain, nausea and vomiting.

Gastric Secretion and Digestion

The stomach secretes hydrogen ions, sodium, potassium, chloride and water, as well as pepsinogen, lipase and varying quantities of mucus into its lumen. Gastric secretion depends on the cellular mass of the various mucosal secretory elements, the action of neural and hormonal agonists and antagonists and the meal volume (Figure 2). Gastric juice is enzyme rich and fluid begins the process of protein and fat digestion in the stomach.

The interdigestive pH of the canine stomach ranges between 3 and 6.5 and usually only falls when acid secretion is stimulated by the sight, smell or taste of food (the cephalic phase of gastric secretion) or by stressful situations (e.g., sepsis, hypoxia, hospitalization, trau-

Table 1 Signs of gastrointestinal disease

Diarrhea
Vomiting
Change in Appetite
Tenesmus
Abdominal Pain
Salivation
Dehydration
Hematochezia
Weight Loss
Shock
Regurgitation
Anemia
Dyschezia
Flatus

ma). This species difference explains, at least in part, the lower incidence of peptic ulcer disease and reflux esophagitis in the dog and cat as compared to man.

Acid is secreted into the gastric lumen by the parietal cells. For each hydrogen ion secreted a molecule of $C0_2$ is generated. This is converted to bicarbonate and enters the interstitial fluid from where it is taken up by mucosal capillaries. Most of this bicarbonate is secreted into the gastric lumen to protect the mucosa from acid damage, while the remainder reaches the systemic circulation and is later excreted in the urine. This is known as the "alkaline tide" (Figure 3).

Gastric secretion has 3 phases: 1) cephalic 2) gastric and 3) intestinal. Secretion is stimulated by a complex interaction between histamine, cholinergic vagal fibers and gastrin-secreting "g" cells in the antrum.

Electrolytes [Na^+, K^+, CI^-] are an important component of gastric secretion, with water passively following secretion of these ions. The proteolytic enzyme pepsinogen is released from Chief cells in the gastric gland. Pepsinogen is converted to pepsin in the lumen, is active at an acid pH, and begins the process of protein digestion. Canine and feline gastric epithelial cells contain lipase that is released into the gastric lumen during the gastric phase of digestion.

Gastric secretion is inhibited by acid (low pH) bathing the antral mucosa. Secretion is also inhibited by acid, fat and hyperosmolar solutions bathing the duodenal mucosa. These feedback mechanisms ensure that gastric secretion is proportional to need.

Acid secretion may be either increased, or decreased in gastric disease. Increased secretion contributes to peptic ulceration which occurs less frequently in domestic animals than in man. Many species are infected with bacteria of the genus Helicobacter which causes acid hypersecretion and ulceration in susceptible individuals. Chronic gastric inflammation, in contrast, reduces the parietal cell mass which results in a corresponding decrease in secretory ability.

The Gastric Mucosal Barrier

The reason why the stomach does not digest itself has intrigued philosophers, physiologists and clinicians for many years. It is attributed to the "gastric mucosal barrier", a phenomenon that has been defined as "the structural and functional protection of the mucosa against its own secreted acid and pepsin".

Structural components include: 1) surface mucus, 2) bicarbonate secreted by gastric epithelial cells, 3) the epithelial cell membranes themselves which possess a relative impermeability to ions, 4) gastric mucosal blood flow, 5) Prostaglandins and cytoprotection, and 6) the basal membrane. Salivary epidermal growth factor also contributes to mucosal strength. Barrier strength has been shown to be increased by some Prostaglandins (PGE_2), an observation that has been used in the development of some anti-ulcer drugs, and with the COX_2 selective non-steroidals. Other substances such as ethanol, bacterial endotoxin, bile salts, and those non-steroidal anti-inflammatory drugs that are

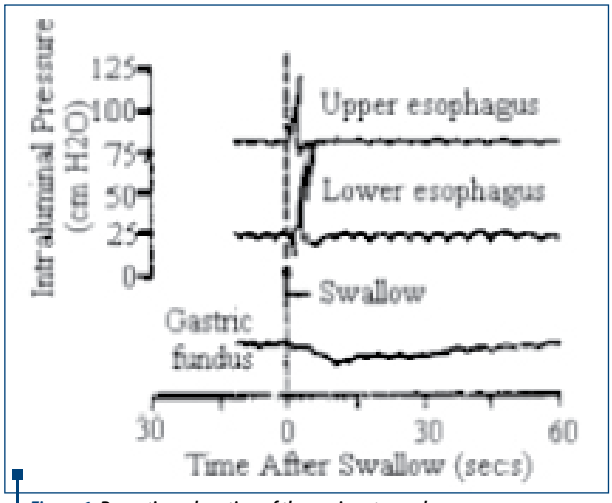

Figure 1. Receptive relaxation of the canine stomach

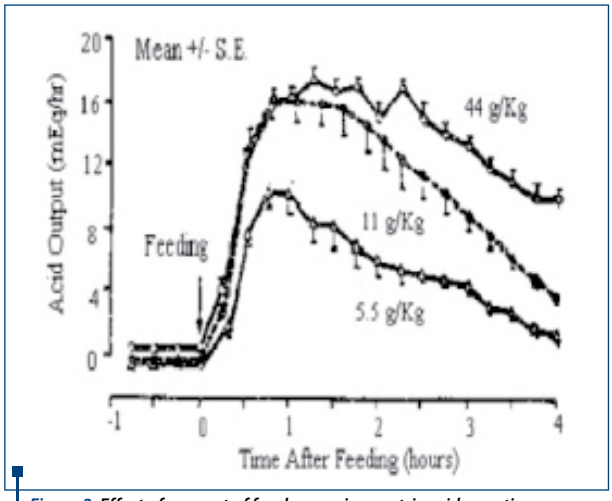

Figure 2. Effect of amount of food on canine gastric acid secretion

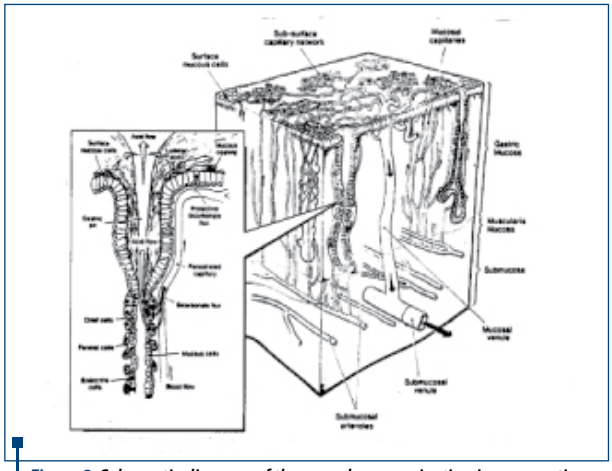

Figure 3. Schematic diagram of the vascular organization in enzymatic mucosa (right) and the proposed mechanism for vascular transport of HCO_3- toward the surface mucus cells from deeper within the mucosa (insert left)

predominantly COX_1 selective (e.g. flunixin, aspirin, indomethacin and ibuprofen) disrupt the barrier. Gastric parasites and foreign bodies disrupt the barrier by direct damage to epithelial cells. Uremia also disrupts the barrier through the cytolytic effect of NH_3 produced by the bacterial degradation of urea.

Mucosal damage allows H^+ to leak back into the mucosa. As H^+ accumulates, intracellular buffers are gradually saturated and cell pH decreases to result in injury and cell death. Damaged mast cells release histamine which disrupts mucosal blood vessels. The result is local ischemia, hypoxia, vascular stasis, and leakage of plasma proteins and blood into the lumen (Figure 4).

Disruption of the barrier causes acid back-diffusion and gastritis (Figure 4) which is associated with decreased secretion, decreased motility, increased resistance to distention, and inhibition of emptying. Vomiting is the almost invariable outcome. Mucosal damage is often severe in the dog and cat as evidenced by the high incidence of hematemesis in these species.

Gastric Motility and Emptying

The stomach receives and stores food, mixes it with gastric secretions and delivers the resulting mixture into the duodenum. As a motor unit, the stomach has two separate components, a proximal receptacle (the fundus and body) and a distal 'pump' (the antrum) which mixes gastric contents and delivers them to the duodenum at a constant rate (Figure 5). The pylorus is a low-pressure sphincter that has little or no role in regulating the emptying of liquids, but is important in preventing large food particles from entering the duodenum.

The stomach empties when intragastric pressure exceeds duodenal pressure and pyloric resistance. The emptying of liquids occurs when intragastric pressure exceeds duodenal pressure and is brought about by slow (about 1 per minute) rhythmic fundic contractions.

The rate of emptying of digestible solids (which must first be broken down to a semi-liquid form) is a function of antral contractile activity, pyloric pressure and duodenal resistance.

Gastric contractile activity begins in the middle of the stomach. The maximum rate of contraction is determined by the slow wave frequency (~ 5/minute in the dog and cat). Contraction is stimulated by a number of factors including gastric distention, neural impulses, and the release of hormones such as cholecystokinin from the small intestine.

Gastric emptying is influenced by the physical and chemical composition of a meal. Digestible and non-digestible solids empty by different mechanisms. Digestible solids are reduced in size to fine particles (less than 2 mm in diameter) by the grinding and mixing of the distal antrum (Figure 6). These particles are suspended in fluid and the resulting liquid suspension is emptied into the duodenum. In a dog fed its caloric requirements once daily the stomach should empty within about 12 hours of eating. Vomiting food after

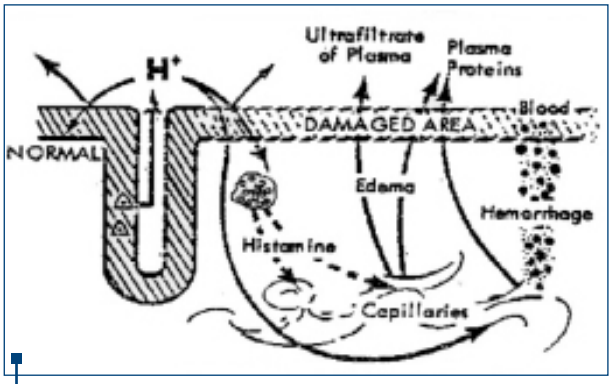

Figure 4. The normal and the broken barrier

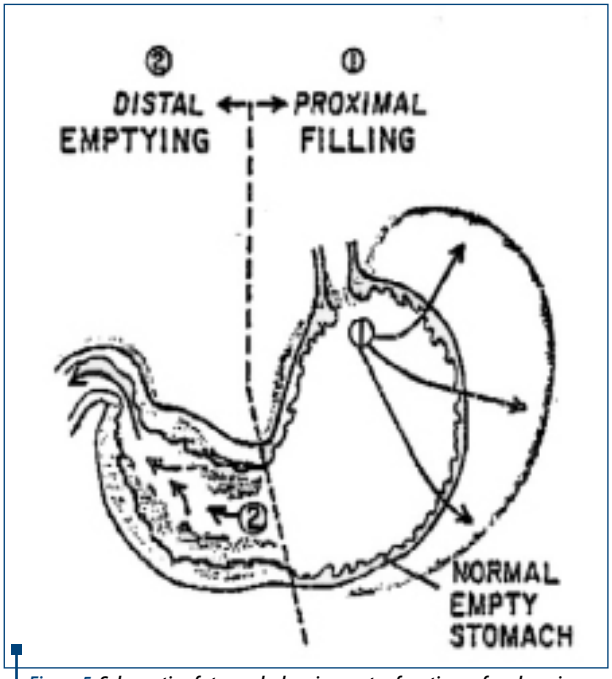

Figure 5. Schematic of stomach showing motor functions of each region. The proximal part is designed to adapt to entrance of food without an increase in pressure, and the distal part is concerned with vigorous motor activity to empty the organ

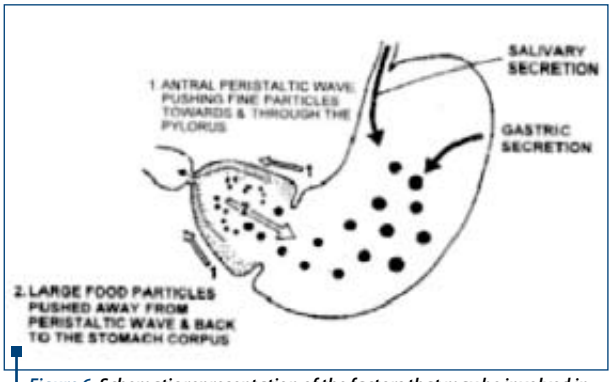

Figure 6. Schematic representation of the factors that may be involved in the intragastric digestion and in the sieving of food by the antropyloric pump

this suggests delayed gastric emptying. Non-digestible solids also trigger antral motility but most are impervious to mechanical grinding and are the last components of the meal to leave the stomach. This is carried out by the very strong contractions of the migrating motility complex, the appearance of which in omnivores and carnivores signals the onset of the fasted interdigestive state.

The rate of emptying is determined by receptors in the proximal small intestine for [H^+], osmolality, fatty acids, and tryptophan. Stimulation of these receptors initiates neurohumoral inhibition of antral motility and pyloric function. The purpose of this complex mechanism is to ensure delivery of energy to the proximal small intestine at a constant rate for subsequent digestion and absorption. Thus, the final determinant of gastric emptying is the caloric content of the meal.

Gastric motility is disrupted in a variety of inflammatory and neoplastic diseases of the stomach and small intestine. This delays gastric emptying and may cause vomiting; gastric outlet obstruction caused by distal antral or pyloric lesions has the same effect.

Vomiting

Vomiting, defined as "the spontaneous ejection of gastric and duodenal contents through the mouth", is a complex reflex act that requires the coordinated effort of the gastrointestinal, musculoskeletal and nervous systems, all of which are mediated through the emetic center in the medulla (Figure 7).

The vomiting center is activated by afferent vagal impulses from many parts of the body. Known stimuli of the emetic center are shown in Table 1. Emetic substances and labyrinthine stimulation (motion sickness) do not stimulate the emetic center directly, but act via the chemoreceptor trigger zone (CRTZ), a group of cells in the floor of the 4th ventricle. The blood-brain barrier is permeable at the CRTZ which is stimulated by emetic substances, such as apomorphine, some chemotherapeutic agents (e.g. adriamycin and cisplatin) and the "toxins" of uremia and ketosis that are carried in the blood.

Some substances such as ipecac, copper sulfate and staphylococcus toxin cause vomiting by direct gastric stimulation.

Vomiting begins with signs of depression or anxiety, followed by hypersalivation and excessive swallowing (nausea). During this time, retroperistalsis forces jejunal and duodenal contents into the stomach. This is followed by retching, which consists of forceful contractions of the abdominal muscles and diaphragm against a closed glottis. During retching the lower esophageal sphincter relaxes and gastric contents pass into the esophagus. The final act consists of expulsion of this material to the exterior. As vomitus passes through the pharyngeal cavity, the glottis remains closed to prevent inhalation and the orifice between the pharynx and nasopharynx closes to prevent nasal regurgitation.

Severe and prolonged vomiting causes dehydration along with loss of sodium, potassium and chloride. Hyponatremia may develop when the patient drinks excessively. Hypokalemia results from excessive renal potassium excretion, loss of gastric and duodenal fluids, and lack of intake. Large quantities of potassium may be lost in the urine in an attempt to conserve body sodium. Muscle wasting and ileus are frequent sequela to hypokalemia.

Acid-base status in the vomiting animal depends on the source of fluid loss. Loss of gastric juice from vomiting in prolonged gastric outlet obstruction is likely to cause major losses of hydrochloric acid. This results in hypochloremia, high serum bicarbonate concentration and metabolic alkalosis. Paradoxical aciduria may be observed as the kidneys preferentially absorb bicarbonate to defend against a chloride deficit. This occurs in only about 10% of vomiting patients. If excessive duodenal fluid is lost, blood pH may be either normal (60%) or low (30%) from the loss of bicarbonate-rich duodenal fluid.

Vomitus usually has an acid pH and may be composed of any combination of food, bile, blood or mucus. The presence of food and its state of digestion will depend on dietary composition, on gastric secretion and motility, and the time that has elapsed since ingestion.

Although most clients associate vomiting with gastric disease, it is important to realize that chronic vomiting is merely a sign, albeit an important one, of a variety of gastrointestinal, intra-abdominal, systemic, metabolic and neurologic disorders (Table 3). In most patients however, vomiting is associated with gastric or proximal small intestinal disease

Chronic vomiting or regurgitation?

Most clients are unable to differentiate vomiting from regurgitation and the vast majority of dogs with regurgitation are presented for diagnosis and treatment of 'vomiting'. Differentiation is critical however, and can usually be accomplished by means of a careful history. Differences between regurgitation and vomiting are summarized in Table 2. The first and most important question in this process is to ask the client to explain in his or her own words exactly what happens when the dog vomits. The clinician should be alert for words that denote an active process, such as retching or heaving, or the premonitory signs of vomiting such as restlessness and ptyalism. In most patients regurgitation is virtually an effortless act and may even occur, for example, during periods of sleep or play.

