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Vol 03 | Issue 03 | June 2016

Gall Bladder Mucocoeles

Management of Working Capital

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Diagnosing FIP

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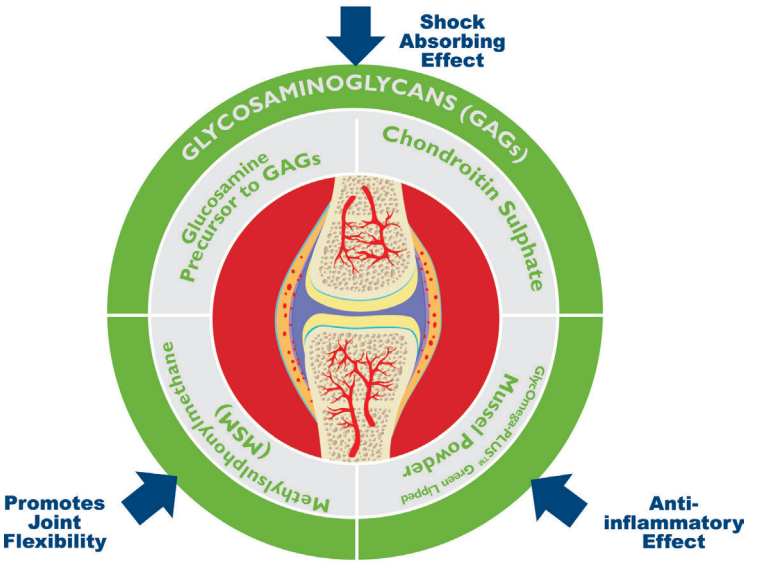

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Editor's Note

I have had requests for back copies of the Vet360. These are available, just email the Vetlink office admin@vetlink.co.za. We are also going to create a bibliography of articles based on the topics. This will be updated after each edition. Printing is however expensive, so this will only be available on the website at vet360.vetlink.co.za.

Over the last few years we are seeing increasing numbers of Scottish Terriers with hepatopathy and showing very elevated ALP concentrations and gall bladder mucocoeles . I have put together several articles to shed some light on this matter.

Feline corona virus is endemic and FIP is a terminal diagnosis. The jigsaw puzzle pieces provided in the article by Dr Hooijberg, an OP graduate, should help you diagnose these cases more accurately.

Regards

Liesel

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- Otitis Externa in Dogs
- Total Ear Canal Ablation Surgery in Dogs

NEXT EDITION: August 2016
- Hyperthyroidism in Cats
- Interdigital and Foot Pad Diseases

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Madaleen Schultheiss

The Management of Working Capital

Andrew Christie
BComm (Industrial Psychology)

Regardless of the type of veterinary practice, access to funds is the difference between a thriving business and one that has to close its doors.

Introduction

Regardless of the type of veterinary practice, access to funds is the difference between a thriving business and one that has to close its doors. This access is particularly important in the short-term as it is required to pay practice expenses (salaries, electricity, etc) and suppliers (consumables and vet foods, etc).

Loans for short-term expenses can be difficult to obtain as banks and other institutions will want to see that the veterinary practice can repay the loan and, of course, if the business had the money available to repay the loan, it wouldn't need to borrow the money in the first place!

So, if a short-term loan is going to be difficult to obtain, this means that cash will be required to pay for day-to-day activities. And the way that cash is obtained is from a business's working capital.

Working Capital = Current Assets – Current Liabilities

Current assets are assets which can be turned into cash within 1 year.

The main current assets in a Vet Practice are typically:

- Stock / Inventory
- Debtors / Accounts Receivable
- Cash

Current liabilities are liabilities which have to be paid within 1 year.

The main current liabilities in a Vet Practice are typically:

- Creditors / Accounts Payable
- Overdraft / Short-term loan

In other words, the main sources of working capital are current assets as these are the short-term assets that the practice can use to generate cash. However, the practice also has current liabilities and these have to be taken into account when calculating how much working capital the practice has at its disposal.

Examining the working capital position enables the practice to foresee any financial difficulties that may arise. Little working capital leads to financial pressure on a practice, increased borrowing (if lucky!), and late payments to suppliers resulting in a lower credit rating.

Analysing Working Capital

Not analysing working capital is the most common reason for businesses failing in South Africa. And the increasing trend for small animal practices selling pet foods and accessories is making them far more susceptible to the risky working capital.

Consider the following figures from a hypothetical start-up Veterinary business:

From the Profit and Loss Statement:	
	R'000
Turnover (Sales)	200
Variable Expenses (Cost of Sales)	-120
Gross Profit	80
Fixed Expenses (Overheads)	-50
Operating Profit	30

From the Balance Sheet:	
	R'000
Current Assets	
Stock (Inventory)	80
Debtors (Accounts Receivable)	50
Current Liabilities	
Creditors (Accounts Payable)	20

The profitability of the business seems pretty good and the current assets are significantly more than the current liabilities.

But before the working capital can be analysed meaningfully, three important key performance indicators (KPI's) need to be calculated:

1. Stock Days

$$\frac{\text{Stock}}{\text{Cost of Sales}} \times 365$$

$$\frac{80}{120} \times 365$$

$$= 243.33 \text{ days}$$

In other words, once an item comes into the practice, it will take an average of 243 days before it can be charged for (surgical needles, gloves, etc) or sold (pet food, etc) and cost recouped.

Analysis:
Naturally one wants the stock days to be as close to zero as possible – not only does a veterinary practice need the money to pay to the owner, but expenses and creditors will need to be paid. If it takes 243 days to get money, it means that the practice will only get money after 8 months!

2. Debtors Collection Period

$$\frac{\text{Debtors}}{\text{Sales}} \times 365$$

$$\frac{50}{200} \times 365$$

$$= 91.25 \text{ days}$$

In other words, once a service has been rendered or a product sold, it takes an average of 91.25 days to collect the money. This doesn't exclude payments made immediately – it is an average of all sales.

Analysis:
Again, it is pretty obvious that the practice wants the money to come in as quickly as possible. If it takes 91 days, it means that the practice would not be able to pay, for example, salaries, for 3 months.

3. Creditors Payment Period

$$\frac{\text{Creditors}}{\text{Cost of Sales}} \times 365$$

$$\frac{20}{120} \times 365$$

$$= 60.83 \text{ days}$$

In other words, it takes an average of 60.83 days to pay all suppliers.

Analysis:
I argue a lot with clients about this. In South Africa we have been raised to pay off debt as quickly as possible. And while this is a good strategy in one's personal life, it is the opposite in a veterinary practice. The reasons for this will become clearer when we look at the Working Capital Cycle.

The Working Capital Cycle

The Working Capital Cycle measures the relationship between trading cash inflows and trading cash outflows.

The KPI's from the previous section create the Working Capital Cycle:

	Days
STOCK DAYS	243
DEBTORS COLLECTION PERIOD	+ 91
TOTAL	334
CREDITORS PAYMENT PERIOD	- 60
	274 days shortfall

What this shows, is that it will take an average of **243 days** to sell an item or use a product in the rendering of a service.

It will take an average of a further **91 days** to collect the money from the client, meaning that from when the practice has bought the stock, it will take a total of 334 days until money is collected.

However, the creditors only need to be paid after an average of **60 days.** This means that the practice will have to find money **274 days** before they receive it, in order to pay their creditors.

So how can this situation be improved?
There are 3 approaches:

1. Reduce stock levels

Compare the stock days at the various stock levels below:

	Stock = 80	Stock = 40	Stock = 20
Stock Days	Stock x 365 / Cost of Sales	Stock x 365 / Cost of Sales	Stock x 365 / Cost of Sales
	80 x 365 / 120	40 x 365 / 120	20 x 365 / 120
	= 243 days	= 121 days	= 61 days

By reducing stock to a quarter of its original level, the stock days have reduced from 243 days to 61 days, a far more manageable period.

Of course, the temptation is always to carry as much stock as possible, ensuring that the practice always has whatever a customer will want. But the bottom line is that by doing this, the practice is being put under additional unnecessary pressure.

2. Reduce Debtors Collection Period
This can only be done in one of three ways – all of which veterinary practices know only too well:

a. Don't let the client out of the practice until they have paid.
b. Don't take on emergency client's that clearly will not be able to pay.
c. Follow a structured debt recovery system. Of these, the one that I find most neglected is the debt recovery system; I strongly encourage practices to send the staff members responsible for collections on a debt collections course, and for a formal strategy to be implemented.

3. Increase Creditors Payment Period
This refers to negotiating and re-negotiating terms with your suppliers. The longer the period you have to pay them, the less pressure your working capital will have. You should fail to meet payment deadlines only in emergencies simply because this will incur penalties and will damage the relationship you have with your suppliers.

4. What Should the working capital cycle be?
Traditionally the KPI's for a veterinary practice are:

	Days
STOCK DAYS	25
DEBTORS COLLECTION PERIOD	+ 5
TOTAL	30
CREDITORS PAYMENT PERIOD	- 30
	0 days shortfall

However, in our current economic climate, this is becoming increasingly difficult as debtors take longer to pay, creditors add pressure for payments and clients take a little longer to make decisions about non-essential treatments and products.

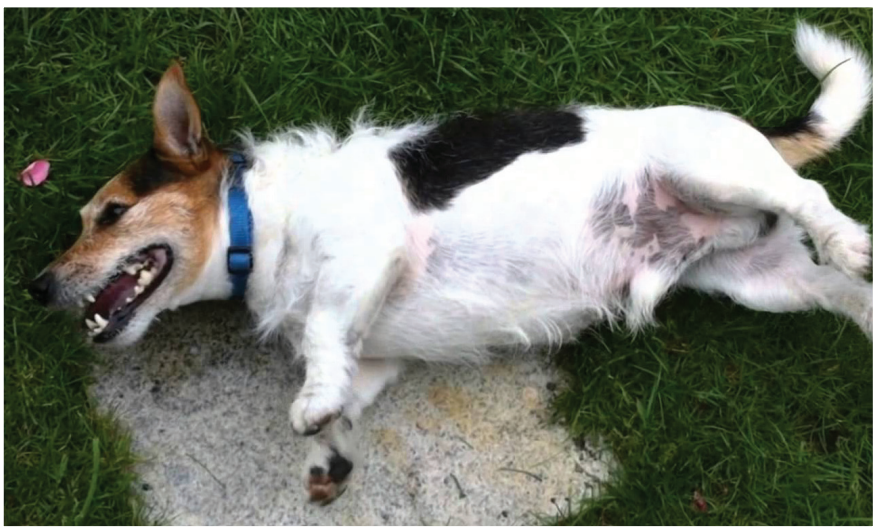
Veterinary practices should:
1. Decrease stock levels to the bare minimum. Arrange with suppliers **better and faster** delivery times. With the reduced prices in logistics, many suppliers should be able to deliver within 2-4 days, meaning that you don't have to carry those items in stock.
2. Improve your debt collection process by making it formal and having a trained member of staff conducting all collections.
3. Try and negotiate better terms with suppliers.

Even if your working capital cycle does not show a shortfall, or even if it shows a surplus, the working capital cycle needs to be continuously improved as both a safety barrier against potential economic fluctuations and as an additional source of revenue by earning interest on the surplus.

Imepitoin, a New Drug to Manage and Control Canine Idiopathic Epilepsy

Efficacy, safety, and tolerability of imepitoin in dogs with newly diagnosed epilepsy in a randomised controlled clinical study with long term follow up. Chris Rundfeldt, Andrea Tipold, Wolfgang Löscher. BioMed Central Veterinary Research (2015) 11:288

Summarised by Dr Liesel van der Merwe, BVSC MMedVet (Med)



Why they did it:

Canine epilepsy is seen in about 0.5 – 5% of the general canine population and is found in a wide range of breeds. Idiopathic epilepsy represents about 60 – 70% of all cases. Current therapy is unsatisfactory with only 15% of patients becoming seizure free and 30% of patients not experiencing significant seizure reduction with phenobarbitone or potassium bromide, the most commonly used anti-epileptic drugs (AEDs).

Imeptitoin, originally developed for epilepsy and anxiety in humans, was withdrawn from further development for humans due to inter-individual pharmacological variability. Initial studies showed the drug was well tolerated in dogs and had good effects as a monotherapy and add-on therapy. The aim of this study was to confirm the safety and efficacy of imepitoin in dogs with idiopathic epilepsy.

How they did it:

A multi-centre randomized double blind clinical field study was aimed at demonstrating the superiority of imepitoin at 30 mg/kg bid (HD) over 1mg/kg bid (LD), the lowest effective dose. In Phase 1, 120 dogs were randomly placed into the HD and LD groups and treated for 12 weeks. Dogs which completed Phase 1 or exited Phase 1 early due to poor response to medication were started on Phase 2, open label follow up at 30 mg/kg bid for another 12 weeks

The sample size was calculated at a minimum of 54 patients per group, which was necessary to pick up a difference of 1 seizure/28 days. Inclusion criteria required at least one of: 2-10 generalised seizures within 3 months, 1 cluster seizure event or status event within 7 days. Dogs were excluded if there was evidence of intracranial disease, more than 10 seizures or any other AEDs used within 3 months of randomisation.

