Case Challenges in Abdominal Medicine

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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

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

"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

WALLACE

Signalment:

A 7 year-old male castrated domestic short-haired cat, weighing 5.8 kg

Current Complaint:

Severe vomiting, acute in onset.

Wallace was presumed to have been hit by a car when he was three years old (hiding under the bed, mild dyspnoea, broken nails), but he recovered uneventfully with basic nursing support. He is fed a high-quality dry cat food and was last vaccinated two years ago.

Physical Examination:

Rectal temperature 39.9°C; pulse 210 (strong, regular); respiration 34 (mild hyperpnoea).

Wallace seems mildly dehydrated. There is mild periodontal disease with tartar over the molar teeth. The third eyelids and oropharyngeal mucosae are noticeably icteric. Abdominal palpation reveals generalized, mild discomfort.

~ ~ 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	5.75	5.5 - 10 x 10^{12}/L
HGB	100	80 - 140 gm/L
PCV	28	24 - 45 L/L
MCV	48.7	40 - 55 fl
MCHC	357	310 - 350 gm/L
WBC	33.5	5.5 – 19.5 x10^9/L
Seg	23.2	2.5 – 12.5 x10^9/L
Band	4.0	0 - 0.3 x10^9/L
Lymph	5.6	1.5 – 7.0 x10^9/L
Mono	0.5	0 – 0.85 x10^9/L
Eos	0.2	0 – 1.5 x10^9/L
Baso	0	Rare

RBC morphology normal. Some WBCs appear ‘toxic’. Platelets appear adequate

Serum Chemistry Profile

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	ND	2.22 – 2.9 mmol/L
GLUCOSE	7.2	3.5 – 9.0 mmol/L
PHOSPHORUS	2.1	1.03 – 2.82 mmol/L
UREA	7.6	5.0 – 10.0 mmol/L
CREATININE	138	74 - 180 µmol/L
TOTAL SERUM PROTEIN	46	60 - 82 g/L
ALBUMIN	23	25 - 39 g/L
CREATINE KINASE	ND	0 - 580 U/L
ALT	260	10 - 75 U/L
ALKP	135	0 - 90 U/L
TOTAL BILIRUBIN	102	0 – 3.93 µmol/L
CHOLESTEROL	5.6	1.50 – 5.1 mmol/L
LIPASE	720	50 – 700 U/L

Urine Analysis

Source	cystocentesis
Volume	4 ml
Colour	Dark yellow
Turbidity	Clear
S.G.	1.046
pH	6.0
Protein	trace
Glucose	negative
Ketone	negative
Bilirubin	3+
Hb	negative

Sediment Exam

Epithelial cells	few
Crystals	none seen
RBCs	1-3/ hpf
WBCs	0-2 / hpf
Debris	small amount
Bacteria	none seen
Casts	occasional fine granular

* hpf = high power field

~ ~ UPDATED PROBLEM LIST? ~ ~

~ ~ UPDATED PLAN?~ ~

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

Because Wallace is not anaemic, you reason that his jaundice is more likely to be hepatic or post-hepatic rather than pre-hepatic. Even if he had been mildly anaemic, you would still have favoured liver disease, because most cats that are jaundiced because of haemolytic anaemia have severe or, at least, moderate anaemia.
You decide to arrange ultrasonographic examination of the liver to see if you can distinguish intrahepatic from post-hepatic disease. [Radiographs in this situation rarely tell you more than does careful abdominal palpation.]

Abdominal ultrasonography

A distended gallbladder with a dilated, tortuous common bile duct. 'Sludge' is present in the gallbladder. Enlarged intrahepatic bile ducts are seen. Diffuse areas of the hepatic parenchyma appear hyperechoic. A small amount of free, hypoechoic fluid is present in the peritoneal cavity.

Coagulation profile

PT	> 30 seconds	[control 16 seconds]
APTT	> 30 seconds	[control 24 seconds]

[Abdominal radiographs]

No abnormalities detected, other than a questionable, mild decrease in intra-abdominal contrast.

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

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

You carry out abdominocentesis with a fine needle on the off-chance that cytological examination of the fluid might be revealing. Examination reveals a modified transudate of low cellularity.
In view of the known association between liver disease and vitamin K-responsive haemostatic disorders, you decide to treat the cat with vitamin K_1 (2.5 mg per kg body weight subcutaneously). You plan to repeat a test of haemostatic function immediately prior to liver biopsy and give fresh frozen plasma if the coagulation test results have not normalized.
In view of the strong evidence that extrahepatic bile duct obstruction is present, you elect to take the cat the surgery rather than carrying out an ultrasound guided needle biopsy of the liver parenchyma.

Surgical findings

The pancreas is oedematous. The liver is slightly enlarged with a distended gallbladder and bile ducts. The bile is cloudy and pale. The precise cause of the extrahepatic bile duct obstruction is not determined.

~ ~ WHAT SHOULD THE SURGEON DO? ~ ~

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

Case progression:

A cholecystojejunostomy was performed. Aspirated bile and a piece of gallbladder wall were submitted for aerobic and anaerobic bacterial culture. Liver biopsies and a piece of gallbladder wall were submitted for histopathological examination and revealed a diagnosis of severe suppurative cholangiohepatitis. *E. coli* was cultured from the bile. Wallace was treated with a 4-week course of oral clavulanate-potentiated amoxicillin, to which the cultured *E. coli* was sensitive. He began to improve on the third day of antibiotic therapy. Other treatment options such as prolonged vitamin K_1 therapy, and ursodeoxycholic acid, were considered but not given.

Two years after surgery, Wallace was in good health.

Summary:

In this case we considered:

- a mental approach to feline jaundice
- differential diagnoses for feline jaundice
- the feline cholangitis / cholangiohepatitis syndrome, one of the most common causes of feline jaundice outside North America
- effects of severe liver disease on haemostasis, and how we might go about managing these
- the association between inflammatory liver disease, pancreatitis, and inflammatory bowel disease in cats.