The age of the patient is also helpful in differentiation in that younger animals, especially those in which the problem develops at or soon after weaning are more likely to have a hereditary disorder that causes regurgitation, such as primary megaesophagus or a vascular ring anomaly. The history might also reveal that the animal has ingested abrasive or caustic material that may have resulted in esophagitis, or that the animal has recently been anesthetized which may have allowed

Table 2 Vomiting or regurgitation? A checklist for differentiation

Regurgitation	Vomiting
Passive process. Sometimes almost effortless expulsion of esophageal contents	Active process usually with vigorous abdominal contractions (retching)
Few additional premonitory signs except ptyalism in esophageal inflammatory or obstructive disease	Premonitory signs pronounced These include: ptyalism, pacing, swallowing, tachycardia (nausea)
Semiformed food material is usually obvious and may smell "fermented"	No characteristic consistency
Often contains mucus (saliva), blood is rare. Never bile-stained	Varies from freshly ingested food to liquid ± bile, blood and mucus May contain grass
pH of esophageal contents variable - unreliable indicator	pH of gastric contents variable - unreliable indicator

Table 3 General causes of chronic vomiting in the dog and cat

Stimulation from higher centers
- Psychogenic vomiting
- CNS tumor
- Increased intracranial pressure
- Encephalitis
- Limbic epilepsy

Stimulation from the cerebellum
- Motion sickness (rotation or unequal input from the labyrinths)
- Vestibular disease

Stimulation from the viscera
- Gastric distention or inflammation
- Intestinal distention and/or obstruction
- Enteritis (duodenitis, jejunitis, ileitis)
- Colitis
- Injury to kidneys, ureters, prostate or uterus
- Torsion of a viscus
- Peritonitis
- Pancreatitis
- Hepatitis
- Ileus
- Constipation and colonic distention
- Gastric, intestinal or pancreatic tumor

Stimulation from extra-visceral sources
- Pharyngitis and tonsillitis
- Heartworm disease (cat)

Effect of drugs or toxins
- Uremia
- Ketosis
- Chemotherapeutic agents (e.g. adriamycin, cisplatin)
- Cardiac glycosides
- Antimicrobials (eg. erythromycin, tetracycline)
- Hepatic encephalopathy

reflux of acid gastric contents into the esophagus. In contrast to animals with megaesophagus, swallowing is very painful in such patients and they are usually anorexic and salivate excessively.

The history should also permit differentiation of regurgitation from the dysphagia and immediate expulsion of ingesta from the pharynx that occurs in animals with pharyngeal motor dysfunction or the uncommon disorder of cricopharyngeal achalasia.

A careful history will also differentiate regurgitation and vomiting from the spurious vomiting associated with tonsillitis. This is a common complaint in miniature and toy breeds which gag or cough up mucus and saliva that is too painful to swallow. Examination of the oropharynx in such patients reveals an accumulation of white frothy mucus in the oropharynx and enlarged hyperemic tonsils.

The vomiting patient

History

A careful history and physical examination are critical in the chronically vomiting patient in that they help to pinpoint the probable cause or at least the appropriate underlying organ system and the direction of subsequent diagnostic and therapeutic maneuvers.

Important questions to the owner of a vomiting dog are given in Table 4. Most important is to establish whether the patient is regurgitating or vomiting, the appearance of the vomitus, and the relationship of vomiting to feeding. The age of the patient may also be of diagnostic benefit (Table 5).

Blood, bile, mucus or food in varying stages of digestion may be found in the vomitus. Blood may be either fresh or digested (coffee grounds) and its presence suggests gastric mucosal damage. Bile, characterized by a yellow or greenish color, is not at all unusual since vomiting begins with jejunal retroperistalsis and small intestinal contents are swept into the stomach before the actual act of expulsion. Mucus in the vomitus is

the rule rather than the exception and comes from either swallowed saliva, or from the gastric or small intestinal mucosa.

The presence of blood, for example, is usually a serious prognostic sign suggesting a bleeding ulcer or tumor, uremic gastritis, or at the least, severe erosive gastritis. The presence of bile is not unusual, but the regular vomiting of bile-stained mucus, especially in the early morning, should suggest reflux gastritis.

A meal sufficient to supply total daily caloric requirements is normally emptied from the stomach within 7-10 hours of ingestion so the presence of food in the vomitus, its state of digestion and most importantly, the time of vomiting after feeding are critical. Food in the vomitus more than 10 hours after a meal suggests the presence of gastric outlet obstruction or a gastric motility disorder and the need for diagnostic studies to be focused on those parts of the gut likely to be involved in the obstructive process, namely the stomach, pylorus, proximal duodenum and pancreas.

The frequency and duration of vomiting may vary from weeks to years. Chronic vomiting of several months duration without some loss of body weight or condition is unusual. The more weight that is lost,

Table 4 Important questions for the owner of the vomiting patient

QUESTION	INTERPRETATION
Describe act	Vomiting or regurgitation?
Appearance of the vomitus	Mucus - salivary or gastric Bile - reflux from small intestine or reflux gastritis Blood - state of digestion; tumor or ulcer if chronic Food - state of digestion Grass - nonspecific sign. If animal has begun to eat a lot of grass, it might suggest abdominal disease
Duration	Acute or chronic?
Frequency	Severity
Relationship to eating	Outlet obstruction or abnormal motility if vomiting consistently more than 7-10 hours post-prandial
Diet and opportunity to eat spoiled food or to scavenge	Dietary sensitivity, toxic gastritis, foreign body

Table 5 Major causes of chronic vomiting in the dog and cat grouped according to age

Young animals
Gastrointestinal parasites (roundworms, hookworms)
Partial intestinal obstruction (foreign body, tumor)
Chronic gastritis
Gastric foreign body
Adrenal insufficiency (dogs)
Chronic enteritis

Mature animals
Gastrointestinal parasites (Physaloptera, Giardia)
Pancreatitis
Chronic gastritis
Gastric or small intestinal foreign body
Enteritis or colitis
Gastric outlet obstruction (dogs)
Gastric or small intestinal tumor
Adrenal insufficiency (dogs)
Abdominal disorders (e.g. peritonitis, prostatitis, pyometra)
Ketoacidosis
Central nervous system disorders (encephalitis, trauma, vestibular disease)
Liver disease
Uremia

Old animals
Uremia
Gastric or small intestinal tumors
Dietary indiscretion
Pancreatitis
Chronic gastritis
Gastric ulcer
Enteritis or colitis
Abdominal disorders (prostatitis, pyometra)
Ketoacidosis
Liver disease
Central nervous system disorders

Table 6 Diagnostic studies in the vomiting patient

Test	Interpretation
Hemogram	
PCV, Hb, RBC	Increased - dehydration; decreased - gastrointestinal blood loss
WBC	Increased inflammation; decreased - sequestration or loss into gut
Blood chemistries	
Na^+, Cl^-	Normal or decreased-outlet obstruction
K^+	Normal in most patients; decreased - outlet obstruction; increased - azotemia or hypoadrenocorticism
Total CO_2	Increased - gastric outlet obstruction; decreased acidosis
BUN	Increased - gastrointestinal bleeding or azotemia; decreased hepatic insufficiency
Creatinine	Increased - azotemia
ALT, ALP	Increased - hepatic disease pancreatitis or intestinal disease
Serum Proteins	Increased - dehydration; decreased - protein losing enteropathy, ascites
Triglycerides	Increased - pancreatitis
Amylase/Lipase	Increased - pancreatitis, azotemia, inflammatory bowel disease
cPLI/fPLI	Increased in pancreatitis
Radiographic Studies	
Survey abdominal films	Gastric distention, foreign body, mass, visceral displacement, ileus, effusion
Upper gastrointestional	Delayed gastric emptying, filling defect, foreign body contrast study
Endoscopy and Mucosal Biopsy	Direct examination, biopsy of lesion, foreign body removal
Exploratory Laparotomy	Definitive diagnosis and/or treatment
Urinalysis	Low specific gravity suggests azotemia
Fecal Exam	Gastrointestinal parasites

then the more severe the prognosis, especially if the frequency is increasing and the appetite continues to decrease.

If the patient has concomitant diarrhea then diagnostic efforts should be focused on the intestinal tract, for it is likely that the vomiting is secondary to underlying intestinal disease.

Physical Findings

The degree of hydration and vital signs may be sufficiently abnormal to necessitate symptomatic and supportive therapy while a diagnosis is pursued. The results of the physical examination will depend upon the underlying cause. The state of nutrition and overall appearance of the patient should suggest the severity and chronicity of the disease process. Abdominal palpation should always be thorough and may reveal a possible cause such as a mass or localized pain, thickened intestinal wall or dilated intestinal loop.

Diagnostic Studies

Diagnostic studies and their possible results are listed in Table 6. Uremia is a common cause of chronic vomiting, especially in the older patient and it is advisable to rule out this diagnosis before pursuing other possibilities. A decrease in serum potassium and chloride and an increase in total CO_2 suggest loss of gastric juice and gastric outflow obstruction.

Survey and upper gastrointestinal contrast studies assume greater importance in the chronically vomiting patient in that they help to diagnose such causes of vomiting as gastric and intestinal foreign bodies and intra-abdominal masses. Contrast studies help to reveal such causes of chronic vomiting as gastric outlet obstruction, gastric nonradiopaque foreign body, gastric ulcer, intestinal tumors and foreign bodies. The most important diagnostic procedure in the clinically vomiting patient, however, is endoscopy. Endoscopy allows direct evaluation of the esophagus, stomach and proximal small intestine. Mucosal abnormalities can be visualized, foreign bodies removed, duodenal aspirates obtained for diagnosis of giardia, and biopsies taken. A mucosal biopsy is essential for diagnosis in the patient that is vomiting as a result of gastric or small intestinal mucosal disease.

An exploratory laparotomy should be considered if an endoscope is unavailable and the clinical conditions warrant it. Laparotomy is a legitimate diagnostic procedure and if biopsies are taken, allows a specific diagnosis to be made.

Treatment

Symptomatic and supportive treatment is indicated if dehydration and vomiting are sufficiently severe, but in most chronically vomiting patients, treatment should be directed at control or elimination of the underlying disease.

Treatment will depend on the diagnosis, but could range, for example, from the use of corticosteroids and cimetidine to treat plasmacytic lymphocytic gastritis, through surgical removal of a foreign body to supportive care of uremia in the patient vomiting because of end-stage renal disease. ■

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Canine and feline liver disease

Colin F. Burrows
BVetMed, PhD, HonFRCVS, DACVIM, College of Veterinary Medicine, University of Florida, Gainesville, FL

Abstract

Acute hepatitis and acute hepatocellular degeneration are characterized by focal or diffuse damage to the hepatocyte. The causes of hepatocellular disruption are many and varied and are often undetermined. Some of the recognized hepatotoxins include chemical solvents like carbon tetrachloride, mycotoxins like aflatoxin, drugs like acetaminophen (in the cat), trimethoprim-sulpha antibiotics, certain mushrooms, viruses, and in tropical and subtropical areas, the false sago palm fruit (clinically important in the Southeastern United States). Clinical signs, biochemical data and histological findings do not usually indicate a specific cause, resulting only in a histologic diagnosis of "toxic" hepatitis.
Keywords: *acute hepatitis, hepatocellular degeneration, hepatotoxins, histologic diagnosis*

The predominant lesion of acute hepatitis is multifocal necrosis of individual hepatocytes associated with abnormal liver enzyme activity.

Clinical presentation

Clinical signs are variable and include inappetence, lethargy and vomiting. Icterus develops if the hepatocellular insult is extensive.

Differential diagnosis

- Acute pancreatitis
- Infectious canine hepatitis
- Acute gastroenteritis
- Severe tryptic enteritis (shock gut)
- Some toxicoses

Diagnosis

The predominate biochemical abnormality is an increase in ALT and AST, the magnitude of which depends on the severity and extent of the hepatocellular damage. The serum alkaline phosphatase (ALP) is usually normal or only slightly increased early in the disease process. An increase in serum bilirubin concentration may occur if a sufficient number of hepatocytes have been damaged or destroyed. The patient will recover if enough functional liver remains to support regeneration.

A liver biopsy is often neither helpful nor essential in the diagnostic evaluation and may be contraindicated by liver-associated coagulopathies. Histologic examination can be useful for assessing the severity and extent of the disease process. A liver biopsy is justified if recurrent abnormal ALT/AST values have been documented (see chronic active hepatitis).

Management

The prognosis is usually good if the inciting cause is removed, permanent loss of functional mass is less than 50%, and the liver has retained the capacity to regenerate.

Chronic inflammatory liver disease

Definition/Overview

The dog and, to a much lesser degree, the cat suffer from a variety of chronic inflammatory liver diseases (Table 1). All have similar clinical signs. In the dog, chronic hepatitis has been associated with leptospirosis, the administration of primidone, dilantin and phenobarbital, abnormal copper metabolism in Bedlington terriers, Dalmatians and West Highland White Terriers, and experimentally induced infectious canine hepatitis. A syndrome of chronic hepatitis with intracellular copper retention has also been recognized in the Doberman Pinscher and American Cocker Spaniel. Chronic hepatitis also has an increased prevalence in Yorkshire Terriers, Laborador Retrievers, Cairn Terriers, Great Danes, Collies, and West Highland White Terriers.

Chronic inflammatory liver disease is less common in the cat and is primarily associated with bile duct inflammation (cholangiohepatitis).

Etiology

The diseases have overlapping histological appearances and are mostly of unknown cause. Chronic inflammation, however, is believed to result from a one time hepatic insult that results in exposure of hepatic antigens and an immune mediated response (Figure 1).

Pathophysiology

Chronic hepatitis is the best known and most frequently diagnosed disorder and is characterized by continuing hepatic inflammation, necrosis and regeneration.

Clinical presentation

Clinical signs are nonspecific: inappetence and lethargy are common complaints. Occasional vomiting, diarrhea and pica may also be reported. Icterus may develop terminally. Signs may be chronic and the disease far advanced before a diagnosis is made.

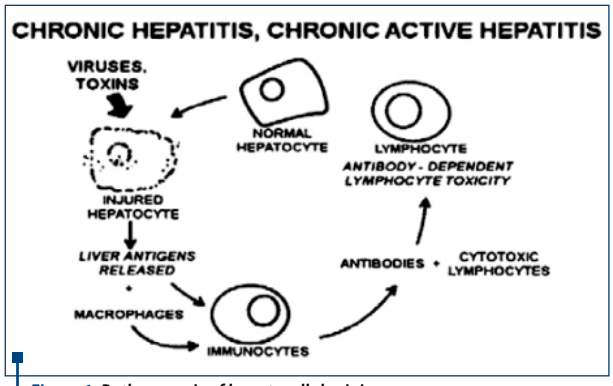


Figure 1. Pathogenesis of hepatocellular injury

Table 1 Inflammatory disorders of the canine and feline liver

Dogs	Cats
Chronic active/persistent hepatitis	Cholangiohepatitis
Breed related e.g.	Chronic portal triaditis
Doberman	Chronic suppurative triaditis
Cocker Spaniel	Chronic suppurative cholangiohepatitis
Labrador retriever	Chronic non-suppurative
Copper storage hepatopathy	Chronic non-suppurative cholangitis
Dalmatian, Bedlington terrier	Chronic major bile duct obstruction
West Highland white terrier	Polycystic liver disease
Chronic non-suppurative hepatitis	Idiopathic hepatic fibrosis
Chronic portal triaditis	
Drug-induced hepatitis	
Chronic cholangiohepatitis	
Chronic cholangitis	
Chronic lobular hepatitis	
Lobular dissecting hepatitis	
Chronic major bile duct obstruction	
Idiopathic hepatic fibrosis	

Differential diagnosis
- Infection (bacterial or viral)
- Drug Induced
- Familial
- Cirrhosis

Diagnosis

The disease is characterized histologically by periportal inflammation and fibrosis. More ominous histologic features suggestive of progression are implied when confluent areas of necrosis are identified that form zones of parenchymal collapse and bridge between portal triads and central veins or span lobules between portal triads. Chronic hepatitis should be suspected when recurrent slight to moderate increases in serum ALT and ALP activity are observed, especially in susceptible breeds. Continuation of the chronic inflammatory process for months to years disrupts normal liver architecture and the earlier biochemical pattern of "low-grade" hepatocellular necrosis progresses to one of hepatic insufficiency. The disease may be difficult to detect biochemically at this stage since serum AST, ALT and ALP activities may be normal or only slightly increased. This is due to the loss of hepatocytes (hepatocellular drop out/destruction and replacement with collagen). A decrease in serum albumin and urea nitrogen concentration are suggestive of advanced liver disease. Earlier indicators of liver insufficiency are marked bilirubinuria and increased serum bile acids. Disruption of clotting factor synthesis may result in bleeding. Histological examination of a liver biopsy sample confirms the diagnosis.

Management

Treatment is symptomatic and supportive (antioxidants, free radical scavengers, immunomodulation, diet).

Cirrhosis

Cirrhosis is a diffuse hepatic disease process characterized by fibrosis and by an alteration of normal hepatic architecture by structurally abnormal regenerative nodules. Cirrhosis is the final, irreversible end stage of many chronic liver diseases.

Etiology

The cause of cirrhosis is varied and is seldom determined.

Pathophysiology

Increased hepatic vascular resistance and portal hypertension result from fibrosis and leads to ascites, hepatic encephalopathy and multiple acquired portosystemic shunts.

Clinical presentation

Clinical signs most often include inappetence, ascites and encephalopathy.

Differential diagnosis
- Metastatic disease
- Nodular regeneration

Diagnosis

Biochemical findings are often subtle. Serum ALT and ALP activities may be normal or only slightly increased because of the decreased hepatic cell mass. Other biochemical abnormalities reflect the reduced functional capacity of the liver and altered hepatic blood flow secondary to changes in architecture. Decreased serum urea nitrogen and a decreased serum albumin concentration may be noted. Increased serum bilirubin concentration is a late indicator of liver insufficiency; persistent bilirubinuria will be detected before jaundice develops. Hematologic changes include anemia (chronic gastric bleeding from gastric ulcers caused by congestive gastropathy) and red cell morphologic changes (poikilocytosis, target cells, spur cells).

Determination of total serum bile acids (fasting and postprandial) is the most reliable test for detecting hepatic insufficiency in the anicteric patient. A reduced number of hepatocytes and an altered portal blood flow result in an increase in peripheral bile acid concentration.

Jaundice may develop in the cirrhotic patient without prior clinical evidence of liver disease and may require differentiation from extrahepatic impairment of bile flow. The increased total serum bilirubin concentration is often lower (usually <6 mg/dL) in the cirrhotic patient than in the patient with extrahepatic biliary obstruction. The absence of a marked increase in ALP activity, a decrease in BUN or a decrease in the serum albumin concentration support a diagnosis of cirrhosis.

Histologic confirmation of cirrhosis is important since on visual inspection cirrhosis may resemble metastatic disease or nodular regeneration.

Management

Supportive measures are aimed at controlling the complications of chronic liver failure, such as hepatic encephalopathy and secondary infection from increased bacterial translocation.

Feline hepatic lipidosis

Definition/Overview

Feline hepatic lipidosis is a syndrome that can be either a primary idiopathic condition or secondary to a variety of common diseases of cats. Hepatic lipidosis is the most common liver disease of the cat accounting for approximately 50% of diagnoses. The disorder accounts for approximately 10% of liver-related deaths in this species. Other names include feline fatty liver syndrome, steatosis and fatty liver.