What they found:

127 dogs were included in the study with 66 dogs in the high dose (HD) group and 61 in the low dose (LD) group. Dogs from 60 different breeds were included. 29 (45%) of dogs in the HD group and 30 (50%) dogs in the LD group completed the study (24 weeks). 100 animals continued to open label treatment in Phase 2. The HD group, despite randomisation, had a significantly higher starting baseline mean seizure frequency. Seizure frequency dropped in both groups, but was more pronounced and in the high dose group, 1.7 ± 2.8 seizures per month, versus 0.8 ± 2 seizures per month in the LD group. This was statistically significant. In the HD group 37.5% of animals became seizure free and 31.7% in the LD group.

There was no change in mean seizure frequency in Phase 2 of the trial - indicating continued efficacy of the drug. The increased dose in Phase 2 for some of the patients resulted in a slight reduction in seizures and an increased number of seizure free dogs. Of the 14 seizure free dogs in the LD group, an additional 8 became seizure free on the higher dose (from 30% in the former LD group to 46.8%). Imepitoin did not result in any significant reduction in cluster seizures. The HD group showed significantly higher numbers of CNS-related adverse events: ataxia, disorientation, hyperactivity and restlessness.

Conclusion:

Imepitoin is an effective monotherapy AED in a clinical setting. The drug is suitable for long term use as tolerance was not shown and adverse effects were mild and transient, in the first weeks of treatment only. Imepitoin is less effective against cluster seizures. Approximately 39% of all animals in Phase 2 became seizure free.

Maropitant as s a Novel Treatment for Chronic Bronchitis in Dogs

Investigation of Neurokinin-1 Receptor Antagonism as a Novel Treatment for Chronic Bronchitis in Dogs
M Grobman, and C Reinero
JVIM 2016; 30: 847-852. *Summarised by Mirinda van Schoor*

Why they did it:

Canine Chronic Bronchitis(CCB) is defined as a self-perpetuating inflammatory disease of the airways in dogs. It is characterized by the presence of a cough of more than 2 month's duration in patients for which cardiac disease and other respiratory causes of coughing were excluded.

The only effective therapy in CCB cases is the lifelong administration of corticosteroids. Substance P and its receptor, neurokinin-1 (NK1-R), have been implicated in peripheral and central sensitization of the cough reflex, as well as in the exacerbation of airway inflammation via their role in the recruitment of airway leukocytes.

Maropitant (Cerenia®, Zoetis) is an NK-1 receptor antagonist which has been anecdotally reported to decrease the frequency of coughing in dogs with CCB. It was hypothesized that maropitant will decrease both coughing and airway inflammation in dogs with CCB, making it the perfect alternative to corticosteroid therapy.

What they did:

Eight client-owned dogs were treated with maropitant at the recommended dose of 2mg/kg once daily. This dose was given on alternate days in order to decrease any side effects that may be associated with chronic treatment. The drug was administered for a period of 2 weeks to ensure that a steady state concentration was reached. Steady state is achieved after 4 doses. The owners were given surveys to complete, together with visual analogue scales to measure clinical signs prior to enrolment, after 1 week of therapy and after 2 weeks of therapy. Airway samples for cytology were collected via bronchoalveolar lavage prior to treatment and again after 2 weeks of treatment. None of the dogs were given corticosteroids, antibiotics or antitussives during the study period.

What they found:

All owners reported significant clinical improvement in their pets' condition and agreed that maropitant was an acceptable long term treatment for CCB. Clients reported 75% decrease in cough frequency and 88% decrease in cough severity. Only one owner reported adverse effects (decreased appetite and activity), but these were not severe enough for the owner to consider discontinuation of the drug.

Airway cytology revealed no significant difference in the percentage of inflammatory cells (neutrophils or eosinophils) between the time of enrolment and after 2 weeks of treatment. This finding renders maropitant unsuitable as a monotherapy drug in the management of CCB.

Take home message:

Although maropitant decreased frequency and severity of coughing in CCB, it did not have a significant effect on airway inflammation. Because the control of airway inflammation is paramount in the management of CCB, maropitant cannot be recommended in the treatment of CCB. However, it's role as an antitussive in other diseases such as tracheal collapse warrants further investigation.

Use of Basal Cortisol to Assess Efficacy of Twice-daily Trilostane Administration

Evaluation of baseline cortisol concentration to monitor efficacy of twice-daily administration of trilostane to dogs with piutuitary dependent hyperadrenocorticism: 22 cases (2008 - 2012) Woolcock, AD, Bugbee AC, Creevy KE. 2016 Journal of the American Veterinary Medical Association Vol 248(7):814 - 821

Summarised : Dr Liesel van der Merwe , BVSc MMedVet(Med)

Why they did it?

Hyperadrenocorticism (HAC)/Cushings syndrome is a common endocrinopathy. Trilostane or mitotane (Lysodren®) can be used to lower circulating cortisol concentrations. Conventionally control of HAC is monitored by regression of clinical signs and the ACTH stimulation test. According the product insert for trilostane a cortisol concentration of 40 – 250 nmol/L as the ideal target range. Synthacten® is however expensive.

A previous study has attempted to prove baseline cortisol as a predictor for post ACTH stimulation cortisol levels but the baseline cortisol did not accurately identify 1.5% of patients with excessive cortisol suppression (< 40nmol/L and at risk of severe side effects. Another study showed a poor correlation between baseline and post ACTH stimulation cortisol levels in dogs treated once a day with trilostane. The purpose of this study was to evaluate the use of baseline cortisol concentrations to predict the post ACTH stimulation cortisol concentrations in dogs with pituitary dependent HAC administered trilostane BID.

What they did.

Retrospective study using medical records. All ACTH test were performed at the standardised 4-6 hours post pilling time slot. Cortisol assays were determined using a validated solid phase chemiluminescent immunoassay (Immulite cortisol assay, Siemens)

What they found.

Only 22 patients of the 65 dogs undergoing ACTH stimulation tests had complete medical records. 109 ACTH stimulation tests were performed on theses dogs. Of these 22 dogs 15 (68.2%) had multiple co-morbidities and 4 (18.2%) had a single co-morbidity. Only 3 (13.6%) did not have a co-morbidity. For the study the mean trilostane dose was 1.98 ±0.73mg/kg/day po split into 2 doses administered approximately 12 hours apart.

The median baseline cortisol concentration was 96 nmol/L (range < 5– 315 nmol/L). The median post ACTH stimulation cortisol concentration was 155nmol/L (range 23 – 588 nmol/L).

Of 109 tests performed 6 (5.5%) had a post stimulation concentration identical to the baseline concentration, 18 (16.5%) had a post stimulation concentration less than the base line cortisol concentration and the remaining 85 (78%) had post stimulation cortisol concentrations greater than the baseline cortisol.

Twenty of the 109 ACTH tests performed (on 22 patients) has a baseline cortisol of <40nmol/L .Of these cases, 10 tests showed post stimulation cortisol concentrations with adequate control (>50nmol/L) and 10 showed over suppression (<50nmol/L).

The remaining 89 tests has basal cortisol levels > 36 nmol/L of which 83 showed signs of adequate control in the post stimulation cortisol result and 6 showed excessive suppression of the adrenal gland. Thus the ability of the baseline cortisol to predict that a dog does not have excessive suppression of the adrenal gland from a baseline cortisol concentration (negative predictive value) was 93% (n=83/89).

Forty-five of the 109 adrenal stimulation tests had a baseline cortisol concentration of ≤ 88nmol/L of which 30 showed adequate control of adrenal gland function post stimulation and 15 showed results of excessive suppression of the adrenal gland. The remaining 64 tests all had a post stimulation cortisol concentration in the adequately controlled range. A baseline cortisol concentration > 88nmol/L predicted that the post ACTH stimulation cortisol would be ≥ 55nmol/L with 100% certainty,

Conclusion:

This study showed that the use of baseline cortisol only to monitor the efficacy of twice daily trilostane treatment provided inadequate information about the extent of suppression of cortisol production. The risk of underestimating over-suppression of the adrenal gland was too great.

Treating The Wounded: New Strategies in Healing

Mar 30, 2016
By Heather Lewellen, DVM
VETTED

Don't just scrape by with your wound care practices. Here's the latest on promoting healthy tissue.

We all want that magic wound-healing bullet—the one treatment or dressing or bandage to use on every wound on every patient. Sure would simplify things, wouldn't it? Unfortunately, wound healing is a complicated process and requires different approaches for optimal care. Fortunately, Emily Miller, DVM, DACVS, from the University of Missouri College of Veterinary Medicine, has laid it all out regarding what's new in wound care.

The 6 basic wound management steps:

1. Prevent further contamination, such as nosocomial infections.
2. Debride necrotic tissue. Devitalised tissue incites inflammation, and as long as inflammation is there, the wound won't progress to the repair phase.
3. Remove foreign debris and contaminants—for the same reason as the necrotic tissue.
4. Ensure that the wound has adequate drainage.
5. Establish a healthy vascular wound bed. Healthy granulation tissue is essential!
6. Select appropriate methods of closing the wound if required.

Flourishing with fluid

Keep in mind that the goal for treating acute wounds is simple—to relieve any roadblocks to uncomplicated wound healing. In a nutshell, Dr. Miller says, "We want to do what we can to foster a happy, healthy wound environment to let this animal's body do its normal wound-healing thing." More and more these days that appears to be fostering a moist wound environment in early healing.

For any wound that needs it, surgical débridement is a must, but autolytic débridement has recently been gaining ground, Dr. Miller says. This is the crux of moist wound healing. It's taking advantage of the body's own capability to débride wounds.

A note about exogenous enzymatic products:

Exogenous enzymes can be used as an adjunct to lavage and surgical debridement. Most will spare healthy living tissues but can be irritating to local tissues. They can break down necrotic tissue, liquefy coagulated thick proteinaceous early wound fluid, and possibly break down biofilm of bacteria. They require contact time to work and each one is different (there is no data on which ones are considered more effective), so follow the manufacturer's recommendations for use and wound contact time.

No longer is wound fluid seen as the enemy. It contains endogenous enzymes that can selectively degrade necrotic tissue, inflammatory cells and phagocytes, Dr. Miller says. Cytokines and growth factors stimulate the formation of granulation tissue, angiogenesis and re-epithelialisation of the wound. And wound fluid also provides an ideal environment for phagocytosis to occur by providing optimal pH and oxygen tension.

Topical antimicrobials: a few of the old favorites

"For some reason, people really love to put things on wounds," says Dr. Emily Miller. **But nothing applied to a wound substitutes for proper wound management.**

Another major caveat? Very little scientific evidence exists for any topical product.

- Zinc bacitracin enhances epithelialisation but delays contraction. The ointment has broad-spectrum antimicrobial activity but is ineffective against Pseudomonas species. It is also better at preventing infection than treating an infection that's already established.
- Silver sulfadiazine has broad-spectrum antimicrobial activity and is effective against some fungi as well. It has been the topical of choice for burn wounds because it can penetrate necrotic tissue, which many topical agents can't. Although it enhances epithelialisation, it's toxic to keratinocytes

and fibroblasts. In addition, it's hydrophilic, which means it promotes moist healing.
- Nitrofurazone has broad-spectrum antimicrobial activity but little efficacy against *Pseudomonas* species. It's hydrophilic but delays epithelialisation and is a known carcinogen.
- Gentamicin sulfate is an appropriate choice when a wound is suspected to be infected with gram-negative bacteria. It also promotes epithelialisation.

Patients that undergo autolytic debridement tend to be more comfortable at the wound site because it's not as painful as surgical or mechanical débridement, Dr. Miller says. However, the disadvantage is significant sometimes—this is a slow process. It may take a couple of days before it becomes noticeably effective.

New avenues of healing

While the old standbys are still around—and will be most likely for some time other approaches have been gaining attention recently. Here are a few:

Biosurgical debridement—AKA medical maggots! These creepy crawlers may make your skin crawl, but they may turn out to be very beneficial in wound management, Dr. Miller says. At the moment they're being studied more in human medicine than veterinary.

Wound-healing enhancers: These two wound-healing enhancers you'd expect to find in your kitchen rather than your wound-care arsenal.
- Honey. Some claim that honey enhances wound debridement, reduces edema (it's hypertonic to the wound, so it draws fluid out of surrounding tissues) and inflammation, promotes granulation and epithelialisation, and has some antibacterial activity. Dr. Miller is reserving judgment. "There is no current data that this is doing better than other wound management strategies," she says. "It sounds great, but is it actually working? I'm not sure." If you do use honey for treating wounds topically, Dr. Miller recommends unpasteurised medicinal grade honey.
- Granulated sugar. Like honey, sugar's benefits seem to arise from its hypertonic character. It's purported to reduce oedema, have some antibacterial properties, accelerate sloughing of any devitalized tissue and promote granulation of the wound. The main challenge? Experts recommend using a 1-cm-thick layer over the wound to be effective. Talk about messy!