Pathogenesis and outcome of extrahepatic biliary obstruction in cats

J Small Anim Pract 2002 Jun;43(6):247-53

Mayhew PD, Holt DE, McLear RC, Washabau RJ.
Department of Clinical Studies, School of Veterinary Medicine, University of Pennsylvania, Philadelphia 19104-6010, USA.

Extrahepatic biliary obstruction (EHBO) was confirmed at surgery or necropsy in 22 cats. Biliary or pancreatic adenocarcinoma was diagnosed by histopathology in six cats and one cat had an undiagnosed mass in the common bile duct. The remaining 15 cats had at least one of a complex of inflammatory diseases including pancreatitis, cholangiohepatitis, cholelithiasis and cholecystitis. The most common clinical signs were jaundice, anorexia, lethargy, weight loss and vomiting. Hyperbilirubinaemia was present in all cases. Distension of the common bile duct and gall bladder was the most commonly observed finding on abdominal ultrasound. Nineteen cats underwent exploratory laparotomy for biliary decompression and diversion. Mortality in cats with underlying neoplasia was 100 per cent and, in those with non-neoplastic lesions, was 40 per cent. Long-term complications, in those that survived, included recurrence of cholangiohepatitis, chronic weight loss and recurrence of obstruction. Based on these findings, the prognosis for EHBO in cats must be considered guarded.

Case Challenge

Chooba

Signalment:

An 6 year old female spayed Crossbreed

History:

Chooba presented to the referring veterinary surgeon with a 24 hour history of anorexia and lethargy. She had also vomited once in the preceding 24 hours.

Chooba had been intermittently unwell for the previous three months with “good and bad days” however her owners had not sought veterinary attention until this point. Her owners felt she may also have been drinking a little more over the same time frame. They did not feel she had lost any weight and she had not previously exhibited any vomiting or diarrhoea. She was fully vaccinated and had been spayed as a young dog.

Physical examination – referring veterinary surgeon

She was reported as being depressed with a subnormal temperature and a mildly uncomfortable abdomen. There was no report of the findings of cardiovascular or respiratory system examination.

WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK REGARDING HISTORY AND PHYSICAL EXAMINATION?

Problem list	Differential diagnosis

Plan???

Please do not turn over yet!!

Haematology

Hct	0.56	
Hb	18.4 g/dL	
RBC	7.26 x 10^{12}/L	
MCV	77 fL	
MCHC	32.9 g/dL	
Total wbc	15.7 x 10^{9}/L	Raised
Neutrophils, segmented	11.1 x 10^{9}/L	
Lymphocytes	3.5 x 10^{9}/L	
Monocytes	6 x 10^{9}/L	Raised
Eosinophils	0.2 x 10^{9}/L	
Basophils	0	

Smear examination: Platelets adequate and some clumps

Biochemistry:

Albumin	29 g/L	
Globulin	45 g/L	
ALP	62 U/L	
ALT	68 U/L	
Tbilirubin	1 μmol/L	
Total calcium	2.7 mmol/L	
Phosphorus	2.44 mmol/L	
BUN	30 mmol/L	Raised
Creatinine	244 μmol/L	Raised
Glucose	3.2 mmol/L	Low
Na	124 mmol/L	Low
K	6.8 mmol/L	Raised

Interpretation?

Further diagnostics?

Treatment plan?

Please do not turn over yet!!

Chooba was started on intravenous fluids of 0.18%NaCl + 4% dextrose at 4ml/kg/hr and dexamethasone was administered intravenously at an unknown dose.

A cortisol level was requested n the previously submitted bloods and results available the following day revealed it to be <28 nmol/L.

Unfortunately Chooba deteriorated overnight and by the following morning was much more depressed to the point where she was unable to stand. She was also reported to be twitching. At this point she was referred for further intensive care. During the journey (approx 2hours) her neurological status deteriorated further such that she was obtunded by the time of arrival.

Evaluation on arrival at referral centre

Cardiovascular: HR 100bpm with good quality pulses. Mucous membranes were pink with a 1.5s CRT.

Respiratory: Unremarkable.

Neurological: Obtunded and weak but no specific abnormalities detected on cranial nerve examination or limb reflexes.

Abdominal palpation: Unremarkable on palpation

Temperature: 37.9ºC

A purulent vaginal discharge was noted. Choob appeared to be moderately dehydrated and also seemed underweight. No other abnormalities were found.

What is your evaluation?

Why has Chooba deteriorated?

Plan?

Please do not turn over yet!!

Venous blood gas

PCV	42%	37-55%
TS	48	55-75
pH	7.466	7.35-7.45
$PvCO_2$	29.5 mmHg	35-55 mmHg
PvO_2	57.3 mmHg	
HCO_3^-	21.5 mol/L	18-24 mmol/L
BE	-2.5	-4 - +4
Na	112.8 mmol/L	140-155 mmol/L
K	3.48 mmol/L	3.5-4.5 mmol/L
Cl	83.5 mmol/L	110-120 mmol/L
iCa	1.04 mmol/L	1.13-1.33 mmol/L
Lactate	0.8 mmol/L	0.5-2.5 mmol/L

Repeat Biochemistry:

Albumin	26.1 g/L	28-39 g/L
Globulin	20.4 g/L	21-41 g/L
ALP	33 U/L	19-285 U/L
ALT	84 U/L	13-88 U/L
Tbilirubin	2.9 μmol/L	0-2.4 μmol/L
Total calcium	1.82 mmol/L	2.13-2.7 mmol/L
Phosphorus	1.64 mmol/L	0.8-2 mmol/L
BUN	4.5 mmol/L	3-9.1 mmol/L
Creatinine	68 μmol/L	98-163 μmol/L
Amylase	482 U/L	176-1245
Lipase	209 U/L	72-1115

Repeat haematology:

Hct	41.9%	37-55
Hb	13.5 g/dL	12-18
RBC	6.07 x 10^{12}/L	5.5-8.5
MCV	69 fL	60-77
MCHC	32.3 g/dL	31-37
Total wbc	13.2 x 10^9/L	6-17.1
Neutrophil, segmented	10.69 x 10^9/L	3-11.5
Eosinophils	0	0-1.3
Basophils	0	0
Lymphocytes	0.66 x 10^9/L	1-4.8
Monocytes	1.85 x 10^9/L	0.15-1.5
Platelets	199 x 10^9/L	150-800

Revised problem list	Differential diagnosis

Ongoing treatment?

