What does this weekend hold in store?

Tonight

- Some short, introductory, warm-up case scenarios
- Brief discussion about well-established sources of diagnostic error
  - to encourage 'self-awareness' and 'fly on the wall' metacognition during the rest of the weekend *(thinking about how we think; thinking about how we go about tackling problems as we tackle them).*

Tomorrow and Sunday

- Small group problem-solving on full cases (suggest groups of 6 to 8)
- Some interspersed, informal 'mini-lectures'
- 'Show and tell' sessions
- Maybe some more short cases
- Some of your cases (if you have them)

Problem-solving in Internal Medicine

Compared to experts, novices tend to...

- Misidentify or miscategorize problems
- Consider fewer differential diagnoses; *i.e.*, narrow down too soon
- Cling more tenaciously to incorrect diagnostic hypotheses, even in the face of very strong 'conflicting' data

"The process of diagnostic reasoning", Kassirer, 1989

Compared to experts, novices tend to...

Everyone jumps to conclusions

But how firmly are those 'conclusions' held?
Are they 'conclusions' or 'working diagnoses'?

"Boris has been passing red urine again Doctor, I saw it in the snow this morning, but this time he wasn't straining…"

"Yes, he was definitely posturing to urinate, not trying to pass stool…"

Compared to experts, novices tend to...

- **Misidentify or miscategorize problems**
- **Consider fewer diagnostic possibilities; *i.e.*, narrow down too soon**
- Cling more tenaciously to their diagnostic hypotheses, even in the face of very strong 'conflicting' data

"Olivia has been throwing up clear foam, Doctor… I wonder if it's her pancreas again…"

"Olivia has been throwing up clear foam, Doctor… I wonder if it's her pancreas again…"

Regurgitation *vs.* vomiting

Expectoration *vs.* vomiting / regurgitation

Compared to experts, novices tend to...

- **Misidentify or miscategorize problems**
- Consider fewer diagnostic possibilities; *i.e.*, narrow down too soon
- Cling more tenaciously to their diagnostic hypotheses, even in the face of very strong 'conflicting' data

Circa 1975

"Clearly this cat has renal failure. It's just a bit of a surprise to me that he's eating so well; I guess they don't all read the textbooks…"

When we're faced with something truly novel (or at least newly appreciated) we're all novices

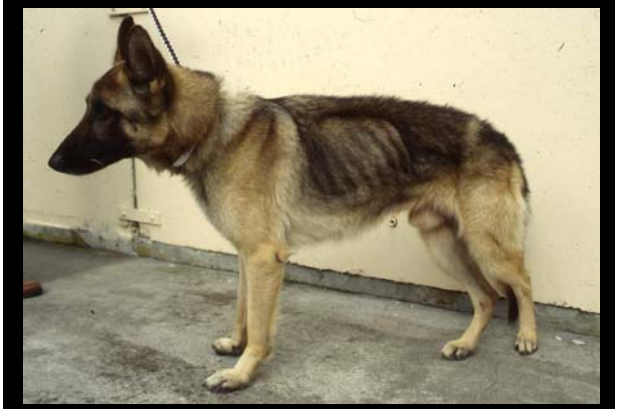
Compared to experts, novices tend to...

- Misidentify or miscategorize problems
- Consider fewer diagnostic possibilities; *i.e.*, narrow down too soon
- **Cling more tenaciously to their diagnostic hypotheses, even in the face of very strong 'conflicting' data**

Problem-orientated medicine *versus* pattern recognition

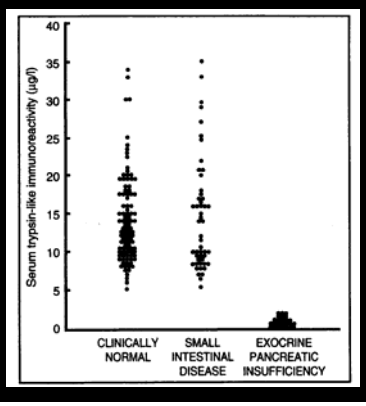
There's nothing intrinsically wrong with pattern recognition, *we all do it*.

A ravenous, young adult GSD with loose stools

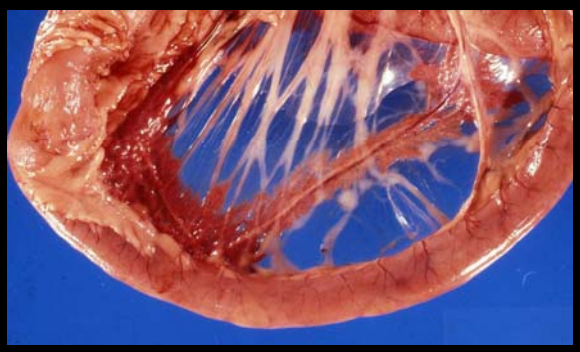


Voluminous greasy stools

TLI test (done after a 12-hour fast)



Exocrine pancreatic insufficiency



There's nothing intrinsically wrong with pattern recognition, *we all do it*.

Rupes

A skinny, 'spaced-out', Yorkshire Terrier
8 months old, male

Rupes – History

- Since the age of 4 months, Rupes has failed to thrive
- He is thinner than he should be
- His appetite is mediocre, but he drinks quite well
- 2-3 hours after eating, he often becomes 'vacant' and stares into space. Sometimes it is difficult to rouse him.

Rupes – P.E.

- Underweight, body condition score 3 or 4 /9
- Distinctly dull for a puppy of this breed and age
- TPR normal
- No abnormal auscultation or palpation findings. Abdomen feels rather empty

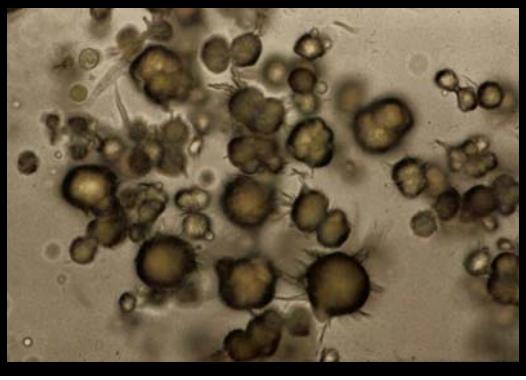
Routine blood work

- Mild, non-regenerative anaemia with slight microcytosis
- Serum albumin, urea and glucose all slightly low

Routine urine analysis

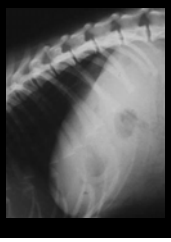
- S.G. 1.013
- Ammonium biurate crystals present in abundance on sediment examination, otherwise unremarkable

Routine urine analysis



Plain abdominal radiographs

- A tiny, 'sliver' of a liver

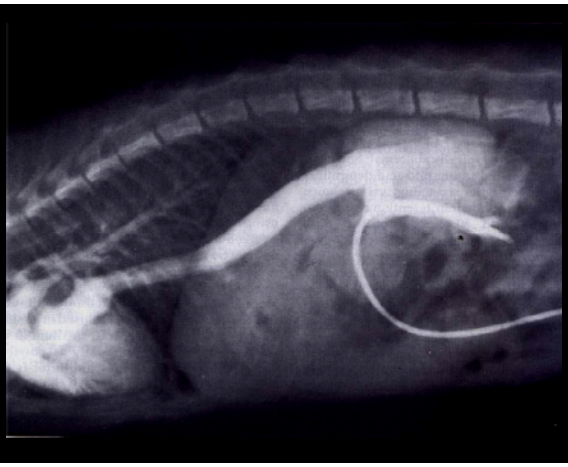


Serum bile acids

- Pre-prandial: slightly raised above normal
- 2 hours post prandial: markedly elevated

Abdominal ultrasonography

- Numerous small bladder stones seen.
  Why not seen on x-ray?
- Very small liver
- A single, large, extrahepatic portosystemic shunt with turbulent blood flow



But what to do if the pattern is, perhaps, not familiar?

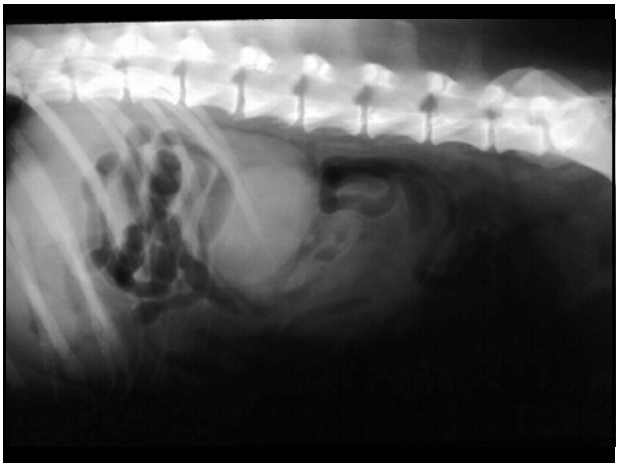
In other words, what if you can't seem to find a conclusion to jump to?

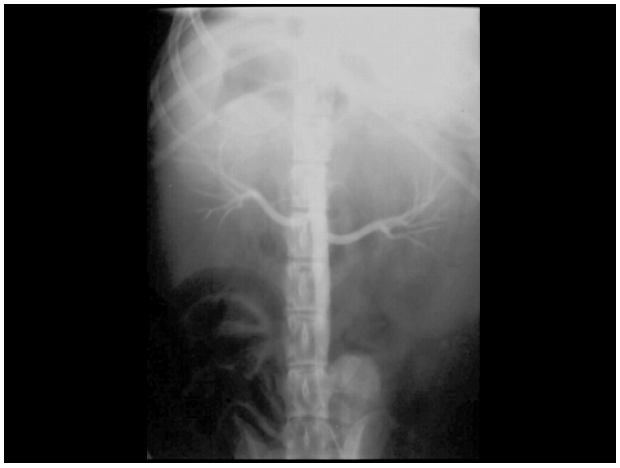
George

- 10 year-old MC black Labrador
- No previous illnesses apart from low grade elbow lameness for several years
- Seizured this morning, apparently for the first time in his life for ~ 5 minutes
- P.E. moist, warm, 'very' red mucous membranes

George

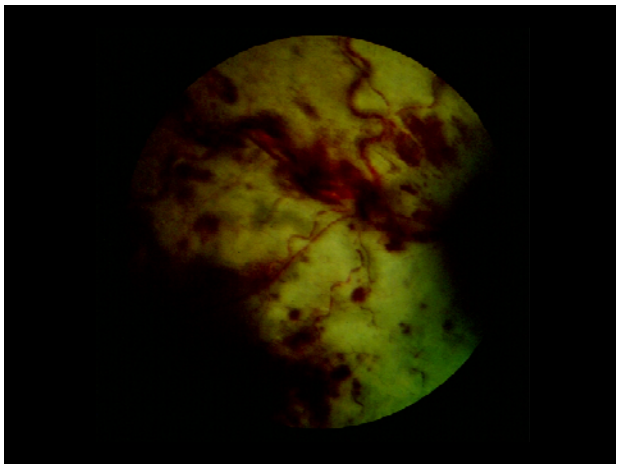
- Large palpable mass or masses in the cranial dorsal abdomen
- PCV 75 TPP 78





Why the seizuring?

- Hyperviscosity of blood
- Poor blood flow, despite high O_2 carrying capacity leads to poor tissue oxygenation
- May also see systemic hypertension, heart failure
- Hyperviscosity affects mainly brain, eye, kidneys, heart

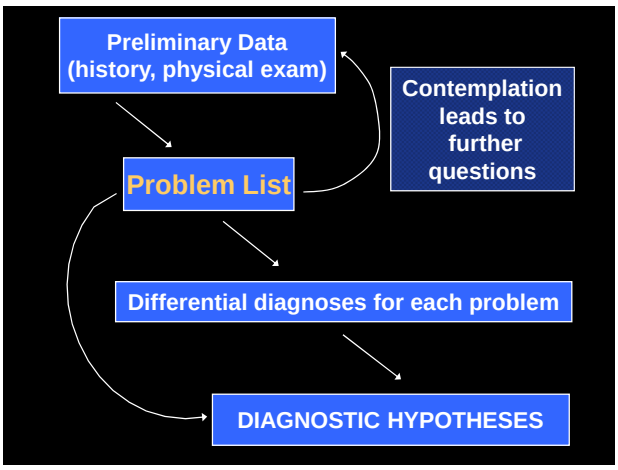


Problem-orientated medicine *versus* pattern recognition

If you don't recognize a pattern (or if you feel dubious about one) you can fall back on a problem-orientated approach.

What is a 'clinical problem'?