Etiology

A variety of factors have been proposed as potential etiologies, none of which, however, have been confirmed. A multifactoral pathogenesis leading to malnutrition is likely. The major risk factor is obesity and a prolonged reduction of food intake but obesity is not universally present in affected cats. Anorexia may be caused by concurrent disease, dietary change, or decreased food intake. Environmental stress is potentially an additional important risk factor.

Pathophysiology

The exact pathogenesis remains to be defined. Decreased caloric intake causes a negative nitrogen balance. Fatty acids are mobilized from tissue stores and transported to the liver. Decreased protein metabolism and amino acid availability means that there is insufficient apoprotein to facilitate its removal and fat accumulates in the hepatocyte. This decreases the ability of the cell to function and liver function slowly decreases.

Clinical presentation

Most afflicted animals are two years old or older. Cats with secondary hepatic lipidosis tend to be older than cats with idiopathic hepatic lipidosis. There is no apparent breed or gender predisposition, but older females seem to be more at risk. Affected cats are commonly obese and/or have experienced a stressful event of some type (e.g. a change in environment or concurrent disease). This is followed by anorexia and rapid weight loss. Anorexia persists and the cat is presented for veterinary evaluation usually between one and three weeks after onset. Jaundice develops in most cats and is usually evident on presentation.

At examination cats are depressed, dehydrated, and icteric and show varying degrees of muscle wasting. Fat pads remain intact, however, reflecting the cat's inability to mobilize fat in this disease and the marked muscle breakdown for gluconeogenesis. Hepatic encephalopathy may be related to severe hepatocellular dysfunction or to a relative deficiency of arginine, to which the anorexic cat is predisposed (cats cannot synthesize arginine and must rely on dietary sources). Abdominal palpation reveals hepatomegaly in the majority of afflicted cats.

Differential diagnosis

- Idiopathic hepatic lipidosis
- Secondary hepatic lipidosis (DM)
- Hepatic lymphoma

Diagnosis

Diagnosis is based on histologic examination of a liver biopsy specimen. Typical laboratory findings are those of cholestasis; total bilirubin values range from normal (0.3 mg/dL to 15 mg/dL or higher). There is usually a mild nonregenerative anemia. Liver enzyme activities are elevated; the ALT is normal or moderately elevated (100 to 450 IU/L), the AST is normal or moderately elevated (50 to 450 IU/L) and the ALP occasionally normal but mostly elevated (75 to 1200 IU/L). Most cats with lipidosis have an ALP between 200 and 600 IU/L. Fasting serum bile acid concentrations are above normal (10 mg/dL) in most cats. The glucose may be elevated, but should not be confused with diabetes.

Abdominal radiographs reveal hepatomegaly in the majority of cats. Ultrasonographic examination of the liver and surrounding structures allows other diseases in the differential diagnosis such as cholangiohepatitis and extra-hepatic bile duct obstruction to be ruled out. The principal ultrasonographic feature of hepatic lipidosis is hyperechogenicity (fat is more dense than water).

Tissue diagnosis can be made by aspiration cytology in some patients, but in the majority of cats is achieved by fine-needle aspiration, percutaneous biopsy or by exploratory laparotomy. Specimens are placed in buffered 10% formalin in which they usually float.

Management

The mainstay of therapy is complete nutritional support and treatment of known concurrent illness.

Cholangitis/cholangiohepatitis

Cholangitis (inflammation of the bile ducts) and cholangiohepatitis (inflammation of the adjacent hepatic parenchyma) is a relatively common liver disease of the cat, but is diagnosed with a lower frequency in the dog.

Etiology

The cause is unknown, but is believed to be due to an ascending bacterial infection of the biliary tract, especially by Gram negative and anaerobic organisms.

Pathophysiology

Bacterial infection causes an initial neutrophilic inflammatory response which later changes to a lymphocytic and plasmacytic infiltrate. Suppurative or mononuclear inflammation of the bile ducts is typical of acute cholangiohepatitis. Periportal hepatocellular necrosis takes place, the limiting plate become disrupted and the inflammatory infiltrate extends into the hepatic parenchyma. The chronic form typically shows a mixed inflammatory response within portal areas and bile ducts.

Clinical presentation

Clinical signs are vague and recurrent; jaundice often develops by the time the liver disorder is recognized. A mild ascites may occur in some cats. Fever and obstructive jaundice may also be present. In dogs an acutely painful abdomen may be detected on palpation especially if the gallbladder is inflamed (cholecystitis). Most afflicted cats are older than four years of age and there is a breed predilection for Persians.

Differential diagnosis

- Congenital biliary tract malformations
- Biliary reconstructive surgery
- Extrahepatic obstruction of the bile duct
- Sinusitis
- Splenic abscess
- Pyelonephritis

Diagnosis

Early laboratory findings of cholangiohepatitis include increased ALT, AST and ALP activities and later hyperbilirubinemia. The ALT and AST levels are almost invariably higher than the ALP and are in the low to mid hundreds (200-600) in the cat, whereas in the dog the ALP is typically higher. Hyperglobulinemia may be present, especially in the cat. In the anicteric patient, the fasting serum bile acid concentration may be normal but the 2 hours post-prandial bile acid concentration is almost invariably elevated. A mild nonregenerative anemia and a neutrophilia with a mild left shift may be noted on the hemogram. Prothrombin and partial thromboplastin time should be measured and vitamin K1 given as needed prior to biopsy.

Either a percutaneous needle biopsy or wedge biopsy taken at laparotomy can be used for histologic confirmation of the diagnosis. Early in the disease process the liver may appear swollen. Later however, as the amount of connective tissue increases, the liver becomes firmer with an irregular surface. Thickened or dilated bile ducts with accentuation of the portal areas may be observed on cut section. A gross similar appearance may be observed subsequent to infection with liver flukes in patients from endemic areas.

Histologically, the feline cholangitis/cholangiohepatitis syndrome can be grouped according to the predominant type of inflammatory cell in the portal area and involvement of the surrounding hepatocytes. The inflammatory cell component may be suppurative (neutrophils) or nonsuppurative (lymphocytes +/- plasma cells), the more common form. In the dog liver biopsy shows a predominantly purulent inflammation. If neutrophils predominate liver tissue and bile should be cultured for aerobic and anaerobic bacteria. Pancreatitis and/or plasmacytic lymphocytic enteritis may be coincidental diseases in the cat.

If a laparotomy is performed to obtain the liver biopsy, the major bile ducts should be evaluated for patency and the gall bladder expressed and palpated for choleliths and inspissated bile.

Management

Primary treatment includes antibiotics on the basis of results of culture and sensitivity testing. In chronic disease glucocorticoids will also be required. Ursodeoxycholic acid is recommended in all cats in whom extrahepatic biliary obstruction has been eliminated. Nutritional support is an important part of treatment.

Breed related copper toxicity

A hereditary metabolic disturbance occurs in certain Bedlington terriers, Dalmatians, West Highland white terriers, and an emerging number of other breeds. A related condition is seen in Dobermans. In Bedlingtons the disease shows an autosomal recessive pattern of inheritance and is similar to hepatolenticular degeneration (Wilson's disease) in humans. The disease is one form of chronic liver disease which progresses to cirrhosis.

Etiology

The disease is caused by an inherited metabolic defect. Copper is abnormally bound with metallothionein in the liver as a result of dysfunctional biliary excretion of copper.

Pathophysiology

Accumulation of copper in the liver leads to major injury as the hepatic mitochondria becomes damaged by oxidants. Acute release of copper from the necrotic hepatocytes may cause hemolytic anemia. With progressive disease, the liver diminishes in size and a mixture of fine and course fibrotic nodules are apparent.

Clinical Presentation

The clinical signs and biochemical findings are similar to these described for chronic active hepatitis.

Differential diagnosis

- Infections
- Drug induced
- Familial
- Lobular dissecting hepatitis
- Idiopathic chronic hepatitis

Diagnosis

The diagnosis is suggested by increased liver enzyme activity and confirmed by copper analysis of a fresh piece of liver.

Management

Asymptomatic animals are best treated by zinc supplements and restriction of dietary copper. Chelator therapy, for example penicillamine, may be required on the basis of periodic monitoring of hepatic copper content. Affected dogs and carriers of the gene should be identified and removed from breeding programs.

Drug-induced liver disease

Drugs can cause liver injury ranging from a transient asymptomatic increase in serum transaminase activity to clinically overt acute or chronic liver disease.

Etiology

Drugs can induce liver injury by two mechanisms: 1) as a direct hepatotoxin or 2) a hypersensitivity reaction.

Pathophysiology

Direct-acting hepatotoxins usually cause acute necrosis when the drug or a metabolite chemically interacts with an essential structural component or metabolic enzyme system of the hepatocyte. An example is acetaminophen (Tylenol).

A drug or its metabolite may also induce liver injury by either altering the regulatory system of the immune response so that reactions to self-antigens are no longer suppressed or by altering hepatocyte antigens so that they are no longer recognized as self-components.

Clinical presentation

A drug history should always be taken. Carprofen (Rimadyl), for example, causes hepatotoxicity in some Labrador Retrievers. Animals may present with several signs, depending on the inciting cause, including lethargy, ataxia, weight loss, anorexia, behavioral changes, coagulopathy, jaundice and ascites.

Diagnosis

A hypersensitivity response is very difficult to prove since challenge exposes the patient to unnecessary risk. Sulfa-containing drugs, anticonvulsants, antielmintics and halothane have been implicated in veterinary medicine.

Differential diagnosis

- Lobular dissecting hepatitis
- Chronic hepatitis

Management

It is prudent to discontinue the inciting drug. If the drug is essential, then the dose should be modified as far as possible.

Extrahepatic biliary obstruction (Extrahepatic Impairment of Bile Flow)

Extrahepatic biliary obstruction refers to the impairment of bile flow in the biliary system between the liver and the duodenum. Acute, total extrahepatic impairment of bile flow is rare.

Etiology

Biliary mucocele, chronic pancreatitis and tumors of the pancreas and the bile duct epithelium are the most common causes, although gallstones lodged in the common bile duct have been reported in both the cat and the dog. Inspissated bile (most often associated with an underlying liver disorder) and liver flukes are additional causes.

Pathophysiology

Biliary mucoceles are caused by mucincus hyperplasia of the gall bladder epithelium. Acute and chronic pancreatitis in the dog and cat may result in sufficient inflammation/fibrosis/fat necrosis to cause an anatomical obstruction involving the common bile duct. Scars usually form around or in the bile ducts.

Clinical presentation

Signs include inappetence, lethargy, icterus and repeated vomiting (if the pancreas is involved). Both cats and dogs may present with a chronic history of anorexia, lethargy and jaundice. Physical findings are usually unremarkable except for icterus and abdominal pain. If a patient with suspected pancreatitis remains icteric beyond 10 to 14 days of symptomatic medical management, an extrahepatic component should be suspected and managed accordingly.

Differential diagnosis

- Parasitism (flukes)
- Neoplasia
- Choleliths

Diagnosis

Extrahepatic cholestasis causes a marked increase in the hepatic synthesis of ALP resulting in a concomitant increase in serum activity. Retained bile acids also cause hepatocellular damage with an associated mild to moderate increase in serum ALT activity. Clotting studies reveal increased PT and PTT which should revert to normal with vitamin K1 therapy. The test for bilirubinuria will be strongly positive. Ultrasonography is a valuable, noninvasive diagnostic tool since it allows visualization of a dilated extrahepatic bile duct.

Acute pancreatitis occasionally causes a mild to moderate increase in serum ALT activity, a marked increase in ALP activity and increased total serum bilirubin concentration. The biochemical pattern may appear similar to extrahepatic biliary obstruction or to chronic active hepatitis with intrahepatic cholestasis. An acute onset of repeated vomiting preceding the development of jaundice suggests acute pancreatitis. Marked increases in serum lipase and amylase activities also suggest the disease, but are not diagnostic. Suggested causes for the transient increase of the liver tests associated with acute pancreatitis include: 1) inflammation of the peripancreatic tissue which compresses the bile duct and 2) the release of proteases into the portal blood which directly damage hepatobiliary tissue.

Management

Surgical correction is the treatment of choice. If no obstruction is found at laparotomy, the liver should be biopsied and the patient managed accordingly.

Gallstones

Gallstones are occasionally found in dogs. Most are serendipitous findings on abdominal radiographs or at necropsy. On rare occasions gallstones can cause obstruction of the common bile duct, but simply finding them on radiographs or on abdominal ultrasound in patients with evidence of liver disease is not justification for removal.

Etiology

Ionized calcium may play a part in the formation of gallstones as it is the main component of pigment gallstones. Choleliths are rare in dogs owing to the absorption of ionized calcium from the gallbladder, reducing the concentration of free ionized calcium in bile.

Pathophysiology

Gallstones in dogs usually contain mucin, calcium and bilirubin.

Clinical Presentation

Gallstones often cause no clinical signs and are incidental findings at necropsy or during imaging. If signs are

apparent they may include vomiting, icterus, anorexia, fever and abdominal discomfort.

Differential Diagnosis

- Parasitism
- Neoplasia
- Non-biliary tract disorders
- Rarely, gallbladder cysts (biliary mucocele)

Diagnosis

Gallstones are typically diagnosed by abdominal ultrasonography. They appear as hyperechoic foci or can be detected by acoustic shadowing originating from the gallbladder. Exploratory laparotomy should be performed and the patency of the common bile duct ascertained.

Management

The usual treatment is surgical excision with follow up therapy with ursodeoxycholic acid.

Rupture of the biliary tract

The common bile duct, distal to the opening of the last hepatic duct seems to be the most common site of ductal rupture. Early diagnosis is necessary to avoid bile peritonitis.

Etiology

Blunt trauma, penetrating wounds, pathology associated with a tumor or infection, biliary mucocele (ruptured gall bladder) and liver biopsies have been associated with rupture of the biliary system.

Pathophysiology

Extravasation of bile elicits a strong inflammatory response and causes transudation of lymph from serosal surfaces.

Clinical presentation

The clinical course is usually protracted unless associated with a biopsy procedure in which any inflammation is self limiting. There is acute abdominal pain for the first 48 hours followed by anorexia, depression, fever, slow abdominal distension and icterus.

Differential diagnosis

- Hepatic tumors
- Bacterial peritonitis

Diagnosis

An exudative, yellow to dark green/black-colored abdominal effusion is found at abdominocentesis. Macrophages containing bile pigment may be noted in a smear of the fluid.

Management

Exploratory surgery is always indicated.

Liver tumors

Primary and metastatic tumors occur in the liver. Metastatic tumors are reportedly twice as frequent as primary tumors. Primary tumors include bile duct adenoma (adenocarcinoma), hepatocellular adenoma, lymphoma, and carcinoma (hepatoma). Hepatocellular carcinomas have been associated with hypoglycemia in the dog. Lymphosarcoma is the most common liver tumor.

Etiology

The cause of primary tumors is not usually determined. Potential causes include nitrosamines, aramite, liver flukes, aflatoxins and radioactive compounds. Metastatic tumors in the dog arise from the pancreas, mammary glands, adrenals, bone, lungs, thyroid, GI tract and spleen whereas in the cat they arise from kidney, pancreas and GI tract. Metastatic hemangiosarcoma is relatively common.

Pathophysiology

Biliary and hepatocellular cancer occur as multifocal nodular or diffuse infiltrations of large areas of liver or as solitary masses.

Clinical presentation

Clinical signs are vague and nonspecific in most patients. They include inappetence progressing to anorexia, weight loss, vomiting, abdominal distention (sometimes marked) and terminal jaundice in some patients.

Differential Diagnosis

- Parasitism
- Hepatobiliary cysts
- Cirrhosis

Diagnosis

Those tumors which do not cause extrahepatic biliary obstruction may cause increases in the serum AST, ALT and ALP activities and total serum bilirubin concentration that may appear similar to the pattern associated with chronic hepatitis. One or more of these tests are abnormal in approximately 50% of the dogs with metastatic tumors but are abnormal in 100% of the dogs with the less common but often massive primary hepatocellular carcinoma.

Abdominal radiographs are often read as normal but may reveal hepatomegaly. Ultrasound guided biopsy or laparotomy and biopsy are essential for diagnosis of neoplastic disease. The gross appearance of neoplastic lesions may appear similar to nodules associated with cirrhosis. Histologic evaluation can differentiate between the two lesions. Microscopic examination of a hepatic aspirate can help diagnose hepatic lymphoma.

Management

Primary tumors confined to a single lobe may be resected surgically. The abdominal cavity should be evaluated for metastatic spread and biopsy specimens of hepatic lymph nodes should be obtained. Advanced therapy may be available through referral services but is of limited value.

Congenital portal systemic vascular anomalies

Congenital anomalies of the portal vascular system (portasystemic shunts-PSS) occur commonly in the dog and are being recognized with increasing frequency in the cat. The anomalous vascular development can involve one or more vessels of the hepatic portal circulation. Single intrahepatic shunts are more common in large breed dogs and single extrahepatic shunts are more common in cats and small breed dogs.

Etiology

The reason(s) congenital portasystemic shunts develop is not known but there appears to be a genetic basis in certain lines of miniature Schnauzers, Irish wolfhounds, Old English sheep dogs, and Cairn terriers. A microvascular dysplasia within the liver has been reported in the latter

breed. Yorkshire terriers and miniature Schnauzers are other breeds that appear to be at increased risk. Mixed breed cats are most commonly affected and of the purebreds, Himalayan and Persian appear to be at increased risk. Most animals develop signs by ten months of age but signs may be subtle and adequately compensated (or accepted by the owner) until more prominent later in life (up to ten years of age).

Pathophysiology

The liver appears grossly small and often mottled, and there is typically atrophy of the hepatocytes with arteriolar hyperplasia and small or absent portal veins. Other features include sinusoidal congestion, biliary hyperplasia, lipogranulomas, increased periportal connective tissue and periportal vacuolization.