Further diagnostics?

Please do not turn over yet!!

Thoracic radiographs

Unremarkable

Abdominal ultrasound

No free abdominal fluid. Liver and both kidneys appeared within normal limits. Spleen has a single hypoechoic nodule in body. Bladder contains echogenic material. The vagina appears dilated and a single uterine horn is visible containing echogenic fluid.

Urinalysis (cysto)

SG	1.020
pH	8
Blood	+++
Protein	++
Glucose	Neg
Ketones	Neg
Sediment	Transitional epithelium and squamous epithelial cells coated in Gram positive cocci
Culture	Submitted

Vaginal discharge

Cytology	Large number of degenerate neutrophils and epithelial cells containing and coated by Gram positive cocci
Culture	Submitted

Faecal

Negative for parasites

Plan?

Please do not turn over yet!!

Treatment

Chooba was started on Hartmanns (131mmol/L Na) at 4ml/kg/hr. She was also started on amoxicillin/clavulanate intravenously at 20mg/kg three times daily and received 0.1 mg/kg dexamathasone IV.

At 7.30pm (6 hours after admission) she had a short seizure.

Venous blood gas and electrolytes at that time showed

PCV	38%	37-55%
TS	45 g/L	60-75 g/L
pH	7.282	7.35-7.45
$PvCO_2$	41 mmHg	35-55 mmHg
PvO_2	62.3 mmHg	
HCO_3^-	19.6 mol/L	18-24 mmol/L
BE	-7.4	-4 - +4
Na	120.3 mmol/L	140-155 mmol/L
K	3.6 mmol/L	3.5-4.5 mmol/L
Cl	94 mmol/L	110-120 mmol/L
iCa	1.12 mmol/L	1.13-1.33 mmol/L
Lactate	8.3 mmol/L	0.5-2.5 mmol/L

Interpretation??

Change in treatment plan?