"Any deleterious deviation from normality, described at the level you currently understand it"



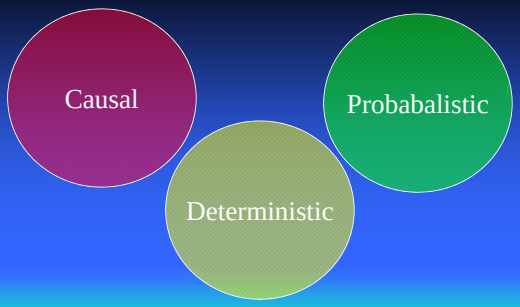
Contemplation leads to further questions

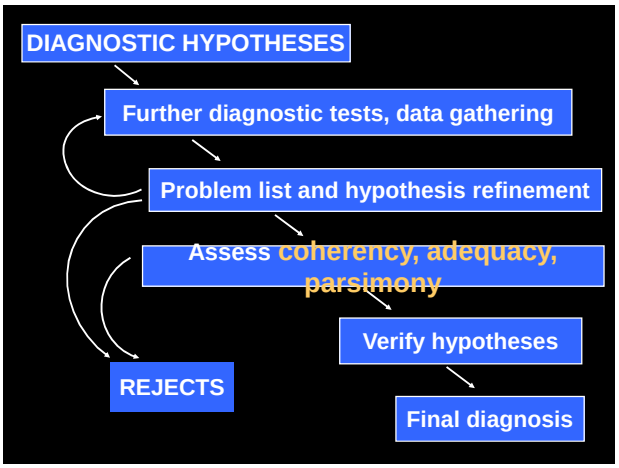
"intellectual framework"

"categories of *X*"

"packaging"

Diagnostic Reasoning





Law of Parsimony

"the assertion that no more causes or forces should be assumed than are necessary to account for the facts"

Etymology
ME f. L parsimonia, parcimonia f. parcere pars - spare

Ockham's razor

"the simplest explanation is usually the correct one"*

* coherent, adequate, parsimonious

So how do we minimize diagnostic errors?

- We don't do 'fancy' diagnostic tests until we've taken a careful history and done a thorough physical exam
- As much as possible, we have clear questions in mind when we request tests, *e.g.*, blood work, radiographs

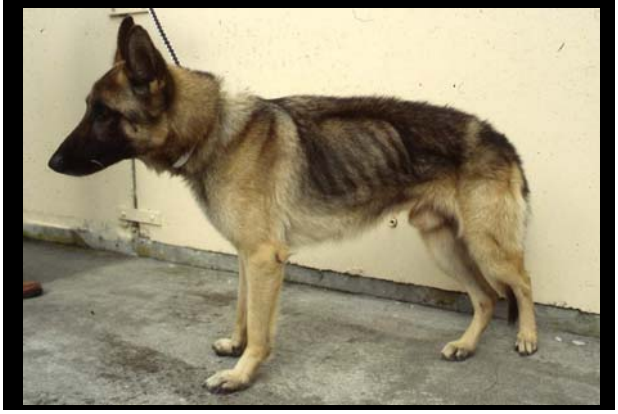
So how do we minimize diagnostic errors?

- If that doesn't work for a particularly challenging patient, we need to be *very* sceptical when we interpret those results

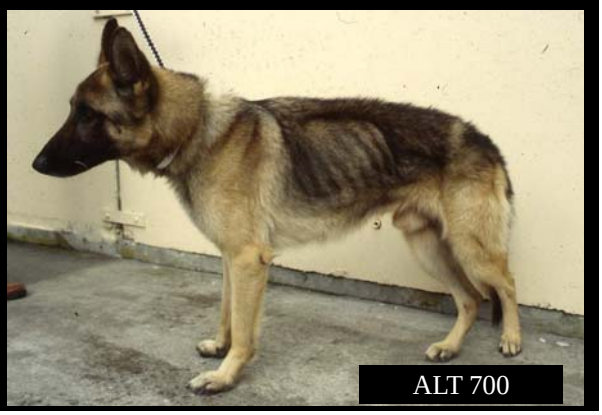
But why *not* go fishing?

- There are just too many ways for artefacts / spurious findings to appear and trick us
- Results aren't at all trustworthy until we ***breathe life into them*** by thinking about what's likely to be meaningful
- 'It's better to go hunting than to go fishing'!

A ravenous, young adult GSD with loose stools



A ravenous, young adult GSD with loose stools



Brer Rabbit and the Tar-Baby, drawing by E.W. Kemble from The Tar-Baby, by Joel Chandler Harris, 1904 Hawthorn Books, Inc.

Human cognition

- We have an innate propensity to err, miss things; we are naturally gullible
- We don't very easily transfer our understanding from one organ system or species to another *'failure of transfer'*
- ***Habit-forming*** helps us to perform much, much better

Referring Vet Consult Calls

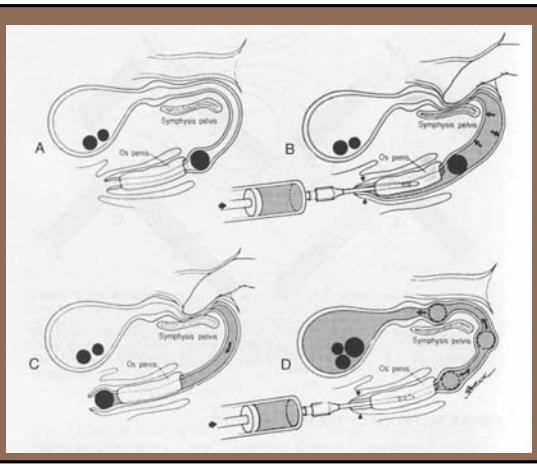
Harriet Syme
BSc BVetMed DACVIM DECVIM-CA MRCVS

2yo MN Bulldog

- ✓Urethral obstruction
- ✓Tried to pass catheter, can't dislodge the stones

Should I do a urethrostomy or should I send it in to you?

Improve CPD. 2003



Retrograde Urohydropropulsion

- ✓Reasons for failure
  - non-anaesthetised animal
  - bladder not decompressed
  - not enough 'helpers'
  - inadequate lubrication
  - inadequate urethral distention
  - rough urolith surface
  - swelling/stricture of the urethra

Improve CPD. 2003

Decompressive - Cystocentesis

- ✓22-gauge needle
- ✓extension set, 3-way tap, syringe
- ✓keep a specimen for urinalysis
- ✓insert needle into caudal bladder so near complete decompression is possible
- ✓can repeat if necessary

Improve CPD. 2003

Risks

- ✓ extravasation of urine
  - leakage of a small amount of urine is quite likely
  - of no great consequence
  - more risk if UTI
- ✓ injury to bladder wall or bladder rupture
  - avoid by good technique
  - only likely if bladder wall devitalised

Improve CPD. 2003

6yo MN Dalmatian

- ✓Cystotomy 2 weeks ago
- ✓Started on allopurinol
- ✓Came in last night with urethral obstruction AGAIN

What new treatments are available?

Improve CPD. 2003

Questions

- ✓Was the composition of the stone analysed?
  - about 95% of stones removed from Dalmatians are urate
- ✓Was post-procedural imaging performed?
  - 1 in 7 dogs and 1 in 5 cats have residual stones following cystotomy

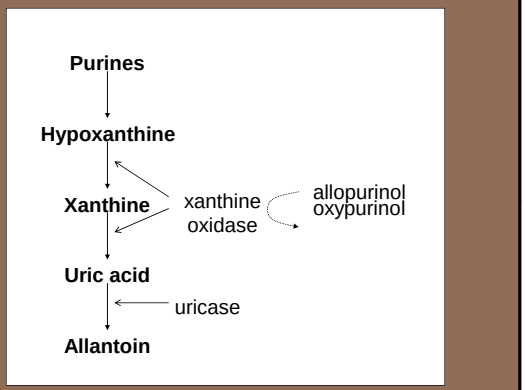
» Lulich et al. (1993) JVIM 7: 124

Improve CPD. 2003

More Questions?

- ✓Is the dog on a low protein diet?
  - Failure to restrict protein intake in dogs on allopurinol may result in xanthine stones forming

Improve CPD. 2003



Recommendations

- ✓ Double contrast cystogram or ultrasound to ensure complete removal of stones
- ✓ Send stones for quantitative analysis
- ✓ U/D diet or similar
- ✓ Allopurinol 15 mg/kg BID for dissolution, 5-10 mg/kg BID for prevention
- ✓ Check owner compliance
  - Lack of crystals - low BUN
  - Low USG - pH <7.0

Improve CPD. 2003

13y MN Yorkie

- ✓Had a cystotomy a year ago to remove calcium oxalate stones. Now it has stones again!

What diet should I feed to prevent stones reoccurring in this dog?

Improve CPD. 2003

60% recurrence rate within 3 years

Questions?

- ✓ are we sure the stones are calcium oxalate?
- ✓ have all the stones now been removed?
- ✓ are there any identifiable (potentially modifiable) risk factors for stone formation in this dog?

Improve CPD. 2003

Risk Factors

- ✓ obesity?
- ✓ acidifying diets
- ✓ diet supplements-chocolate, peanuts, vitamin C
- ✓ hyperadrenocorticism
- ✓ primary hyperparathyroidism

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Dietary Therapy

- ✓ controversial!
- ✓ recommendations are changing
- ✓ none of the commercial diets are supported by trials demonstrating efficiency in the field

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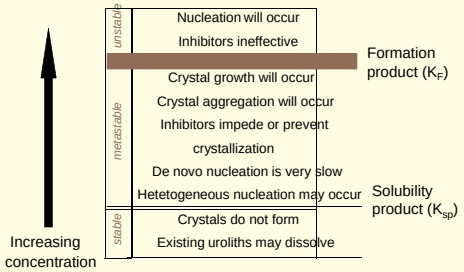

Dietary Recommendations

- ✓ increase water intake- probably least controversial and single most useful recommendation
  - evidence from epidemiological studies
- ✓ sodium supplementation?
  - previously recommended sodium restriction, since increased sodium leads to increased calcium excretion
  - but concentration may actually decrease

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Phosphorus and Protein

- ✓previous recommendations were to restrict these ~~hence feed k/d~~
  - epidemiological studies do not support this
  - dietary phosphorus restriction should be avoided, since this increases intestinal calcium absorption and hence urinary excretion
  - pyrophosphate inhibits calcium oxalate crystal formation

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Restrict Calcium

- ✓intuitive!

(reduce urinary excretion)

- ✓should not give calcium supplements between meals
- ✓dietary restriction actually increases oxalate absorption and so increases risk of stone formation

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Alkalinise Urine

- ✓acidifying diets (for struvite dissolution) increase the risk of CaOxalate stones
- ✓due to promotion of hypercalciuria
- ✓increase the pH too much and calcium phosphate stones form

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Recommendations?

- ✓ Waltham® Canine S/O Urinary Tract Support Diet
  - reduced relative supersaturation
    - » Stevenson et al (2002) 16: 377
- ✓ Hills W/D with potassium citrate
  - achieve a urine pH 6.5-7.0
  - citrate complexes calcium in urine reducing its availability and reducing CaOxalate formation

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Any Other Management Recommendations?

(-13 yo MN Yorkie with recurrent calcium oxalate stones)

- ✓can the stones be left in the bladder?
- ✓are there any non-surgical methods for removing the stones?

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Voiding Urohydropropulsion

- ✓larger stones can be passed in females, but it is possible to void small stones in males
- ✓what if it gets lodged?
  - retrograde urohydropropulsion to return stones to the bladder
  - CaOxalate not ideal as often have irregular surface

Improve CPD. 2003

How?

- ✓determine stones are small enough to try- can pass urinary catheter to estimate urethral width
- ✓GA
- ✓fill bladder with saline, suspend dog so that stones fall into the trigone and express
- ✓radiograph to ensure removal

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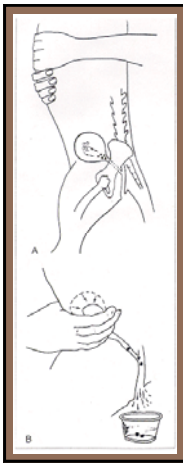

Urohydropropulsion

- ✓fill bladder with saline
- ✓position so urethra vertical
- ✓agitate
- ✓allow stones to settle
- ✓initiate voiding
- ✓continue pressure to keep brisk urine flow
- ✓3 days antibiotics

When not to…

- ✓urethral obstruction
- ✓untreated UTI
- ✓recent cystotomy

Improve CPD. 2003

Case Challenge

BRIDGET

Signalment:

An 8½ year-old female spayed English Springer Spaniel

Current Complaint:

Over the past 6 weeks, Bridget's owners have noticed that she has been drinking and urinating much more than usual. She has started to leak urine while lying down asleep. In the last week, she seems to have been having some difficulty seeing in the dark.