Clinical presentation

This is one liver disorder in which the signalment and history provide important clues to the diagnosis. Affected animals may be small for their age, less active than their littermates and may be labeled 'poor doers'. Neurologic signs are common in the dog and include seizures and personality changes. Polydipsia/polyuria, ptyalism or recurrent formation of urinary calculi are also common historical findings. Renal, cystic, and urethral calculi may be the first clinical indication of an underlying congenital portasystemic shunt; urolithiasis develops commonly. Small stature, recurrent behavioral changes and ptyalism highlight the clinical features in young cats. Intermittent depression, disorientation, aggression, head pressing, blindness, mydriasis and seizures have also been observed. Signs may or may not be associated with eating. Unexpected prolonged recovery from general anesthesia or an exaggerated response to tranquilizers have been noted.

Findings at physical examination are often unremarkable; occasionally an inappropriately small body stature is present. Slightly enlarged kidneys have been palpated in some cats. Some cats have a copper colored iris.

Differential diagnosis

- Hepatic microvascular dysplasia
- Congenital enzyme deficiency of the urea cycle
- Infectious diseases (CDV, FIP, diseases related to FeLV and FIV, toxoplasmosis)
- Idiopathic epilepsy
- Metabolic disorders (for example, hypoglycemia, thiamine deficiency)
- Hydrocephalus
- Toxicity

Diagnosis

A missed diagnosis can easily occur since few abnormalities are evident on the biochemical profile. Serum ALT and ALP activities may be normal or only slightly increased. Serum albumin and urea nitrogen concentrations are decreased in many patients with PSS. Decreased concentrations occur because of hepatic atrophy and insufficient portal blood flow. These two parameters are valuable indicators of liver insufficiency.

A mild hypoglycemia may also be detected. The combination of a compatible history and biochemical abnormalities indicate the need for a function test such as serum bile acids or blood ammonia. Ammonium biurate crystals may be observed in the urine sediment and evaluation of the hemogram may reveal a slightly decreased MCV. Serum iron is decreased in over 50% of dogs with confirmed portasystemic shunts.

Survey radiographs may reveal microhepatica in some patients. Visual confirmation of the portal vascular anomaly requires ultrasonography, scintigraphy, computerized tomography, a cranial mesenteric angiogram, splenoportography or jejunal vein portography.

Histologically, the liver may appear normal architecturally or there may be subtle changes of hepatic cord atrophy, small or absent portal veins, and an increase in arteriolar structures. Other nonspecific findings that may be reported include periportal or midzonal vacuolization, mild to moderate biliary hyperplasia, mild periportal fibrosis, and foci of iron-rich fatty macrophages (lipogranulomata). The latter finding is rare in dogs under the age of 6 years and should raise the suspicion of an underlying congenital portasystemic shunt.

Management

Occlusion of the anomalous vessel either surgically or non-invasively using fluoroscopic guidance is the treatment of choice in the dog and cat. If surgery is not an option, medical management with a low quantity/high quality protein diet plus lactulose and neomycin or metronidazole to ameliorate signs of encephalopathy may provide satisfactory results for months to several years depending on the severity of the vascular insufficiency.

Glucocorticoid hepatopathy

This is a unique and idiosyncratic response of the dog to either exogenous administration or endogenous overproduction of corticosteroids. The disorder is characterized by hepatocellular vacuolization, with associated hepatomegaly and a variety of clinical and laboratory signs. Afflicted dogs are never icteric.

Etiology

Exogenous administration of glucocorticoids and naturally occurring hyperadrenocorticism can induce morphological changes in hepatocytes and abnormal clinicopathologic test results in the dog.

Pathophysiology

While most dogs receiving glucocorticoids develop hepatocellular vacuolization, not all develop the same degree of change. Some dogs develop focal necrosis and severe hepatomegaly whereas others exhibit almost no response. The reason for this individual variation is unknown.

Clinical presentation

Animals usually present with the complaint of polydipsia and polyuria, restlessness (nocturnal wandering), panting, and an increased appetite. Physical examination may reveal hepatomegaly.

Differential diagnosis

These include all causes of hepatomegaly including hepatic tumors and hepatic lipidosis.

Diagnosis

The serum ALP is the most consistently increased test. The increased serum ALP activity is predominantly due to

an isoenzyme of ALP produced by the liver in response to glucocorticoids. Serum ALP activity may be dramatically increased, similar to the increases associated with extrahepatic biliary obstruction and cholangitis. In contrast, serum bilirubin concentration remains normal in dogs with glucocorticoid hepatopathy. Also, while ALT may show a mild to moderate increase, the AST usually remains close to normal. The serum bile acid concentration may be mildly increased (usually less than 25 mmol/L in the fasted state).

Hepatic aspiration cytology or liver biopsy differentiates the glucorticoid induced hepatopathy from other hepatic disorders.

Management

Remove the inciting cause (stop steroid treatment or treat Cushing's).

Special issues in feline hepatology

Anatomy

The blood supply to the liver, including the hepatic portal vein, is similar to the dog. In contrast to the dog, however, the cat has a common bile and pancreatic duct. Some cats may also have a second pancreatic duct. Pancreatitis or pancreatic disease that impairs bile flow therefore causes cholestasis much more readily in the cat than in the dog.

Excretion

The cat has a relative deficiency of glucoronyltransferase when compared to the dog. This explains why cats have a decreased ability to excrete compounds such as salicylates, acetaminophen, NSAID's, some of the narcotics as well as a variety of other drugs. As a result of this deficiency, the cat is also slower to excrete bilirubin than the dog.

Energy Requirements

In contrast to the dog, cats utilize protein to satisfy part of their basic energy needs. The gluconeogenesis pathway therefore never shuts down in the cat as it does in the fasting dog. If the cat fasts, there is an ongoing protein catabolism which is why cats with hepatic lipidosis, for example, lose muscle mass so quickly while fat is conserved. Cats are also unable to synthesize arginine and develop signs of hepatic encephalopathy when ingesting arginine deficient diets. Taurine is used to conjugate bile acids in the cat and this is why the cat has a higher basal taurine requirement. Glutathione is also important in the cat as a mechanism for the prevention of intoxication.

Liver Enzymes

As in the dog, ALT elevation suggests hepatocyte membrane damage. AST is more difficult to interpret in the cat than in the dog and elevations are less dramatic. The fasting cat, when it breaks down muscle for energy, releases AST. This is why we focus more on the ALP and ALT in terms of total evaluation in this species.

ALP is not a leakage enzyme, but is produced by the hepatocyte and the bile duct epithelium in response to accumulating bile acids. In the cat, elevation of ALP strongly suggests hepatic disease. Mild elevations of ALP (150-200), which are non-specific and often the result of reactive hepatopathy in the dog, may well be associated with underlying liver problems in the cat because the cat has 1/3 of the total ALP concentration in its body and feline ALP has a much shorter half-life (6 hours versus 72 hours).

Bilirubin

The processes of bilirubin production, conjugation and excretion are the same as in the dog. An increase in serum bilirubin concentration indicates an obstruction to bile flow (extrahepatic obstruction or liver disease and intrahepatic cholestatic disease). Cats tend to develop jaundice earlier than dogs because of their reduced glutathione concentrations and resulting decreased secretory ability. Cats also develop mild elevations in bilirubin from non-hepatic causes. Examples of diseases that may cause bilirubin elevation include: renal disease, panleukopenia, inflammatory bowel disease, sepsis, FIP, lymphoma, diabetes, and occasionally hyperthyroidism. Anorexia may be associated with increased serum bilirubin concentrations. This is because when protein intake decreases, the concentration of both glutathione transferase and intrahepatic bilirubin transfer proteins are decreased and bilirubin metabolism is impaired. There is an associated increase in serum bilirubin.

The renal threshold for bilirubin is nine times higher in the cat than in the dog and bilirubinuria is therefore a reliable sign of hepatic disease in this species.

Clotting Function

Bile is essential for fat-soluble vitamin K absorption and since Vitamin K is critical for synthesis of prothrombin, clotting function may be impaired in patients with cholestasis. If the PT is elevated in cats with obstructive jaundice it may be returned to normal after parenteral administration of Vitamin K.

Liver Function

Bile acids are a sensitive test of liver function in the cat. The blood ammonia test is also used in the same way as in the dog. Serum protein concentrations are much slower to change in the cat with liver disease than in the dog. Hypoalbuminemia is extremely rare in cats with liver disease. Hyperglobulinemia is seen in a variety of feline diseases and it is not uncommon to see a cat with liver disease and increased serum globulin concentration. ■

Concepts of liver disease

Colin F. Burrows
BVetMed, PhD, HonFRCVS, DACVIM, College of Veterinary Medicine, University of Florida, Gainesville, FL

Abstract

Liver disease is a relatively uncommon presentation in small animal practice. In contrast, elevated liver enzyme due to extra-hepatic disease and an associated reactive hepatopathy is a more common finding. Liver disease can be a challenge to diagnose and differentiate from reactive hepatopathies. Clinical signs are often subtle and diagnosis can be difficult because the liver has an extensive functional reserve, 70-80% of its mass may be surgically excised before clinical and laboratory evidence of dysfunction is detected. The liver also has impressive regenerative capabilities provided that blood supply, bile drainage and stromal framework are adequate. Under controlled conditions the organ can, for example, regenerate up to 75% of its original mass within a few weeks. Signs of hepatic disease or failure are thus seldom clinically evident until the disease process is severe or well advanced.
Keywords: *liver disease, enzyme, reactive hepatopathy*

The purpose of this chapter is to relate liver anatomy and physiology to disease and to provide a format for a diagnostic approach to liver disease.

Anatomy

An understanding of hepatic disease begins with an understanding of hepatic anatomy.

The liver is composed of four anatomical units: 1) hepatocytes, 2) the biliary network, 3) the vascular system and 4) the Kupffer cells. These are all integrated into a lobule, the functional unit of the liver.

The lobule

The lobule is composed of a central vein surrounded by hepatocytes and with portal tracts at the periphery (Figure 1). The extensive vascular and sinusoidal network reflects the dependency of the hepatocytes on an adequate perfusion of blood to maintain functional integrity.

In addition to its vascular network, the liver is richly laced with an intrahepatic biliary network which progressively increases in size and ultimately unifies to form the extrahepatic bile duct. The flow of bile is opposite to that of the blood, i.e. bile flows away from the central vein toward the portal tract. This pattern explains why a severe lesion around the central vein (centrilobular) may not be associated with jaundice while a relatively mild lesion around the portal tract (periportal) may cause sufficient impairment to the flow of bile such that jaundice develops. Focal mechanical obstruction of small bile ducts, e.g., tumor metastasis, seldom results in detectable obstruction of bile flow since the intrahepatic canaliculi do not simply run between hepatocytes in a straight line but form an extensive three-dimensional network.

Kupffer cells, a component of the monocyte phagocytic system (reticuloendothelial system), line the sinusoids adjacent to the endothelial cells (Figures 2, 3 and 4). These cells are involved in the clearing of living microorganisms and 'toxins' of gut origin from the blood. They may also be involved in the mediation of chronic inflammatory liver disease.

Portal vascular system

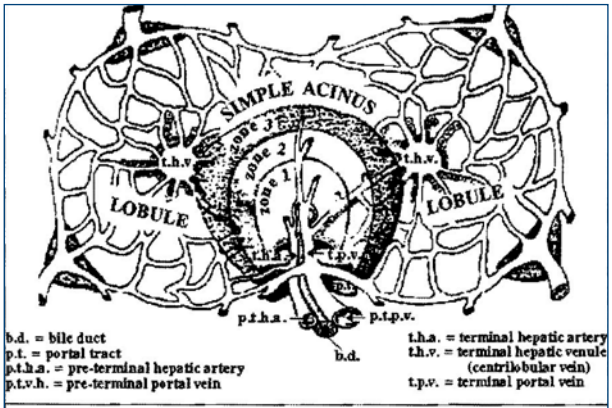
Blood flows from all the organs of the digestive system and the spleen, through the portal vascular system to the liver (Figure 5). This enables the liver to act as a metabolic filter and detoxify ingested substances. The process of detoxification releases cytokines which can damage the hepatocytes and cause an increase in liver enzyme activity.

Congenital anomalies of the portal vascular system and acquired portal systemic shunts (PSS), which develop secondarily to hepatic cirrhosis, occur frequently in the dog and to a lesser extent in the cat. In both disorders, portal blood does not flow through the liver so that products of intestinal digestion 'bypass' hepatic metabolism and exert 'toxic' effects on the central nervous system. This can result in a variety of abnormal neurologic signs that are collectively called **hepatic encephalopathy**.

The Hepatocyte

The hepatocyte contains many different kinds of specialized ultrastructural features (organelles) that reflect the numerous metabolic functions of the organ. No one hepatocyte is typical of the rest, but several general statements can be made about their structure and enzyme content.

The hepatocyte has two surfaces bordered by sinusoids. A portion of each of the inter-hepatocyte surfaces called the **canaliculus** is further modified to secrete bile (Figure 6). The cytoplasm also contains organelles which synthesize albumin and prothrombin. These two biochemical parameters can be used as indicators of liver function.



Diagrammatic representation of the relationship between the hepatic acinus and the hepatic lobule

Figure 1

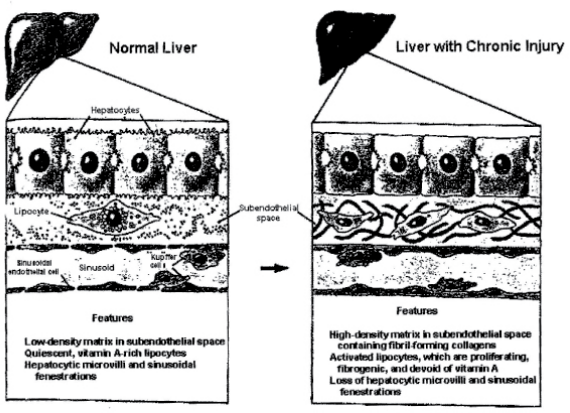


Figure 2. Diagrams of normal liver showing microanatomy of the vascular and sub-vascular space and the changes that occur in these areas as a result of injury.

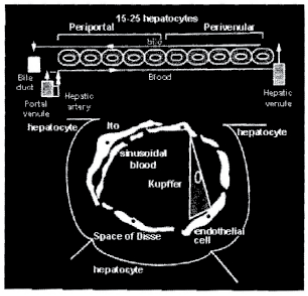


Figure 3. Cross sectional diagram of sinusoid showing anatomical arrangement along with a diagram that shows bidirectional flow of bile and blood.

Figure 2 and Figure 3

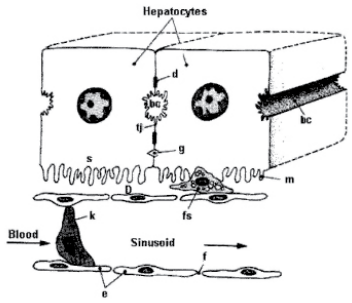


Figure 4. Hepatocellular plasma membrane domains and spaces. Two hepatocytes (h) are represented to illustrate that the hepatic plasma membrane has a sinusoidal or basolateral domain (s), an intercellular domain in which tight junctions (tj) desmosomes (d), and gap junctions (g) can be recognized, and finally a bile canalicular domain (bc). The bile canaliculus is formed by the junction of two hemicanaliculi (c) each provided by one hepatocyte. Microvilli (m) project into the extra-cellular-extra-vascular space of Disse, also called perisinusoidal space (D). Perisinusoidal liver cells such as the fat-storing cell (fs), also called stellate cell, lipocyte, or Ito cell, are represented. The hepatic sinusoid wall is formed by endothelial cells (e) which have discontinuities called fenestrations (f) allowing the free exchange of solutes between the vascular compartment and hepatocytes. Kupffer cells (k) are intra-sinusoidal, participating in the removal of large particles.

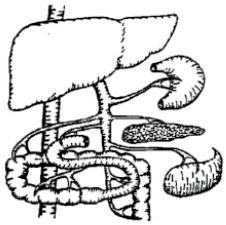


Figure 5. The portal vascular system receives blood from all parts of the gastrointestinal tract, pancreas and spleen.

Figure 4 and Figure 5

A large number of enzymes are located in both the organelles and the cytoplasm of the hepatocyte. Knowledge of the enzyme location in the cell and within the hepatic lobule allows for the recognition of a particular disease process. Factors such as the number of hepatocytes, hepatic concentration, intercellular distribution, permeability changes of the cell membrane, and plasma half-life times contribute to the serum enzyme activity measured for each species.

Hepatic Function

The liver has myriad of functions, all of which may be disrupted in hepatic disease. The liver, for example, plays a central role in the metabolism of proteins, fats, carbohydrates, vitamins and minerals.

Hepatic glycogen stores provide the body with a readily available source of glucose. Glycogen stores are depleted in severe liver insufficiency (associated with cirrhosis or congenital portosystemic vascular anomalies) and can predispose to hypoglycemia. The deficiency may be accentuated by decreased food intake which further depletes already inadequate glycogen stores.

All amino acids except branched-chain amino acids (BCM) are degraded by the liver (BCM are degraded in muscle, gut wall and kidney). The liver is also the site of conversion of ammonia and amines into urea. Hepatic insufficiency may result in encephalopathy as a result of disturbed protein metabolism (hepatic encephalopathy).

The liver is involved with many aspects of lipid metabolism. Triglycerides accumulate to cause a fatty liver when the rate of hepatic synthesis exceeds the rate of lipid dispersal. Hepatic triglycerides are produced from the intrahepatic esterification of fatty acids derived from the systemic circulation (from dietary and storage sources). Fatty acids within the liver are esterified to triglycerides and packaged with apoprotein B, phospholipids and cholesterol to form very low density lipoproteins (VLDL). They may also be esterified to phospholipids and cholesterol esters or undergo oxidation within the liver.

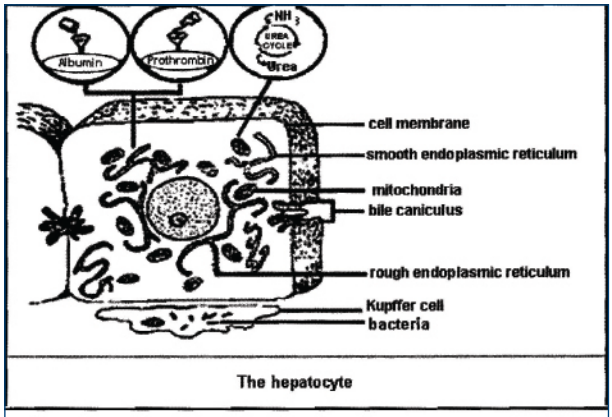


Figure 6. Diagram of a hepatocyte showing representative functions.