Circling the drain? Another goal for wound-healing therapy is to provide adequate drainage. What's new here is … ready for it? Vacuum-assisted drainage! This technique uses open-cell sterile polyurethane foam that can be trimmed to the wound size. This is then sealed to the wound with an adhesive drape to which a vacuum is attached. The vacuum-generated pressure draws the wound fluid into a reservoir.

Applying sub-atmospheric pressure (negative 125 mm Hg to be exact) is thought to increase blood flow to the wound tissues, increase the speed with which granulation tissue forms and reduce micro-organism numbers (although this effect has not been reproduced). A significant benefit is that this method of wound treatment allows for bandage changes every two days or so, depending on how exudative the wound is.

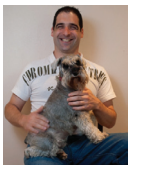
Bandages: So what's new in bandages? Two words: interactive dressings.

These primary (closest to the wound) layers are semi-occlusive and non-adherent. They're hydrophilic, helping create the desired moist healing environment, but can cause maceration of normal skin, so they should be applied only to the wound bed. Some can modulate cell activity and growth factor release. They are highly absorbent, which allows a longer time between bandage changes—and that's a beautiful thing.

There are two getting attention right now:
- Calcium alginate. Made from seaweed, calcium alginate comes in ropes and sheets for different wound types. It stimulates granulation and epithelialisation. Fair warning: When it's doing its job, over time it turns into a viscous, jelly-like substance against the wound and secondary bandage layer, so it needs to be rinsed off. It can be used during inflammatory and repair phases of healing. Depending on how exudative the wound is, bandage changes should occur every one to five days.
- Polyurethane foam. This material is even more absorbent than calcium alginate, is comfortable for the patient, and promotes epithelialisation and wound contraction. It conforms well to wounds and can be used as a filler in deep wounds. However, it can result in reduced granulation tissue formation. It also can be used in the inflammatory and repair phases of wound healing and again, depending on the amount of exudate a wound is producing, this dressing needs to be changed every three to seven days.

Nutrition: Cancer and Patient Care

Dr Anthony Zambelli BSc(Hons) BVSc DiplSenMgmt MMedVet(Med),
Email: info@inandavets.com Tel: 031 762 1816
Inanda Veterinary Hospital & Specialist Referrals



Key Points

- Malignancy creates profound metabolic changes in patients, which can persist into remission
- Treatments and dietary manipulation can blunt or exaggerate these metabolic alterations
- A cancer treatment plan that does not address dietary issues is incomplete and harms patient welfare and outcomes
- It appears that a high protein, high fat, low carbohydrate, moderate insoluble fibre diet high in ω3 fatty acids is ideal for cancer patients

Introduction

The management of a veterinary cancer patient may include one or more of: surgery (sometimes quite aggressive), chemotherapy, radiation therapy, immunotherapy as well as control of concurrent illnesses (comorbidities) that influence overall health e.g. obesity, osteoarthritis, organ dysfunction (e.g. kidney or cardiac disease). The veterinarian embarking on even the most basic oncotherapy should include a nutritional needs assessment as part of the minimum database, and manage the patient's needs on an ongoing basis.

This recognises the profound and fundamental influences a malignancy has on metabolism and thus the response to treatment. A basic understanding of cancer nutritional metabolic pathology is adequate but necessary to manage this class of patient.

Cancer and metabolism in a nutshell

Various cancers have been demonstrated to affect the patient's metabolism in manifold ways. It is safe to say that all malignancies can be underfed or misfed, and any the minority are properly fed. Although can-

cer patients have a tolerance range for dietary intake of various nutrients e.g. lactate[1], this homeostatic reservoir is narrower than for healthy patients.

Essentially, malignancy causes alterations in the major nutritional axes as follows:

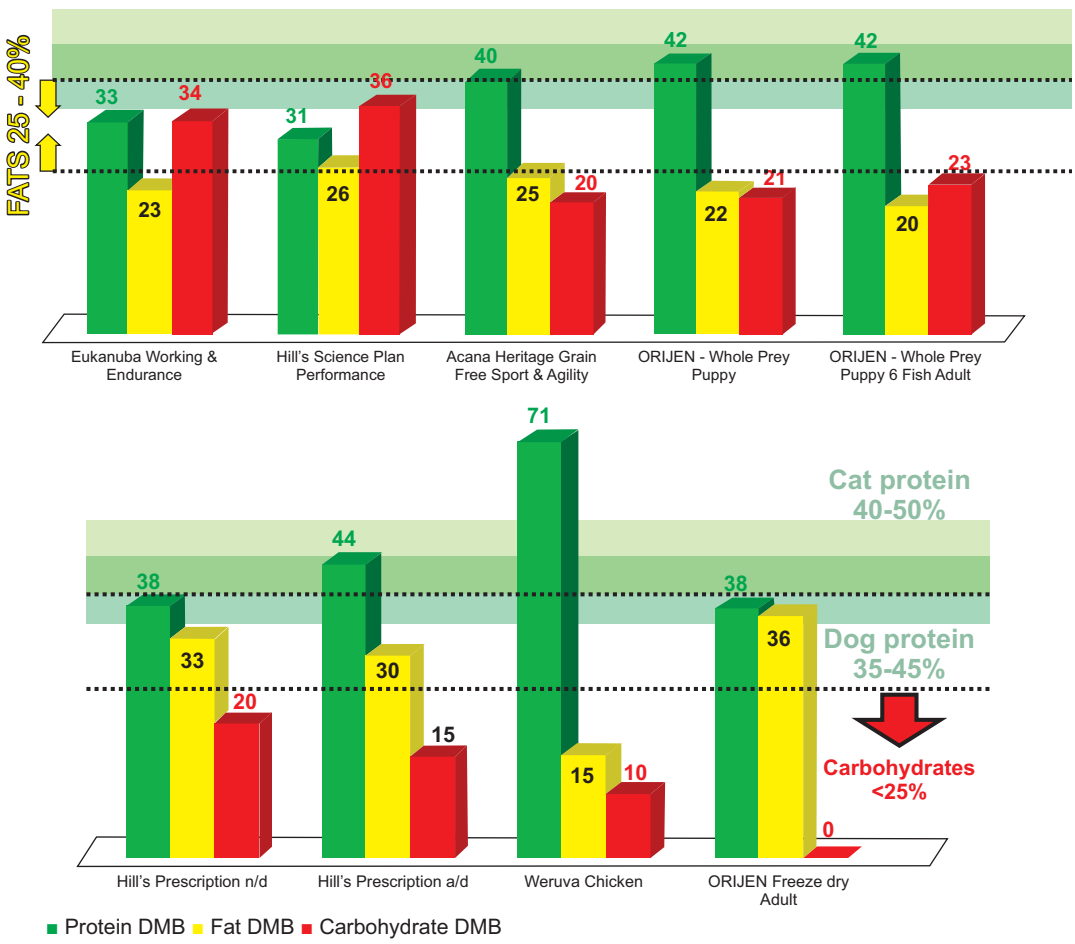
- PROTEINS – patients have skeletal muscle loss due to increased activity of proteolytic (catabolic) pathways and processes, even though liver protein synthesis increases. This causes protein metabolism to be shifted toward the tumour(s), and not replacing the muscle loss (sarcopenia). The altered liver metabolism is typical of the "acute phase protein response" seen in many inflammatory diseases.
  - Clinical consequence – if protein intake does not match requirements, poor wound healing and immunity and altered GIT function result.

- FATS/LIPIDS – the cachexia seen with malignancy is also due to reduce fat synthesis with concurrent lipolysis, driven by various cytokines and chemicals. Insulin resistance caused by various cytokines and tumour-derived factors may be an important driver of this altered lipid state.[2] In addition, the finding that some tumour cells have only a limited ability to utilise fats for gluconeogenesis (the generation of glucose from non-carbohydrate sources) leads some to believe a higher fat diet is physiologically appropriate to cancer patients. In fact, dogs allowed to self-select foods according to the satisfaction of energy intake, will tend to select a diet that is 30% protein, 63% fat and only 7% carbohydrate.[3] In addition, the type of fat eaten seems to be important, with diets higher than 5% in ω3 fatty acids advised by veterinary nutritionists.[4]
  - Clinical consequence – patients with inadequate lipid intake may experience alterations in energy status, increased usage of muscle amino acids to drive gluconeogenesis (further sarcopaenia), altered lipid-soluble vitamin homeostasis (Vit A, D, E and K) and greater susceptibility to oxidative damage of other organs.

- CARBOHYDRATES – at the centre of many arguments about cancer metabolism are carbohydrates (CHO). In this respect we are not discussing fibre, which is discussed below. Carbohydrate is an energy source used by all cells, but less effectively in cancer, due to insulin resistance at the cell membrane. This is aggravated by a relative or absolute hyperlactataemia which may be worsened by administration of Ringer's Lactated intravenous solutions[1, 5] Malignancy sufferers express embryonic versions of certain enzymes and have

Protein, Fat & Carbohydrate Percentage Dry Matter Contents of Various Diets

Recommended Values from Roudebush et al (2004)



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defective Cori-cycles which promote the formation of lactate, and furthermore rob the patient of energy in the form of expenditure required to correct this very abnormality.
Tumour tissue consumes glucose anaerobically, leaving a net gain of only 2 moles of ATP, versus normal metabolism using the Krebs Cycle, which generate 38 moles of ATP from the same amount of glucose.[1, 5-8]

- Clinical consequence – hyperlactataemia and metabolic acidosis. This affects the function of almost every body system, enzyme and membrane by altering the ionisation and movement of electrolytes, the pKi of enzymes (as proteins their binding to substrate is specific to a pH and temperature band) and broader processes such as vascular tone and muscle and nerve impulse conduction. Hyperlactataemia can suppress appetite, further worsening cachexia.

BCS is graded 1 to 5, with 2.5 – 3.0 being considered "normal", 1 cachexic and 5 grossly obese.

MCS is graded from 0 to 3, with 3 = no wasting, 2 = mild, 1 = moderate, and 0 = severe.

The nutritional needs assessment

Why is a nutritional assessment necessary?
It appears that the majority of patients presenting to vets with cancer are in an abnormal body (BCS) or muscle condition score (MCS).[9]

It is important to grade BOTH BCS and MCS to have a fuller appreciation of the patient's needs. Do this at each visit, along with the weigh-in and TPR.

For example, a patient may have BCS 4.5 and MCS 1, if it had a functional adrenocorticotrophic tumour – fat but with little muscle. Some practical considerations for such a patient might include:

- Poor wound healing.
- Increased infection rate.
- Increased friability of the skin.
- Fewer sites for safe or comfortable intramuscular injection.
- Poor muscle strength resulting in slower return to mobility after anaesthesia, and increased risk of tendon or ligament rupture.
- Osteopenia and joint pain from excessive weight without matching supportive strength.
- Easy spread of infection along muscle planes e.g. injection site reactions.

What would be appropriate here, in this patient?

This is the line of reasoning the vet would need to pursue, which requires an understanding of metabolic physiology, diet characteristics and so forth.

Another relevant example might be a rostral mandibulectomy for a fibrosarcoma.

- The patient would require a temporary oesophagostomy tube and then transition onto solid food after 2 weeks.
- The non-nutritive characteristics of the diet are therefore important. Can it be tube fed? What is its water content? What is the patient's water requirement?
- Water intake (in the form of liquidised food) reduces the caloric and nutrient density of the diet. Is this taken into account?
- What is the patient's stomach capacity?
  - An adult cat can tolerate 300ml of stomach fluid given over 10+ minutes.
  - A dog of 15kg can tolerate 450ml; a dog of 55kg+ up to 3L over the same period.

Never feed a sick animal >5% of its body weight at a time if it has not eaten properly in over 3 days; transition it back with small, frequent meals. For dogs undergoing chemotherapy, the prophylactic use of antiemetics (maropitant, ondansetron, metaclopramide) before therapy, and feeding on the morning of chemotherapy, helps offset catabolism from repeated and extended periods of hyporexia some drugs cause.