Bridget is up to date on her routine vaccinations. There is no history of previous illness or surgery apart from the ovariohysterectomy. She has no known allergies. Her appetite is excellent. There has been no vomiting, diarrhoea, coughing or sneezing.

Physical Examination:

T 38.3°C, P 90 (strong), R Panting
Bright, alert, well hydrated
Dilated pupils, hyper-reflective tapetal fundi, direct and consensual pupillary light responses intact.
Retinal exam: diffuse retinal atrophy, abnormally small retinal vessels.
Abdomen slightly tense on palpation. No abnormalities on rectal exam.

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION? ~ ~

~ ~ WHY IS A RECTAL EXAM PARTICULARLY IMPORTANT IN THIS CASE? ~ ~

PROBLEM LIST	DIFFERENTIALS

~~WHAT IS YOUR INITIAL PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET !~ ~

Complete blood count

Test	Patient	Reference Range
WBC	6.9	6.1 -17.4 x 10^9/L
Seg	4.899	3.0 -11.5 x 10^9/L
Band	0	0.0 - 1.0 x 10^9/L
Lymph	1.173	1.0 - 4.8 x 10^9/L
Mono	0.483	0.15 -1.35 x 10^9/L
Eos	0.345	0.1 - 1.25 x 10^9/L
Baso	0	Rare
RBC	7.51	5.5 - 8.5 x 10^{12}/L
HGB	185	120 - 180 gm/L
HCT	0.52	0.37 – 0.55 L/L
MCV	67	66 - 77 fl
MCHC	360	310 - 340 gm/L
Plasma Protein	72	50 - 75 gm/L
Platelets	Adequate	145 - 440 x 10^9/L

Serum Chemistry Profile

Test	Patient	Reference Range
SODIUM	144	145 - 158 mmol/L
POTASSIUM	3.7	3.6 - 5.5 mmol/L
CHLORIDE	112	105 - 122 mmol/L
TOTAL CO2	19	18 - 30 mmol/L
TOTAL CALCIUM	2.52	2.2 - 2.75 mmol/L
PHOSPHORUS	0.83	0.80 - 1.6 mmol/L
GLUCOSE	3.0	3.9 - 6.1 mmol/L
UREA NITROGEN	4.64	3.6 - 7.1 mmol/L
CREATININE	88.4	50 - 110 µmol/L
TOTAL SERUM PROTEIN	69	50 - 75 g/L
ALBUMIN	38	22 - 35 g/L
ALKP	670	0 - 200 U/L
CREATINE KINASE	80	0 - 460 U/L
AST	78	10 - 50 U/L
ALT	498	0 - 130 U/L
TOTAL BILIRUBIN	6.84	0 - 6.9 µmol/L
CHOLESTEROL	6.32	2.58 - 5.85 mmol/L

Urine Analysis

Source	cystocentesis
Volume	7 ml
Colour	light yellow
Turbidity	clear
S.G.	1.008
pH	8.0
Protein	negative
Glucose	negative
Ketone	negative
Bilirubin	positive
Hb	2+ (mod)
Urobilinogen	0.2

Sediment Exam

Epithelial cells	few
Crystals	few amorphous urates
RBCs	too numerous to count
WBCs	0 - 3 / hpf
Debris	large amount

Notes:

~ ~ PLEASE DO NOT TURN OVER JUST YET !~ ~

Abdominal Radiographs

Your initial interpretation: stomach appears somewhat abnormal in position, perhaps partially twisted. Increased radio-opacity in the cranial dorsal quadrant. Perhaps hepatomegaly is present.

(Radiographs will be projected during the session)

~~WHAT IS YOUR UPDATED PROBLEM LIST?~~

~~WHAT IS YOUR UPDATED PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

You are considering the possibility that Bridget's urinary incontinence is perhaps being exacerbated, or made manifest, by her polydipsia/polyuria. Given the blood test results, renal insufficiency (but not failure), liver disease or Cushing's disease are reasonable differential diagnoses. Diabetes mellitus is ruled out. In fact, the low blood glucose is a little troubling. You plan to repeat a blood glucose measurement on a fresh, appropriately-handled sample; and to carry out further investigations of liver and adrenal function.

Repeat blood glucose:

3.1 mmol/L (normal 3.9 - 6.1)

Low Dose Dexamethasone Suppression Test

Resting level	221	(normal 83 - 221 nmol/L)
4 hours post	69	(normal < 30 nmol/L)
8 hours post	83	(normal < 30 nmol/L)

Serum bile acids (pre & post prandial)*

Pre	14.7	(normal <12.25)
Post	53.9	(normal < 36.75)

* *Done by a colleague one day after an episode of bloating. Bridget was brought to the practice out-of-hours because of a distended abdomen. A stomach tube was passed and the distension was relieved easily. The next day, serum bile acids were measured. Several days later, Bridget bloated a second time. Again, a stomach tube was passed easily and the gastric distension was resolved.*

~~WHAT IS YOUR UPDATED PROBLEM LIST AND PLAN?~~

~ ~PLEASE DO NOT TURN OVER JUST YET!~ ~

Repeat LDDST:

Resting	70 nmol/L	(normal 83 - 221 nmol/L)
4 hour	63 nmol/L	(normal < 30 nmol/L)
8 hour	154 nmol/L	(normal < 30 nmol/L)

Repeat Abdominal Radiographs

An 8 cm diameter soft tissue mass is present in the cranial, dorsal, right abdomen. It displaces the stomach into an abnormal position, so that the stomach appears to be partially twisted.

Abdominal Ultrasound findings

A mass is present within the caudate lobe of the liver. It is about 10 cm in diameter. It is very close to the caudal vena cava, but does not appear to be invading that vessel. The remainder of the liver is of normal echogenicity, except for one or two hypoechoic nodules, consistent with nodular hyperplasia, or some other infiltrative process.

Diagnosis: Consider neoplastic liver mass, primary or secondary.

Chest Radiographs

Normal thorax

~~WHAT IS YOUR UPDATED PROBLEM LIST AND PLAN?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Surgical Pathological Findings

When the abdominal cavity was opened, the stomach was in an abnormal position, displaced by a liver mass. A moderately firm, 10 cm diameter, round purple mass occupied the caudate lobe of the liver. This lobe was resected with some difficulty, since it extended to the pedicle of that lobe, very close to the caudal vena cava. Resection was considered to have been incomplete. The mass was submitted for histopathological examination. The pancreas was palpated: it felt normal. The adrenal glands were inspected. A small mass was found on the right adrenal gland. This was biopsied. The left adrenal gland was normal. A gastropexy was performed. Histologically, the liver mass was reported to be a hepatoma and the adrenal mass was reported as adrenal cortical hyperplasia.

Follow up (2 weeks post op)

Urinary incontinence and polydipsia / polyuria resolved completely within days of surgery.

Glucose	6.1 mmol/L
ALKP	267 U/L
LDDST	normal suppression at 4 and 8 hours.

~~ HOW WOULD YOU MONITOR THIS CASE IN FUTURE? ~~

~~ WHAT DEVELOPMENTS MIGHT YOU PREDICT? ~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Conclusions:

Bridget's polydipsia, polyuria and hypoglycaemia were probably a consequence of her liver tumour. The retinal atrophy was considered to be an unrelated problem.

The urinary incontinence resolved once the PU/PD was controlled. In the author's practice, about half of all patients with urinary incontinence have an underlying PU/PD problem.

Bridget's failure to suppress in two LDDSTs was probably a consequence of the persistent hypoglycaemia. Hypoglycaemia is a powerful stimulus for cortisol secretion. A low dose of dexamethasone may be insufficient to suppress cortisol secretion in a hypoglycaemic patient. Arguably, the right adrenal cortical hyperplasia may have played a role in the failure to suppress in the LDDST, but it is noteworthy that the left adrenal gland was not atrophic.

Bridget's liver tumour was incompletely resected. She was monitored for tumour regrowth by abdominal ultrasound examinations every three months. After 18 months, tumour regrowth was noted on ultrasound exam. Hypoglycaemia was noted to have recurred at the same time. Despite this, Bridget remained free of any related clinical signs. A repeat laparotomy was done. The surgeon could not resect the tumour because of its location. Bridget was sent home to be fed frequently and monitored for signs of hypoglycaemia. After a further six months, she showed signs of hindlimb weakness and was found to be severely hypoglycaemic. Ultrasound examination showed that the liver tumour had not invaded or compressed the abdominal aorta or caudal vena cava. Glucocorticoid therapy was started (prednisone, 1 mg/kg bid po). Hypoglycaemia improved and the hindlimb weakness resolved. Bridget remained in apparent good health for a further 8 months. At that time, signs of hypoglycaemia recurred despite continuing glucocorticoid therapy. The owners elected euthanasia at that stage.

Summary:

- ***In this case we encountered an unusual cause of PU/PD, raised liver enzymes and failure to suppress in the low dose dexamethasone suppression test.***
- ***We saw that positive endocrine test results should be evaluated critically in light of all available clinical information.***
- ***We recognized that urinary incontinence is often exacerbated by PU/PD. Treatment of the PU/PD will often resolve such incontinence, without need for specific pharmacotherapy.***

Massive hepatocellular carcinoma in dogs: 48 cases (1992-2002).

Liptak JM, Dernell WS, Monnet E, Powers BE, Bachand AM, Kenney JG, Withrow SJ.

J Am Vet Med Assoc. 2004 Oct 15;225(8):1225-30.

Animal Cancer Center, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO 80523, USA.

OBJECTIVE: To determine clinical signs, diagnostic findings, outcome, and prognostic factors in dogs treated surgically for massive hepatocellular carcinoma (HCC) and compare survival times of surgically and conservatively treated dogs. DESIGN: Retrospective study. ANIMALS: 48 dogs. PROCEDURE: Medical records were examined for clinical signs, diagnostic and surgical findings, and postoperative outcome. Dogs were allocated into surgery and nonsurgery groups depending on whether curative-intent liver lobectomy was performed. Data from the surgical and nonsurgical groups were analyzed to identify prognostic factors and determine and compare rates of tumor control and survival time. RESULTS: 42 dogs were treated surgically, and 6 were managed conservatively. In the surgery group, intraoperative mortality rate was 4.8% with no local recurrence, metastatic rate was 4.8%, and median survival time was > 1,460 days (range, 1 to 1,460 days). High alanine aminotransferase and aspartate aminotransferase activities were associated with poor prognosis. Median survival time for the nonsurgery group was 270 days (range, 0 to 415 days), which was significantly less than that of surgically treated dogs. CONCLUSIONS AND CLINICAL RELEVANCE: Liver lobectomy is recommended for dogs with massive HCC because tumor-related mortality rate was 15.4 times higher in dogs in the nonsurgery group, compared with the surgery group. Tumor control was excellent after surgical resection with no local recurrence and a low metastatic rate. Prognostic factors were identified, but their clinical relevance was uncertain because only 9.5% of dogs in the surgery group died as a result of their disease.

Hypoglycemia associated with intra-abdominal leiomyoma and leiomyosarcoma in six dogs.

Bagley RS, Levy JK, Malarkey DE.

J Am Vet Med Assoc. 1996 Jan 1;208(1):69-71.

Department of Clinical Sciences, College of Veterinary Medicine, Washington State University, Pullman 99164-6610, USA.

Intra-abdominal leiomyoma or leiomyosarcoma was diagnosed in 6 dogs that had hypoglycemia (resting blood glucose concentration < 50 mg/dl). Tumors were large (12 to 24 cm) and arose from intra-abdominal structures including the jejunum, pylorus, duodenum, stomach, and liver. Four dogs had a leiomyoma, and 2 dogs had a leiomyosarcoma. In those dogs in which the tumor was successfully removed at surgery, blood glucose concentration returned to the reference range after tumor resection. Four dogs lived for at least 12 months after tumor resection, without redeveloping hypoglycemia.

Case Challenge

MOLLY

Signalment:

A 6½ year-old female spayed mixed breed (setter cross) dog

Current Complaint:

Molly is presented with a 3 year history of stiffness and difficulty rising from a prone position. The problem has been partially responsive to prednisolone, which was provided by another veterinarian. More recently, she has developed polydipsia and polyuria.

Molly was last vaccinated 2 years ago. She has had no previous serious illnesses. Her ovariohysterectomy was carried out uneventfully three years ago. She has no known allergies. There has been no coughing, sneezing, vomiting or diarrhoea.