Figure 6

The release of nascent VLDL particles depends on several subcellular events. Triglyceride accumulation may occur secondary to a deficiency of apoprotein, an abnormality in the subcellular assembly process or defective release from the hepatocyte. A syndrome is recognized in the cat in which hepatic fat mobilization is so severely impaired that liver insufficiency develops and may be reflected by the development of icterus (feline hepatic lipidosis).

The liver also plays an important role in immune function in which the Kupffer cells play a key role. The liver also plays an important role in the storage of a variety of compounds including both water and fat soluble vitamins, glycogen, iron, copper and fat. The liver is also important in the detoxification and excretion of bilirubin, steroids, ammonia and many drugs.

Important hematologic functions include the synthesis of plasma procoagulants: factor I (fibrinogen), II (prothrombin), V, VII, VIII, IX, and X. Factors II, VII, IX, and X depend on vitamin K for normal hepatic synthesis. A prolonged PT and/or PTT occur in severe or extensive liver disorders. The major differential diagnoses for prolonged coagulation tests are diseases that cause severe hepatocellular damage and impair coagulation factor synthesis. Parental administration of vitamin K_1 does **not** return the PT to normal in these situations (cells are too damaged). Since vitamin K_1 is fat-soluble, its absorption is reduced in bile duct obstruction. Parenteral vitamin K_1 therefore, **does** return the PT to normal within 48 hours in these disorders. The PT may also be prolonged (and corrected by parenteral vitamin K administration) in patients with nutrient malabsorption.

Disseminated intravascular coagulopathy (DIC) may develop in severe, fulminating hepatic disease. Release of thromboplastin-like substances from damaged hepatocytes and decreased hepatic clearance of clot-promoting factors are partly responsible for initiation of the hemorrhagic diathesis. The most important digestive function is the synthesis and excretion of bile acids.

Diagnosis of liver disorders

Early diagnosis of liver disease is often impaired by the lack of any specific signs and by paucity of findings at physical examination. Unless jaundice is present or the patient exhibits overt signs of encephalopathy, clinicopathologic abnormalities are frequently the first indication of a liver disorder. Biochemical recognition of primary liver disease is complicated, however, by the fact that disorders which involve the liver secondarily as well as certain drugs can cause abnormal liver tests. In addition to liver tests, radiographs, ultrasonography and the histologic examination of liver tissue are employed in the evaluation of the hepatobiliary system.

History

Signs associated with liver disorders are usually nonspecific and may suggest primary disease of another organ system except when jaundice and/or dark urine is observed in relation to a normal packed cell volume.

A breed predisposition for certain liver disorders is recognized in dogs and may place a liver disorder higher up the list of differential diagnoses (e.g. Bedlington Terrier, Doberman, American Cocker Spaniel).

Encephalopathy secondary to liver insufficiency does not occur as often in the cat as in the dog. Dysfunction of the central nervous system, including behavioral changes and seizure activity, may be a postprandial observation in this species but may occur independent of food ingestion. Ptyalism is a relatively common manifestation of hepatic encephalopathy in the cat. Lethargy and gastrointestinal signs such as vomiting and diarrhea may be early, but less consistent, manifestations of liver insufficiency in both cats and dogs.

Physical examination tips in suspected liver disease

The soft palate, sclera and pinna should be carefully inspected for yellow discoloration since jaundice is often first apparent in these tissues. The liver should be carefully palpated. In the cat, symmetrical hepatomegaly is associated with many of the diseases which result in jaundice. In the dog, a symmetrically enlarged liver may be noted in non-icteric animals and is usually secondary to the administration of glucocorticoids (glucocorticoid hepatopathy).

In the dog, portal hypertension secondary to hepatic fibrosis and hypoproteinemia may be of sufficient magnitude to result in the formation of a low-protein transudate and the formation of ascites. The finding of a high-protein modified transudate (> 2.5 g/dL, predominantly mesothelial cells) is more commonly associated with right-sided cardiac insufficiency and makes the diagnosis of primary liver disease less likely. Ascites is uncommon in feline liver disease, but a small volume of protein rich fluid (exudate) may be seen in cats with cholangiohepatitis.

In the cat suspected of liver disease, an ophthalmologic examination may reveal lesions suggestive of systemic disease such as feline infectious peritonitis. Palpation of the thyroid gland, as well as measuring the T4 concentration, is important in older cats because hyperthyroidism can be associated with clinical and biochemical findings suggestive of liver disease.

Laboratory evaluation

The early recognition of liver disease may be serendipitous; abnormal laboratory test results may be revealed, for example, while investigating vague clinical signs. Clinicopathologic assessment routinely includes a hemogram, biochemical profile, urinalysis, fecal examination and in cats, FeLV and FIV tests. Depending on the results, a liver function test may be needed in the non-jaundiced patient.

Table 1 The signs of liver disease

Often waxing and waning and vague
Decreased appetite
Lethargy
Swollen abdomen (ascites or hepatomegaly)
Jaundice
Ptyalism
Vomiting
Neurologic signs (encephalopathy)

Liver Enzyme Tests

Serum liver enzyme tests are traditionally divided into those which reflect hepatocellular leakage: alanine aminotransferase (ALT), aspartate aminotransferase (AST) and those which increase secondary to accelerated production: alkaline phosphatase (ALP/SAP) and gamma glutamyltransferase (GGT). Impaired bile flow and certain drugs are the most common causes of moderate to markedly increased hepatic synthesis of ALP and GGT.

Most chronic liver diseases have sufficient cellular damage or accelerated enzyme synthesis to result in an increase in serum enzyme activity. Enzymes can therefore be considered biochemical "markers" of liver diseases but are not usually indicative of the type or severity of the pathologic process.

ALT is abundantly located in the cytoplasm while AST is located in both cytoplasm and mitochondria. Hepatocellular membrane disruption would allow ALT and some AST leakage while mitochondrial disruption (in more severe liver disease) is necessary for the additional release of AST. Since mitochondrial disruption is a more severe pathologic process, increased serum AST activity is suggestive of more severe hepatic damage. In the dog, both ALT and AST activities increase after experimentally induced hepatic necrosis. The magnitude of serum ALT activity increase, however, is greater than the increase in AST and the AST returns to normal faster than the ALT. Fasting and associated muscle breakdown results in the release of AST in the cat.

In the dog (but not the cat), glucocorticoids most consistently increase the activity of ALP. These drugs cause a histologically characteristic vacuolar change in the hepatocyte secondary to the accumulation of glycogen and appear to stimulate either the production of a novel ALP isoenzyme or a delayed clearance of endogenous ALP.

Another major cause of increased ALP activity are bile acids, which reach high concentrations in the hepatocyte during cholestasis. This induces ALP synthesis with a subsequent increase in serum ALP activity. Bile salts also damage the bile duct lining in cholestatic disease and perpetuate hepatocellular damage.

Anticonvulsant medications frequently produce abnormal liver enzyme activities in the dog without causing liver disease per se. Chronic active liver disease occasionally develops, however, in dogs receiving primidone or diphenylhydantoin, and on rare occasions, phenobarbital. This poses a diagnostic dilemma; do the increased serum enzyme activities simply reflect drug-stimulated production or the presence of inflammatory liver disease? The only way to be sure is to obtain a hepatic biopsy.

There is a species variation in the magnitude of increased serum ALP activity. The cat does not develop the marked increases of serum ALP activity seen in the dog because of a dramatically shorter plasma half-life (6 hours vs 66 hours) and has a lesser capability for the hepatic production of ALP. Thus, even a mild increase in serum ALP activity is more reliably suggestive of liver disease in the cat than it is in the dog.

Remember! The most common cause of liver enzyme elevation in the dog and cat is extrahepatic disease.

Liver Function Tests

Liver function tests are more specific than enzyme tests and indicate the degree of hepatocellular function. Some are more sensitive than others.

1) Bilirubin (serum). An increase in serum bilirubin concentration results in yellow discoloration of tissues. In a patient without increased erythrocyte destruction, hyperbilirubinemia is caused by either intrahepatic disease or extrahepatic bile duct obstruction. Total serum bilirubin is a relatively insensitive test of liver function and another more sensitive test of liver function is therefore unnecessary in an icteric patient.

Determination of the percentage of conjugated and unconjugated bilirubin is of no help in differentiating between intrahepatic and extrahepatic cholestatic diseases. In the non-anemic animal, increased total serum bilirubin indicates severe liver disease or bile duct obstruction.

Hyperbilirubinemia may also occasionally develop in patients without hemolysis or primary liver disease. Hyperbilirubinemia may occur in sepsis and in a variety of other disorders, particularly in the cat. Mild increases (<3.0 mgL/dL) may be also seen in cats with diabetes, hyperthyroidism, panleukopenia, FeLV infection, renal disease, FIP, inflammatory bowel disease, pancreatitis and lymphoma.

2) Ammonia. Increased concentration of plasma ammonia is associated with severe liver insufficiency. An ammonia tolerance test (oral or rectal ammonium chloride) may be indicated if hepatic disease is suspected and the fasting ammonia concentration is normal. Measurement of the plasma ammonia concentration is most useful for diagnosis of portosystemic shunts in dogs and cats and for documenting hepatic encephalopathy in patients manifesting central nervous dysfunction. Ammonium chloride should not be administered to a patient manifesting signs suggestive of hepatic encephalopathy before the fasting plasma ammonia concentration is ascertained.

3) Bile Acids. Measurement of total serum bile acids is a very useful liver function test and is available through most commercial veterinary clinical pathology laboratories. Bile acids are a convenient test of liver function because 1) no exogenous dyes need to be given, 2) they have not been shown to contribute to hepatic encephalopathy, therefore a tolerance test can be performed without fear of exacerbating central nervous system dysfunction and 3) they are stable in the serum, thereby alleviating the need for special sample management. Total serum bile acids are also a sensitive indicator of liver insufficiency especially if a 2 hours post-prandial bile acid concentration is measured (referred to as a bile acid tolerance test). The fasting and 2 hours post-prandial bile acid concentrations are normally less than 5 µm/L and 10 µm/L in the cat and less than 5 µm/L and 15 µm/L in the dog.

Total serum bile acids help in the differential diagnosis of portosystemic shunts and elevated levels support the need for liver biopsy in the non-jaundiced patient. Liver biopsy is most rewarding in cats and in dogs when the fasting total serum bile acid concentrations are greater than 20 and 30 µmoles/L, respectively. It should be emphasized, however, that while measurement of total serum bile acids is a sensitive test of liver insufficiency, the test does not indicate the type

or severity of liver disease nor does it differentiate between primary and secondary liver disorders.

4) Bilirubin (urine). Moderate to marked bilirubinuria occurs when there is an increase in the plasma conjugated bilirubin concentration. Unconjugated bilirubin and most of the conjugated bilirubin are bound to albumin which prevents their glomerular filtration. The excretion of conjugated bilirubin by the kidney depends on a small fraction that is not bound to albumin. In the dog, the detection of increased bilirubin in the urine precedes the recognition of jaundice and may be an early indication of liver insufficiency. A liver function test can be used to assess the functional integrity of the liver if the total serum bilirubin is normal in such patients.

The canine kidney has the enzymatic capacity to metabolically alter hemoglobin to bilirubin and a small amount of bilirubin (1+) can be a normal finding in canine urine. Bilirubinuria in the cat, however, even in concentrated urine, is abnormal and should be investigated. Bilirubinuria is a reliable sign of liver disease in the cat.

5) Albumin. Albumin is produced by the liver and decreased concentrations may occur in severe liver insufficiency, reflecting a decrease in hepatocyte numbers. When other potential causes of hypoalbuminemia are eliminated in the non-jaundiced patient, a more sensitive liver function test (e.g. bile acids) should be used to confirm liver insufficiency.

6) BUN. The formation of blood urea nitrogen (BUN) or serum urea nitrogen (SUN) is related to ammonia metabolism. Ammonia is absorbed from the intestinal tract after intraluminal protein metabolism and is carried to the liver via the portal blood. Urea is formed by the incorporation of ammonia into the urea cycle. A low BUN results when portal blood is "shunted" around the liver or when there is a deficiency of one or more of the enzymes of the urea cycle. This is associated with cirrhosis (insufficient functional hepatic mass) and subsequent development of hyperammonemia.

Hemogram

The hemogram usually provides limited information in the differential diagnosis of liver disorders. Target cells have been associated with portosystemic vascular anomalies in dogs and poikilocytes and acanthocytes (abnormally shaped erythrocytes) occur in chronic inflammatory liver disease. The mean cell volume (MCV) is decreased in approximately 70% of dogs with portosystemic shunts and is attributed to abnormal iron metabolism. Many dogs with severe liver disease have gastric ulcers which can bleed and cause chronic low grade microcytic anemia.

Test of clotting function should also be routine in patients with liver disease, especially if a biopsy is contemplated. This may include an activated clotting time (ACT), bleeding time, platelet count and prothrombin time.

Fecal Examination

A test of the feces for occult blood can be performed in patients with anemia and liver disease since chronic GI blood loss is common.

Supplementary tests and procedures

Abdominocentesis

An abdominal effusion may develop subsequent to chronic liver disease. In the dog, a transudative ascites (protein < 2.5 g/dl, few cells) is the most common finding. Ascites secondary to liver disease is uncommon in the cat. However, a high protein effusion occurs in approximately 50% of cats with cholangiohepatitis. Ascites may also occur in cats with hepatic lymphoma. An abdominal effusion should always be assessed for total protein and cellular content in an effort to recognize the findings associated with neoplasia, feline infectious peritonitis, infection (bacterial, fungal), hemorrhage and ruptured gallbladder.

Radiographs

Survey abdominal radiographs provide limited information in the evaluation of the hepatobiliary system. Liver size and symmetry may be assessed, opacities within the biliary system may be noted, and the presence of abdominal fluid may be detected before it is clinically recognized.

Ultrasound

Ultrasonographic examination of the hepatobiliary system is a noninvasive procedure that can provide a great deal of useful diagnostic information. The technique can detect dilated bile ducts suggestive of extrahepatic obstruction, detect focal and diffuse differences in hepatic densities suggestive of neoplasia or cirrhosis, help guide the needle during percutaneous liver biopsy, identify loculated abdominal fluid, and localize portosystemic vascular anomalies.

Cytology

Cytologic specimens can be obtained from the liver by needle aspiration, especially when there is hepatomegaly, and from touch imprints made from the liver biopsy specimen. Vacuolated hepatocytes suggest hepatic lipidosis in the icteric cat. Lymphoma (lymphosarcoma) may also be identified cytologically in both species.

Biopsy

Histologic examination of liver tissue is frequently necessary in patients with unexplained increases in liver tests and/or hepatomegaly. Assessment for bleeding disorders is recommended before biopsy because of the role of the liver in the synthesis of most coagulation factors. An activated coagulation test (ACT) is a simple and convenient test. A value greater than 125 seconds for the dog and 65 seconds for the cat should be further investigated by means of a prothrombin time and activated partial thromboplastin time. Platelet numbers should also be evaluated since a defect in platelet function has been documented in patients with chronic liver disease.

The pursuit of a liver biopsy in a patient with abnormal coagulation tests is addressed on an individual basis. In predominantly cholestatic disorders subcutaneous administration of vitamin K_1, (2 mg/kg for the dog and 5 mg/cat every 12 hours) may return the coagulation tests to normal within 24 to 48 hours. Administration of vitamin K_1 is unlikely to correct the deficiency, however, if the coagulopathy is secondary to insufficient liver function. Transfusion with fresh blood or plasma is required before surgery in a patient which fails to respond to vitamin K_1. ■

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Inflammatory Bowel Disease and other causes of chronic diarrhea

Colin F. Burrows
BVetMed, PhD, HonFRCVS, DACVIM, College of Veterinary Medicine, University of Florida, Gainesville, FL

Abstract

Signs of chronic small intestinal disease (SID) are usually nonspecific (vomiting, weight loss and/or diarrhea) and vary in severity from occasional passage of semisolid feces that is merely an annoyance or concern to the owner, to severe progressive weight loss accompanied by explosive watery diarrhea.
Keywords: *small intestinal disease, vomiting, diarrhea*

A number of specific disease entities cause such chronic problems in dogs and cats, and histologic examination of a small intestinal biopsy specimen is required for definitive diagnosis. Clearly this expensive and invasive approach to diagnosis is not possible (or indicated) in all cases. There are no definitive tests for the diagnosis of SID. Even histologic examination of intestinal biopsies may be of limited practical value since in many cases there are no well-defined specific therapeutic measures for histologically defined diseases.

It is often necessary to make a presumptive diagnosis based on history, physical examination, and results of limited laboratory tests. The safest and least expensive treatments that are most likely to be effective are then started.

It is also important to realize that adequate and time-consuming communication with the client is essential when dealing with cases or suspected SID. Clients otherwise get frustrated very quickly when there is no instant diagnosis and no specific treatment. It is essential to explain the limitations of the various approaches to diagnosis and patient management. There are no magic answers, but patience and a logical approach are usually rewarded with a successful outcome and a happy client.

Inflammatory Bowel Disease

Inflammatory Bowel Disease (IBD) is the most common cause of chronic vomiting and diarrhea in dogs and cats. It actually involves a group of idiopathic chronic gastrointestinal tract disorders characterized by the infiltration of the lamina propria by lymphocytes , plasma cells, eosinophils, macrophages, neutrophils or a combination of these cells. IBD is diagnosed most frequently in middle aged or older dogs and cats, but the disease can occur in animals of any age. It is not unusual, for example, for the disorder to be diagnosed in kittens as young as 4 months of age, particularly in those that have suffered from diarrhea since weaning. There is no apparent sex predisposition. Some dog breeds, however, appear to be more susceptible to certain types of IBD. The Basenji, for example, suffers from an immunoproliferative form of IBD and the Boxer has a predilection for histiocytic ulcerative colitis.