Diet Selection

In general, aim for a diet with:

- A high quality protein at 35 – 45% DMB (dog) or 40 – 50% (cat) which is readily available and diverse in amino acid profile
- Low readily-soluble carbohydrates (<25%), but dietary fibre of 2.5%+
- Fats 25 – 40%, with ω3 fatty acids >5%

Adjust this according to comorbidities e.g. chronic kidney disease, joint disease. Avoid excessive antioxidant supplementation directly before or during radiation therapy, as this reduces the efficacy of treatment. Some suitable diets of various manufacturers have been graphically represented as examples of diets you can use in SA. This list is not all-inclusive. (Fig 1)

NEVER use raw meats or unwashed vegetables in patients with cancer as they have less tolerance to the myriad of potential parasites and infections carried in unprocessed food, which also has NO improvements in digestibility or nutrient profile over premium ingredient, quality-assured commercial diets. Client compliance with recipes provided for home cooked diets is also abysmally poor; it cannot be recommended at all.[10-16]

The FIP Jigsaw-Puzzle

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Feline infectious peritonitis is a systemic disease caused by a mutated form of the feline enteric coronavirus (FCoV). Although the prevalence of enteric coronavirus infection is high, around 90% in catteries, only 5% of these cats, at the most, will go on to develop FIP.[1]

The pathogenesis of FIP is related to an aberrant immunological and inflammatory response to this virulent mutated virus (FIPV). There are two forms of FIP. The "wet" form occurs as a result of inflammation of serosal surfaces (e.g. pleura, peritoneum) and is associated with the presence of body cavity effusions. The "dry" form is characterised by granulomatous lesions in organs like the kidney, intestines, abdominal lymph nodes, liver, eyes and CNS.[2] The wet form is more common, with ascites usually present.[3] The wet and dry forms represent two extremes of one disease, and patients may present with clinical signs and lesions anywhere on the continuum between them. FIP is invariably fatal.

There is unfortunately no simple definitive test for FIP. The diagnosis of FIP can be compared to making a jigsaw puzzle, where various puzzle pieces need to be fitted together to complete the picture.

The important corner pieces

Signalment: Although FIP can occur in cats in of any age, 50-70% of FIP cases occur in cats under one year of age.

Cats from multi-cat households, catteries or rescue centres with high population densities are exposed to high viral loads of FCoV in faeces and easily become infected. Stressors occurring during this time, like re-homing or neutering, decrease the chance that the animal will eliminate the virus. Cats with immune-suppression are also predisposed. Purebred cats appear to be more susceptible, however the at-risk breeds vary in different studies in different regions and susceptibility may in fact be related to specific bloodlines within a breed rather than the breed itself.[2] Male cats and intact individuals are predisposed.[4]

Effusion: Ascites, thoracic and/or pericardial effusion characterise the wet form of FIP. It should be kept in mind however that less than half of effusions in cats are caused by FIP, and it is the characteristics of the effusion, not the mere presence, that make up this important piece of the FIP puzzle.[1]

Results from effusion analysis, in particular total protein, albumin:globulin (A/G) ratio and PCR, have a higher diagnostic value than tests performed on blood.[3] The following are typical for an FIP effusion:

- Appearance: Usually straw-coloured and clear – typical for FIP but NOT diagnostic
- Cell count: Low (<2x10^9 /L) with the cell population consisting of a mixture of non-degenerate neutrophils and macrophages with lower numbers of lymphocytes
- Protein: High (>35 g/L) due to the presence of gamma globulins. If possible, the albumin concentration of the fluid should be determined (this can be performed on a bench-top analyser, do not attempt if the fluid is very viscous and thick). The globulin fraction is calculated by subtracting the albumin from the total protein concentration. The A/G ratio can then be calculated. An A/G ratio <0.4 has a high predictive value for the presence of FIP and a ratio of >0.8 a high predictive value for the absence of FIP.[3]
- Rivalta test: This is a simple, inexpensive test that is useful to demonstrate a high content of inflammatory proteins in the effusion. A transparent tube of 10-20 mL is filled with 7-8 mL of distilled water and a drop of 98% acetic acid is added and mixed well in order to acidify the solution. A drop of effusion is placed carefully onto the surface of the mixture. If this drop disappears and the mixture remains clear, the result is negative. If the drop remains formed and slowly sinks to the bottom of the mixture like a jellyfish, the test is positive. A negative result has been found to have a high predictive value for the absence of FIP. A positive result could indicate FIP, but may also be seen in effusions due to lymphoma or bacterial infection.[1,5]

An effusion with a high cell count (<2x10^9 /L), predominance of cells other than neutrophils and macrophages, low protein concentration (<30 g/L), A/G ratio >0.8 and negative Rivalta test is **highly unlikely** to be from FIP and other causes should be investigated – i.e. this piece belongs to another puzzle.

Serum albumin: globulin ratio: The serum A/G has a higher diagnostic value than serum total protein or gamma globulin concentrations. Cats with an A/G ratio >0.8 are highly unlikely to have FIP, cats with an A/G ratio <0.6 are highly likely to have FIP.[4]

Histology and Immunostaining: Identification of the FCoV virus in effusion macrophages or in tissue sections (dry form) using immunohistochemistry provides definitive proof of FIP.

- Effusion: Immunohistochemical staining can be used to demonstrate the presence of the virus within macrophages in the fluid and should be performed on all effusions fitting the criteria for FIP. The sample required is at least 12 mL of effusion fluid in EDTA tubes (i.e. at least three filled 4 mL EDTA collection tubes), which should be submitted as soon as possible. (This test is performed in the **Pathology Laboratory at the Faculty of Veterinary Science** in South Africa, and commercial laboratories can forward samples to them.) Positive results are 100% predictive for the presence of FIP, however a negative result does not rule out FIP (the negative predictive value is only 57%). This is because false negatives can occur due to insufficient numbers of macrophages in the sample examined.[6]
- Tissue sections: FIP lesions exhibit a typical histopathological pattern, which is considered to be the gold standard test. The demonstration of FCoV in organ biopsies using immunohistochemistry is also 100% predictive for FIP. These techniques involve invasive sampling but are the only way to confirm FIP in animals without effusions. False negatives may occur.

Pieces that complete the picture, particularly in the dry form, where the "effusion" piece is not present

Clinical signs:[3]

- Wet form: Moderate pyrexia, distended abdomen, dyspnoea
- Dry form: Variable and non-specific but include moderate refractory pyrexia, weight loss, lethargy, intraocular changes (uveitis), neurological signs and enlarged abdominal lymph nodes.

Haematology: Anaemia (both regenerative and non-regenerative) is present in about 50% of cases. A recent study found microcytosis in one third of cats with FIP, 40% of which were not anaemic.[4] This suggests that microcytosis in a non-anaemic cat can increase the suspicion of FIP if other pieces of the puzzle are also present. Lymphoenia is a common finding.[2]

Hyperbilirubinaemia: This a common finding in FIP cats and FIP is the most common cause of this change in cats under 3 years of age.[2,4] Usually there are no concurrent increases in liver enzyme activity. The increase in bilirubin may result in icterus.

PCR in effusions and tissues: Detection of the FCoV virus in effusion or tissue means in theory that there is systemic spread – i.e. the virus is FIPV and FIP disease is present. Studies suggest that this is a useful test to run, particularly on effusions, where it could replace the immunofluorescence test. A consensus as to whether this is an important corner piece or just a useful piece of the FIP puzzle has not yet been reached in the current literature, but updates are expected in the next few years. The RT-PCR test itself needs to be run to high technical standards in order to provide a good diagnostic performance.[7]

The confusing or unhelpful "blue sky" pieces

Coronavirus antibody titres: The antibodies detected in these tests are against feline coronaviruses and are NOT specific for FIP. The vast majority of cats with FCoV titres do not have FIP. **More cats have probably been euthanased based on a positive anti-**

body titre than have died from FIP. Conversely, some cats with acute FIP will not have an antibody titre and some patients with terminal disease may test negative asdall antibody is all bound to viral antigen.[3] The diagnosis of this disease should never ever be based on antibody titres alone. Very high titres of > 1:1600 can be used as a very small piece of the puzzle only.[2]

PCR in blood: Routinely offered PCR detection of coronavirus in blood is not useful, as the test cannot distinguish between FCoV and FIPV. In addition, false negatives occur commonly.[3] A mutation in the spike protein (a protein on the viral envelope that assists with cell invasion) of the FCoV has recently been identified which appears to be associated with systemic spread of the virus (and therefore may be associated with FIPV).

A RT-PCR which detects this mutation is commercially available in Europe, but the presence of the mutation is still not 100% specific for the presence of FIP.[7] PCR of faeces should only be used to identify FCoV shedders for the purposes of controlling viral spread in catteries.

Serum electrophoresis: Electrophoresis patterns may show either a monoclonal or polyclonal increase in gamma globulins, and are not specific for FIP. An alpha-2 globulin increase may be present, representing the increase in acute phase proteins, but is also a non-specific change. Additionally the serum A/G ratio has a higher diagnostic value than the gamma globulin concentration.[6] Electrophoresis is therefore not a useful puzzle piece for the diagnosis of FIP.

Acute phase proteins: Acute phase proteins like serum amyloid A, haptoglobin and alpha(1)-acid glycoprotein (AGP) are elevated in cats with FIP, but increases are also expected in any systemic inflammatory disease. AGP however has been investigated in detail and increases above 1.5-2 mg/mL can potentially discriminate cats with FIP from those without FIP but with clinical signs consistent with the disease.[8] This suggests that AGP is a useful puzzle piece but this test is unfortunately not available in South Africa.

Conclusion

The diagnosis of FIP is not always straightforward and a combination of findings, or pieces of the puzzle, need to be considered together. Finding more pieces will give a clearer picture, and some pieces make a bigger contribution than others.

FIP is NOT contagious:

Cats shed FCoV, which has a possibility of mutating to FIPV in specific cats - those with immunocompromise or immature immune systems, and those in high stress, multi-cat environments

FIP is an SYSTEMIC disease - not an ENTERIC disease - and virus is not shed in the faeces.

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2. A review of sterilisation practices and impact on the individual animal, in dogs and cats. Drs K de Cramer and K May SMS code = a26581
1. Rehabilitation of neurological patients. Dr Megan Kelly, SMS code = a97907

CPD Questions

AC/1504/16

1. Which one of the following statements about FIP is INCORRECT?
   a. FIP is caused by the intestinal corona virus FCoV.
   b. FIP is caused by a virulent mutation of the intestanal corona virus.
   c. FIP is caused by an aberrant response of the host to the mutated corona virus.
   d. FIP is caused by the mutated virus FIPV.
   e. FIP virus is present in 90% of the feline population.

2. Which one of the following statements regarding the prevalence of FIP is INCORRECT?
   a. The prevalence of enteric corona virus can reach 90% in breeding catteries.
   b. Less than 5% of cats infected with FCoV will go on to develop FIP.
   c. 50 – 70% of FIP occurs in cats older than one year of age.
   d. Pure breed cats appear to be more susceptible.
   e. High density population in shelters is a risk.

3. Which one of the following statements regarding effusions in FIP is INCORRECT?
   a. Less than half of the effusions occurring in cats are due to FIP
   b. Ascites and pleural effusion characterise the wet form of FIP
   c. The effusion due to FIP has a characteristic appearance
   d. Tests performed on the effusion may be more diagnostic than those performed on blood.
   e. Effusions in a young cat are almost diagnostic for FIP

4. Which one of the following clinical signs is NOT typical of the dry form of FIP?
   a. Intra-ocular changes such as uveitis
   b. Refractory pyrexia
   c. Dyspnoea
   d. Enlarged abdominal lymphnodes
   e. Neurological signs

5. Which of the following statements regarding FIP effusions is INCORRECT?
   a. The cell count of the effusion is generally low (<2x109/L).
   b. The fluid is generally clear and straw coloured.
   c. The fluid had a high protein content making it quite viscous.
   d. The major contribution to the protein content is albumin from vascular leakage
   e. The Rivalta test is a simple test to show high protein content of fluid.

6. Which of the patterns of effusion characteristics listed below is MOST UNLIKEY to be FIP.
   a. An effusion with a high cell count, a predominance of macrophages, low protein concentration , A/G ratio >0.8 and negative Rivalta test
   b. An effusion with a low cell count, predominance of cells other than neutrophils and macrophages, a low protein concentration and a A/G ratio of >0.8 and a positive Rivalta test
   c. An effusion with a low cell count, a predominance of cells other than neutrophils and macrophages, a high protein concentration , and A/G ratio of >0.8 and a positive Rivalta test
   d. An effusion with a low cell count, a predominance of cells other than neutrophils and macrophages, a high protein concentration , and A/G ratio of >0.6 and a positive Rivalta test
   e. An effusion with a high cell count , predominance of cells other than neutrophils and macrophages, low protein concentration (<30 g/L), A/G ratio >0.8 and negative Rivalta test is

7. Which one of the following statements regarding immunohistochemical staining for FIP is INCORRECT ?
   a. Immunohistochemical staining demonstrates presence of virus in the plasma
   b. Immunohistochemical staining demonstrates presence of virus in the macrophages in the effusion
   c. Immunohistochemical staining demonstrates presence of virus in the macrophages in tiisue biopsies
   d. Immunohistochemnistry is 100% predictive and specific for FIP
   e. Immunohistochemistry is not 100% sensitive - false negatives can occur

8. Which of the following statements regarding FeCoV titres is INCORRECT?
   a. You can get false negative titres in the terminal phase of the disease
   b. Serum corona titres are a good screening test for FIP infection
   c. Serum corona titres test for feline corona virus not FIPV
   d. Serum titres are only significant if titres are very high (1: 1600) with a classical clinical presentation
   e. Titres can be tested in blood and effusion fluid.