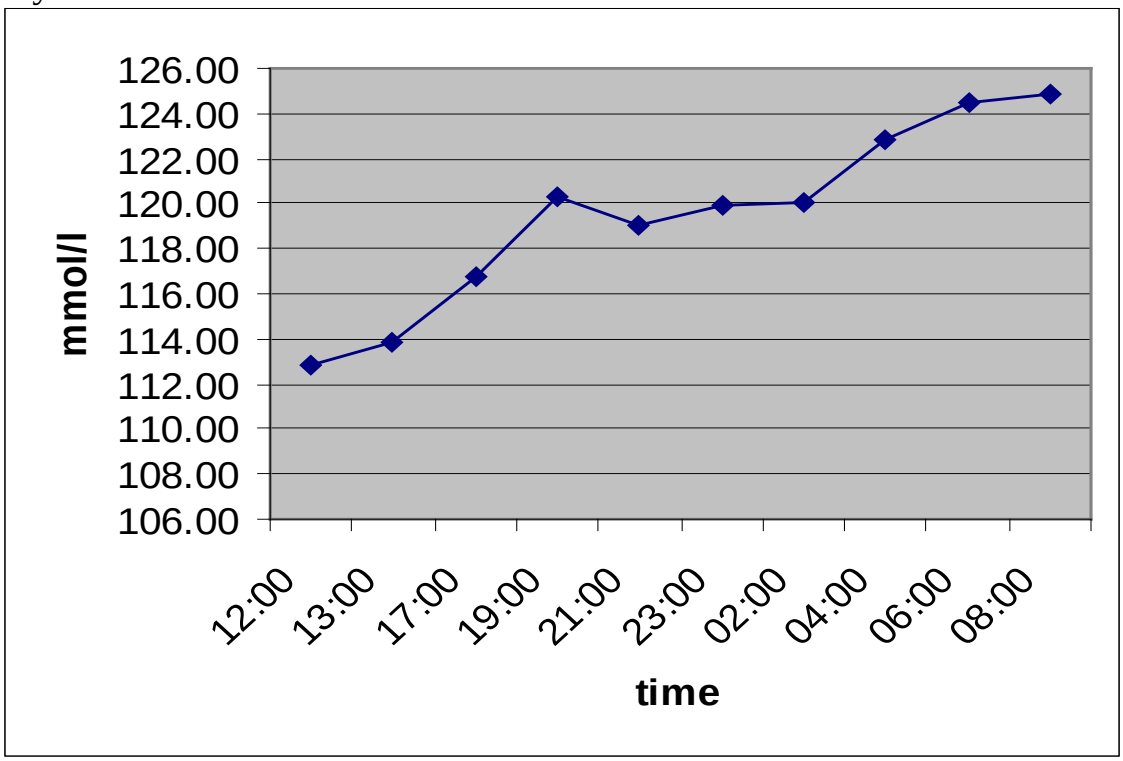
Please do not turn over yet

Progression

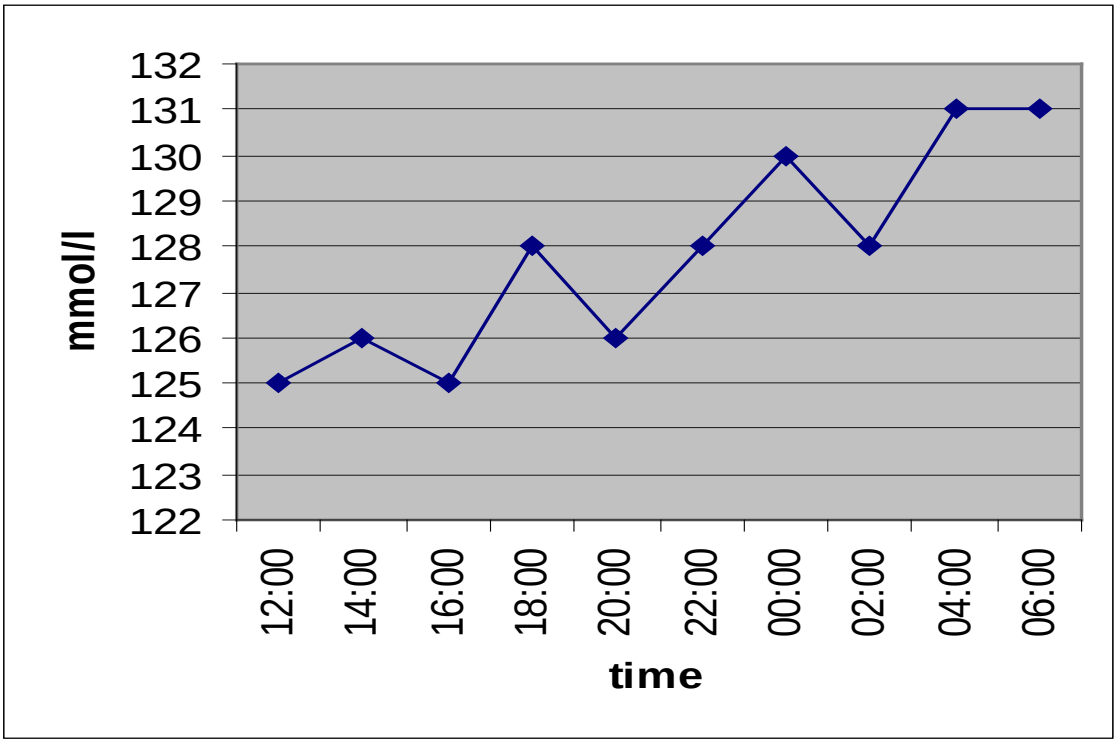
Overnight Chooba received 0.45% NaCl at 4 ml/kg/hr and by 6am Na had reached 124 mmol/L

At that point fluid therapy was changed to 0.45%NaCl at 2 ml/kg/hr.

Day 1



Day 2



At noon on day 2, fluid therapy was switched to Hartmanns at 2 ml/kg/hr and this was reduced to 1 ml/kg/hr at midnight.

Fludrocortisone was introduced on day 2 at 0.01mg/kg PO once daily.

Chooba's mentation improved steadily such that she was able to stand by the end of day 2.

A full neurological examination at that time showed

- Tetraparesis with right worse than left
- Right sided proprioception deficits
- Blindness in right eye

On day 3, there continued to be an improvement in Chooba's neurological state and she started to eat voluntarily.

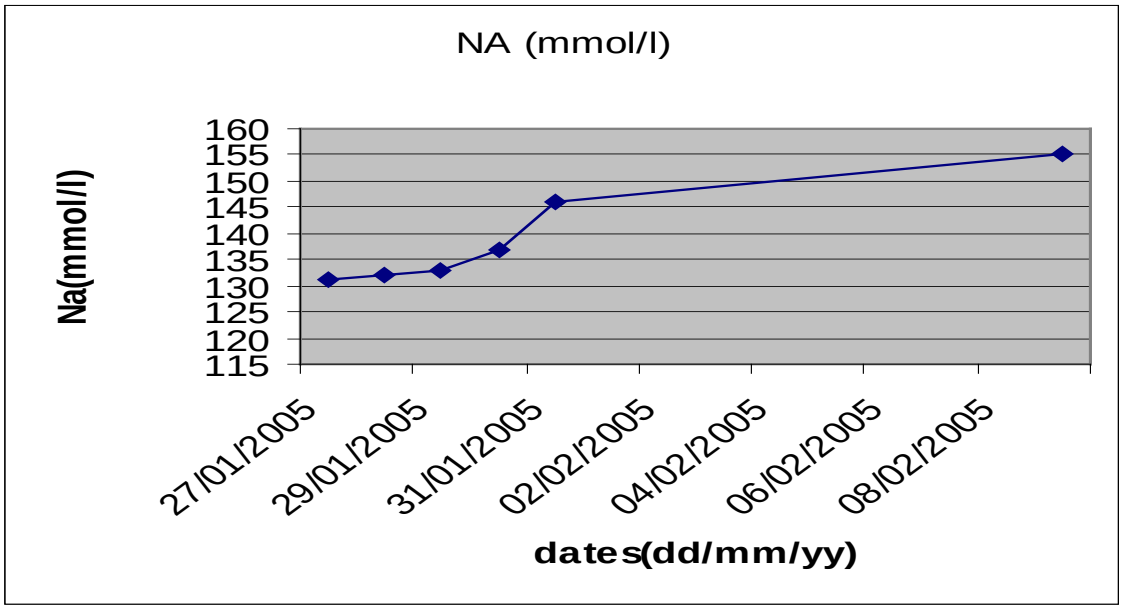
Urine and vaginal swab culture results were received and both yielded a growth of *Staphylococcus intermedius* sensitive to amoxicillin/clavulanate.

Further diagnostic evaluation?

Further treatment?

Please do not turn over yet!!

Chooba continued to make steady progress. A repeat abdominal ultrasound on day 4 did not show any abnormalities.The enlarged uterine horn could not be identified. Blood work was also repeated on day 4 – the biochemistry panel was unremarkable. The haematology panel showed a mild anaemia (hct 30%) and an inflammatory leukogram with 19.9 x 10^9/L with 1% bands. The vaginal discharge also resolved.



Chooba was discharged on day 8 on fludrocortisone (0.1mg PO sid), amoxicillin/clavuanate (250mg PO bid) and prednisolone (5mg PO sid).
Her appetite was good and her demeanour and strength were continuing to improve.