Pathogenesis

IBD may be associated with many conditions including giardiasis, campylobacteriosis, histoplasmosis, bacterial overgrowth, dietary sensitivity, lymphangiectasia, regional enteritis and lymphosarcoma, but in the majority of cases no obvious cause can be identified. It is therefore important to be aware that IBD is merely a histologic description, and may represent a single manifestation of disease with many causes. The clinician should consider the probability that the infiltrate is an immune-mediated inflammatory reaction directed against antigens from the intestinal lumen. Immunologic reactions in IBD may involve a variety of hypersensitivity reactions accompanied by the release of inflammatory mediators.

History and clinical signs

Inflammatory Bowel Disease (IBD) can involve either the small intestine, the large intestine or both organs simultaneously. It is important therefore to appreciate that a wide spectrum of clinical signs can be associated with this disease. Also, clinical signs in IBD are relatively nonspecific and usually relate to the degree and extent of intestinal pathology.

Dogs with IBD most often present with small or large bowel diarrhea, whereas vomiting is the most common sign in cats. The spectrum of signs associated with IBD in dogs and cats are listed in Table 1.

Diarrhea in IBD can be either small bowel or large bowel in character (Table 2) and even when both the small and large intestine are involved in the disease process, signs associated with one organ will usually predominate over the other. Diarrhea may be the sole clinical sign or may occur in conjunction with vomiting and other signs. Diarrhea may also be acute or chronic,

Table 1 Signs associated with inflammatory Bowel disease in dogs & cats

Dogs	Cats
Diarrhea Vomiting Weight loss Anorexia Lethargy Edema Ascites Borborygmus Tenesmus Hematochezia Halitosis Polydipsia and polyuria Abdominal pain	Vomiting Diarrhea Weight loss Lethargy Depression Hematochezia

Table 2 Differentiation of small intestinal from large intestinal diarrhea

Parameter	Small intestine	Large intestine
FECES		
Volume	Increased	Normal or increased
Mucus	Rarely present	Frequent
Melena	Occasional	Absent
Hematochezia	Absent	Fairly common
Steatorrhea	Occasional	Absent
Undigested food	Occasional	Absent
DEFECATION		
Urgency	Uncommon	Common
Tenesmus	Absent	Greater than 3 times normal
Frequency	2-3 times normal	Occasional
Dyschezia	Absent	Occasional
ANCILLARY SIGNS		
Weight loss	May occur in malabsorption	Rare
Vomiting	Can occur	Can Occur
Flatulence	Can occur	Absent
Halitosis	Can occur	Absent

but most patients are evaluated because of chronic diarrhea that is unresponsive to routine antidiarrheal treatment. In some cats with IBD, diarrhea does not occur until the animal experiences a stressful episode such as change in environment or queening.

The first stage in approaching the patient with diarrhea is to decide whether the process involves the small intestine, the large intestine or both. This helps determine the direction of future diagnostic steps and is achieved by combining information from the history, results of the physical examination and characteristics of the feces (Table 2).

Small bowel diarrhea is most often characterized by large volumes of soft, semi-formed, bulky or watery feces whereas large bowel diarrhea is characterized by small volume mucoid feces that are often blood stained.

Chronic intermittent and listless behavior are the most consistent signs of IBD in cats and may occur with or without diarrhea. When taking the history careful attention should be paid to patterns of vomiting observed by the owner. Vomiting in feline IBD is often recognized as an intermittent occurrence for weeks, months or years. The frequency of vomiting may increase as the disease progresses and other signs, such as listlessness and anorexia, may cause the owner to seek veterinary attention. Occasionally a cat with a history of an acute onset of vomiting and lethargy and with no history of chronic disease may be presented with moderate or even severe inflammatory changes eventually being found in intestinal biopsy specimens.

The history of vomiting in dogs with IBD may resemble that in the cat, but vomiting usually develops after the animal has developed diarrhea.

Vomiting episodes in both species are usually associated with retching, are nonprojectile, and usually produce only clear fluid, bile or foam. Vomiting of food, either fresh or partially digested, may also occur, but vomiting in IBD is usually unrelated to eating. Hematemesis is rare and may indicate concurrent gastric involvement (eg. erosions, foreign body, gastritis, neoplasia) or more likely, superficial erosive changes in the proximal small intestine. Vomiting hairballs or their accumulation in the stomach is an important sign of the disease in cats.

Animals with moderate to severe disease are lethargic and inappetent on days in which more than one vomiting episode occurs, but many animals with mild IBD go about their daily routine showing no untoward effects from any of the vomiting episodes.

Vomiting and associated clinical events may be cyclical in nature, particularly in cats. Signs may be evident on one of several days and then spontaneously disappear, indicating that untreated IBD runs a course characterized by exacerbations and remission. Therapeutic success should not therefore be automatically attributed to the symptomatic treatment that is often given to these patients.

Other signs of IBD include varying degrees of listlessness, inappetence or anorexia, loss of body weight and condition, polydipsia and polyuria, abdominal pain, borborygmus, halitosis and flatus. Some patients with IBD exhibit an increased, even ravenous appetite, probably in response to nutrient malabsorption. Dogs but not cats with severe small intestinal disease may develop protein losing enteropathy and may present with ascites or peripheral edema. Dogs with IBD (particularly eosinophilic gastroenteritis) occasionally present with cyclical bouts of vomiting and abdominal pain, with or without diarrhea, that may mimic acute pancreatitis in their severity.

Physical findings

Findings at physical examination in dogs with IBD vary from the usually unremarkable to an animal with a palpably thickened intestine, cachexia and edema or ascites. Hypoproteinemia most frequently appears to cause peripheral edema in large breed dogs, whereas ascites develops more frequently in small breed dogs. Findings may be more subtle in cats with only weight loss and perhaps a thickened intestine. Hypoproteinemia with associated edema or ascites is uncommon in IBD in this species.

Abdominal palpation, however, is usually normal, especially in dogs and cats with large intestinal involvement. A palpably thickened intestine with mesenteric lymphadenopathy develops in cats with severe disease.

Differential diagnosis

The differential diagnoses of IBD in cats and dogs are listed in Table 3. Major differential diagnoses in dogs include exocrine pancreatic insufficiency, small intestinal bacterial overgrowth and intestinal tumors, particularly diffuse intestinal lymphoma. Hyperthyroidism must always be excluded as a cause of vomiting, weight loss, polyphagia and diarrhea in cats older than 5 years.

Diagnosis

A definitive diagnosis of IBD can only be made after identification of typical histologic changes in gastric, intestinal or colonic biopsy specimens. A stepwise approach to diagnosis should initially consider or eliminate intestinal pathogens and parasites, partial intestinal obstruction, systemic disorders and exocrine pancreatic insufficiency.

Recommended tests in all patients with suspected IBD include a complete blood count, biochemical profile, urinalysis, fecal examination for parasites. In cats, serum cobalamin, T4, and FeLV and FIV tests should be run. A TLI test can also be run if clinically indicated. Diagnostic tests in dogs should include measurement of serum TLI, folate and cobalamin concentrations.

Baseline test results are often normal or negative in dogs and cats with IBD, especially in patients with signs of large bowel involvement. Abnormalities that may be identified however, include mild nonregenerative anemia, leukocytosis (20-50,000 cells/L) without a marked left shift, and eosinophilia in some animals with eosinophilic enteritis or colitis. Lymphopenia may be seen in patients with secondary lymphangiectasia. A hyperproteinemia may occasionally be identified that is attributable to an increased globulin fraction in Basenjis with IBD and in some cats with the disease. Hypoproteinemia (total protein <55 g/L) with albumin and globulin fractions proportionately decreased, may occur in dogs with small intestinal IBD but seldom in cats with similar lesions. The reasons for this distinct species difference are unknown. Panhypoproteinemia in cats is rare and is more often associated with diffuse intestinal lymphoma than with IBD. Serum calcium will also be depressed if serum albumin is reduced. Intestinal lymphoma should be suspected if the albumin is low and calcium concentration is normal or increased.

In advanced cases the mucosal infiltrate can cause secondary lymphangiectasia with a subsequent loss of plasma protein into the gut lumen. Diarrhea is almost invariably present in these situations because plasma protein concentrations less than 45 g/L exert an insufficient oncotic pressure to allow water absorption and there is a net loss of fluid into the intestinal lumen.

Mild to moderate elevations of liver enzyme activity, particularly in ALT and AST, are seen in both dogs and cats with IBD. The AP may also be elevated in dogs, but rarely in cats with the disorder. Serum amylase and lipase activity may also be increased in acute exacerbations of severe disease to the extent that it may be difficult to clinically differentiate IBD from acute pancreatitis.

Survey abdominals are unremarkable in both dogs and cats with IBD.

The only means to definitively diagnose IBD is by gastric, intestinal or colonic biopsy. Intestinal biopsy specimens can be obtained either by endoscopy or by exploratory laparotomy. One of the many advantages of endoscopy is that it is a rapid and relatively noninvasive procedure. Multiple biopsy specimens can also be obtained from the stomach, proximal small intestine and colon. It is also

Table 3 Differential diagnosis of inflammatory Bowel disease in dogs & cats

Dogs	Cats
SMALL BOWEL Exocrine pancreatic insufficiency Small intestinal bacterial overgrowth Intestinal lymphoma Giardiasis Intestinal histoplasmosis Acute pancreatitis Adverse drug reaction (erythromycin)	**SMALL BOWEL** Hyperthyroidism Intestinal lymphoma Giardiasis Bacterial overgrowth Exocrine pancreatic insufficiency Feline infectious peritonitis Acute and chronic pancreatitis Adverse drug reactor (tetracycline, ampicillin)
LARGE BOWEL Diffuse colonic lymphoma Colonic fungal infection Cecal inversion Trichuriasis infection	**LARGE BOWEL** Colonic lymphoma

possible to obtain ileal biopsies at colonoscopy in some dogs and cats. It is important to obtain biopsy specimens from as many areas as possible since disease may not be uniformly distributed along the length of the gut.

The gross appearance of the intestine and colon at endoscopy in IBD can range from normal through varying degrees of mucosal irregularity to extreme changes with a cobblestone appearance and hemorrhage that resemble lymphoma. The mucosa may be extremely friable and both the jejunum and colon may bleed from direct contact as the endoscope tip is advanced.

If an endoscope is not available, full thickness biopsies must be taken from the small intestine at exploratory laparotomy. A minimum of 5 specimens should be taken, 1 from the stomach, 1 from the duodenum, 2 from the jejunum and 1 from the ileum. The colon is seldom biopsied at laparotomy since diagnosis by colonoscopy (even using a rigid colonoscope) is relatively straightforward. It is important to realize that the intestine appears grossly normal in most patients with IBD. A lymph node biopsy should also be taken if there is any degree of lymphadenopathy since it is important to distinguish between lymphoma and IBD. Both dogs and cats with small intestinal lymphoma may have a reactive mesenteric lymphadenopathy and vice versa.

Pathologic findings

It is important for the clinician to be able to interpret the pathologist's description of the lesions. Increased numbers of inflammatory cells are invariably present in the lamina propria of patients with IBD. Either single cell types or mixed cell infiltrations may be reported. The most common findings are lymphocyticplasmacytic enteritis or colitis, but eosinophilic enteritis or colitis may also be reported. Neutrophils are not commonly identified in the small intestinal mucosa of patients with IBD, but when present, probably indicate a response to a luminal microbial component.

Other abnormalities that may be reported include mucosal atrophy, villus atrophy or fusion, epithelial erosion or fibrosis. Secondary lymphangiectasia and crypt abscesses may also be reported.

It is important to ascertain whether the changes are described as mild, moderate or severe, since this determines the approach to treatment as well as the prognosis.

Mild changes may simply indicate a reaction to intestinal parasites or to small intestinal bacterial overgrowth. Moderate to severe changes on the other hand are much more significant and usually mandate aggressive treatment.

Severe cases of plasmacytic lymphocytic enteritis can be difficult to differentiate histologically from lymphosarcoma, especially in endoscopic biopsy samples. If the diagnosis is unclear it may be necessary to repeat the endoscopic biopsy procedure or to obtain full thickness biopsy specimens.

Dietary management of inflammatory bowel disease

Diet has an important role in the management of IBD, because the major presumed cause of the disorder involves hypersensitivity to luminal dietary or microbial antigens. Treatment is therefore aimed at removing any antigenic cause of inflammation and suppressing the cell-mediated inflammation in the gut.

The approach to the treatment of most gastrointestinal diseases involves a combination of pharmacologic and dietary therapy. Restriction or manipulation of individual dietary components, particularly fat content and protein source, may be the most important factor in the dietary management of dogs with IBD.

Drug treatment of inflammatory bowel disease

Small intestinal disease

While it may be possible to successfully treat patients with mild histologic change by removing any underlying disease and manipulating the diet, patients with

moderate to severe disease almost invariably require immunosuppressive therapy for successful control. Clients, moreover, are usually unwilling to wait for weeks or months for the results of dietary change to become apparent and demand treatment that will resolve signs within a reasonable time. Treatment of IBD almost always involves varying combinations of glucocorticoid and antimicrobial therapy.

Glucocorticoids

Judicious and appropriate use of prednisone (or prednisolone) is the most important component in the successful treatment of IBD of the small intestine.

The drug should be started at an initial dose of 0.5-1 mg/kg Q12H for 3-4 weeks. The dose can be reduced soon after signs subside in 50% decrements every 2-3 weeks until a maintenance dose of 0.5 mg/kg every other day is achieved.

The drug may be withdrawn after 8-12 weeks if signs do not recur. Higher doses of prednisone (1-2 mg/kg Q12H) may be indicated if the words "moderate" or "severe" appear on the biopsy report. The same decremental withdrawal regimen should be followed as described above.

Most patients respond well to this treatment regimen but clients should be cautioned about the possibility of an exacerbation of signs, particularly diarrhea for the first 3-4 days after treatment. The cause of this diarrhea is unclear, but may be attributable to the side effects of the rapid lysis of mucosal infiltrates of plasma cells and lymphocytes.

Methylprednisolone acetate (Depo-Medrol) can be used to treat both canine and feline inflammatory bowel disease, but consistent control of clinical signs is difficult to attain. The approach is warranted however, in cats which the owner has difficulty medicating. Initially 20 mg of the drug is given intramuscularly and is repeated at 1-2 week intervals for 2-3 doses. Injections are then given every 2-4 weeks as needed for control.

In refractory cases it is appropriate to try a course of cyclosporine.

Antimicrobial Therapy

Oral antimicrobial therapy is another important component of treatment of diarrhea in IBD. Metronidazole is the drug most widely used, but it is not known if the apparently beneficial effects of treatment are due to control of secondary bacterial overgrowth or to the purported effect of metronidazole to modulate cell mediated immunity.

An oral dose of metronidazole of 10-20 mg/kg Q12H appears to be effective in controlling diarrhea. Side effects appear uncommon at this dose and the drug may be continued indefinitely (but for a minimum of 2-4 weeks). In some patients, particularly cats with IBD, long term control of diarrhea may be achieved with as low a dose as 75 mg (¼ 250 mg tablet) once daily.

Other antibiotics that are effective in the control of secondary bacterial overgrowth in IBD are tetracycline 10 mg/kg Q12H and tylosin 10-20 mg/kg Q12H.

Azathioprine

If remission is not maintained on a combination of prednisone and metronidazole, azathioprine (Immuran) should be introduced into the treatment regimen. The oral dose in dogs is 1 mg/kg once daily and in cats 0.3 mg/kg once daily. Since the drug is only available as 50 mg tablets it must be reformulated to appropriately treat cats. For this one 50 mg tablet is usually crushed and dissolved in syrup or a multivitamin solution and dosed as an oral suspension. The drug should be continued for at least 4 weeks since it takes time for azathioprine to influence the immune system. The WBC should be monitored weekly and treatment stopped if the total white cell count falls below 5000. Treatment with azathioprine is usually continued for 3-9 months.

Adjunctive Therapy

The total serum protein concentration seems to have an important effect on maintaining the integrity of the gastric mucosal barrier in the dog. Gastric erosions are often noted at endoscopy when serum proteins are < 30 g/L, a situation that is exacerbated if prednisone is used to treat the disease. For these reasons it is important to protect the gastric mucosa with either sucralfate (0.25-0.5 g) Q8H, or H2 receptor antagonists such as cimetidine (5 mg/kg) Q8H, ranitidine (2 mg/kg) Q12H, or famotidine (2 mg/kg) Q24H. Treatment should be continued until serum proteins return to normal.

Adjunctive vitamin therapy is also beneficial in severe disease. Oral folic acid (eg. 5 mg/day for 1-6 months) and parenteral cobalamin (eg. 500 g/month) for 6 months may help mucosal repair. Cobalamin therapy may be particularly useful in cats with chronic IBD and diarrhea since cobalamin malabsorption appears to exacerbate the disease. Severe disease is also associated with fat malabsorption and fat soluble vitamin deficiency. Parenteral fat soluble vitamins should therefore be part of the initial treatment plan in patients with severe disease.

Colonic disease

Immunosuppressive Therapy

Despite the knowledge of symptomatic and nutritional therapy for colitis, the basis of treatment still involves the judicious use of drugs that modulate mucosal immune function. The most important and widely used of these agents are sulfasalazine, prednisone (or prednisolone), and azathioprine. Such drugs as methotrexate, cyclosporin, and some new drugs that specifically influence individual cytokine function are now being used in the treatment of human inflammatory bowel disease. It is possible that these drugs may be useful in the treatment of canine colitis.

Sulfasalazine

Sulfasalazine (salazopyrine) (25 to 40 mg/kg Q8H for three to four weeks in dogs, and 10-20 mg/kg Q12H for 2-3 weeks in cats) is the preferred drug for treating chronic plasmacytic-lymphocytic colitis, the most common type of idiopathic colitis. The active ingredient is 5-aminosalicylate (mesalamine). The exact mechanism or mechanisms of action of 5-aminosalicylate are unclear, but current evidence suggests that it has various bene-

ficial effects. It has been demonstrated, for example, to decrease interleukin-1 production, inhibit 5-lipogenase, reduce platelet-activating factor, act as a free radical scavenger, and inhibit mucosal mast cell histamine release and prostaglandin production.