9. Which one of the following statements is INCORRECT?
   a. False negatives occur frequently with PCR in blood
   b. Routine PCR can distinguish between FCoV and FIPV
   c. Serum electrophoresis generally shows a polyclonal or even a monoclonal gammopathy, but is not specific for FIP
   d. Acute phase proteins are elevated in cats with FIP – but are also once again, non specific.
   e. AGP (alpha 1 – acid-glycoprotein) is elevated to a greater degree in cats with FIP versus cats with other causes of systemic inflammation
10. A problem with FIP in a cattery can be managed by doing which one of the following?:
    a. Testing the blood of all the cats for FCoV titres
    b. Doing PCR on blood of all the cats
    c. Doing PCR of the faeces of all the cats to detect shedders, and separating.
    d. Euthanasing cats who are positive shedders
    e. Doing electrophoresis to check for acute phase proteins in all the cats

Management of FIP in High Density Households and Catteries

Breeding catteries are high risk environments and FCoV is endemic in many or most. The virus is transmitted primarily via the faecal-oral route and thus hygiene is most important. Corona virus is maintained by continued cycles of infection and re-infection. Young cats are predominantly infected - 40% of infections occur in cats 6m-2yrs old. The incidence of FIP is <4% if cats are older than 3 years.

The virus can stay viable for long periods in the cat litter. Gross and microscopic litter dust contains high numbers of virus particles. Cats contaminate their paws and fur as they use the litter box and then ingest virus particles when they groom themselves. Cats with an indoor outdoor lifestyle seldom get FIP. The risk of transmission is reduced if smaller groups of cats, ≤ 3, are kept per room, have separate airflow, and have outdoor access to bury their faeces.

Faecal shedding:

Attempts to control viral spread by segregation of faecal shedders and non-shedders has been suggested - but is still controversial.

- Faecal PCR is used to determine if the cat is shedding FCoV and non-shedders and shedders are separated.
- Shedders will be retested after 3 months as most natural infections will stop shedding after this time.
- 15% of cats are persistent shedders and these will need to be permanently separated.
- Cats with immunosuppressive conditions or other illness shed more virus for longer:
  - FIV : Shedding is 100X increased, and duration is prolonged.
  - FeLV: Proportion of cats become persistent shedders.
  - Stressed individuals = susceptible individuals.
    - Shelter environment amplifies shedding massively (10^6) due to stress.
    - Sick animals and kittens shed higher levels .
- The primary stage of infection lasts 7-18 months - when shedding is at the highest levels.

Prevention of infection of Kittens:

Kittens typically develop FIP signs post weaning and after rehoming.

Isolation and early weaning:

Most kittens are protected from FCoV infection from maternal derived immunity after until they are 5-6 weeks of age. Separating the queen from other cats and removing their kittens to a clean environment at 5-6 weeks of age will help prevent transmission. Good hygiene is essential to prevent transmission of viruses on food and litter items and clothing.

References:

Addie D, Belák S, Boucraut-Baralon C, et al. Feline Infectious Peritonitis: ABCD Guidelines on Prevention and Management. Journal of Feline Medicine and Surgery. 2009;11:594-604.

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Nail Diseases

By .Patrick Hensel, Dr.med.vet., DACVD
CVC IN WASHINGTON, D.C. PROCEEDINGS

Concurrent claw problems are not unusual in many skin diseases, but rare as the only dermatological problem. The most common disease affecting single claws are: trauma, bacterial or fungal infections; the most common condition affecting multiple claws would be onychodystrophy.

The claw is important for the pet for grasping and holding, moving and used as a defense tool. For this reason it is important that the claws are regularly trimmed and healthy. Diseased claws will predispose to trauma, abnormal locomotion, pain, lameness, and pododermatitis. Various medical terms are used to describe the claw lesion such as: Paronychia (inflammation or infection of the claw fold), Onychodystrophy (abnormal claw formation), Onychogryphosis (hypertrophy and abnormal curvature of claw), Onychomadesis (sloughing of claws), Onychomalacia (softening of claw), Onychomycosis (fungal infection), and Onychoschizia (splitting of claw). If a patient is presented with a claw disease, a detailed history (e.g. vaccine can induce vasculopathy; started with one claw and is spreading typical for symmetric lupoid onychodystrophy) should be obtained and a thorough inspection of the affected claw should be performed.

Cytology is very important and simple test to identify bacteria and yeast, which are very often a cause of secondary infection in abnormal claws. Bacterial and fungal culture is required if initial antimicrobial therapy did not cure the infections. Biopsies are usually required if multiple claws are affected and systemic and immune-mediated diseases or neoplasia are suspected.

Trauma

Trauma is the most common cause for claw diseases in dogs and sometime in cats. Typically one or few claws are affected. In rare cases claws on all four feet are affected, which can occur due to excessive running on asphalt, concrete and gravel, or the use of infected nail clippers. Untreated claw trauma will often result in secondary bacterial infections. The distal part of the affected nail should be removed and the foot bandaged if necessary. In older lesions with suspected infection, foot soaks with disinfectants, and oral antibiotics in more severe cases is recommended.

Bacterial claw diseases

Bacterial claw infections are common and are considered a secondary problem. The most common underlying cause is trauma. However, systemic diseases such as hypothyroidism, hyperadrenocorticism, diabetes mellitus, hypersensitivities, immune-mediated diseases and onychodystrophy may cause claw infections. Chronic severe infections of the nail bed can result in permanent defective claw growth. Swelling of the nail bed area, pain and pus formation is typical.

Cytology is the preferred diagnostic method. If the infections do not respond to initial antibiotic therapy a bacterial culture & sensitivity may be necessary. To treat infected claws it is best to remove as much of

the affected claw as possible. Topical antibiotics or in more severe cases, oral antibiotics should be given until the lesion is healed and enough until the normal claw is covering the previously affected area.

Fungal claw diseases

Fungal infections are rare and organisms which have been reported in claw and often nail bed diseases are: Malassezia, dermatophytes (especially Trichophyton), followed by blastomycosis, cryptococcosis, sporotrichosis. Dogs with Malassezia infections show brown-red discoloration of the claw with brown-colored waxy exudate on the proximal aspects of the claws. This condition is very often seen in dogs with allergies. Diagnostic tests, which will help to identify the organisms, are: cytology, Wood's lamp, fungal culture, and biopsy. Most of these fungal infections (except Malassezia) require aggressive systemic anti-fungal therapy. Itraconazole and terbinafine have been shown to accumulate in keratin, horn, and hairs.

Parasitic diseases

Parasites such as Demodex do not affect the claws, directly but if not treated appropriately, will result in secondary changes due to persistent inflammation and secondary infections. Demodex mites are often difficult to find on skin scrapes from pedal skin, due to their deep follicular localisation in those areas.

Sometimes skin biopsies are necessary to confirm a demodicosis of the feet (due to thickening of the epidermis...*Ed)* Other parasites which have been reported to cause onychogryphosis are Leishmania and hookworms, which may require specific laboratory tests to confirm the disease.

Symmetrical lupoid onychodystrophy

Based on the clinical presentation, inflammatory pattern and response to therapy, symmetric lupoid onychodystrophy should be considered an immune-mediated disease or vasculopathy. This disease can occur in different breeds (personally seen in Greyhound, Schnauzer, Labrador Retriever), but German Shepherds are considered predisposed to this disease.

The problem usually starts with one claw, but within a couple of weeks other claws become affected. The animals appear otherwise healthy, but once the claws start to slough off they become painful, Show lameness, and secondary infections. The claw horn continues to grow, but the horn quality is poor. The claw appears short, discoloured, deformed, soft and crumbly and tends not to attach well to the underlying nail bed. (Figure 1) Histopathology (hydropic and lichenoid interface dermatitis) is usually required to confirm the diagnosis. Because the histological lesions are located very deep at the claw base, amputation of the 3rd phalanx may be necessary to be able to diagnose the disease.

Treatment of this disease requires immunosuppressive therapy with steroids and high doses of omega-3 and

Figure 1: Symmetrical Lupoid Onychodystrophy demostrating short, deformed, crumbling, soft nails.

omega-6 fatty acids (e.g. Derm Caps or Omegaderm) with Vit E, or tetracycline with niacinamide. Improvement and claw regrowth should be noticed within 3-4 months.

Auto-immune diseases

Very rarely auto-immune diseases such as pemphigus vulgaris, foliaceus or erythematosus may affect claws besides other skin areas. A biopsy of the nail bed is usually required to diagnose the disease. As with auto-immune diseases in general, immune-suppressive therapy is required to manage this disease.

Neoplasia

Among the most common neoplasia involving the claws, squamous cell carcinoma, melanoma and mast cell tumors should be considered. Other differentials such as inclusion cysts, keratocanthoma, and inverted papilloma should be ruled out. In cats, nail bed tumors are rare, but metastasis of primary lung carcinoma, hemangiosarcoma and squamous cell carcinoma have been reported. Tumors usually form solitary lesions. Melanoma and mast cell tumors may be aggressive and tend to metastase. All neoplasia should be diagnosed by histopathology and aggressive excision or amputation of the affected digit is usually curative.

Miscellaneous rare claw diseases

Idiopathic onychodystrophy affect multiple claws in older dogs. This condition appears to be predisposed in Siberian Husky, Dachshund, Rhodesian Ridgeback, Rottweiler and Cocker Spaniel. Biotic and gelatin may help to improve the horn quality of the claws.

Idiopathic onychomadesis has been reported in German Shepherd, Whippet, and English Springer Spaniel. Secondary claw infections are not uncommon. Anecdotal reports suggest the use of pentoxifylline.

Anecdotal reports of diseases such as epidermolysis bullosa, dermatomyositis, drug eruption, ergotism, thallotoxicosis, linear epidermal nevi, nutritional deficiencies, disseminated intravascular coagulation, and necrolytic migratory erythema do exist.

References available on www.vet360.vetlink.co.za

Vacuolar Hepatopathy in the Scottish Terrier

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Reviewed by Dr Marlies Böhm

Vacuolar hepatopathy (VH) is a common and frustrating diagnosis often without a specific aetiology. When hepatocytes become injured, one response is for them to swell and become vacuolated. Hepatocellular vacuoles distending the cytosolic compartment may contain, fat, glycogen, intracellular water, metabolic waste products or metabolic intermediates.

Vacuolar hepatopathies may occur in conjunction with hydropic degeneration in which there is cytosolic swelling but devoid of distinct vacuoles. Vacuolar hepatopathies can be caused by a number of conditions - or lesions may develop because of secondary chronic stress (presumed to be endogenous steroid induced) resulting from concurrent disease.

In dogs there are several ALP isoenzymes : bone , liver, and steroid induced, specific to dogs, being some. In dogs, elevated serum ALP activity may reflect inflammatory, neoplastic, or cholestatic disorders involving the liver, biliary tree, or pancreas. A common cause for VH in dogs is the endogenous overproduction of steroidogenic hormones or treatment with cortisone[2]. Increased hepatic production of ALP and its release into the systemic circulation accompanies VH, with the corticosteroid-induced ALP iso-enzyme predominating. Greater than a 3-fold elevation in serum ALP activity is common in dogs with VH.

Traditionally VH is considered a benign lesion, however, progressive VH can lead to diffuse hepatic remodelling resulting in the formation of parenchymal nodules and intra-sinusoidal hypertension secondary to hepatic remodelling. In its extreme manifestation, VH can result in splanchnic hypertension, acquired portosystemic shunts, ascites, and hepatic insufficiency.

Although degenerative VH can develop in any dog, it is recognized as a progressive VH in the Scottish terrier, a breed which also shows a high incidence of hepatic carcinoma. One study in healthy Scottish terriers showed a relationship between elevated serum ALP activity and increased adrenocorticosteroid hormone concentrations following ACTH stimulation as well as a correlative association between older age and concentration of androstenedione.

In a retrospective study, it was shown that VH accompanied by a progressive increase in serum ALP activity was found in the Scottish terrier. These increases are marked - reaching the 2000-3000u/L range . Sequential monitoring over years in individual dogs has confirmed the progressive nature of this syndrome, with increases in serum ALP activity coinciding with development of diffuse increased hepatic parenchymal echogenicity and numerous small hypoechoic parenchymal nodules evident on ultrasonographic examination. Although ultrasonographic detection of features such as hyperechoic or coarse-appearing hepatic parenchyma, hypoechoic nodules contrasting against hyperechoic parenchyma, and discrete mass lesions is common, ultrasound cannot defini-

tively confirm VH. Ultrasonographic features reflect hepatic parenchymal remodelling and progression of non-degenerative to degenerative VH with formation of regenerative nodules sometimes associated with dysplastic hepatocellular foci. It is possible that these foci precede the development of hepatic carcinoma as occurs in humans.