Physical Examination:

T 38.9°C P 100 (strong) R 24

Bright, responsive, slightly underweight, very stiff gait
Periarticular soft tissue thickening and reduced range of motion in the carpi, tarsi and elbows.
Crepitus in the stifles, carpi, elbows.
Perhaps some joint effusion in stifles, carpi and tarsi.
Pain on extension of the hips.
On rectal, a smooth, rounded, fluctuant, 4 cm diameter mass is palpated about 10 cm cranial to the anus. It is in the midline, ventral to the rectum.

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION? ~ ~

PROBLEM LIST	DIFFERENTIALS

~~ WHAT IS YOUR INITIAL PLAN OF ACTION? ~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Complete Blood Count

Test	Patient	Reference Range
WBC	15.7	6.1 -17.4 x 10^9/L
Seg	12.55	3.0 -11.5 x 10^9/L
Band	0.1	0.0 - 1.0 x 10^9/L
Lymph	2.1	1.0 - 4.8 x 10^9/L
Mono	0.65	0.15 -1.35 x 10^9/L
Eos	0.2	0.1 - 1.25 x 10^9/L
Baso	0	Rare
RBC	6.86	5.5 - 8.5 x 10^{12}/L
HGB	181	120 - 180 gm/L
HCT	50	37 - 55 L/L
MCV	73	66 - 77 fl
MCH	26	19.9 - 24.5 pg
MCHC	360	310 - 340 gm/L
Plasma Protein	70	50 - 75 gm/L
Platelets	Adequate	145 - 440 x 10^9/L

Serum Chemistry Profile

Test	Patient	Reference Range
SODIUM	144	145 - 158 mmol/L
POTASSIUM	4.8	3.6 - 5.8 mmol/L
CHLORIDE	118	105 - 122 mmol/L
TOTAL CO2	23	18 - 30 mmol/L
TOTAL CALCIUM	2.65	2.25 - 2.95 mmol/L
PHOSPHORUS	3.29	0.80 - 1.6 mmol/L
GLUCOSE	5.44	3.9 - 6.1 mmol/L
UREA NITROGEN	36.8	3.6 - 7.1 mmol/L
CREATININE	477	50 - 110 µmol/L
TOTAL SERUM PROTEIN	71	50 - 75 g/L
ALBUMIN	24	22 - 35 g/L
ALKP	189	0 - 200 U/L
CREATINE KINASE	139	0 - 460 U/L
AST	23	10 - 50 U/L
ALT	45	0 - 130 U/L
TOTAL BILIRUBIN	3.42	0 - 6.9 µmol/L
CHOLESTEROL	9.78	2.58 - 5.85 mmol/L

Urine Analysis

Source	cystocentesis
Volume	8 ml
Colour	light yellow
Turbidity	clear
S.G.	1.011
PH	7.0
Protein	2+
Glucose	negative
Ketone	negative
Bilirubin	negative
Hb	negative
Urobilinogen	0.2

Sediment

Epithelial cells	few transitional
Crystals	few amorphous
RBCs	2 - 4 / hpf*
WBCs	0 - 2 / hpf
Debris	small amount
Bacteria	none
Casts	few hyaline

* high power field

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Skeletal Radiographs

Soft tissue swelling and marked periosteal new bone formation around all joints examined. Advanced degenerative joint disease.

Abdominal Radiographs / Ultrasound

The kidneys were slightly small, with hyperechoic cortices. A soft tissue mass is present dorsal to the neck of the bladder, ventral to the rectum/colon junction. It appears to be part of the remnant genital tract and contains fluid of mixed echogenicity. Suspect stump pyometra or cystic neoplastic mass.

~~ WHAT IS YOUR UPDATED PROBLEM LIST? ~~

~~ WHAT IS YOUR UPDATED PLAN OF ACTION? ~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Molly appears to be in chronic renal failure. On the basis of urine dipstick and sediment findings, she may have significant (glomerular) proteinuria. You decide to check a urine protein:creatinine ratio (UP:UC). You also decide to do several joint taps and submit synovial fluid for cytology and culture.

UP:UC:

6.5

Joint taps:

Cytology:

Increased cell count, a mixed population of mononuclear and polymorphonuclear leukocytes. No organisms seen. *Diagnosis*: Chronic, suppurative and mononuclear inflammation. Culture recommended.

Aerobic bacterial culture:

No growth

Repeat rectal examination:

Gentle pressure on the fluctuant mass led to passage of dark green, viscid pus from the vulva.

~ ~ CAN YOU TIE TOGETHER THE GENITAL DISEASE, JOINT DISEASE, RENAL FAILURE AND PROTEINURIA? ~ ~

Conclusions:

Molly's stiffness began abruptly three weeks after her ovariohysterectomy. She also had a fever at that time. The problem was substantially, but not completely, responsive to prednisone. She has received steroids on and off since then. One possible explanation for the observations three years later, is that she developed immune complex mediated glomerular and joint disease as a consequence of bacterial antigens leaching from a stump pyometra. Glomerular injury leads over time to glomerular loss, and eventually (when the overall glomerular filtration rate falls sufficiently) to overt renal failure. The periosteal new bone formation is a likely consequence of chronic joint inflammation.

Molly was taken to surgery for resection of the stump pyometra. Anaesthetic precautions were taken in an attempt to avoid acute exacerbation of the chronic renal failure. A renal biopsy was taken: it showed glomerulonephritis and chronic interstitial nephritis. Post operatively, Molly was managed for chronic renal failure and arthritis.

Summary:

- ***A case is presented with chronic arthritis, glomerulonephritis, renal failure and a stump pyometra. The arthritis began abruptly three weeks after the ovariohysterectomy.***
- ***Whenever immune-mediated disease (e.g., affecting joints, skin, glomeruli, or blood cells) is diagnosed; strenuous efforts should be made to identify an underlying cause.***
- ***Recent drug therapy or vaccination might provide an antigen that could contribute to the process. Infections or neoplasia are other frequently encountered sources of novel antigens that can contribute to the development of immune-mediated diseases.***

Urinary tract infection as nidus for systemic spread and septic arthritis.

Morrow M.

Can Vet J. 1999 Sep;40(9):666-8.

Ontario Veterinary College, University of Guelph.

A 12-year-old bearded collie was diagnosed with septic arthritis. The same beta-hemolytic streptococcus was cultured from the joint, blood, and urine. With arthritis, it is important to search for an inciting cause (this case, urinary tract infection) and to differentiate infectious from immune-mediated disorders, as treatment may be very different.

Type I immune-mediated polyarthritis in dogs: 39 cases (1997-2002).

Clements DN, Gear RN, Tattersall J, Carmichael S, Bennett D.

J Am Vet Med Assoc. 2004 Apr 15;224(8):1323-7.

Department of Veterinary Clinical Studies, University of Glasgow Veterinary School, Bearsden, Glasgow, Scotland, G61 1QH, UK.

OBJECTIVE: To determine clinical signs, laboratory findings, relationship to vaccination, and response to treatment for type I immune-mediated polyarthritis (IMPA) in dogs. DESIGN: Retrospective study. ANIMALS: 39 dogs PROCEDURE: Clinical records and radiographic reports from 3 university referral hospitals were reviewed. Clinical signs, laboratory and investigative findings, relationship to vaccination, and response to treatment were evaluated. RESULTS: Clinical signs and initial laboratory and clinical investigative findings were frequently abnormal but were nonspecific and not associated with likelihood of recovery. Time of vaccination was not associated with onset of disease. Chemotherapeutic immunosuppression resulted in complete cure in 56% of dogs. Continuous medication was required in 18% (7/39) of dogs, relapses were treated successfully in 13% (5/39) of dogs, and 15% (6/39) of dogs died or were euthanatized as a result of disease. CONCLUSIONS AND CLINICAL RELEVANCE: The possible involvement of vaccination in type I IMPA was not made clear from this study because of the small population size. Signalment, clinical signs, and results of diagnostic tests other than multiple synovial fluid analyses were generally nonspecific. Most dogs with type I IMPA responded to initial immunosuppressive treatment, but 31% (12/39) of dogs relapsed, required further treatment, or both.

Case Challenge

MONTY

Signalment:

A 9 year-old, male castrated, domestic shorthaired cat weighing 7.3 kg

Current Complaint:

A firm mass dorsally, between the shoulder blades

Past history:

Monty has been in his current owner's' possession since he was seven weeks old. He is a much-loved indoor/outdoor pet cat. He was castrated when he was about seven months of age. Overall, he has been remarkably free of illnesses, having suffered only a mild right stifle lameness (which lasted for about one month) about two years ago. It was managed with a non-steroidal anti-inflammatory drug.

Monty is up-to-date on his routine vaccinations and receive a flea preventative medication during the summer months.

Physical Examination:

Rectal temperature 38.2°C; pulse 190 (strong, regular); respiration 26 (normal pattern). Mild dental tartar.

The 1 x 1.5 x 2cm mass is subcutaneous, roundish, firm and non-painful. It is located dorsally, between the scapulae. It can be moved relative to the skin and, to a lesser extent, relative to the deeper tissues.

Mild crepitus is present in the right stifle, but no pain can be elicited on manipulation.

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION? ~ ~

PROBLEM LIST	DIFFERENTIALS

~ ~ WHAT IS YOUR INITIAL PLAN OF ACTION?~ ~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Complete blood count

Test	Patient	Reference Range
RBC	ND	5.5 - 10 x 10^{12}/L
HGB	111	80 - 140 gm/L
PCV	33.7	24 - 45
MCV	ND	40 - 55 fl
MCHC	329	310 - 350 gm/L
WBC	6.8	5.5 – 19.5 x10^{9}/L
Grans	4.1	2.5 – 12.5 x10^{9}/L
Lymph / Mono	2.7	1.5 – 7.0 x10^{9}/L
Platelets	412	175 - 500 x 10^{9}/L

No morphological abnormalities noted

Serum Chemistry Profile

Test	Patient	Reference Range
SODIUM	150	150 - 165 mmol/L
POTASSIUM	4.38	3.7 - 5.8 mmol/L
CHLORIDE	ND	112 - 129 mmol/L
TOTAL CO_2	ND	14 - 26 mmol/L
TOTAL CALCIUM	2.5	2.22 – 2.9 mmol/L
GLUCOSE	7.7	3.5 – 9.0 mmol/L
PHOSPHORUS	1.47	1.03 – 2.82 mmol/L
UREA NITROGEN	13.05	5.0 – 10.0 mmol/L
CREATININE	121	74 - 180 µmol/L
TOTAL PROTEIN	73.6	60 - 82 g/L
ALBUMIN	31.2	25 - 39 g/L
CREATINE KINASE	415	0 - 580 U/L
ALT	41	10 - 75 U/L
ALKP	13	0 - 90 U/L
TOTAL BILIRUBIN	0.7	0 – 3.93 µmol/L
CHOLESTEROL	2.32	1.50 – 5.1 mmol/L

Urine analysis

Not done

Fine needle aspirate of mass

The sample had a low cellularity. Many “naked” nuclei were present. The intact cells present had ovoid-elongated nuclei, fine chromatin, single nucleoli and a small amount of tapering, basophilic cytoplasm. Mild anisocytosis / anisokaryosis was noted.

Diagnostic Impression: The mesenchymal population may be neoplastic. Excision is recommended.

Chest radiographs

Nothing abnormal detected

Incisional biopsy of mass

The mass is comprised of neoplastic spindle cells, studded with small aggregates of lymphocytes. The tumour cells are arranged into irregular whorls.

Diagnosis: Schwannoma

Comment: In some areas, the mass has more of the appearance of a fibroma / fibrosarcoma, and it can sometimes be difficult to differentiate between this and a schwannoma. However, schwannomas frequently contain lymphoid aggregates.

~ ~ UPDATED PROBLEM LIST, DIFFERENTIALS AND PLAN? ~ ~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Second opinion on the biopsy

The lesion is a spindle cells sarcoma comprising interwoven bands of fibroblastic cells with variable pleomorphism and production of a light, collagenous matrix. There are multiple focal areas of infiltrative lymphoid cells, sometimes forming follicular aggregates. Occasional histiocytic cells containing granular material are present amongst the tumour cells. This lesion is typical of vaccinal sarcoma.

You telephone the pathologist to discuss the biopsy report. She thinks that the granular material in some of the histiocytes may be vaccine adjuvant. You decide to take Monty to surgery for excision of the mass. The mass appears well circumscribed and surgery seems to go well. You use India ink to mark the deep margin of the resected mass.