Most patients respond to sulfasalazine alone or in combination with dietary change within one to four weeks of the onset of treatment. The diagnosis must be reexamined if signs fail to improve after four weeks of treatment or if they recur when the drug is withdrawn. Long-term therapy is contraindicated because sulfasalazine causes keratoconjunctivitis sicca (KCS) induced by sensitivity to sulfapyridine.

Prednisone and Azathioprine

Prednisone has been widely used to treat plasmacytic-lymphocytic enteritis in dogs and cats, but is apparently not as effective as sulfasalazine in the treatment of plasmacytic-lymphocytic colitis. The indication for corticosteroids in the treatment of colitis has not been clarified and remains empirical. Some dogs with colitis reportedly deteriorate when treated with corticosteroids; other patients apparently benefit. Because chronic colitis is an inflammatory disease of unknown cause, corticosteroid use in conjunction with sulfasalazine is occasionally beneficial in patients that are unresponsive to more conventional treatment.

Eosinophilic colitis seldom responds to dietary change alone. Immunosuppressive doses of prednisone or prednisolone (2 to 4 mg/kg once daily for two weeks and then tapered for 6 to 10 weeks) are the preferred treatment for this disease. If mucosal changes are severe, prednisone can be combined with azathioprine (1 mg/kg Q24H for 4-6 weeks in the dog, 0.3 mg/kg Q24H in the cat). Occasional 7 to 14 days courses of azathioprine alone can be used to control signs in patients with plasmacytic-lymphocytic or eosinophilic colitis that are apparently resistant to other forms of treatment.

Histiocytic colitis responds to treatment with enrofloxacin.

Conclusion

With appropriate therapy, the prognosis for most patients with IBD is good. The prognosis should become even better in the future, based on a better understanding of mucosal immune regulation, colonic metabolism, and the effect on colonic mucosal health by short-chain fatty acids produced by microbial fermentation of nondigestible carbohydrate. Nevertheless, there is still a long way to go in understanding this complex and enigmatic group of diseases.

Intestinal lymphangiectasia

Lymphangiectasia refers to a group of small intestinal disorders, the outstanding feature of which is marked dilatation of submucosal intestinal lymphatics. The cause may be obstruction to lymphatic flow secondary to granulomatous or neoplastic disease of the mesenteric lymph nodes or ducts, or less commonly, secondary to cardiac failure. Lacteal dilatation may also be seen in chronic enteritis where it probably arises secondary to restriction of submucosal lymph flow by inflammatory infiltrates. The disease may also be a primary hereditary absence or deficiency of intestinal lymphatics, as has been reported in Norwegian Lundehunds.

In the dog the disease most commonly appears to be secondary to a chronic inflammatory response in the small intestinal lymph nodes and vessels. The inciting cause is unknown. This may occur in any breed, but prevalence seems to be high in Yorkshire Terriers.

Obstruction of the lymphatics results in a characteristic dilatation of the lacteals, disruption of the villus and a loss of lymph (containing protein and lymphocytes) into the intestinal lumen.

Signs may include diarrhea (not always present), weight loss, edema, ascites, and pleural effusion. Panhypoproteinemia (decreased albumin and globulin), hypocholesterolemia and (in about 50% of cases) lymphopenia are commonly seen. Microcytic or hypochromic anemia may also be present.

The diagnosis is made from the clinical and laboratory findings and exclusion of hepatic and renal causes of hypoalbuminemia. Intestinal biopsy may reveal a specific underlying disease. If available, quantitate measurement of fecal protease activity may be used to assess the severity of intestinal protein loss.

Treatment is with a low fat diet, medium chain triglycerides oral antibiotics and prednisone. The prognosis is guarded to poor.

Bacterial overgrowth syndrome

Although well defined in man, overgrowth of bacteria (which refers to overgrowth in the upper small intestine, where few bacteria are normally present) is less well documented in the dog. Normal dogs and cats appear to have bacterial populations which would be compatible with overgrowth in other species. Overgrowth has been associated with a specific enteropathy in the German Shepherd Dog, as well as in many dogs with exocrine pancreatic insufficiency. The disorder probably occurs secondary to a variety of gastrointestinal diseases, but in many cases may be subclinical. It is also suspected that rather than overgrowth (i.e. an increase in number per se) there may be an overgrowth of one species, especially anaerobes, thus strictly, it is an imbalance in numbers. Since many dogs and cats with chronic diarrhea and presumed bacterial overgrowth respond to oral antibiotic therapy. The term "antibiotic responsive diarrhea" is preferred by some.

Classically, bacterial overgrowth is associated with a surgically induced "blind loop" of small intestine, but it may occur with diseases in which there is a failure of normal intestinal motor function, failure of gastric acid secretion (achlorhydria – rare in dogs and cats), pancreatic exocrine insufficiency or abnormal intestinal immune function. Bacteria directly injure the mucosa by production of damaging enzymes. In addition, they change the intraluminal environment by deconjugation and dehydration of bile salts and hydroxylation of fatty acids.

Signs include chronic diarrhea and/or weight loss, perhaps accompanied by borborygmus, flatulence or pica, including coprophagia. Definitive diagnosis requires culture of intestinal content which is clearly impractical in most practices. Increased serum folate and/or decreased serum cobalamin provide indirect evidence for the presence of bacterial overgrowth.

Attempts should be made to eliminate any underlying disease, although in many cases this may not be possible. Even if the cause of bacterial overgrowth cannot be identified and remedied, the overgrowth itself represents a potentially treatable component of different intestinal diseases. A single course of antibiotic therapy may produce long-term improvement in some dogs, while other animals require periodic re-treatment when signs recur. Tetracycline is the recommended antibiotic of choice (10-20 mg/kg Q12H for 1-2 weeks). In some patients metronidazole at 10-20 mg/kg Q12H for 1-2 weeks may be more effective because of its action against obligate anaerobic bacteria. Tylosin at ¼-1 teaspoonful/meal (20 mg/kg Q12H) is an economical choice in some animals that require chronic antibiotic therapy to control signs of gastrointestinal disease. However, some patients reject food treated with tylosin and it may be necessary to place the drug in gelatin capsules.

Small intestinal tumors

Leiomyosarcoma, leiomyoma, lymphosarcoma and adenocarcinoma are the most common tumors of the small intestine in the dog and cat. Signs include vomiting, diarrhea and weight loss. These may be related to malabsorption and protein losing enteropathy secondary to infiltration of the intestinal wall and bacterial overgrowth secondary obstruction.

Intestinal tumors are more common in older dogs or cats, although lymphosarcoma occurs sporadically in younger animals. Radiographs and ultrasound may be helpful for diagnosis when abdominal masses cannot be palpated. Exploratory laparotomy and intestinal and mesenteric node biopsy are diagnostic. Adenocarcinomas may cause signs of intermittent obstruction and may metastasize to the liver or pancreas and produce signs of secondary organ dysfunction. Pulmonary metastasis, however, is rare.

The prognosis is guarded with canine intestinal lymphoma since the alimentary type responds poorly to treatment. The prognosis is better in the cat. Adenocarcinoma has a poor prognosis in both species unless diagnosed early, in which case resection of a localized lesion can produce marked clinical improvement for a period of time - recurrence may be slow (up to 2 years), particularly in cats. Leiomyomas/sarcomas are often slow-growing tumors with an excellent prognosis after complete surgical excision.

Short Bowel Syndrome

This syndrome occurs after extensive surgical resection of the small intestine. The pathophysiology is complex and includes bacterial overgrowth, failure of digestion and absorption, hypersecretion of gastric acid, and secondary changes due to malnutrition. However, the bowel remaining after resection can hypertrophy and increase its absorptive capacity; there can therefore be clinical improvement with time. Supportive care may however be required in some patients and includes a low fat highly digestible diet supplemented with medium chain triglyceride oil, vitamin and mineral supplementation (including parenteral replacement in some patients), elemental dietary supplements, pancreatic enzyme supplements, oral antibiotics, frequent small meals, and cimetidine to inhibit gastric acid secretion.

Mycotic enteropathies

These are rare, but some (e.g. Histoplasmosis) may be more commonly encountered in specific geographical regions.

Histoplasma Capsulation often produces a disseminated infection, and in some patients weight loss and diarrhea may be prominent signs. Other findings depend on the organs involved. There may be panhypoproteinemia. Organisms may be seen in various tissues/organs (blood, respiratory tract) but may also be evident in colonic scrapings or fecal cytologic specimens. Treatment with itraconazole, ketoconazole and/or amphotericin B may be effective.

Intestinal Phycomycosis due to *Pythium sp.* (and others) is fairly common in States bordering the Gulf of Mexico (especially Louisiana). Vomiting and weight loss due to proximal small intestinal involvement are the prominent signs, but more extensive intestinal involvement may occur and result in diarrhea. The intestinal wall may be thickened (an abdominal mass can be palpable in some patients). Phycomycosis should be included in the differential diagnosis of dogs with intestinal masses in the SE USA.

Diagnosis requires intestinal biopsy and identification of fungal elements; surgical resection of the affected bowel is indicated since successful medical treatment is not reported. The prognosis is poor to hopeless.

Aspergillosis and candidiasis have been reported rarely as gastrointestinal pathogens. Immunosuppression of the host has to be suspected in these patients. Treatment with ketoconazole, itraconazole, nystatin or amphotericin B may be effective.

Bacterial enteropathies

Bacterial causes of chronic SID are not well documented. *Yersinia enterocolitica*, *Campylobacter sp.* and *Salmonella sp.* are generally believed to be causative agents in rare instances of acute diarrhea, and have also been cited as cause of chronic diarrhea in some dogs and cats. These cases are of importance because they are potential sources for zoonotic transmission to human beings. *Clostridium difficile* and its cytotoxin have been isolated from dogs with chronic diarrhea but no weight loss; the diarrhea responded to metronidazole therapy, but relapses necessitated repeated therapy. *Clostridium perfringens* can be associated with chronic small and large intestinal diarrhea in dogs. ■

Opriți năpârlirea excesivă!

NUTRIVET - ulei de somon sălbatic (Omega 3 - 30%)!

NUTRIVET

Omega 3 este o necesitate, nu o opțiune, pentru o sănătate optimă. Blana lucioasă și pielea sănătoasă sunt cei mai vizibili indicatori ai stării de sănătate a câinelui sau pisicii noastre. O dietă completă și echilibrată contribuie din plin la menținerea unei stări de sănătate optime pentru animalul dvs., dietă din care trebuie să facă parte și acizii grași esențiali Omega 3 și Omega 6.

Acizii grași esențiali nu pot fi produși de organism și trebuie obținuți din hrană, urmând apoi a fi metabolizați. Omega 3 și Omega 6 sunt cele două tipuri de acizi grași esențiali care trebuie să fie prezenți în nutriția câinelui sau pisicii dvs. Deși nutriția „tradițională" nu pune accentul pe suplimentarea hranei cu acizi grași esențiali, studiile recente arată din ce în ce mai mult importanța acestor elemente pentru starea de sănătate a animalelor de companie. Cantitatea corespunzătoare de Omega 3 în dietă are un impact determinant asupra dezvoltării neuronale și creșterii puilor de câine și de pisică, asupra sistemului imunitar al puilor și adulților deopotrivă, precum și în alte arii - sistemul cardiovascular și reglarea colesterolului, reducerea suferințelor, articularea, buna funcționare a sistemului urinar și a rinichilor, precum și (cel mai vizibil efect) îmbunătățirea calității blănii și a sănătății pielii, mai ales în cazul dermatitelor sau al altor probleme dermatologice.

Cei mai importanți acizi grași Omega 3 sunt ALA (acid alfalinoleic), EPA (acid eicosapentanoic) și DHA (acid docosahexaenoic). Organismul câinilor poate converti ALA în EPA și DHA, dar cu o eficiență limitată, depinzând printre altele și de sursa de proveniență a acizilor grași metabolizați (vegetală/plante sau animală/pește); de aceea, este ideal ca toți acizii grași Omega 3 să fie prezenți în suplimentele administrate. Pisicile, pe de altă parte, nu au capacitatea de a converti ALA în EPA și DHA, deci în cazul lor este esențial ca toți acizii grași să fie oferiți prin alimentație.

În mod similar, cei mai importanți acizi grași Omega 6 sunt LA (acidul linoleic) și AA (acidul arachidonic). Ca și în cazul acizilor grași Omega 3, LA poate fi convertit în AA de organismul câinilor, dar nu poate fi sintetizat de cel al pisicilor, ceea ce face ca în cazul acestora din urmă să fie necesară prezența ambilor acizi pentru o alimentație completă.

Multe din alimentele comerciale destinate animalelor noastre de companie susțin că au în compoziție diferite cantități de Omega 3, dar sursa acestora este în mod primar ALA (obținut din uleiuri vegetale), care poate fi convertit în acizi grași cu catena mai lungă doar în mod limitat. De aceea, este important să urmărim prezența DHA și EPA în aceste alimente, precum și proporția de Omega 3 și Omega 6.

Modernizarea și industrializarea agriculturii au introdus în dieta noastră (și a animalelor noastre) o cantitate prea mare de acizi grași Omega 6. În cazul oamenilor, o dietă tipică din statele moderne conține de circa 10 ori mai mulți acizi grași Omega 6 decât Omega 3 și o situație similară este și pentru mâncarea pentru animalele de companie. Cu toate acestea, studiile de specialitate demonstrează că o proporție echilibrată de Omega 6:Omega 3 de aproximativ 5:1 este cea mai indicată și de dorit. În cantități prea mari, acizii grași Omega 6 (în special AA) pot avea un efect inflamator și pot accentua stările de boală, în mod particular în cazul dermatitelor, al problemelor articulare și renale.

Sintetizând cele spuse mai sus, trebuie să reținem următoarele - că acizii grași esențiali sunt extrem de importanți pentru organism și că, în cazul animalelor de companie, aceștia nu pot fi metabolizați și transformați, deci trebuie să fie introduși prin alimentație (mai ales în cazul pisicilor). În al doilea rând, raportul de Omega 6:Omega 3 trebuie să fie de maximum 5:1 pentru a permite organismului să se dezvolte armonios. În al treilea rând, sursa de proveniență a acizilor grași este foarte importantă, întrucât de ea depinde capacitatea de metabolizare a acestora și, în aceste condiții, sursa animală (adică peștele) este clar de preferat celei vegetale (adică plantele). Mai mult, în cazul surselor animale, adică al peștelui, este de preferat să alegem peștele sălbatic celui de fermă, deoarece acesta sintetizează acizi grași esențiali direct din sursa de hrănire și nu conține produsele toxice care se regăsesc în majoritatea animalelor de fermă (antibiotice, pesticide, coloranți). ■

Arin Christu
SC CAROSHEL Import-Export SRL
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SĂNĂTATEA ANIMALULUI TĂU CONTEAZĂ!

Flapsul mușchiului flexor carpo-ulnar

Ioana Manea
Clinica Veterinară Petvet Ploiești

Abstract

Soft tissue defects occurring after trauma, can be corrected with surgical reconstruction techniques, depending on the site where are located. One of these techniques is to use flaps. Flexor carpi ulnaris muscle flap is used to reconstruct wounds involving tissue from the antebrachial, carpal and metacarpal areas.
Keywords: *reconstruction, antebrachial, flexor carpi ulnaris muscle flap*

Rezumat

Defectele de țesut moale apărute în urma unor traumatisme pot fi remediate cu ajutorul unor tehnici chirurgicale de reconstrucție, în funcție de zona unde sunt localizate. Una din aceste tehnici constă în utilizarea flapsurilor. Flapsul mușchiului flexor carpo-ulnar se folosește la reconstrucția rănilor care implică țesuturi din zona antebrațului, zona carpiană și metacarpiană.
Cuvinte-cheie: *reconstrucție, zona antebrațului, flaps mușchi flexor carpo-ulnar*

În ianuarie anul trecut a fost adus la cabinet un câine luat de către o iubitoare de la stăpânul lui, care nu era dispus să îi acorde îngrijiri medicale. Surpriza nu a a fost acest lucru, ci faptul că animalul avea o fractură distală de radius, deschisă, veche de aproape două săptămâni. În acest timp câinele s-a lins, lăsând vizibile razele osoase implicate în fractură. Nu am ales varianta amputării, ci am decis să tratăm plaga infectată și să remediem fractura. După remedierea fracturii m-am confruntat cu o problemă: închiderea plăgii. Din cauza musculaturii slab reprezentate în zonă și a țesutului cutanat foarte puțin mobil, marginile plăgii nu se puteau afronta corect, lăsând un defect de țesut moale destul de consistent. Am decis să efectuăm un flaps muscular pentru a acoperi porțiunea osoasă, flaps la care am utilizat mușchiul flexor carpo-ulnar, urmat apoi de o grefă de piele.

Fragmentul de os care a rămas neacoperit de musculatură era de aproximativ 3 cm lungime. Pentru efectuarea flapsului am folosit porțiunea humerală a mușchiului flexor carpo-ulnar, flaps care de obicei se folosește la reconstrucția rănilor care implică țesuturi din zona antebrațului, zona carpiană și metacarpiană.