Approximately 40% of Scottish terriers with VH have a broad range of historical, physical, and clinic-pathologic features consistent with typical hyperadrenocorticism. Finding elevated serum ALP activity or VH in a Scottish terrier may thus lead to adrenal function testing that may show an exaggerated adrenal hormone production, which then may be interpreted to represent typical or atypical hyperadrenocorticism. However, one study in 13 Scottish terriers undergoing conventional therapy for hyperadrenocorticism showed no positive outcomes. All treatments were either ineffective or resulted in adverse reactions, such as glucocorticoid insufficiency, hepatotoxicity, and even death. The apparent frailty of the Scottish terrier with VH treated with conventional therapy for hyperadrenocorticism remains unexplained.

As the underlying metabolic abnormality causing VH in the Scottish terrier may be associated with sex hormone–related adrenal gland hyperactivity, treatment with trilostane may thus be ill-advised, considering that studies in dogs have shown that trilostane fosters accumulation of sex hormone precursors, fails to control overproduction of androstenedione, and leads to adrenomegaly. Adrenal gland hyperactivity associated with VH in the Scottish terrier predominantly involves sex hormones (most notably progesterone and androstenedione) and may be accompanied by overt adrenomegaly (unilateral or bilateral). Despite adrenomegaly, various studies have shown that cortisol assessments (baseline and post-ACTH stimulation testing, low-dose dexamethasone suppression test, urine cortisol-to-creatinine ratio) inconsistently verify typical hyperadrenocorticism.

It remains possible that adrenal hyperplasia secondary to chronic stress or illness can account for the increased serum concentrations of cortisol and sex hormones. However, it is plausible that the adrenal gland hyperactivity and associated VH reflect a breed-related genetic disorder affecting adrenal steroid genesis that leads to ALP induction, hepatocellular glycogen accumulation, progressive VH, and ultimately formation of hepatic carcinoma in some dogs. In some Scotties, VH is not always benign, as it may rapidly progress to liver failure with development of small nodular liver, portal hypertension, and ascites. Yet, in other dogs with VH, chronically elevated serum ALP activity (3-20 fold) can persist for > 10 years without apparent clinical deterioration.

The magnitude of elevated serum ALP and ALT activities does not differentiate between dogs with VH with and without hepatic carcinoma and does not predict early death from hepatic failure. Consequently, considering the variable age of syndrome onset and rate of progression, it is impossible to predict relative risk for hepatic failure or development of hepatic carcinoma without sequential patient monitoring.

In conclusion, the Scottish terrier has a breed-specific VH containing glycogen and moderately to severely elevated serum ALP activity. Affected dogs generally have no clinical signs; show elevated ALP activity; and increases in one or more non-cortisol steroid hormones can be demonstrated. It is thus recommended that Scottish terriers with elevated serum ALP activity undergo biannual monitoring of serum liver enzyme activities and liver function as well as a yearly hepatic ultrasonographic examination.

Increased surveillance frequency is advised for dogs with sudden marked increases in serum ALP activity or transitioning to a nodular hepatopathy on ultrasonographic evaluation. This strategy would allow early detection of hepatic mass lesions likely to represent surgically excisable hepatic carcinomas and recognition of a subclinical gallbladder mucocele that may allow for uncomplicated cholecystectomy.

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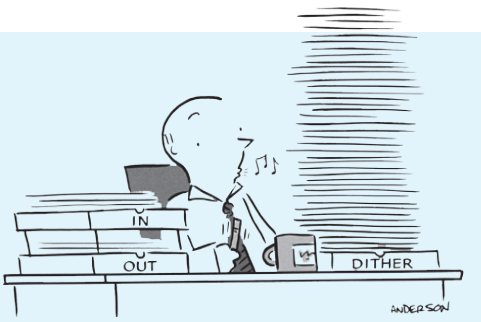
In Summary

From the desk of Dr Marlies Bohm



- Scotties have higher ALP levels than other breeds.
- In Scotties, ALP levels increase with age.
- The pesky bit - Scotties also have a higher prevalence of diseases associated with high ALP levels - Cushing's, pancreatitis, cholestatic liver disease, diabetes.
- When Scotties have these diseases their ALP levels are much more elevated than that of dogs of other breeds affected with the same disease. But then they also show the typical changes on history, clinical exam and bloods for these diseases.

In your happy high ALP Scotties, ALT levels are typically mildly increased (usually < 3x), urine SG is decreased (typically in the mid teens) and cortisol levels may be increased post ACTH in about 1/3 of dogs.

One retrospective study of 114 Scotties with high ALP showed that 34 eventually developed a hepatocellular carcinoma. But the dogs with a liver tumour lived just as long as the dogs that had just the vacuolar hepatopathy.

In your happy high ALP Scotties, ALT levels are typically mildly increased (usually < 3x), urine SG is decreased (typically in the mid teens) and cortisol levels may be increased post ACTH in about 1/3 of dogs.

So:

- If a Scottie has high ALP levels and is otherwise well, leave him be.
- If you suspect pancreatitis, diabetes or Cushing's you need to ignore the ALP in your work-up.This is particularly important with Cushing's.
- If you think he has another hepatobiliary disease you're going to need more than an elevated ALP to suspect it and a biopsy to prove it.
- Dogs with focal hepatocellular carcinoma can do very well after resection, so if you have a client that would operate on their dog serial ultrasounds could be considered.

An Update on Gallbladder Mucocoeles in Dogs

Apr 01, 2009
By Audrey K. Cook, BVM&S, MRCVS, DACVIM, DECVIM-CA, Rebecca Quinn, DVM, DACVIM
VETERINARY MEDICINE

Once thought to be rare in dogs, gallbladder mucocoeles are now frequently diagnosed. And although primarily thought to be best treated surgically, mucocoeles may be effectively managed medically in some patients when caught early.

Before 2000, gallbladder mucocoeles were rarely reported in dogs. However, they are now considered one of the more common causes of extrahepatic biliary disease. While gallbladder mucocoeles were initially treated by cholecystectomy, case-based evidence indicates that some may resolve with medical therapy.[1]

In this article, we review gallbladder anatomy and physiology and discuss the pathophysiology of mucocele development. We also describe the clinical presentation, diagnosis, and management of dogs with gallbladder mucocoeles.

Gallbladder Anatomy

The gallbladder is an excretory organ found between the quadrate and right medial liver lobes. It is pear-shaped and composed of a fundus, body, and neck. From the gallbladder's neck, the hepatic ducts join the cystic duct to form the common bile duct, which leads to the duodenum.

The gallbladder wall has five layers: epithelium, mucousa, tunica muscularis externa, tunica serosa, and tunica adventitia.[2]

1. The epithelium is simple columnar and highly absorptive. It plays an important role in gallbladder function because it secretes mucin, immunoglobulins, and acid.
2. The mucousa is a combined layer consisting of the lamina propria and tunica submucousa.[2] These layers of the mucousa are indistinguishable and contain a dense population of lymphocytes and plasma cells.
3. The tunica muscularis externa consists of scant, randomly organized smooth muscle fibers.
4. The tunica serosa is a membranous layer surrounding the gallbladder that faces away from the liver.
5. The tunica adventitia is the outermost gallbladder layer and faces the liver.

Gallbladder Functions

The gallbladder plays many important roles in digestive health and function, including storing and concentrating bile. Within the gallbladder, water, electrolytes, lipids, and proteins are absorbed from the bile. This absorption allows for a five- to 20-time increase in bile bilirubin and bile salt concentrations. The gallbladder also acidifies bile through epithelial acid secretions and adds mucin to bile by bile acid stimulation of the mucousa.

Gallbladder Physiology

Bile

Bile is required for the successful digestion and absorption of nutrients. It aids in fat digestion and absorption by emulsifying large fat particles into smaller ones that are more susceptible to pancreatic lipase. Bile also enhances intestinal absorption of digested fats and aids in the excretion of waste products, including cholesterol and bilirubin.

Bile is produced by hepatocytes and collected in the hepatic canaliculi. From the canaliculi, bile drains into interlobular ducts. These ducts progressively merge to form hepatic ducts, which join the cystic duct to form the common bile duct.

The common bile duct enters the duodenal lumen at the major duodenal papilla; this junction is commonly referred to as the sphincter of Oddi. The pancreatic duct also empties into the small intestine at this point.

The pH of bile ranges from 5.9 to 7.8. Bile is made of water, bile acids, bilirubin, cholesterol, lecithin, and electrolytes.[3] Although some bile flows directly into the small intestine from the liver, most of it is temporarily stored in the gallbladder.

Substantial bile modifications occur within the gallbladder: sodium, chloride, and water are removed and hydrogen ions are added. After a meal, the gallbladder also secretes bicarbonate-rich mucin, which mixes with the stored bile. The gallbladder contents become inspissated (sludgelike) when more fluid is reabsorbed or more mucin is added.

Biliary Sludge

Biliary sludge is a mobile mixture of precipitated cholesterol crystals, bile pigments, bile salts, and mucin. In people, inspissated bile is considered abnormal and is associated with cholestasis, choleliths, cholecystitis, biliary infection, fasting, and partial parenteral nutrition.[4] In dogs, biliary sludge may be associated with disease but is also often seen in clinically normal geriatric dogs.[5] At present, the significance of biliary sludge in dogs is unknown.

Gallbladder Contraction

The gallbladder musculature contracts in response to cholecystokinin; this hormone is released from enterocytes after the ingestion of a fatty meal. In particular, proteoses, peptones, and long-chain fatty acids stimulate cholecystokinin release. Cholecystokinin concentrations peak about 20 minutes after a meal and may remain elevated for almost two hours. Once the gallbladder contracts, its contents are emptied into the duodenum within about 60 minutes.[3] Cholecystokinin also causes relaxation of the sphincter of Oddi and the release of pancreatic digestive enzymes.

Other factors that contribute to gallbladder contraction include parasympathetic stimulation by the vagus nerve, sympathetic inhibition of the splanchnic nerve, neurotensin, and substance P.[6-8]

Gallbladder relaxation

The gallbladder relaxes in response to somatostatin, vasoactive intestinal polypeptide, nitric oxide, and pancreatic polypeptide.[3,6,9] The relaxation phase is associated with bile storage and modification. During this period, the sphincter of Oddi is closed.

Gallbladder Mucocele Pathophysiology

A gallbladder mucocele is an abnormally distended gallbladder containing a buildup of luminal mucus. Grossly, the gallbladder appears enlarged (unless it is ruptured) and contains gelatinous material that may be congealed mucus, a mucus cast, or inspissated bile (Figures 1 & 2).

Before 2000, mucocoeles were considered rare.[10] In fact, most were noted as incidental necropsy findings.[11] Given the diagnostic tools available at the time, the true clinical impact and incidence of mucocoeles before the 1990s are unknown.

Predisposition to mucocoeles may be associated with dyslipidemias in particular breeds such as Shetland

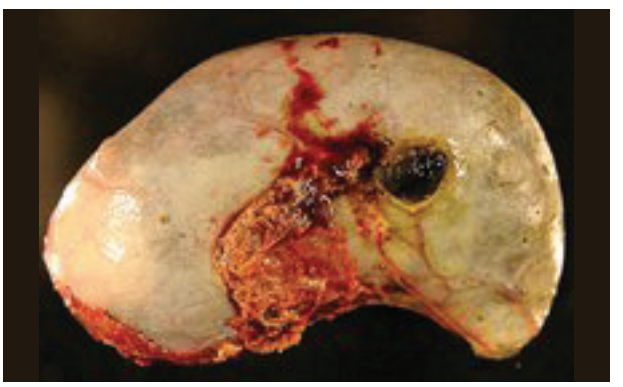


Figure 1. A surgically removed canine gallbladder with a mucocele. The cystic duct has been ligated and removed, and the resultant opening allows visualisation into the distended gallbladder. (Image courtesy of Dr. Roy R. Pool, Texas A&M University's Department of Veterinary Pathobiology.)

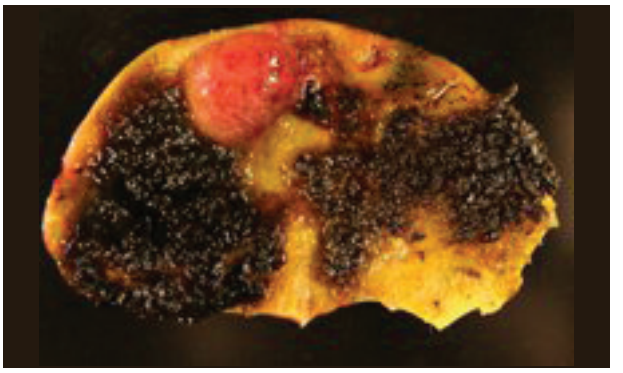


Figure 2. A longitudinal cross section of the gallbladder shown in Figure 1. The yellow material is the mucocele, and the black material is bile. The pink area is a hematoma that developed during surgical excision. (Image courtesy of Dr. Roy R. Pool, Texas A&M University's Department of Veterinary Pathobiology.)

sheepdogs.[12] While extrahepatic biliary duct obstruction is a recognized cause of mucocele formation in people, prospective studies in dogs do not support this finding.[13] Conversely, it appears that extrahepatic biliary duct obstruction is secondary to mucocele formation.[14]

Other suggested causes of canine mucocele formation include progestational therapy or progestational compounds, cholecystitis, and glucocorticoid excess.[4,15,16] To date, none of these theories has been widely supported.