She was re-examined 7 days following discharge. Her owners reported her to be very well at home and still making steady progress. Her neurological examination was now normal and there was no vaginal discharge.
Haematology was all within normal limits. An electrolyte panel revealed a sodium of 155 mmol/L and a potassium of 4.6 mmol/L – there were no other abnormalities on the biochemistry screen.
Abdominal ultrasound was unremarkable. Cystocentesis showed urine with a SG of 1.015 and pH8 but no blood or protein. Urine culture was negative.
She was discharged for conti ued management by her own veterinary surgeon. A telephone follow-up 6 months after her initial presentation showed her to be doing very well and receiving fludrocortisone only.

Case summary

This case highlights

- The severe physiological consequences of rapidly changing serum sodium concentrations
- The concerns regarding use of hypotonic fluids
- The diagnosis and treatment of hypoadrenocorticism

Relevant papers

Brady CA, Vite CH, Drobatz KJ (1999) Severe neurologic sequelae in a dog after treatment of hypoadrenal crisis. J Am Vet Med Assoc **215**:222-5, 210.

A 3-year-old mixed-breed dog was evaluated for lethargy, weakness, anorexia, and vomiting. The dog was dehydrated, hyponatremic, hypochloremic, and hypoglycemic. Results of an ACTH stimulation test indicated hypoadrenocorticism. Treatment to restore cardiovascular stability and serum electrolyte balance caused serum sodium concentration to increase by 32 mEq/L within 48 hours, and the dog developed severe neurologic signs that persisted for approximately 3 weeks. Magnetic resonance imaging revealed cerebrocortical lesions on day 6 and more severe lesions, including diffuse atrophy of the cerebral hemispheres, at 23 weeks after initial evaluation; however, the dog recovered complete neurologic function. Serum sodium concentration should be monitored during treatment for hypoadrenal crisis to avoid rapid increases that can cause CNS damage.

Peterson ME, Kintzer PP, Kass PH. (1996) Pretreatment clinical and laboratory findings in dogs with hypoadrenocorticism: 225 cases (1979-1993). J Am Vet Med Assoc **208**:85-91.

OBJECTIVE--To evaluate clinical and laboratory findings in 225 dogs with naturally occurring hypoadrenocorticism diagnosed over a 14-year period. DESIGN--Retrospective case series. ANIMALS--220 dogs with primary hypoadrenocorticism and 5 dogs with secondary hypoadrenocorticism (primary ACTH deficiency). PROCEDURE--We reviewed medical records of all dogs with naturally occurring hypoadrenocorticism examined at The Animal Medical Center between 1979 and 1993 or at Tufts University, Foster Hospital for Small Animals, between 1987 and 1993. RESULTS--Dogs ranged from 4 months to 14 years old. Most (71%) were female, and female dogs had a significantly higher relative risk of developing hypoadrenocorticism than did males. Great Danes, Portuguese Water Dogs, Rottweilers, Standard Poodles, West Highland White Terriers, and Wheaton Terriers had a significantly higher relative risk of developing hypoadrenocorticism than did dogs of other breeds. Common owner complaints included lethargy, poor appetite, and vomiting, whereas lethargy, weakness, and dehydration were common abnormalities detected on physical examination. Serum

biochemical testing at the time of diagnosis revealed moderate-to-severe azotemia and hyperphosphatemia in most dogs. In 99 of 172 (57.6%) dogs that had a pretreatment urinalysis performed, urine specific gravity was < 1.030 even though dogs were azotemic. Serum electrolyte changes included hyperkalemia (n = 215), hyponatremia (183), hypochloremia (94), and hypercalcemia (69). Five of the 220 dogs with primary hypoadrenocorticism and the 5 dogs with secondary hypoadrenocorticism did not have hyperkalemia at time of diagnosis. In all dogs, ACTH stimulation testing revealed a low to low-normal baseline serum cortisol concentration with little to no rise after ACTH administration. Endogenous plasma ACTH concentration measured in 35 dogs with primary hypoadrenocorticism was markedly high; whereas ACTH concentration was undetectable to low in the 5 dogs with secondary hypoadrenocorticism. CLINICAL IMPLICATIONS-- hypoadrenocorticism is a rare disease in dogs, most commonly affecting young to middle-aged females; some breeds are at greater risk of developing the disease than others. In general, clinical signs are nonspecific and similar to manifestations of more common diseases. Serum electrolyte disturbances of hyperkalemia and hypernatremia are characteristic in dogs with primary hypoadrenocorticism, but concentrations may be normal in dogs with early or mild primary or secondary hypoadrenocorticism. Diagnosis of hypoadrenocorticism is best confirmed by demonstration of a low baseline serum cortisol concentration with a subnormal or negligible response to ACTH administration. Determination of endogenous plasma ACTH concentrations is valuable in differentiating primary from secondary hypoadrenocorticism, particularly in dogs with normal serum electrolyte concentrations.

Case Challenge

Holly

Signalment:
A 10 year old female spayed Rough Collie

History:
Holly is a previously healthy dog who presents to you having been found collapsed in the kitchen when her owners return from a day out. She has been alone since about 10am (it is now 6pm) but had seemed well that morning. She is still responsive to her owners but unable to stand.

She is fully vaccinated. She has been becoming a little slower on her walks over the previous 6-12 months and receives occasional oral meloxicam therapy for presumed hindlimb osteoarthritis. She last received meloxicam three days prior to this presentation.

Physical examination – major body systems

Cardiovascular system: HR 180 beats per minute with weak femoral pulses. Mucous membranes are bright pink with a CRT of 2.5s

Respiratory system: Tachypnoeic, however lung fields sound within normal limits

Neurological system: Quiet but responsive. Unable to stand unaided but proprioception and reflexes seem unremarkable when she is supported.

Abdominal palpation: Appears painful especially in mid-dorsal abdomen, however no specific masses or fluid thrill palpable

Both the abdomen palpation and neurological exam should be interpreted with the knowledge that Holly is obese and you feel the examination is compromised by this.