Histopathology of the resected mass

The tumour consists of densely packed whorls of proliferating fibroblasts, which are invading the dermis, subcutis and adjacent muscle tissue. The tumour margins are reasonably well defined but extend to involve nerves and small blood vessels. The deep margin is only two connective tissue bundles in thickness. The tumour cells are well differentiated but occasional anaplastic cells and mitotic figures are present. Mitotic rate = about 10 / 10 hpf.

Diagnosis: Fibrosarcoma

Comment: The tumour appears to have been completely resected, however the involvement of blood vessels is a concern, although no tumour emboli were observed in these sections.

~ ~ UPDATED PROBLEMS, DIFFERENTIALS, AND PLAN? ~ ~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Case progression:

Adjuvant radiation therapy was not acceptable to the owner of this cat. He went home and suffered a rupture of his right cruciate ligament 3 months later, which was managed surgically. Two months after that, the interscapular tumour was noticed to have recurred. After some weeks, he was represented to a referral centre, at which stage the tumour was 8 x 3 x 3cm. The was no evidence of regional or distant metastatic disease, so a further 'debulking' surgery was carried out. The surgeon was unable to achieve decent margins, because the tumour was so much larger and more invasive than previously. This time, the pathologist saw tumour cells extending right to the inked margin of the resected tissue. Monty went home and his surgical skin wound healed nicely, but his residual tumour continued to grow locally and eventually caused him unmanageable discomfort. He was euthanased at that stage.

Summary:

In this case we considered:

- Risk factors for development of injection-site (or vaccinal) sarcoma;
- Prevention and management of these tumours;
- Evidence for and against changing booster revaccination intervals.

Case Challenge

PUSS

Signalment:

A 7 month-old female intact domestic short haired cat, weighing 1.6 kg

Current Complaint:

For the last five days, Puss has been inappetent, depressed and has become progressively weaker.

Past history:

Puss was vaccinated against feline panleucopenia virus, feline herpesvirus type 1, and feline calicivirus as a kitten. She is an indoor/outdoor pet cat who eats a high-quality complete cat food intended for cats up to one year of age. On one occasion, about two months ago, she was found at the doorstep with bilateral epistaxis, which quickly resolved. The owners assumed that she may have been involved in a catfight.

Physical Examination:

Puss is small for her age. Although responsive to external stimuli, she is reluctant to move around. Rectal temperature, pulse rate and quality, and respiratory rate and pattern are normal. Capillary refill time and mucous membrane colour are likewise normal. You judge Puss to be normally hydrated. Puss's nose is directed down towards the tabletop; and although she can move her head sideways, she does not seem to be able to extend her neck and lift her head.

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION? ~ ~

PROBLEM LIST	DIFFERENTIALS

~~WHAT IS YOUR INITIAL PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Complete blood count

Test	Patient	Reference Range
RBC		5.5 - 10 x 10^{12}/L
HGB		80 - 140 gm/L
HCT	0.53	24 - 45 L/L
MCV		40 - 55 fl
MCH		13 - 17 pg
MCHC		310 - 350 gm/L
WBC	12.3	5.5 – 19.5 x10^9/L
Seg	5.1	2.5 – 12.5 x10^9/L
Band	0	0 - 0.3 x10^9/L
Lymph	7.0	1.5 – 7.0 x10^9/L
Mono	0	0 – 0.85 x10^9/L
Eos	0.2	0 – 1.5 x10^9/L
Baso	0	Rare

RBC & WBC morphology normal. Platelets appear adequate

Serum Chemistry Profile

SODIUM	215	150 - 165 mmol/L
POTASSIUM	4.7	3.7 - 5.8 mmol/L
CHLORIDE	129	112 - 129 mmol/L
TOTAL CO_2	14	14 - 26 mmol/L
TOTAL CALCIUM	2.9	2.22 – 2.9 mmol/L
GLUCOSE	4.8	3.5 – 9.0 mmol/L
PHOSPHORUS	2.7	1.03 – 2.82 mmol/L
UREA NITROGEN	19.3	5.0 – 10.0 mmol/L
CREATININE	212	74 - 180 µmol/L
TOTAL SERUM PROTEIN	81	60 - 82 g/L
ALBUMIN	42	25 - 39 g/L
CREATINE KINASE	479	0 - 580 U/L
ALT	37	10 - 75 U/L
ALKP	44	0 - 90 U/L
TOTAL BILIRUBIN	2.1	0 – 3.93 µmol/L
CHOLESTEROL	4.17	1.50 – 5.99 mmol/L

Urine Analysis

Source	cystocentesis
Volume	4 ml
Colour	dark brown
Turbidity	turbid
S.G.	1.069
pH	5.0
Protein	4+
Glucose	negative
Ketone	negative
Bilirubin	negative
Hb	4+

Sediment Exam

Epithelial cells	few transitional
Crystals	none seen
RBCs	too numerous to count
WBCs	
Debris	large amount
Bacteria	none seen
Casts	occasional granular

[FeLV / FIV]

Both negative

[Toxoplasma IgG titre]

Negative

Notes:

~~WHAT IS YOUR UPDATED PROBLEM LIST AND LIST OF DIFFERENTIALS?~~

~~WHAT IS YOUR UPDATED PLAN (DIAGNOSTIC, THERAPEUTIC, COMMUNICATION)?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

You consider that Puss's neck ventroflexion is most likely a manifestation of neuromuscular weakness. You remember that hypokalaemia has been associated with a non-inflammatory polymyopathy and neck ventroflexion. However, Puss is not hypokalaemic. You make a mental note to read up about the causes of neck ventroflexion in cats. Meanwhile, the extreme hypernatraemia is startling, especially since Puss does not seem clinically dehydrated. You decide to correct the hypertonicity gradually, without lowering the serum sodium concentration too rapidly.

~~FLUID TO BE INFUSED?~~

~~ROUTE OF ADMINISTRATION?~~

~~INFUSION RATE?~~

~~ MONITORING STEPS YOU PUT IN PLACE?~~

After two days of fluid therapy, Puss's serum sodium has declined to 172 mmol/L. Her strength and willingness to move have also improved substantially.

~~FURTHER DIAGNOSTIC OPTIONS AT THIS STAGE?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Electromyography

Abnormalities were detected in all muscle groups studied. There was prolonged insertional activity, fibrillation potentials, positive sharp waves, and bizarre high-frequency discharges.

Nerve conduction velocities

Normal

Muscle biopsies

Normal, as assessed by light microscopy

Puss was sent home with instructions to allow unlimited, convenient access to water. She was examined 3 days later, when she was found to be clinically normal, but once again hypernatraemic.

~ ~ FURTHER MANAGEMENT OPTIONS? ~ ~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Discussion:

Puss's hypernatraemia was diagnosed as being a consequence of hypodipsia, perhaps secondary to previous hypothalamic injury. Perhaps Puss was hit by a car on the day she returned home with bilateral epistaxis. Head trauma can certainly cause obstructive hydrocephalus and might conceivably have also injured the hypothalamic thirst centre. Puss's problems with hypernatraemia were completely resolved by feeding her canned food mixed in with sufficient water.

Hypothalamic injury might also explain Puss's small size for her age (*how?*). Cases similar to this one may manifest other kinds of adenohypophyseal dysfunction such as hypoadrenocorticism, hypothyroidism, and failure to mature sexually.

Summary:

In this case we considered:

- a range of differential diagnoses for neck ventroflexion in cats
- some different categories of dehydration
- problems that may be encountered when serum hyperosmolality is corrected too rapidly
- a very unusual consequence of (suspected) head injury

Acknowledgements:

This case is based on one owned by New Zealand veterinarian Neal Marshall. However, many of the ideas presented here are derived from a case report by Steven Dow and associates.

Hypodipsic hypernatremia and associated myopathy in a hydrocephalic cat with transient hypopituitarism.

Dow SW, Fettman MJ, LeCouteur RA, Allen TA.

J Am Vet Med Assoc. 1987 Jul 15;191(2):217-21.

Hypodipsic hypernatremia developed in association with hypopituitarism and hydrocephalus in a 7-month-old cat. Initial clinical signs (generalized weakness, cervical ventroflexion) were related to a hypernatremia-induced polymyopathy. Forced water intake and dietary sodium restriction corrected the hypernatremia and signs of muscle dysfunction. After restoration of eunatremia, secretion of pituitary hormones normalized. It was speculated that hypothalamic dysfunction, possibly related to hydrocephalus, induced both hypodipsia and transient hypopituitarism.

Case Challenge

ROSIE

Signalment:

A 5 year-old female intact Rhodesian Ridgeback.

Current complaint:

Rosie is presented to you for assessment of chronic back pain and vomiting once daily for the past 2 to 4 weeks.

Rosie is up-to-date on her routine vaccinations. She is normally fed a Hill's maintenance diet. She had her last litter of puppies two years ago and has not been seen in oestrus since then.

Rosie may have suffered a mild episode of back trauma 2 to 3 years ago, but the owner cannot remember any details.

One year ago, Rosie underwent an exploratory laparotomy for suspected pyometra because she vomited twice, manifested pain upon standing up and was noticed to have a mucoid vulvar discharge. At that time, she appeared well hydrated; PCV was 43%, TPP 100 g/L, neutrophil count was 28.3 x 10^9/L and lymphocyte count was 7.63x10^9/L. At laparotomy, no abnormalities were noted and ovariohysterectomy was not done. Oral antibiotic therapy was prescribed.

Five months later (7 months ago), Rosie was referred to a surgeon because of decreased activity, back pain, and unwillingness to go up stairs. Discospondylitis L1-2 and L2-3 was diagnosed. A Brucella titre was negative. The surgeon curetted the relevant disc spaces and submitted material for histopathology and culture. This revealed moderate hypertrophy of the annulus fibrosus and mild ossification, chondrification and mineralization of the nucleus pulposus. There was no growth on aerobic or anaerobic bacterial culture. Fungal culture reportedly grew a contaminant: *Aspergillus flavus*. No medications were provided post-operatively. Rosie was no better 2 months later.

Three months ago, Rosie was started on NSAID therapy. This helped relieve some of her discomfort.

About a month ago, vomiting began. Vomiting was not observed by the owner; it occurred about once daily and no "coffee grounds" were seen. Two to 3 weeks ago the dog became somewhat inappetent and began to visit her water bowl more often. She has lost some weight in the past month and her back pain seems to be more severe, despite the NSAID treatment.

Physical examination:

Temperature, pulse, respiration and mucous membrane colour are normal.

Arched back, tense abdomen, very painful upper lumbar spine.

Stiff hind limb gait. Reduced lumbar epaxial muscle mass. No neurological deficits noted.

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION? ~ ~

PROBLEM LIST	DIFFERENTIALS

~~WHAT IS YOUR INITIAL PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Complete blood count

Test	Patient	Reference Range
WBC	31.3	6.0 - 17.4 x10^9/L
Seg	26.6	3.0 - 11.5x10^9/L
Band	0	0 - 0.3 x109/L
Lymph	2.817	1.0 - 4.8 x10^9/L
Mono	1.878	0.15 - 1.35 x10^9/L
Eos	0	0.1 - 1.25 x10^9/L
Baso	0	Rare
RBC	6.84	5.5 - 8.5 x 10^{12}/L
HGB	148	120 - 180 gm/L
HCT	42	0.37 – 0.55 L/L
MCV	62	66 - 77 fl
MCH	22	19.9 - 24.5 pg
MCHC	350	310 - 340 gm/L
Plasma Protein	118	50 - 75 gm/L
Platelets	Adequate	145 - 440 x10^9/L

Serum Chemistry Profile

Test	Patient	Reference Range
SODIUM	149	145 - 158 mmol/L
POTASSIUM	4.0	3.6 - 5.8 mmol/L
CHLORIDE	113	105 - 122 mmol/L
TOTAL CO_2	18	18 - 30 mmol/L
TOTAL CALCIUM	2.6	2.20 - 2.58 mmol/L
PHOSPHORUS	1.13	0.8 - 1.6 mmol/L
GLUCOSE	6.9	3.9 - 6.1 mmol/L
UREA NITROGEN	3.57	3.6 - 7.1 mmol/L
CREATININE	115	50 -110 µmol/L
TOTAL SERUM PROTEIN	105	50 - 75 g/L
ALBUMIN	30	22 - 35 g/L
ALKP	107	0 - 200 IU/L
CREATINE KINASE	47	0 - 460 U/L
AST	14	10 - 50 U/L
ALT	30	0 - 130 U/L
TOTAL BILIRUBIN	5.13	0 - 6.9 µmol/L
CHOLESTEROL	4.54	2.58 - 5.85 mmol/L

Urine Analysis

Source	cystocentesis
Volume	5 ml
Colour	light yellow
Turbidity	slightly hazy
S.G.	1.009
pH	6.5
Protein	negative
Glucose	negative
Ketone	negative
Bilirubin	negative
Hb	2+
Urobilinogen	0.2

Sediment Exam

Epithelial cells	few transitional
Crystals	none
RBCs	15 – 20 / hpf*
WBCs	0 – 2 / hpf
Debris	none
Bacteria	none
Casts	rare granular

* hpf = high power field

Notes:

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Aspergillus titre

Negative

Urine & blood bacterial cultures

Negative

Intervertebral disc aspirate

Bacterial culture negative

Abdominal Ultrasound

(Ultrasound images will be projected during the session)

~~ WHAT IS YOUR UPDATED PROBLEM LIST? ~~

~~ WHAT IS YOUR UPDATED PLAN OF ACTION? ~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Progression:

Rosie was in the ICU receiving intravenous fluid therapy, metoclopramide and cephazolin pending further blood culture results. She fell over suddenly, as if seizuring. Her tongue was reported to be cyanotic. External chest resuscitative efforts were unsuccessful.