Se incizează pielea în porțiunea caudolaterală a antebrațului, pornind de la cot până la 1-2 cm distal lângă pisiform pentru a expune mușchiul (figura 1). Se continuă cu incizia fasciei antebrahiale și a fasciei carpiene, pentru identificarea mușchiului flexor carpoulnar, mușchi format dintr-o porțiune humerală (inserată pe epicondilul medial humeral pe creasta flexorilor) și o porțiune ulnară (inserată pe olecran). Porțiunea humerală se află între flexorul carpoulnar (caudal) și mușchiul ulnar lateral (în lateral). Se disecă tendonul porțiunii ulnare a flexorului, pentru expunerea completă a părții humerale. După identificarea corectă și disecția acestei părți humerale

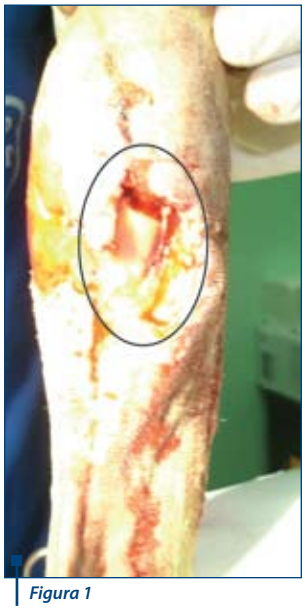


Figura 1

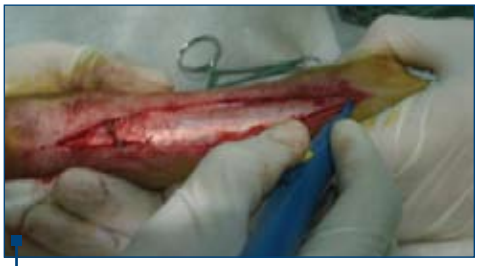
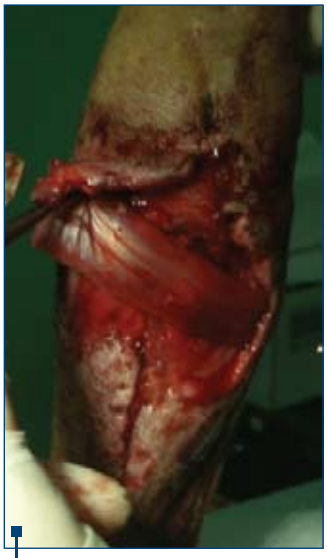


Figura 2


Figura 3


Figura 4

Figura 5



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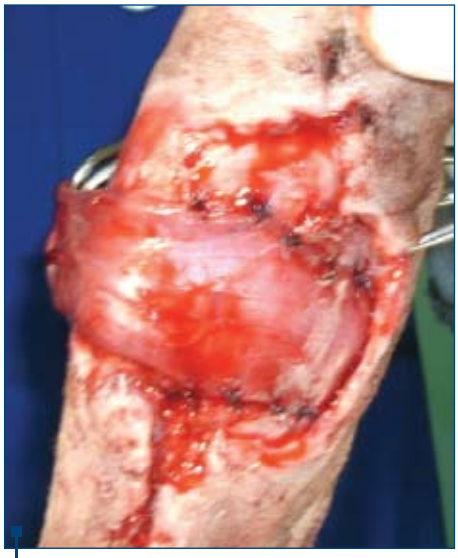

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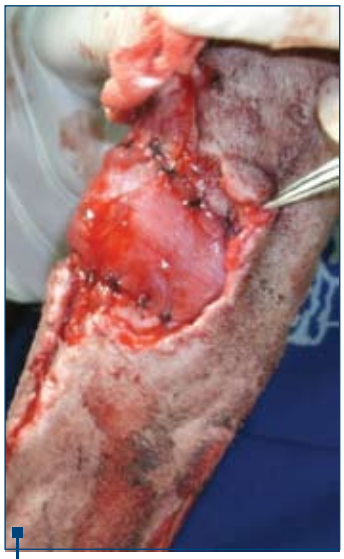

Figura 8

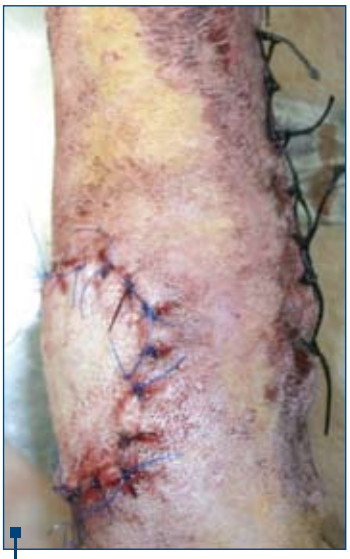

Figura 9

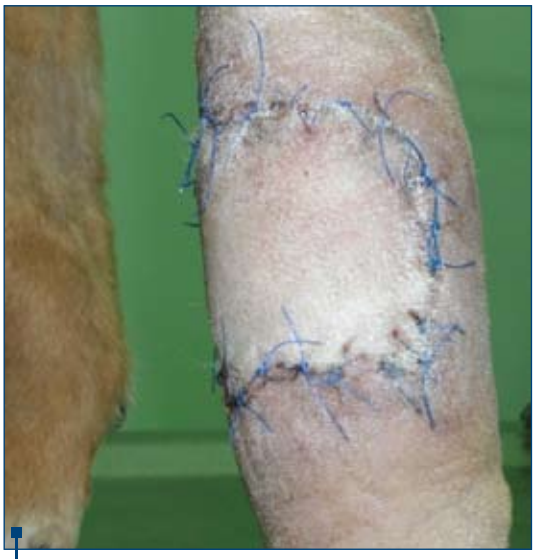

Figura 10

Figura 11. Câinele la un an după intervenție

(figura 2), se secționează mușchiul între treimea proximală și cea mijlocie (figura 3). Se poate face o incizie punte pentru a uni defectul existent cu incizia țesutului cutanat sau se poate dilacera pielea pornind de la incizia cutanată până la nivelul defectului, creând un tunel suficient de lat ca mușchiul să poată fi rotit și adus în plaga care trebuie acoperită (figurile 4, 5). Se afrontează mușchiul cu țesutul viabil (figurile 6, 7) și se plasează dren dacă este necesar. Dacă mușchiul nu este acoperit imediat de o grefă de piele, se aplică bandaje umede, neaderente. Pentru închiderea completă a defectului am efectuat o grefă cutanată (figura 8, imediat după operație, figura 9 - a doua zi). ■

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Care este rolul dietei în boala inflamatorie intestinală a câinelui?

Conferinţă în cadrul ÎNTÂLNIRII LA NIVEL ÎNALT NESTLE PURINA, 22-24 martie 2012 în Lisabona

Prof. Kenny Simpson
Universitatea Cornell, SUA

Ca practicieni veterinari, dacă ne confruntăm cu un caz de boală intestinală inflamatorie canină, de obicei optăm pentru o nouă dietă, pe lângă medicamentele antimicrobiene, antiinflamatorii şi imunosupresive. Dar ce argumente susţin schimbarea dietei? Este dieta un factor predispozant în IBD la câini, şi este schimbarea dietei întotdeauna benefică? Acest articol va examina rolul dietei în etiopatogeneza şi tratamentul IBD la câini.

Boala intestinală inflamatorie (IBD) reprezintă un termen colectiv aplicat unui grup de enteropatii cronice caracterizate prin simptome gastrointestinale (GI) persistente sau recurente şi de inflamaţia tractului GI. Este acceptat la scară largă faptul că IBD este cauzată de interacţiunea dintre genetica gazdei, microflora intestinală (în mod special bacteriile şi constituenţii dietetici), sistemul imunitar şi factorii de mediu „declanşatori" ai inflamaţiei intestinului. Oricum, paşii caracteristici care conduc la IBD, motivele variaţiei fenotipice şi răspunsul imprevizibil la tratament nu sunt cunoscute. În cazul pacienţilor cu IBD caracterizată prin inflamaţie granulomatoasă sau neutrofilică suntem nevoiţi să muncim din greu pentru a detecta agenţii etiologici infecţioşi şi pentru a-i eradica printr-o terapie adecvată. În orice caz, modificările patologice cel mai des observate la câinii cu IBD sunt infiltraţia mucoasei cu limfocite şi celule plasmatice, atrofia vilozităţilor şi dilataţia vaselor limfatice. Acesta este cazul în care de obicei recomandăm dieta. Având acestea în vedere, ce date confirmă într-adevăr rolul dietei în IBD la câini?

Ce dovezi susţin rolul dietei în IBD la câini?

I. Răspunsul clinic la dietă în enteropatiile cu specificitate de rasă

- Seterii Irlandezi sunt predispuşi să dezvolte enteropatie sensibilă la gluten, cunoscută a fi o boală autozomală recesivă.
- Reacţii adverse la porumb, tofu, brânză de vacă, lapte, făină integrală de grâu şi miel au fost descrise la Wheaten Terrierii cu blană moale (SCWT) ce prezintă enteropatie cu pierdere de proteine (PLE) şi nefropatie cu pierdere de proteine (PLN). Analiza pedigriului a 188 de SCWT a dezvăluit un strămoş comun mascul, deşi modul de transmitere este necunoscut. Autoanticorpii asociaţi cu colita ulcerativă la oameni au fost de asemenea observaţi în număr mare la SCWT cu PLE şi preced hipoalbuminemia în medie cu 2,4 ani.
- În cazul a numeroase rase de câini au fost de asemenea găsite niveluri ridicate de autoanticorpi la de două ori mai mulţi câini cu enteropatii determinate de hrană decât IBD care nu au legătură cu hrana.

Î: Pot prevedea care câini cu IBD vor răspunde cel mai probabil la dietă?

R: Pacienții mai tineri și cei cu un nivel normal de albumină par a fi mai receptivi la un tratament bazat numai pe dietă.

II. Răspunsul clinic la dietele comerciale cu număr limitat de antigeni

■ În studii controlate la 65 de câini cu IBD și diaree, 39 de câini au fost receptivi la o dietă pe bază de somon și orez (PURINA VETERINARY DIETS® DRM Derm® Canine Formula). Doar la opt câini boala a recidivat în momentul confruntării cu hrana lor inițială și nici unul nu a fost sensibil la testele cu carne de vită, pui sau lapte. Câinii care au răspuns la dietă erau mai tineri și aveau niveluri mai ridicate de albumine serice decât cei care nu au răspuns la dietă. Aceștia din urmă au fost tratați cu steroizi. Interesant este că histopatologia intestinală nu era diferita în cazul câinilor receptivi la dietă, dar nici în al celor receptivi la steroizi, înainte și după tratament.

■ Într-un studiu realizat pe 13 câini ce prezentau colită plasmocitică limfocitară, semnele clinice au dispărut la toți câinii în decurs de 2 - 28 de luni de hrănire cu o dietă săracă în reziduuri, ușor de asimilat și relativ hipoalergenică.

■ Ca o comparație interesantă între specii, în cazul a 55 de pisici cu afecțiuni GI, 49% au răspuns la schimbarea cu o dietă cu conținut limitat de antigeni. Semnele clinice au reapărut la 16 din 26 de pisici care s-au confruntat cu hrana inițială. Grupul dominant de antigene care au determinat apariția unui răspuns a fost reprezentat de: cereale (grâu, gluten de porumb, orz) și proteine din carne (carne de vacă, pui, miel), iar 50% dintre pisici prezentau multiple alergii.

III. Răspunsul clinic la dietele comerciale cu proteine hidrolizate

■ Șase câini cu IBD au primit o dietă hipoalergenică disponibilă în comerț, conținând soia hidrolizată drept singură sursă de proteină (PURINA VETERINARY DIETS® HA Hypoallergenic® Canine Formula). Cinci din șase câini nu au fost receptivi la diete controlate, iar patru nu au răspuns la nici un mijloc de tratament. Numai terapia dietetică a determinat ameliorarea semnelor clinice la patru câini, dintre care doi având nevoie de terapie medicală concomitentă, unul din ei prezentând insuficiență pancreatică exocrină. La cinci câini s-a observat de asemenea o îmbunătățire ușoară spre moderată din punct vedere histologic la biopsiile duodenale de după terapie.

■ Într-un studiu recent, 26 de câini cu semne de afecțiune gastrointestinală cronică (șase cu patologie normală GI) au fost hrăniți fie cu o dietă intestinală, fie cu una pe bază de soia sau pui hidrolizat. Răspunsul inițial la dietă s-a observat la 88% dintre cazuri în ambele grupuri și aproximativ 66% dintre câinii din ambele grupuri au recidivat la contactul cu hrana inițială. În orice caz, după o perioadă de trei ani, doar unul din șase câini hrăniți cu dieta intestinală a rămas în recidivă, comparativ cu 13 sau 14 câini supuși la dieta hidrolizată.

■ Într-un studiu aflat în derulare s-au semnalat răspunsuri pozitive la o dietă hidrolizată pe bază de soia

(PURINA VETERINARY DIETS® HA Hypoallergenic® Canine Formula) la 18 din 24 de câini cu IBD plasmocitică-limfocitară şi nivel normal de albumine serice şi cobalamine. Acei câini care nu au răspuns numai la dietă au reacţionat la hrană + antibiotice (n = 2) sau imunosupresive (n = 3). Un câine nu a răspuns la terapie. Interesant este că 9 din 24 de câini au prezentat semne dermatologice concomitente, iar la 7 dintre aceştia s-au semnalat răspunsuri pozitive dermatologice la tratamentul cu hrană sau hrană + antibiotice.

Î: Ar trebui să aleg o dietă hidrolizată sau una cu număr limitat de antigeni pentru cazurile mele de IBD?

R: A fost demonstrat că ambele prezintă beneficii pentru câinii cu IBD. Într-un studiu, câinii hrăniţi cu o dietă hidrolizată au fost mai predispuşi la recidivă, în comparaţie cu cei hrăniţi cu o „dietă intestinală".

Care sunt bazele răspunsului clinic la intervenţiile dietetice în IBD?

A fost mult timp recomandat ca intervenţia dietetică în cazul IBD să fie bazată pe o anamneză minuţioasă a dietei, punându-se accentul pe determinarea expunerii la proteine, mai ales la cele de origine animală. Intervenţia dietetică a fost astfel direcţionată spre hrănirea cu o dietă ce conţine proteine care nu au mai fost administrate în prealabil. O abordare mai recentă s-a bazat pe hidrolizarea proteinelor, pentru a ajunge la o greutate moleculară care să nu le permită să lege IgE de celulele ţesutului conjunctiv (aproximativ 4,5-10kDa). Ambele abordări sunt bazate pe ipoteza că inflamaţia intestinală este determinată de hipersensibilitate sau alergia la o dietă pe bază de proteine, frecvent de origine animală. În orice caz, observaţia că mulţi câini nu prezintă recidive în momentul confruntării cu hrana iniţială pune sub semnul întrebării rolul alergiei în IBD la câini. Până când un patomecanism relevant va fi elucidat, termenii de diagnostic „receptiv la hrană" sau „intolerant la dietă" par a fi mai potriviţi decât „alergie la hrană", dat fiind faptul că o bază imunologică a afecţiunii nu a fost identificată. Studiile pe Setteri Irlandezi au sugerat că proteinele din cereale, precum glutenul, ar trebui de asemenea luate în vedere în geneza inflamaţiei intestinale. Este de notat faptul că ingredientele bazate pe cereale pot fi în aceeaşi măsură ca şi proteinele de origine animală răspunzătoare pentru sensibilitatea la hrană a pisicilor cu probleme GI. Ratele ridicate de răspuns la diete care diferă considerabil prin compoziţie (ex: soia hidrolizată versus somon), dar sunt formulate din relativ puţine ingrediente ridică posibilitatea că poate fi vorba mai mult de absenţa unor anumite ingrediente, decât de modificarea sau substituirea proteinei dietetice, care are un efect benefic.

Concluzie

Ratele răspunsului clinic la 60% până la 80% dintre câinii cu IBD plasmocitică limfocitară hrăniţi cu o dietă cu număr limitat de antigeni sau una hidrolizată indică faptul că modificarea dietei este un instrument terapeutic important în controlarea IBD la câini. O descoperire pozitivă neaşteptată a studiilor recente este că puţini câini au nevoie de tratament continuu cu corticosteroizi sau alţi agenţi imunosupresivi. Mecanismul care susţine răspunsurile pozitive la manipularea dietetică a IBD la câini urmează a fi elucidate şi este important să considerăm alte posibilităţi în afară de hipersensibilitatea mediată de IgE la proteine de origine animală.

Î: La câţi dintre pacienţii mei canini cu IBD să mă aştept că vor fi receptivi la o schimbare de dietă?

R: Luate ca un întreg, studiile relevă răspunsuri la dietele cu restricţie de antigeni sau la cele hidrolizate de la 60% până la 88% din câinii cu IBD plasmocitică limfocitară. Rate mari de receptivitate au fost observate la câinii cu nivel normal de albumină. ■

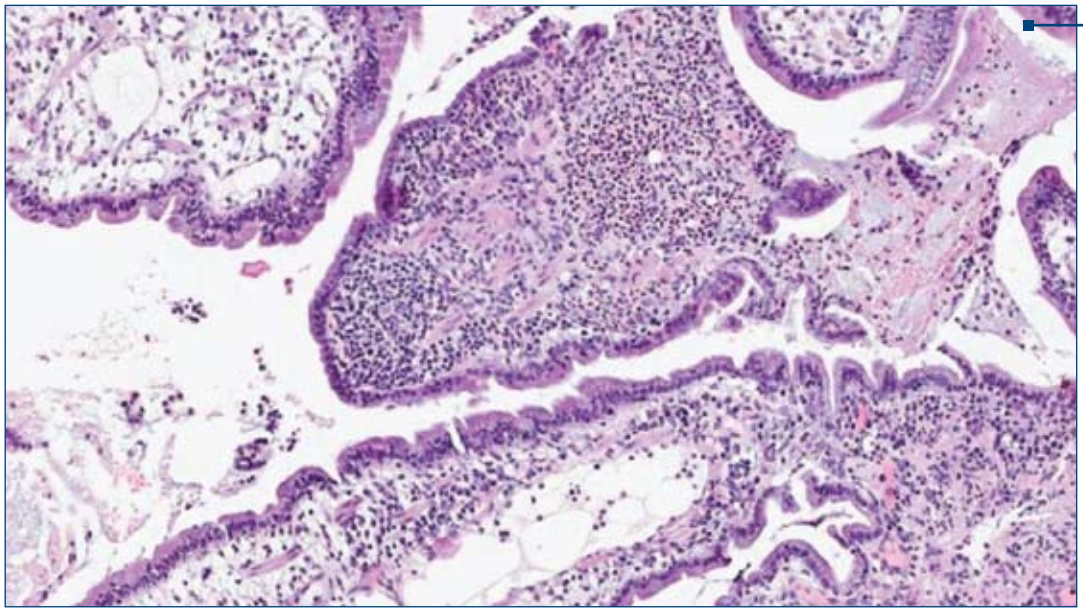

Figura 1.
IBD severă cu limfangiectazie şi infiltraţie limfocitică-plasmocitică şi atrofia vilozităţilor

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