Temperature: 40.2ºC

WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK REGARDING HISTORY AND PHYSICAL EXAMINATION?

Problem list	Differential diagnosis

Initial plan?

Please do not turn over yet!!

Initial database

PCV	55%	37-55%
TS	80 g/L	60-75 g/L
pH	7.27	7.35-7.45
$PvCO_2$	28 mmHg	35-55 mmHg
PvO_2	54 mmHg	
HCO_3^-	12 mol/L	18-24 mmol/L
BE	-8	-4 - +4
Na	143 mmol/L	140-155 mmol/L
K	4.8 mmol/L	3.5-4.5 mmol/L
Cl	112 mmol/L	110-120 mmol/L
iCa	1.27 mmol/L	1.13-1.33 mmol/L
Lactate	4.3 mmol/L	0.5-2.5 mmol/L

Blood smear evaluation;

Red blood cell morphology appeared normal. Platelet count adequate. Neutrophil count moderately increased with 4% band cells present.

In house coagulation:

PT 13s (reference 12-17s)
PTT 112s (reference 70-102s)

Biochemistry (vetscan):

Albumin	26 g/L	25-44 g/L
Globulin	34 g/L	23-52 g/L
ALP	113 U/L	20-150 U/L
ALT	86 U/L	10-118 U/L
Amylase	1003 U/L	200-1200 U/L
Tbilirubin	5 μmol/L	2-10 μmol/L
Total calcium	2.20 mmol/L	2.15-2.95 mmol/L
Phosphorus	1.88 mmol/L	0.93-2.13 mmol/L
Creatinine	120 μmol/L	27-124 μmol/L
BUN	12.1 mmol/L	2.5-8.9 mol/L
Glucose	3.6 mmol/L	3.3-6.1 mmol/L

UA SG 1.050 with 2+ protein and 2+ blood. Urine submitted for culture

Interpretation?

Please do not turn over yet!!

Revised problem list	Differential diagnosis

What other diagnostic tests would you like to perform?

Please do not turn over yet!!

Abdominal ultrasound

No free fluid seen. Bladder appears intact

Abdominal radiographs

These will be displayed during the presentation

Interpretation?

Ongoing treatment?

Further diagnostics?

Please do not turn over yet!!

Specialist abdominal ultrasound

Confirmed no free peritoneal fluid and intra peritoneal organs appear unremarkable. The left kidney appears abnormal with a mass in the left cranial pole and calculi in the renal pelvis. Small pockets of fluid are seen in the retroperitoneal space.

This fluid is aspirated and reveals a serosanguinous fluid

Retroperitoneal fluid analysis

	Peripheral blood	Retroperitoneal fluid
Urea (mmol/L)	7.5	7.8
K (mmol/L)	3.6	3.4
Glucose (mmol/L)	5.5	3.6
Lactate (mmol/L)	1.5	4.5

How do you interpret these results?

What is your next step?

Please do not turn over yet!!!

Cytology on the retroperitoneal fluid also reveals some neutrophils with intracellular bacteria.

Holly has received 2 crystalloid fluid boluses, the first of 40ml/kg and the second of 25 ml/kg during the time you have been pursuing your diagnostics. She is also started on intravenous cefuroxime at 20mk/kg tid and morphine analgesia (0.2 mg/kg im PRN)

A repeat database shows

PCV	35%	37-55%
TS	48 g/L	60-75 g/L
pH	7.47	7.35-7.45
$PvCO_2$	26 mmHg	35-55 mmHg
PvO_2	70 mmHg	
HCO_3^-	17 mol/L	18-24 mmol/L
BE	-4	-4 - +4
Na	140 mmol/L	140-155 mmol/L
K	4.0 mmol/L	3.5-4.5 mmol/L
Cl	110 mmol/L	110-120 mmol/L
iCa	1.24 mmol/L	1.13-1.33 mmol/L
Lactate	2.7 mmol/L	0.5-2.5 mmol/L

How would you interpret these results?

Is Holly a surgical candidate; if so, when?

Please do not turn over yet

Exploratory laparotomy

The left kidney appeared grossly enlarged with a distinct mass about 3cm in diameter in the cranial pole. The left ureter was dilated with multiple calculi. The retroperitoneal space contained fluid and necrotic fat. A left ureteronephrectomy was performed

What would you like to monitor post-operatively?

How should the patient be instrumented post-operatively?

Progression

For the first 24 hours, Holly received fluid therapy (replacement crystalloids and colloids) with monitoring of perfusion parameters, blood lactate and urine output. Antibiosis was continued and analgesia was provided with a fentanyl CRI.

On day 2 she developed worsening tachypnoea and thoracic radiographs revealed a moderate volume pleural effusion - on thoracocentesis this proved to be a modified transudate.

Histopathology

Was received on day 4 and showed ruptured renal haemangioma

Culture of urine and retroperitoneal fluid

Was received on day 3 and revealed a multi drug resistant *E. coli* sensitive only to amikacin, imipenem and polymixin B.

Further progression

Antibiosis was changed to intravenous imipenem. Holly remained in the Intensive Care Unit and received oxygen therapy, fluid therapy, analgesia and antibiosis. Unfortunately she continued to slowly deteriorate with worsening pleural effusion and peripheral oedema. On day 5 her owners elected for euthanasia.

What do you think the course of deterioration was? With the benefit of hindsight would you change any of the treatment or management decisions?

Case summary

This case highlights

- The need to cross reference perfusion parameters to evaluate the presence of distributive shock as opposed to simple hypovolaemic shock
- The use of fluid analysis to help establish the diagnosis of a septic fluid
- Concerns regarding the presence of multi drug resistant bacteria already being present on admission to the hospital.