Post mortem finding

Disseminated *Aspergillus terreus* infection with death caused by a ruptured abdominal aortic aneurysm.

Summary:

- **Aspergillus terreus *can affect breeds other than German Shepherd dogs***
- ***The fungus is vasculotropic, favouring bone, kidneys and eyes***
- ***Aortic aneurysms have now been seen in several infected dogs***
- ***If an organism is reported as a contaminant, but the disease seems to be infectious, consider the possibility of mis-reporting and contact your clinical pathologist.***

Multifocal Aspergillus terreus discospondylitis in two German shepherd dogs.

Berry WL, Leisewitz AL.

J S Afr Vet Assoc. 1996 Dec;67(4):222-8.

Department of Medicine, Faculty of Veterinary Science, University of Pretoria, South Africa.

Multifocal fungal (Aspergillus terreus) discospondylitis was diagnosed in 2 German shepherd dogs. In one dog, the aetiology was established by means of fluoroscopic-guided disc aspiration, cytology and culture of disc material and urine. Disseminated aspergillosis was confirmed at necropsy and A. terreus cultured from numerous organs in this dog. The aetiology in the other dog was not established until therapeutic failure forced surgical curettage of disc material from which the fungus was cultured. Ketoconazole therapy failed to effect an improvement, and at necropsy, disease was localised to the spinal column, with A. terreus cultured from the affected discs and associated vertebrae. Immunodeficiency was suspected in both cases. In the case of disseminated disease a reduced lymphocyte blastogenic response was demonstrated. Reduced IgA was shown in both cases. The German shepherd breed seems to be predisposed to Aspergillus infections and IgA deficiency.

Mycotic aneurysm of the thoracic aorta due to Aspergillus terreus: case report and review. [IN A HUMAN]

Silva ME, Malogolowkin MH, Hall TR, Sadeghi AM, Krogstad P.

Clin Infect Dis. 2000 Nov;31(5):1144-8.

Department of Pediatrics, University of California-Los Angeles School of Medicine, Los Angeles, CA 90095, USA.

Mycotic aneurysms of the aorta caused by fungi are uncommon. We describe an unusual case of aortic aneurysm infection caused by Aspergillus terreus, which most likely spread from an adjacent pulmonary focus. Successful treatment included partial pneumonectomy, resection of the aneurysm with graft repair, and prolonged sequential administration of amphotericin B and itraconazole. A review of the published experience with aortic aneurysms caused by Aspergillus species is also presented. When invasive aspergillosis is suspected in proximity to areas with major vascular structures in immunocompromised patients, further investigation to rule out vascular invasion may be warranted. If the diagnosis is confirmed, aggressive and prompt treatment with antifungal agents combined with surgical debridement is essential to improve outcome.

Case Challenge

SAMANTHA

Signalment:

A 3 year-old female Akita

Current Complaint:

Samantha is presented to you because both of her third eyelids are protruded to the extent that they seriously impair her ability to see. She also has loud inspiratory stridor. These problems have been going on for at least a year. To some extent, both problems have been responsive to the prednisolone therapy prescribed by a colleague.

Samantha is up to date on her routine vaccinations. She receives heartworm preventative medication year round. She has no known allergies. There has been no diarrhoea or sneezing. She has been vomiting yellow liquid or partially-digested food once or twice a week for as long as the owner remembers. She coughs occasionally.

Has been off prednisolone 3.5 weeks

Physical Examination:

T 38.5°C P 60 (strong, regular) R 40 (harsh, stridorous)

Samantha was in good to obese body condition. She was unable to see until the third eyelids were moved aside. Mild enophthalmia and severe protrusion of both 3rd eyelids and mild ptosis. Slight atrophy of the muscles of mastication. Bilateral mild, mucopurulent oculonasal discharge. Laboured breathing, especially on inspiration

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION?~ ~

Neurological Examination

Unable to assess cranial nerves II, III, IV, VI.

Reduced masticatory muscle mass, reduced corneal sensation: suspect trigeminal paresis / paralysis

Absent palpebral reflex OU, ptosis, but normal mobility of ear and nostrils: suspect partial facial nerve paresis / paralysis

Reduced phonation, gag, swallow: suspect reduced function of cranial nerves IX, X

Other cranial nerves, spinal reflexes and postural reactions normal.

PROBLEM LIST	DIFFERENTIALS

~~WHAT IS YOUR INITIAL PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Complete blood count

Test	Patient	Reference Range
RBC	7.39	5.5 - 8.5 x 10^{12}/L
HGB	148	120 - 180 gm/L
HCT	0.43	0.37 – 0.55 L/L
MCV	57	66 - 77 fl
MCH	20	19.9 - 24.5 pg
MCHC	340	310 - 340 gm/L
Plasma Protein	70	50 - 75 gm/L
Platelets	Adequate	145-440 x10^{9}/L
WBC	11.7	6.0-17.4 x10^{9}/L
Seg	8.07	3.0-11.5x10^{9}/L
Band	0.12	0-0.3 x10^{9}/L
Lymph	2.57	1.0-4.8 x10^{9}/L
Mono	0.35	0.15-1.35 x10^{9}/L
Eos	0.58	0.1-1.25 x10^{9}/L
Baso	0	Rare

Serum Chemistry Profile

SODIUM	148	145 - 158 mmol/L
POTASSIUM	4.3	3.6 - 5.8 mmol/L
CHLORIDE	116	105 - 122 mmol/L
TOTAL CO_2	19	18 - 30 mmol/L
TOTAL CALCIUM	2.6	2.20-2.58 mmol/L
PHOSPHORUS	1.39	0.8-1.6 mmol/L
GLUCOSE	5.11	3.9-6.1 mmol/L
UREA	0.357	3.6-7.1 mmol/L
CREATININE	106	50-110 µmol/L
TOTAL SERUM PROTEIN	60	50 - 75 g/L
ALBUMIN	31	22 - 35 g/L
ALKP	45	0 - 200 U/L
CREATINE KINASE	58	0 - 460 U/L
ALT	51	0 - 130 U/L
TOTAL BILIRUBIN	2	0 - 6.9 µmol/L
CHOLESTEROL	3.8	2.58 - 5.85 mmol/L

Urine Analysis

Source	Cystocentesis
Volume	8 ml
Colour	Light yellow
Turbidity	Clear
S.G.	1.037
pH	7.0
Protein	neg
Glucose	neg
Ketone	neg
Bilirubin	pos
Haemoglobin	neg
Urobilinogen	0.2

Sediment Exam

Epithelial cells	none
Crystals	few triple phosphate
RBCs	0-2 / hpf
WBCs	0-1 / hpf
Debris	Small amount
Bacteria	None
Casts	None

Resting T4

21 nmol/L (normal 12–50 nmol/L)

ANA

Not done

Chest Radiographs

Prominent unstructured interstitial and bronchial lung markings throughout the lung fields. Fluid-filled oesophagus in the caudal thorax.

Findings consistent with bronchitis. Significance of fluid-filled oesophagus unknown. May be physiologic.

~~WHAT IS YOUR UPDATED PROBLEM LIST?~~

~~WHAT IS YOUR UPDATED PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

You decide to anesthetize Samantha to examine the larynx. You suspect upper airway obstruction because of the stridor. You plan to examine the vocal folds carefully for laryngeal paralysis, since this is a common cause of stridor in a large breed dog. You plan to have a surgeon ready to help relieve upper airway obstruction if necessary. You plan to do a transtracheal wash, electromyogram, cerebrospinal fluid tap, and muscle biopsies (if EMG suggests this would be appropriate) under the same anaesthetic.

CSF analysis:

Pressure appeared normal

RBCs	10 / µl	
WBCs	none seen	(normal 0 - 5 /µl)
Protein	11 mg/dl	(normal 10-30)

5 - 10 mononuclear cells seen on cytospin

No abnormalities noted

EMG:

Abnormal, excessive spontaneous electrical activity in the muscles of the larynx and in the masticatory muscles.

Muscle biopsy:

Biopsies were taken from the temporalis muscle and one of the intrinsic laryngeal muscles. Results are pending.

Nerve conduction velocities and nerve biopsy

Not done

Transtracheal wash:

Specimen contains very few cells. Those present are epithelial cells, with a few mononuclear cells and wisps of mucus.

No evidence of airway disease.

Laryngeal exam:

The larynx was examined under very light general anaesthesia. The larynx appeared symmetrical and normal in morphology. No movement of the arytenoids or vocal folds was seen on inspiration or expiration, despite vigorous inspiratory efforts.

~~WHAT IS YOUR UPDATED PROBLEM LIST?~~

~~WHAT IS YOUR UPDATED PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Laryngeal paralysis was diagnosed and Samantha was taken to surgery for lateralization of the left arytenoid cartilage. It was immediately evident post operatively that the results of surgery were unsatisfactory . Therefore, the right side was lateralized. Samantha was somewhat improved after the second procedure. She remained in the critical care unit overnight, but her stridor was worse that it had been pre-operatively by the next morning.

~~WHAT IS YOUR UPDATED PLAN AT THIS STAGE?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET!~ ~

We planned to take Samantha back to surgery for re-evaluation of her larynx and possible arytenoidectomy. Prior to this, repeat thoracic radiographs were made to check for evidence of aspiration.

Repeat thoracic radiographs:

Previously noted fluid-filled oesophagus is now replaced by an obvious gastric fundus and fluid/gas distended thoracic oesophagus, which is causing ventral deviation of the trachea.

Focal areas of alveolar disease are seen in the most ventral portion of the left cranial lung lobe.

Diagnosis:

Hiatal hernia with acute secondary megaoesophagus and aspiration pneumonia.

~~WHAT IS YOUR UPDATED PLAN AT THIS STAGE?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Because of continuing stridor, Samantha was taken to surgery for partial arytenoidectomy. At this surgery, it was evident that severe laryngeal fibrosis / ankylosis was the reason for the failure of the previous attempts at arytenoid lateralization. The constituent parts of the larynx could not be moved one relative to another. Partial arytenoidectomy was carried out uneventfully. Samantha's stridor was much improved immediately post-operatively.

~~WHAT COMPLICATIONS WOULD YOU MONITOR FOR IN THE POST-OPERATIVE PERIOD?~~

~~WHAT WOULD YOU DISCUSS WITH THE OWNER?~~

~~WHY DO YOU THINK SAMANTHA HAD LARYNGEAL FIBROSIS?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Conclusion:

Samantha was managed aggressively for suspected low grade aspiration pneumonia. She went home several days later. The masticatory muscle biopsy showed mild lymphocytic myositis. The laryngeal muscle biopsy revealed mild atrophy of type 2 fibres. We hypothesized that chronic reflux of acid gastric contents secondary to hiatal hernia was the cause of laryngeal fibrosis in this case. Hiatal hernia was also the most likely explanation of Samantha's chronic vomiting problem. The origin of the neural deficits remained unclear. Samantha was not found to be hypothyroid. The steroid responsiveness of these problems suggested an inflammatory or immune-mediated basis for the neurological disease.

Several weeks later Samantha was seen by a surgeon for correction of her hiatal hernia. Ovariohysterectomy was done at the same time as hernia repair. The surgery was uncomplicated, but immediately post-operatively Samantha developed severe pneumonia and died. Although appropriate airway protective precautions were apparently taken, necropsy revealed that aspiration pneumonia was the cause of death.

Summary:

An usual case of polyneuropathy, laryngeal fibrosis, and hiatal hernia is described.

The diagnostic approach to polyneuropathy and stridor is discussed.

Management of laryngeal paralysis and aspiration pneumonia is considered.