It has been proposed that mucocele formation is the result of progressive biliary sludging.[17] As biliary sludge forms and progresses, gallbladder motility may be decreased, resulting in biliary stasis and increased water absorption. As the gallbladder continues to absorb water, its contents become more solid and immobile.

The most widely supported theory of canine mucocele formation implicates mucus-secreting cell proliferation and dysfunction.[18] In this condition, cystic mucinous hyperplasia of the gallbladder epithelium occurs, and the gallbladder epithelial cells secrete excessive mucus into the gallbladder lumen. This condition may be a primary defect (an inherent disorder of the mucous cells) or a secondary defect (exposure to excessive bile salts).[19]

Diagnosing Gallbladder Mucocoeles

History and signalment

Most patients with mucocoeles are older (average age of 9 years), and no sex predilection has been established.[11] Mucocoeles are usually reported in small or medium-sized dogs, and Shetland Sheepdogs and Cocker Spaniels are overrepresented.[20] In one study, 66% of Shetland Sheepdogs with gallbladder disease had confirmed mucocoeles.[12]

Clinical Signs

About 77% of patients with mucocoeles are clinically ill, often with an acute onset of signs.[21] Vomiting, anorexia, lethargy, polyuria, polydipsia, and diarrhea are most often reported.[21] Cumulatively, studies have shown that almost one-quarter of patients with mucocoeles were asymptomatic.[10,14,17,18,22]

Physical Examination Findings

The most common physical examination findings in patients with mucocoeles are abdominal pain and icterus. A small percentage of patients may be febrile or have abdominal distention.[18]

Diagnostic Test Results

Laboratory evaluation and radiographic findings may vary, but common results are listed in Table 1. It may be difficult to appreciate the differences between biliary sludge and a true mucocele on ultrasonographic examination. Biliary sludge is movable and gravity-dependent, whereas a mucocele is immobile and displays a distinctive striated or stellate (kiwi fruit or starfish) pattern (Figures 3 & 4).[17]

If differentiation is difficult, a second ultrasonographic examination after the administration of a cholagogue (an agent that promotes increased bile flow from the gallbladder, such as cholecystokinin octapeptide given intravenously over one minute at a dose of 0.04 µg/kg23) may be helpful. If a patient has biliary sludge, gallbladder contraction occurs within 10 minutes, and

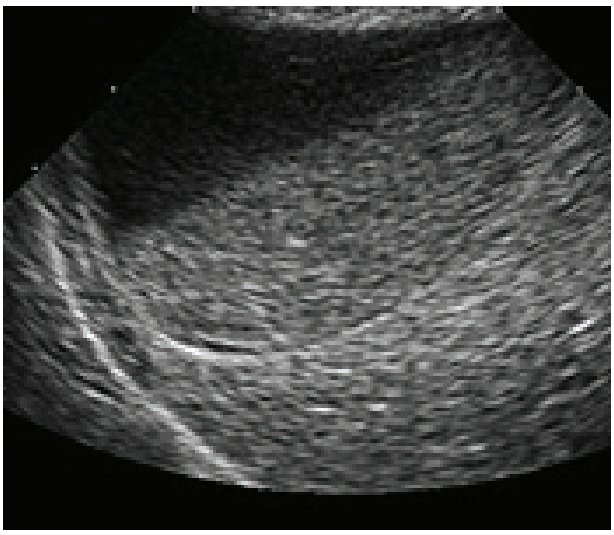


Figure 3. A transverse ultrasonogram of a canine gallbladder containing normal biliary sludge. The echogenic material within the gallbladder has fallen dorsally, and the supernatant of more normal bile can be appreciated ventrally as an anechoic area. (Image courtesy of Dr. Benjamin D. Young, Texas A&M University's Large Animal Clinical Sciences Department of Radiology.)

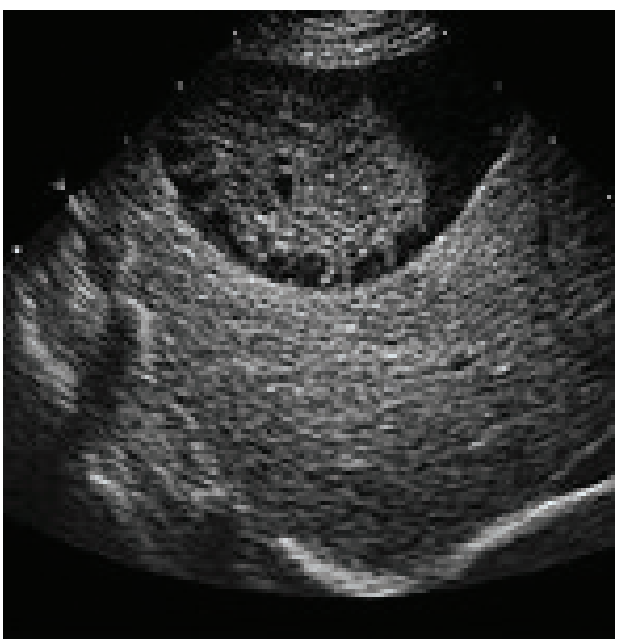


Figure 4. A transverse ultrasonogram of a canine gallbladder containing a mucocele. This image demonstrates the classic stellate pattern and nongravity-dependent gallbladder contents pathognomonic for mucoceles. (Image courtesy of Dr. Benjamin D. Young, Texas A&M University's Large Animal Clinical Sciences Department of Radiology.)

Tabel 1: Common Diagnostic Test Results in Dogs with Gallbladder Mucoceles

Diagnostic Test	Result
Complete blood count	Normal results or neutrophilic leukocytosis or non-regenerative anemia
Serum chemistry profile	Elevated alkaline phosphatase, alanine transaminase, and gamma-glutamyltransferase activities; increased total bilirubin concentration
Urinalysis	Normal results or bilirubinuria
Abdominal radiography	Normal findings or cranial abdominal mass effect
Abdominal ultrasonography	Enlarged gallbladder; increased gallbladder volume; distended common bile duct; immobile or echogenic bile, striated or stellate biliary pattern; normal, thick, or disrupted gallbladder wall

a substantial (about 40%) reduction in gallbladder volume will be evident.[23]

cholecystocentesis may be performed, as well as aerobic and anaerobic bacterial cultures of the bile. Anywhere from 9% to 43% of patients with mucocoeles may have biliary bacterial infections, and bacterial populations are often mixed.

The most common isolates are *Escherichia coli* and *Enterobacter*, *Enterococcus*, and *Clostridium* species.[24] The bacterial infections are thought to ascend from the intestines but may also be hematogenous in origin.

Histopathology

A true mucocele has hyperplastic mucus-secreting glands and may also demonstrate inflammatory infiltrates.[17] In one study, gallbladder wall necrosis was diagnosed in 80% of patients with mucocoeles, and hepatic pathology was often present.[17] The most common hepatic changes include cholestasis, neutrophilic inflammation, and bacterial infection.[20]

Treatment Options

Emergency surgery is necessary if the gallbladder has ruptured or a patient has septic peritonitis. Surgery is also indicated if a dog is clinically compromised or has evidence of extrahepatic biliary duct obstruction, but the patient may need to be stabilised with fluids, intravenous antibiotics (Table 2), antiemetics, and analgesics before anesthesia.[21] Time to stabilisation will vary greatly for each patient and may require one to three days. If the patient has biochemical changes but is asymptomatic or has only mild clinical signs, medical management appears to be an appropriate choice.[1]

Surgical Management

Preoperative diagnostic tests, including a complete blood count, serum chemistry profile, urinalysis, and coagulation panel, are indicated. Treat all patients with appropriate intravenous fluids, and any electrolyte derangements must be addressed before surgery. Vitamin K_1 therapy is also recommended (Table 2) in all dogs, even if the results of preoperative coagulation panels are normal.[21] Start subcutaneous vitamin K_1 therapy 24 hours before surgery to allow adequate time for the production of vitamin K-dependent co-

Table 2: Drugs Commonly Used in the Perioperative and Medical Management of Dogs with Gallbladder Mucoceles

Drug	Dosage and Frequency	Route	Comment
Ampicillin sodium-sulbactam sodium (Unasyn - Pfizer)*	20-30mg/kg every 8 hours	IV	Broad-spectrum IV antibiotic
Metronidazole	15 mg/kg every 12 hours or 7.5 mg/kg every 12 hours if hepatic insufficiency is present	PO, IV	Antibiotic - effective against anaerobes; antiprotozoal
Enrofloxacin	5-20 mg/kg every 24 hours	PO, IV	Broad-spectrum antibiotic-effective against gram-negative organisms
Amoxicillin trihydrateclavulanate potassium	15-22 mg/kg every 12 hours	PO	Broad-spectrum oral antibiotic
Vitamin K_1	0.5 mg/kg every 12 hours	SQ	Counteracts Vitamin K deficiency
Ursodiol	7.5 mg/kg every 12 hours or 10-15 mg/kg every 24 hours	PO	Choleretic, hepatoprotectant, antioxidant, anti-inflammatory
Silymarin	20-50 mg/kg every 24 hours	PO	Choleretic, hepatoprotectant, antioxidant, anti-inflammatory
S-adenosylmethionine	20 mg/kg every 24 hours	PO	Antioxidant

**Reference: Plumb DC. Plumb's Veterinary Drug handbook. 6th ed. Ames, Iowa: Blackwell Publishing, 2008;60-62.*

agulation factors. Continue therapy until bile flow is normal, typically two to four days after surgery.

During surgery, a complete abdominal exploratory is recommended so that concurrent or occult problems can be identified and addressed. While several surgical techniques are described, cholecystectomy has many advantages, as removal of the gallbladder prohibits secondary gallbladder infection and rupture.[18] Other surgeries that have been described include cholecystotomy, cholecystoduodenostomy, and cholecystojejunostomy.[25] Cholecystotomy is a suboptimal choice given the disease process and possible associated gallbladder wall compromise.

The surgical evaluation should also include expressing or cannulating the common bile duct, performing liver biopsy, and collecting bile and liver samples for aerobic and anaerobic culture. In patients with biliary rupture, extensively flush the peritoneal cavity, and place abdominal drains.

Perioperative care includes broad-spectrum antibiotics (adjusted based on culture results), hepatoprotectants, and a low-fat diet. The most common complications of cholecystectomy include pancreatitis and bile peritonitis; death is also common.[14,17,18] Chronic vomiting occurs infrequently. The perioperative mortality rate associated with cholecystectomy is moderate, with 22% to 40% of patients dying within 14 days of surgery.[14,17,18]

Patients that survive this period have excellent long-term survival rates. In general, the morbidity and mortality rates of patients with extrahepatic biliary duct obstruction undergoing biliary diversion procedures are higher than those undergoing cholecystotomy.26 Unfortunately, no reliable predictors for survival exist.

Medical Management

Medical management can be considered in asymptomatic and mildly symptomatic patients without evidence of extrahepatic biliary duct obstruction or gallbladder rupture, as long as the clinician and client are aware of the potential complications.[1,21]

Antibiotics. Because some mucocoeles are associated with bacterial infection, performing an ultrasound-guided cholecystocentesis to obtain a sample for aerobic and anaerobic bacterial culture is recommended. Although considered relatively safe, rare complications of percutaneous cholecystocentesis may include bile leakage, bradycardia due to a vasovagal reaction, bacteremia, and local hemorrhage.27,28 Studies have indicated that risks associated with percutaneous cholecystocentesis in normal dogs and those with cholecystitis are minimal, but complication rates have not been determined in dogs with mucocoeles.[27,29] If bacteria are isolated, a six- to eight-week course of antibiotics is recommended. Gram-negative anaerobes are the most common bacteria isolated, but infections may be mixed. For this reason, antibiotic therapy with a combination of two medications is often pursued. In cases in which ultrasound-guided cholecystocentesis is not feasible, empirical antibiotic therapy should be prescribed (Table 2).[21] Use caution when prescribing antibiotics in patients with hepatic insufficiency since they may be unable to metabolize certain antibiotics appropriately.