Bonczynski JJ et al Comparison of peritoneal fluid and peripheral blood pH, bicarbonate, glucose, and lactate concentration as a diagnostic tool for septic peritonitis in dogs and cats. Vet Surg. 2003 Mar-Apr;32(2):161-6.
OBJECTIVE: To establish a reliable diagnostic tool for septic peritonitis in dogs and cats using pH, bicarbonate, lactate, and glucose concentrations in peritoneal fluid and venous blood. STUDY DESIGN: Prospective clinical study. ANIMALS: Eighteen dogs and 12 cats with peritoneal effusion. METHODS: pH, bicarbonate, electrolyte, lactate, and glucose concentrations were measured on 1- to 2-mL samples of venous blood and peritoneal fluid collected at admission. The concentration difference between blood and peritoneal fluid for pH, bicarbonate, glucose, and lactate concentrations were calculated by subtracting the peritoneal fluid concentration from the blood concentration. Peritoneal fluid was submitted for cytologic examination and bacterial culture. Peritonitis was classified as septic or nonseptic based on cytology and bacterial culture results. RESULTS: In dogs, with septic effusion, peritoneal fluid glucose concentration was always lower than the blood glucose concentration. A blood-to-fluid glucose (BFG) difference > 20 mg/dL was 100% sensitive and 100% specific for the diagnosis of septic peritoneal effusion in dogs. In 7 dogs in which it was evaluated, a blood-to-fluid lactate (BFL) difference < -2.0 mmol/L was also 100% sensitive and specific for a diagnosis of septic peritoneal effusion. In cats, the BFG difference was 86% sensitive and 100% specific for a diagnosis of septic peritonitis. In dogs and cats, the BFG difference was more accurate for a diagnosis of septic peritonitis than peritoneal fluid glucose concentration alone. CONCLUSIONS: A concentration difference > 20 mg/dL between blood and peritoneal fluid glucose concentration provides a rapid and reliable means to differentiate a septic peritoneal effusion from a nonseptic peritoneal effusion in dogs and cats. CLINICAL RELEVANCE: The difference between blood and peritoneal fluid glucose concentrations should be used as a more reliable diagnostic indicator of septic peritoneal effusion than peritoneal fluid glucose concentration alone.

Case Challenge

Humphrey

Signalment:
An 8 year old male castrated Abyssinian

History:

Humphrey presented to his referring veterinary surgeon for evaluation of 36 hours of inappetence and progressive lethargy. He had vomited bile once during this time period. Defecation and urination were unknown as he had still been going outside for this purpose. This evening, he has started open mouth breathing which has prompted the owner to bring him in as an emergency.

Humphrey is an outdoor cat. He is fully vaccinated and wormed. He had a road traffic accident when he was 1 year old and was hospitalised for a few days but the owner is unsure of the exact nature of his injuries. Otherwise he has been a very healthy cat.

Physical examination – referring veterinary surgeon

Cardiovascular: HR 144 bpm with moderate to poor quality pulses. Metatarsal pulse just palpable. Mucous membranes appear pale and dry – it is very difficult to assess CRT. No murmur or gallop audible.

Respiratory: Tachypnoeic but no obvious dullness of crackles on auscultation. Occasional episodes of open mouthed breathing especially when stressed.

Neurological: Very depressed and lying in lateral recumbency most of the time but no abnormalities on cranial nerve examination.

Abdominal palpation: Small bladder palpable. No obvious mass lesions or fluid thrill.

Temperature: 34.6ºC

WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK REGARDING HISTORY AND PHYSICAL EXAMINATION?

Problem list	Differential diagnosis

Plan???

Please do not turn over yet!!

Haematology

Hct	15.5%	27-50
Hb	5.3 g/dL	8-15
RBC	3.56 x 10^{12}/L	5.5-10
MCV	44 fL	40-55
MCHC	34.2 g/dL	31-34
Total wbc	8.3 x 10^{9}/L	4-15
Granulocytes	7.2 x 10^{9}/L	1.2-6.8
Lymphocytes	0.8 x 10^{9}/L	1.2-3.2
Monocytes	0.3 x 10^{9}/L	0.3-0.8
Platelets	14 x 10^{9}/L	200-600

Smear examination: Platelets appear to be genuinely low

Biochemistry:

Albumin	20.9 g/L	21-39
Total Protein	54.4 g/L	54-78
ALP	6 U/L	0-40
ALT	2324 U/L	0-20
Tbilirubin	0.9 μmol/L	0-10
Total calcium	2.29 mmol/L	1.6-3
Phosphorus	1.63 mmol/L	1.4-2.6
BUN	18.7 mmol/L	6-10
Creatinine	210 μmol/L	80-180
Glucose	14.1 mmol/L	4.3-6.6
Na	139 mmol/L	120-155
K	3.6 mmol/L	3.6-5.6

FeLV/FIV	Negative

Interpretation?

Plan?

Please do not turn over yet!!

Initially Humphrey was started on 0.9%NaCl at 6ml/kg/hr for 4 hours. This was then reduced to 2ml/kg/hr and Oxyglobin® was started at 1ml/kg/hr. He also received burpernorphine at an unknown dose and enrofloxacin at 10mg/kg slow intravenous once.

By the following morning, Humphrey appeared a little brighter. His heart rate was 200bpm with tall and narrow pulses. His temperature was still subnormal at 35.2ºC. Systolic blood pressure was measured by Doppler at this time and was found to be 90mmHg.

He was then referred for further management.

Evaluation on arrival at referral centre

Cardiovascular: HR 160bpm with tall and narrow pulses. Mucous membranes were pale.

Respiratory: Tachypnoeic at 60 breaths per minute with unremarkable uasultation. He still exhibited open mouth breathing when stressed.

Neurological: Depressed but otherwise unremarkable.

Abdominal palpation: Abdomen felt slightly “doughy”.

Temperature: 36.8ºC

Examination was otherwise unremarkable.

What is your evaluation?

Plan?

Please do not turn over yet!!