Esophageal hiatal hernia and megaesophagus complicating tetanus in two dogs.

Dieringer TM, Wolf AM.

J Am Vet Med Assoc. 1991 Jul 1;199(1):87-9.

Department of Small Animal Medicine and Surgery, College of Veterinary Medicine, Texas A&M University, College Station 77843.

Two dogs with tetanus developed transient megaesophagus and hiatal hernia associated with gastroesophageal reflux and regurgitation. The megaesophagus and hiatal hernia were diagnosed radiographically and resolved with resolution of the tetanus. These 2 cases, plus previously reported cases, indicate that tetanus can cause megaesophagus and esophageal dysfunction. Therefore, thoracic radiography should be included as part of the diagnostic evaluation of dogs suspected of having tetanus.

Case Challenge

"SPAN"

Signalment:

An 8 year-old female Fox Terrier weighing 8 kg.

Past history:

No previous illnesses of significance. Had 3 litters of puppies over her lifespan. May have been mated 2 weeks ago.

Current Complaint:

Reported to have developed profound depression and anorexia over 24 hours. Haemorrhagic, mucoid diarrhoea began after 6 hours.

Physical Examination:

Rectal temp 39.0°C Very tense abdomen.

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION? ~ ~

PROBLEM LIST	DIFFERENTIALS

~ ~ WHAT IS YOUR INITIAL PLAN OF ACTION? ~ ~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Haemogram (day 1)

Test	Patient	Reference Range
RBC	5.5	5.5 - 8.5 x 10^{12}/L
HGB	137	120 - 180 gm/L
PCV	0.41	0.37 - 0.55 L/L
MCV	75	66 - 77 fl
MCH	24.9	19.9 - 24.5 pg
MCHC	334	310 - 340 gm/L
Plasma protein	67.7	50 - 75 gm/L
Platelets	Adequate	145 - 440 x10^9/L
WBC	143	6.0 - 17.4 x10^9/L
Seg	124.4	3.0 - 11.5x10^9/L
Band	11.44	0.0 - 0.3 x10^9/L
Lymph	2.86	1.0 - 4.8 x10^9/L
Mono	0	0.15 - 1.35 x 10^9/L
Eos	4.29	0.1 - 1.25 x 10^9/L
Baso	0	Rare

Morphology: Some WBCs show 'toxic' changes.

Serum Chemistry Profile

Test	Patient	Reference Range
SODIUM	ND	150 - 165 mmol/L
POTASSIUM	ND	3.7 - 5.8 mmol/L
CHLORIDE	ND	112 - 129 mmol/L
TOTAL CO_2	ND	14 - 26 mmol/L
TOTAL CALCIUM	2.4	2.22 - 2.9 mmol/L
GLUCOSE	ND	3.5 - 9.0 mmol/L
PHOSPHORUS	1.08	1.03 - 2.82 mmol/L
UREA	4.5	5.0 - 10.0 mmol/L
CREATININE	67	74 - 180 µmol/L
TOTAL SERUM PROTEIN	67.7	60 - 82 g/L
ALBUMIN	31.8	25 - 39 g/L
CREATINE KINASE	338	0 - 580 U/L
ALT	22	10 - 75 U/L
ALKP	87	0 - 90 U/L
TOTAL BILIRUBIN	1.4	0 - 3.93 µmol/L
CHOLESTEROL	5.1	1.50 - 5.1 mmol/L
AMYLASE	946	350 – 920 U/L
LIPASE	345	14 - 252 U/L

From days 2 – 4, Span remained at the referring vet's practice. She received fluid therapy, enrofloxacin and cephalexin. Anorexia and mucoid, haemorrhagic diarrhoea continued, varying in severity. Intermittent vomiting began. The rectal temperature remained < 38.5°C throughout this period. Chest radiographs were taken and were interpreted as showing a small pleural effusion. Nothing abnormal was seen on abdominal radiographs.
On day 4, Span was referred. Physical examination revealed the findings previously described, plus muffled lung sounds.

~ ~ UPDATED PROBLEM LIST? ~ ~

~ ~ UPDATED PLAN?~ ~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Repeat Haemogram (day 4)

Test	Patient	Reference Range
RBC	4.6	5.5 - 8.5 x 10^{12}/L
HGB	110	120 - 180 gm/L
PCV	0.33	0.37 - 0.55 L/L
MCV	72	66 - 77 fl
MCH	23.9	19.9 - 24.5 pg
MCHC	333	310 - 340 gm/L
Plasma protein	52.1	50 - 75 gm/L
Platelets	Adequate	145 - 440 x10^9/L
Absolute Retics	9.1	20 - 60 x 10^9/L
WBC	252.0	6.0 - 17.4 x10^9/L
Seg	214.2	3.0 - 11.5x10^9/L
Band	22.68	0.0 - 0.3 x10^9/L
Lymph	7.56	1.0 - 4.8 x10^9/L
Mono	5.04	0.15 - 1.35 x 10^9/L
Eos	4.29	0.1 - 1.25 x 10^9/L
Baso	0	Rare

Morphology: Many neutrophils appear ‘toxic’. Some lymphocytes appear ‘active’

Repeat Serum Chemistry Profile (day 4)

Test	Patient	Reference Range
SODIUM	159	150 - 165 mmol/L
POTASSIUM	2.4	3.7 - 5.8 mmol/L
CHLORIDE	111	98 - 107 mmol/L
BICARBONATE	26.3	13 - 29 mmol/L
TOTAL CALCIUM	2.33	2.22 - 2.9 mmol/L
GLUCOSE	ND	3.5 - 9.0 mmol/L
PHOSPHORUS	1.45	1.03 - 2.82 mmol/L
UREA	2.8	5.0 - 10.0 mmol/L
CREATININE	55	74 - 180 µmol/L
TOTAL SERUM PROTEIN	52.1	60 - 82 g/L
ALBUMIN	22.1	25 - 39 g/L
CREATINE KINASE	1016	0 - 580 U/L
ALT	25	10 - 75 U/L
ALKP	232	0 - 90 U/L
TOTAL BILIRUBIN	11.4	0 - 3.93 µmol/L
CHOLESTEROL	4.8	1.50 - 5.1 mmol/L
AMYLASE	1976	350 – 920 U/L
LIPASE	3538	14 - 252 U/L

Urine analysis

Unremarkable. SG 1.044

Faecal culture:

Pending

Abdominal radiographs

Poor contrast / serosal detail. Suspect peritoneal fluid is present in the cranial to mid-abdomen.

Chest radiographs

Bilateral pleural effusion, especially cranially, plus a small amount of free pleural air. Suspect cranial mediastinal mass. Cranial portion of R & L cranial lobes poorly aerated / collapsed.

(Radiographs will be projected during the session)

Abdominal ultrasonography

Gastric wall thickening (muscularis). Fluid-filled small intestine and colon. Intestinal wall layers appear normal.

Pleural fluid analysis

Massive, suppurative inflammation. Neutrophils in various stages of degeneration. Strongly suspect a septic process.

FNAB of cranial mediastinal mass

Massive numbers of neutrophils in various stages of degeneration. In two areas where there are fewer neutrophils there are small numbers of large cells with eccentric, hyperchromatic nuclei, prominent nucleoli and variable quantities of basophilic cytoplasm.

Interpretation: septic inflammation of the cranial mediastinum.

Comment: because of the ragged, degenerative nature of the neutrophils, together with their sheer number, I would favour a septic process rather than a neoplastic disorder.

~ ~ UPDATED PROBLEM LIST, DIFFERENTIALS, AND PLAN? ~ ~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

The peripheral blood neutrophilia is so extreme in this case that, despite the comments of the clinical pathologist, you favour a diagnosis of paraneoplastic extreme neutrophilia rather than sepsis. After all, there is a well circumscribed mass in the cranial thorax and Span has not had a fever. Faecal culture results become available and reveal no Salmonella or Campylobacter infection. Culture of the pleural fluid is so far not growing anything. You decide to discuss the case with a surgeon.

~ ~SHOULD THIS DOG BE TAKEN SURGERY?~ ~

~ ~IF YOU DO DECIDE TO TAKE THIS DOG TO SURGERY, WHAT WOULD YOU DO IN PREPARATION?~ ~

~ ~ WHAT SHOULD YOU ASK THE SURGEON TO DO? ~ ~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Case progression:

CT revealed pleural effusion, a small, right-sided pneumothorax and the cranial mediastinal mass. Span was cross-matched and taken to surgery.

A cranial median sternotomy was done. The cranial mediastinal mass was removed along with part of the left chest wall and the left cranial lung lobe.

The histological findings were strongly reminiscent of malignant fibrous histiocytoma of humans. This tumour has been associated with extreme neutrophilia. The tissues of the tumour are themselves, often, intensely infiltrated with neutrophils.

Postoperatively, Span was managed with local and systemic analgesics and a chest tube. After several days the chest tube was removed. Span was able to leave the hospital, but unfortunately her respiratory status was judged, after a few weeks, to be incompatible with a good quality life. She was euthanased at the owner's request.

Summary:

In this case we considered:

- Causes of extreme neutrophilia, particularly paraneoplastic causes
- Challenges associated with diagnosing pancreatitis
- The unreliability of 'toxic' changes as an indicator of bacterial infection

Neutrophilic leucocytosis in a dog with a rectal tumour.

Knottenbelt CM, Simpson JW, Chandler ML.

J Small Anim Pract. 2000 Oct;41(10):457-60.

Department of Veterinary Clinical Studies, Royal School of Veterinary Studies, Easter Bush Veterinary Centre, Roslin, Midlothian.

A nine-year-old cocker spaniel was presented with a three-year history of intermittent haematochezia and a palpable rectal mass. Routine haematological examination revealed a marked mature neutrophilia (86.04 x 10(9) neutrophils/litre). A friable mass in the middle portion of the rectum was detected on colonoscopy. Histopathological examination of mucosal pinch biopsies collected from the mass confirmed a diagnosis of adenomatous tubulopapillary polyp. Some evidence of malignant transformation was observed. Palliative treatment with piroxicam suppositories at a dose of 1.4 mg/kg administered rectally every third day was instituted. On re-evaluation, 47 days after starting medical therapy, the owner reported a significant reduction in haematochezia and tenesmus; however, frequency of defecation had remained unaltered. Routine haematology revealed a reduction in the mature neutrophil count (33.67 x 10(9) neutrophils/litre). This report describes a case of a rectal tumour associated with a neutrophilic leucocytosis, which responded to palliative therapy with piroxicam suppositories.

Clinical outcome and associated diseases in dogs with leukocytosis and neutrophilia: 118 cases (1996-1998)

Lucroy MD, Madewell BR.

J Am Vet Med Assoc. 1999 Mar 15;214(6):805-7.

Veterinary Medical Teaching Hospital, School of Veterinary Medicine, University of California, Davis 95616, USA.

OBJECTIVE: To describe diseases, prognosis, and clinical outcomes associated with leukocytosis and neutrophilia in dogs. DESIGN: Retrospective study. ANIMALS: 118 dogs with leukocytosis and neutrophilia. PROCEDURE: Medical records from 1996 to 1998 were examined for dogs with WBC > or = 50,000 cells/microliter and neutrophilia > or = 50%. Signalment, absolute and differential WBC counts, body temperature, clinical or pathologic diagnosis, duration and cost of hospitalization, and survival time were reviewed. RESULTS: Mean age was 7.7 years, WBC count was 65,795 cells/microliter, and absolute neutrophil count was 53,798 cells/microliter. Mean duration of hospitalization was 7.4 days and cost of hospitalization was $2,028.00. Forty (34%) dogs were febrile, and 73 (62%) dogs died. Overall median survival time was 17 days. Dogs with neoplasia or fever were more likely to die than dogs that were hospitalized or had systemic or local infections. CLINICAL IMPLICATIONS: Leukocytosis and neutrophilia were associated with high mortality rate and have prognostic value. Given the mean duration and cost of hospitalization, frank discussion with an owner at first recognition of leukocytosis and neutrophilia may be warranted.

Case Challenge

STOOGIE

Signalment:

A 9½ year-old male castrated mixed breed dog, weighing 13.2 kg.

Current Complaint:

Over the past 18 months Stoogie's owner has noticed polydipsia / polyuria. There have been occasional episodes of constipation over the same time period. Over the past six months the owner has noticed progressive lethargy, anorexia and weight loss. There has been occasional vomiting. A colleague in your practice recently palpated a small (~0.5 cm) nodule in the right anal sac.