Choleretics. Choleretics, drugs that stimulate hepatic bile excretion, should be administered (Table 2). Ursodiol (ursodeoxycholic acid) is a naturally occurring bile acid that increases bile flow by decreasing the cholesterol content of bile and thinning biliary secretions by producing bicarbonate-rich enhanced bile flow. Ursodiol is also considered hepatoprotective since it reduces the hepatotoxic effects of bile salts and protects liver cells from endogenous hydrophobic bile acids such as lithocholate and deoxycholate.[30] It is important to note that ursodiol is contraindicated in cases of extrahepatic biliary duct obstruction. Anecdotally, mucocoeles have recurred after ursodiol discontinuation.

Other hepatoprotectants. Silymarin (milk thistle) should also be instituted (Table 2). This nutraceutical alters the composition of hepatocyte membranes and limits the entry of hepatotoxins into cells.[31] It also stimulates protein synthesis and hepatic regeneration.[31] Silymarin contains flavonoids such as silybin, silydianin, and silychristin, which increase glutathione concentrations and provide antioxidant effects.[31] Inhibition of the inflammatory effects of leukotrienes is also reported.[31]

S-adenosylmethionine (SAMe) is a precursor to glutathione and has antioxidant effects (Table 2).[32] It also enhances DNA repair.[32] While studies clearly indicate that milk thistle and SAMe protect hepatocytes from damage, studies have not confirmed that either is useful in dogs with mucocoeles.[31,32]

Monitoring. Re-examine dogs undergoing medical therapy for mucocoeles after four to six weeks. The recheck should include abdominal ultrasonography, a complete blood count, and a serum chemistry profile. In one report involving two dogs, alanine aminotransferase and alkaline phosphatase activities and total bilirubin concentrations remained elevated for weeks to months after ultrasonographic resolution of the mucocele.[1] These persistent elevations are most likely associated with the extension of pathological processes from the gallbladder to the liver or from a concurrent hepatic or endocrine (e.g. hyperadrenocorticism, hypothyroidism, diabetes mellitus) condition.

Prognosis. The prognosis for patients treated medically appears to be variable. In one study, seven of 25 patients with mucocoeles were treated with ursodiol and SAMe. Two died within two weeks, two were lost to follow-up, and three survived without complications for at least six months.[12]
In a recent case study, two clinically ill patients with

mucocoeles were successfully managed with medical therapy.[1] One dog was treated with SAMe, omega-3 fatty acids, famotidine, ursodiol, and levothyroxine. The second dog received ursodiol, fenbendazole, and levothyroxine and was fed a hypo-allergenic diet. Levothyroxine was used to treat hypothyroidism, and fenbendazole was used for prophylactic gastrointestinal parasite therapy. These patients showed complete ultrasonographic resolution of the mucocele after two and three months.[1]

Surgical intervention should be recommended in all dogs that fail to improve with medical therapy, including those with unresolved clinical signs, worsening laboratory findings, and progressing ultrasonographic abnormalities.

Summary

Gallbladder mucocoeles are being diagnosed with increasing frequency in dogs, but their true incidence remains uncertain. The underlying cause of this condition is still controversial, but there is a strong association with mucous gland hyperplasia within the gallbladder epithelium. While surgical management has been the historic treatment of choice, recent case-based evidence suggests that some patients may respond to medical management.

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Surgical Management of Biliary Tract Disease

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Surgical Anatomy

The gall bladder is a pear shaped duct sitting in the right cranial quadrant of the abdomen in a fossa between the right medial and quadrate lobes of the liver. The gall bladder drains into the cystic duct, the cystic duct connects the gallbladder to the common bile duct. The hepatic ducts drain the liver lobes into the common bile duct. The common bile duct allows bile to be transported into the gall bladder for storage or allows bile to be transported into the duodenal lumen via the major duodenal papilla.(fig 1)

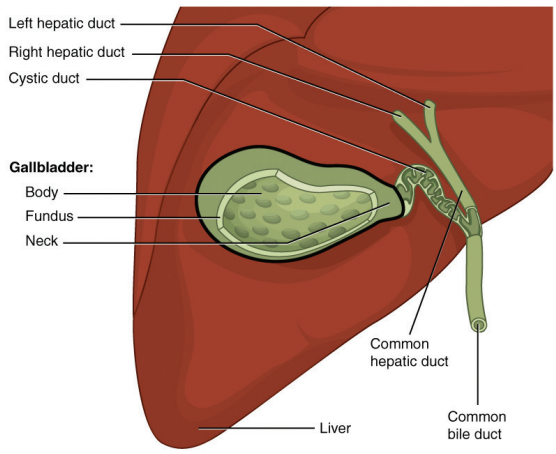

Fig 1: Anatomy of the biliary tract of the dog.

Surgery of the Biliary Tract

Many patients presented for surgery of the biliary tract are systemically compromised and require stabilisation prior to any surgery. These patients either have obstruction of the biliary tract, rupture of the biliary tract or gallbladder mucocoeles. Fluid therapy and initial stabilisation are crucial in most cases prior to surgical treatment.

Obstructive Biliary Tract Disease

Obstruction of the biliary tract from pancreatitis is a common cause and can often be managed medically on current evidence. However if the patient is deteriorating, showing progressive dissention of the biliary tract and worsening of the hyperbilirubinaemia then surgical decompression is advised. Obstruction of the biliary tract from a choleolith should be addressed surgically as soon as possible. Obstruction due to neoplastic disease carries a poor prognosis but generally needs an exploratory celiotomy to investigate, diagnose and palliate the condition.

Obstructive Biliary Disease

There are 3 main techniques for dealing with obstructive biliary disease.

1. Cholecystoenterostomy

This is the most common technique used to re-establish bile flow into the intestine. The cholecystoduodenostomy is the ideal technique as it anatomically is the most correct at re-establishing bile flow.

This technique should be used if you are unable to catheterise the common bile duct at the duodenal papilla. This is performed through a small enterotomy on the anti-mesenteric surface of the duodenum in the area of the duodenal papilla. The papilla is visualised on the internal mesenteric surface of the duodenum. A small feeding tube is then introduced into the papilla and gently advanced into the gallbladder. If this tube is able to pass into the gallbladder and bile flows through the tube choledochal stenting should be performed. If the tube cannot be passed and the obstruction cannot be removed then a cholecystoduodenostomy should be performed.

On the rare occasion that there is a choleolith in the common bile duct this can be removed via a small incision into the bile duct over the obstruction and sutured closed with fine monofilament absorbable sutures. The common bile duct should be stented to divert bile from the incision site while it heals. To perform a cholecystoduodenostomy the gallbladder is bluntly dissected out of the fossa between the respective lobes of the liver usually using gentle digital manipulation. Care must be taken not to damage the first cystic ducts that run into the common bile duct. Severe haemorrhage can be controlled by application of a haemostatic agent such as Surgiceltm or Perclottm. The freed gallbladder is placed on the enterotomy previously performed to ensure there is no tension on the intended anastomosis site. This enterotomy should be in the region of around 3-5 cm as stoma smaller than 2,5cm are predisposed to recurrent

obstructions from stricture formation. The gallbladder is now sutured to the far side of the enterotomy as the surgeon looks at the site with a continuous monofilament 3-0 or 4-0 absorbable. An incision is now made in the gallbladder to match the enterotomy. The near side of the gallbladder incision and enterotomy are now sutured in the same fashion. The area should be checked for leaks and a liver biopsy taken. Any abnormal lesions in the area of the bile duct, duodenal papilla should be biopsied to rule out neoplastic disease as a cause of the obstruction or confirm pancreatitis. Complications include haemorrhage, incisional breakdown and peritonitis, stricture of the stoma, ascending cholangitis and gastric ulceration.

2. Choledochal Stenting

This is a very simple technique that can be used if the common bile duct can be catheterised through the enterotomy. This is ideal for traumatic tears of the common bile duct, reversible obstructions or removal of choleoliths in the common bile duct. A small feeding tube is passed into the duodenal papilla. There should be good flow of bile through the tube. The largest size that will fit should be chosen but not too large to damage the papilla. The tip is cut leaving 3-4 cm of tube in the duodenum. Always place a stay suture in the end of the tube before cutting it as to not lose the end in the common bile duct. The duodenal end is now sutured to the submucosa of the duodenum to keep it in place. An absorbable monofilament should be used. The theory behind this is that once the cause has been reversed or the common bile duct has healed the suture will dissolve and pass out in the stool. The stent can be removed via endoscopy if it has not passed out in 2-3 months. The stent can lead to ascending cholangiohepatitis if not removed or passed out.

3. Cholecystostomy Tube

This diverts bile from the biliary system to an external collecting system. This can be done rapidly in compromised patients and does not alter the anatomy of the biliary system. It can only be used as a temporary solution in patients with permanent obstruction or in patients with a temporary obstructive disease. It should only be done when the gallbladder wall is considered to be healthy. A Foley's catheter is placed through the right lateral wall of the abdomen. This Foley's is then placed through a stab incision in the apex of the gallbladder. A purse string is placed around the incision and the cuff inflated of the Foley's. This is connected to a collection system externally and bile is diverted.

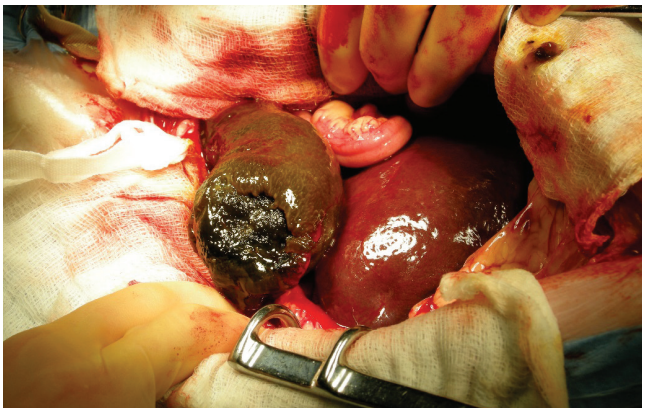

Fig 2: Ruptured gallbladder mucocoele

Gallbladder Disease

Cholecystectomy

Gallbladder mucocoele is the most common disease affecting the gallbladder. It appears to be caused by cystic mucosal hyperplasia. This can lead to extra hepatic biliary obstruction or bile peritonitis from rupture of the gallbladder. The procedure of choice is a cholecystectomy. This entails removal of the entire gallbladder. Before performing a cholecystectomy the patency of the common bile duct must be assessed. This is performed through an enterotomy as described in the previous section on cholecystoenterostomy. The gallbladder is then bluntly dissected as described until the junction of the cystic duct, gallbladder and common bile duct can be seen. The common bile duct is now ligated above the section where the cystic ducts enter and the gallbladder removed. The fossa where the gallbladder was removed is now assessed for haemorrhage, which can be controlled. The gallbladder should be submitted for histopathological examination.

Cholecystotomy

There are few indications for cholecystostomy, they include removal of choleoliths, biopsy and any procedure requiring access to the lumen of the gallbladder. This should never be performed if the gallbladder wall is unhealthy. The gallbladder has a poor suture holding ability when diseased and trying to suture can lead to leakage and peritonitis. The gallbladder is packed off with sterile swabs and an incision is made in the gallbladder. The bile should be suctioned out and the choleolith removed. The gallbladder is then sutured with a monofilament absorbable suture and the area lavaged.

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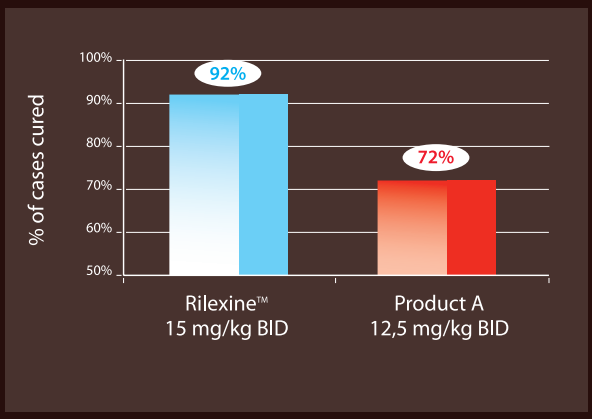

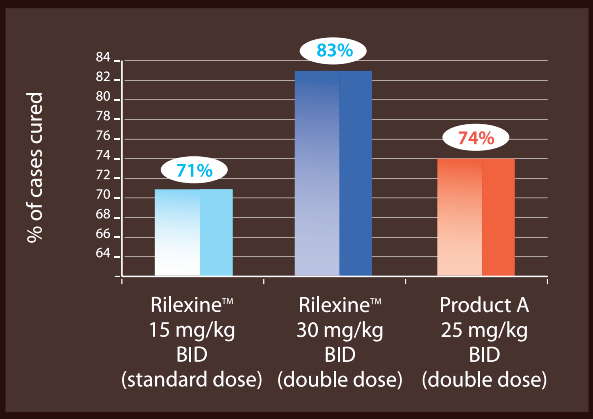

Reference1: Gauguère E. et al. Proceedings of the 3rd World Congress of Veterinary Dermatology, Edinburgh, 1996.

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