Stoogie is up to date with his routine vaccinations. There is no history of previous serious illness or surgery apart from the castration. He has no known allergies. There has been no coughing or sneezing. A year ago Stoogie was evaluated at another veterinary practice for seborrhoea. Blood work at that time was normal, except for the presence of hypercalcaemia (3.75 mmol/L). No further diagnostic work was done at that time.

Physical Examination:

Temperature, pulse, respiration normal.

Stoogie is rather thin and quiet. A spherical, well-circumscribed, ~7 mm diameter nodule is present in the right anal sac. The muscles of the abdominal wall seem to have less tone than is normal for a relaxed dog.

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION? ~ ~

PROBLEM LIST	DIFFERENTIALS

~~WHAT IS YOUR INITIAL PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Complete blood count

Test	Patient	Reference Range
WBC	12.3	6.0 - 17.4 x10^9/L
Seg	9.9	3.0 - 11.5x10^9/L
Band	0	0 - 0.3 x109/L
Lymph	1.9	1.0 - 4.8 x10^9/L
Mono	0.35	0.15 - 1.35 x10^9/L
Eos	0.15	0.1 - 1.25 x10^9/L
Baso	0	Rare
RBC	7.03	5.5 - 8.5 x 10^{12}/L
HGB	160	120 - 180 gm/L
HCT	0.48	0.37 – 0.55 L/L
MCV	68	66 - 77 fl
MCH	23	19.9 - 24.5 pg
MCHC	330	310 - 340 gm/L
Plasma Protein	67	50 - 75 gm/L
Platelets	Adequate	145 - 440 x10^9/L

Serum Chemistry Profile

Test	Patient	Reference Range
SODIUM	153	145 - 158 mmol/L
POTASSIUM	4.4	3.6 - 5.8 mmol/L
CHLORIDE	120	105 - 122 mmol/L
TOTAL CO_2	18	18 - 30 mmol/L
TOTAL CALCIUM	3.82	2.20 - 2.75 mmol/L
PHOSPHORUS	2.1	0.8 - 1.6 mmol/L
GLUCOSE	3.39	3.9 - 6.1 mmol/L
UREA NITROGEN	33.92	3.6 - 7.1 mmol/L
CREATININE	424	50 -110 µmol/L
TOTAL SERUM PROTEIN	67	50 - 75 g/L
ALBUMIN	30	22 - 35 g/L
ALKP	188	0 - 200 IU/L
CREATINE KINASE	456	0 - 460 U/L
AST	12	10 - 50 U/L
ALT	54	0 - 130 U/L
TOTAL BILIRUBIN	3.42	0 - 6.9 µmol/L
CHOLESTEROL	3.97	2.58 - 5.85 mmol/L

Urine Analysis

Source	cystocentesis
Volume	6 ml
Colour	light yellow
Turbidity	hazy
S.G.	1.011
pH	6.0
Protein	negative
Glucose	negative
Ketone	negative
Bilirubin	positive
Hb	2+ (moderate)
Urobilinogen	0.2

Sediment Exam

Epithelial cells	few transitional
Crystals	few amorphous
RBCs	too numerous to count
WBCs	15 - 30 / hpf
Debris	large amount
Bacteria	many rods
Casts	none seen

Notes:

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Abdominal Radiographs

These show severe nephrocalcinosis bilaterally. The kidneys are easily seen on plain radiographs of this thin dog, because they are speckled with small “snowflake-like”, mineral dense structures.

(Radiographs will be projected during the session)

Abdominal Ultrasound

Supported the radiographic findings, but did not add substantial information. The renal opacities were seen as echodense structures present throughout the renal parenchyma.

(Ultrasound images will be projected during the session)

~~WHAT IS YOUR UPDATED PROBLEM LIST?~~

~~WHAT IS YOUR UPDATED PLAN OF ACTION?~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

You are considering anal sac adenocarcinoma as a possible cause of Stoogie's hypercalcaemia. However, the mass is very small, especially if it has really been there for a year. Other likely differential diagnoses include lymphosarcoma (lymphoma), primary hyperparathyroidism and chronic renal failure. The calcium is a bit high for a case of uncomplicated chronic renal failure.

Urine culture & sensitivity:

Heavy growth of *E. coli* with a broad range of sensitivity.

PTH assay & ionized calcium:

PTH	48 pmol/L	(Normal 2 - 13 pmol/L)
Ionized Ca^{2+}	1.53 mmol/L	(Normal 1.25 - 1.4 mmol/L)

Lymph node aspirate:

Normal cytological picture

~ ~ ARE YOU CONSIDERING TAKING STOOGIE TO SURGERY? IF SO, WHAT SURGICAL PROCEDURE IS APPROPRIATE? WHAT PRE-OPERATIVE STEPS ARE APPROPRIATE?~ ~

Chest radiographs:

No sign of tumour metastases. Normal heart and lungs.

Surgical pathological findings:

A single parathyroid nodule was found and resected. Histologically, it was a parathyroid adenoma.

The right anal sac was resected without difficulty. Histologically, the mass it contained was an apocrine gland adenocarcinoma.

~ ~ WHAT POST-OPERATIVE COMPLICATIONS WOULD YOU ANTICIPATE IN THIS CASE? HOW WOULD YOU PLAN TO MANAGE THEM? ~ ~

Conclusions:

Stoogie's hypercalcaemia, ignored a year previously, was probably a real finding. There was no evidence that it was an artifact. Although he had at least three possible reasons to be hypercalcaemic, it is likely that primary hyperparathyroidism was his most long-standing problem. The anal sac adenocarcinoma may have started to grow more recently. Without measuring PTH-related protein, it is difficult to assess how much the anal sac carcinoma was contributing to the overall hypercalcaemia.

Stoogie was already in renal failure by the time he was presented. The purpose of surgery was to attempt to control the hypercalcaemia and to slow down the progression of the renal failure. Control of the urinary tract infection would also have helped in this regard.

Predictably, Stoogie developed severe hypocalcaemia in the post-operative period. He was managed with oral and intravenous calcium supplementation, oral dihydrotachysterol, intravenous calcitriol and oral hydrochlorothiazide.

He went home on post-operative day 9 and was managed for his irreversible chronic renal failure.

Summary:

- ***In this case we contemplated our diagnostic approach to polydipsia / polyuria and hypercalcaemia.***
- ***We considered the value of ionized calcium and PTH assays in the differential diagnosis of hypercalcaemia.***
- ***We anticipated post-operative problems, rather than reacting to them after manifestations became obvious.***
- ***We avoided a "blinkered" approach, and considered other possible differentials when the most obvious one (staring us in the face) did not seem to be entirely sufficient to explain the facts.***

Case Challenge

YOSHI

Signalment:

A 2 year-old female-spayed Japanese Akita

Current Complaint:

The owner complains that Yoshi has had a productive cough for five months. There has also been some sneezing and nasal discharge. The cough has gradually been getting worse over time. Several antibiotics (cephalexin, sulpha-trimethoprim, oxacillin) have been prescribed by a colleague and administered by the owner, with no apparent beneficial effect. Butorphanol and steroids have been given intermittently and they do seem to reduce the severity of the coughing. However, the owner has not been using steroids consistently.

Yoshi is up-to-date on her routine vaccinations. Peripheral blood eosinophilia has been observed intermittently. There is no history of previous illness or surgery apart from the ovariohysterectomy. Yoshi has no known allergies. There has been no vomiting or diarrhoea.

Physical Examination:

T 39°C P 70 (strong) R Panting Borderline obese

Increased expiratory effort. Spontaneous and easily elicited soft coughing. Harsh bronchial sounds. Occasional crackles in the dorsal lung fields at the peak of inspiration.

~ ~ WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK CONCERNING THE HISTORY AND PHYSICAL EXAMINATION? ~ ~

PROBLEM LIST	DIFFERENTIALS

~~ WHAT IS YOUR INITIAL PLAN OF ACTION? ~~

~ ~ PLEASE DO NOT TURN OVER JUST YET! ~ ~

Complete Blood Count

Test	Patient	Reference Range
WBC	10	6.1 -17.4 x 10^9/L
Seg	5.5	3.0 -11.5 x 10^9/L
Band	0.1	0.0 - 1.0 x 10^9/L
Lymph	3.5	1.0 - 4.8 x 10^9/L
Mono	0.4	0.15 -1.35 x 10^9/L
Eos	0.5	0.1 - 1.25 x 10^9/L
Baso	0	Rare
RBC	7.45	5.5 - 8.5 x 10^{12}/L
HGB	157	120 - 180 gm/L
HCT	0.46	0.37 – 0.55 L/L
MCV	60	66 - 77 fl
MCH	21	19.9 - 24.5 pg
MCHC	340	310 - 340 gm/L
Plasma Protein	72	50 - 75 gm/L
Platelets	Adequate	145 - 440 x 10^9/L

Thoracic Radiographs

Increased bronchial markings. No evidence of pneumonia. Improved compared with a previous referral film.

Trans-tracheal wash / B.A.L.

Cytology:

The specimen is very cellular. There are many neutrophils and macrophages present, as well as numerous columnar epithelial cells and rare mast cells. There are also many bacterial cocci (some within PMNs, mostly extracellular) and many sepatate fungal hyphae present. The background contains a few erythrocytes, a moderate amount of mucus and a few Curschman's spirals.

Chronic, active inflammation - septic due to bacterial cocci and fungal elements.

Aerobic bacterial culture and Mycoplasma culture:

No growth

Fungal culture:

Results pending

~~ WHAT IS YOUR UPDATED PROBLEM LIST? ~~

~~ WHAT IS YOUR UPDATED PLAN OF ACTION? ~~

Yoshi had been receiving prednisone during the period prior to her appointment. After the results of TTW/BAL cytology were seen, this was discontinued. Itraconazole (5 mg/kg po sid) was started pending fungal culture results. **Paecilomyces** *was eventually cultured. Yoshi's cough deteriorated over the next month. Repeat physical examination revealed marked worsening of lung sounds with prominent crackles throughout the lung fields.*

~~ WHAT IS YOUR UPDATED PLAN? ~~

Repeat radiographs:

Worsening of the bronchial disease. No evidence of fungal or bacterial pneumonia.

Repeat TTW:

Cytologically the specimen is very cellular. There are many eosinophils present, as well as many non-degenerate neutrophils, a moderate number of macrophages, a few ciliated columnar epithelial cells, and rare mast cells. The background contains abundant mucus, a few broken cells, and a few Curschman's spirals. No organisms seen.

On culture, no bacteria or fungi were grown.

~~ WHAT WOULD YOU DO AT THIS STAGE? ~~

Progression:

Eosinophilic bronchitis, complicated by *Paecilomyces* infection was diagnosed. Yoshi was sent home on aminophylline and continuation of itraconazole, pending culture results. Once culture results were available; and since there was no evidence of current *Paecilomyces* infection; prednisone was reinstituted to treat the eosinophilic bronchitis. The owner was instructed to monitor rectal temperature and respiratory pattern closely. Yoshi improved rapidly, once prednisone was reinstituted. After a second negative fungal culture, itraconazole was discontinued. Serial TTWs and BALs have shown no evidence of recurrent fungal respiratory infection. The prednisone has been weaned to 0.25 mg/kg eod. Aminophylline has been continued. Yoshi is in excellent health one year later.

Summary:

- *A challenging case of eosinophilic bronchitis complicated by fungal respiratory infection is presented.*
- *The possible primary and secondary contributions of Paecilomyces to allergic respiratory disease are discussed.*
- *The importance of monitoring for intercurrent infections in patients receiving high doses of glucocorticoids is emphasized.*
- *The confounding effects of glucocorticoids on various diagnostic tests are mentioned.*

Paecilomycosis in dogs and horses and a review of the literature.

Foley JE, Norris CR, Jang SS.

J Vet Intern Med. 2002 May-Jun;16(3):238-43.

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We reviewed 14 cases of paecilomycosis in a tertiary care veterinary hospital and all reports of the disease in the veterinary literature. Paecilomycosis is a rare disease primarily of dogs, horses, reptiles, and humans. Clinical manifestations in veterinary patients vary but include disseminated disease and diskospondylitis, particularly in dogs: pneumonia in dogs, horses, and reptiles; keratitis in horses; and miscellaneous local infections. It is important to have an appropriate index of suspicion because the diagnosis can be difficult, particularly in localized disease where it is difficult to determine whether a positive culture represents an etiology or a contamination with an environmental saprophyte. Spinal radiographs, transtracheal washes, histopathology, and fungal culture have proven to be valuable diagnostic tools. The prognosis for paecilomycosis is poor, although some treatment success has been reported, and success rates could improve if additional information were available regarding fungal species occurring in veterinary patients and drugs to which these fungi are susceptible.