Venous blood gas

PCV	14%	24-45%
TS	Not possible due to Oxyglobin	
pH	7.325	7.35-7.45
$PvCO_2$	25.8 mmHg	35-55 mmHg
PvO_2	40.7 mmHg	
HCO_3^-	13.5 mol/L	18-24 mmol/L
BE	-12.7	-4 - +4
Na	142.4 mmol/L	140-155 mmol/L
K	4.67 mmol/L	3.5-4.5 mmol/L
Cl	115.6 mmol/L	110-120 mmol/L
iCa	1.21 mmol/L	1.13-1.33 mmol/L
Lactate	7.4 mmol/L	0.5-2.5 mmol/L

Repeat Biochemistry (submitted by referring vet – sample drawn pre Oxyglobin®):

Albumin	27.6 g/L	28-42 g/L
Globulin	26.5 g/L	25-46 g/L
ALP	19 U/L	11-58 U/L
ALT	2390 U/L	25-130 U/L
Tbilirubin	0.6 μmol/L	0-3 μmol/L
Total calcium	2.15 mmol/L	2.07-2.8 mmol/L
Phosphorus	1.59 mmol/L	0.92-2.16 mmol/L
BUN	15.4 mmol/L	6.1-12 mmol/L
Creatinine	199 μmol/L	107-193 μmol/L

Repeat haematology:

Hct	15.1	24-45
Hb	5.00 g/dL	8-15
RBC	3.29×10^{12}/L	5-10
MCV	46 fL	39-55
MCHC	33.2 g/dL	30-36
Total wbc	7.19×10^9/L	5.5-19.5
Neutrophil, segmented	6.76×10^9/L	2.5-12.5
Neutrophil, band	0.58×10^9/L	0
Eosinophils	0	0-1.5
Basophils	0	0-0.4
Lymphocytes	0.29×10^9/L	1.5-7
Monocytes	0.14×10^9/L	0-1.5
Platelets	19.4×10^9/L	200-800
Reticulocytes	0.8%	

Smear evaluation: Red cell morphology; rare polychromatophils seen, little anisocytosis. 1nrbc/100wbc. Neutrophils left shifted, rare reactive lymphocyte.

Revised problem list	Differential diagnosis

Ongoing treatment?

Further diagnostics?

Please do not turn over yet!!

Blood type

A

Coagulation screen

PT and APTT both within normal limits

In saline agglutination

Negative

Abdominal ultrasound

Liver appeared abnormal with irregular outline and generally mixed echogenicity. There is a large amount of echogenic free fluid.

Urinalysis (free catch)

Discoloured with Oxyglobin® - SG >1.060

Plan (it is now Friday afternoon)?

Please do not turn over yet!!

Abdominocentesis

Haemorrhagic fluid aspirated with PCV of 21% and TS of 50. Cytological examination of the fluid revealed just red cells with no platelets and white cells consistent with peripheral blood.

Treatment

Humphrey was continued on Oxyglobin® at 1 ml/kg/hr whilst a transfusion of fresh whole blood was organised. This was then given over the subsequent 6 hours. Vitamin K was started at a dose of 0.5 mg/kg sc twice daily and ampicillin (20mg/kg iv tid) and metronidazole (10mg/kg bid) were administered.

He was monitored closely through the weekend. His demeanour improved throughout this time such that by the Mon morning he was eating well and was quite bright. His heart rate was 150bpm with good pulses and pale pink mucous membranes. He no longer exhibited open mouth breathing and he was maintaining a normal body temperature.

It is now Monday morning – what is your plan for Humphrey's further evaluation and treatment?

Please do not turn over yet!!

Repeat biochemistry (3 days post Oxyglobin®):

Albumin	41.8 g/L	28-42 g/L
Globulin	42.5 g/L	25-46 g/L
ALP	79 U/L	11-58 U/L
ALT	1216 U/L	25-130 U/L
Tbilirubin	4.2 μmol/L	0-3 μmol/L
Total calcium	1.99 mmol/L	2.07-2.8 mmol/L
Phosphorus	0 mmol/L	0.92-2.16 mmol/L
BUN	7.1 mmol/L	6.1-12 mmol/L
Creatinine	184 μmol/L	107-193 μmol/L
Ammonia	40 μmol/L	0-60 μmol/L

Repeat haematology:

Hct	27.8	24-45
Hb	10.2 g/dL	8-15
RBC	6.25×10^{12}/L	5-10
MCV	44.5 fL	39-55
MCHC	36.5 g/dL	30-36
Total wbc	12.2×10^{9}/L	5.5-19.5
Neutrophil, segmented	10.7×10^{9}/L	2.5-12.5
Eosinophils	0.85×10^{9}/L	0-1.5
Basophils	0	0-0.4
Lymphocytes	0.24×10^{9}/L	1.5-7
Monocytes	0.37×10^{9}/L	0-1.5
Platelets	97.5×10^{9}/L	200-800

Smear evaluation: Platelets aggregated and adequate

Repeat abdominal ultrasound

Minimal volume free fluid remaining. Hepatic parenchyma appears less abnormal but still of mixed echogenicity.

Interpretation??

Further diagnostics/treatment?

Please do not turn over yet!!

Progression

Following discussion of further diagnostics including hepatic aspirate or biopsy, Humphrey's owners elected to take him home. He was discharged with a further 7 day course of oral antibiotics (ampicillin and metronidazole). He was rechecked by his own practice after one week at which time he was doing well and his blood work was nearly normal other than a slightly elevated ALT.

Three months following the initial presentation, he started to exhibit similar clinical signs and his referring vets confirmed the recurrence of haemoabdomen. At this time his owners elected for euthanasia.

Post mortem a sample of liver was taken and confirmed hepatic amyloidosis.

Case summary

This case highlights some differences between the assessment and management of cats as opposed to dogs with severe intra-abdominal disease

- Free fluid can be much harder to detect on physical examination
- In collapsed cats, hypothermia and inappropriately slow heart rates (in the 120-150 range) should raise be a cause for concern
- Cats with haemoabdomen may present simply with lethargy and tachypnoea

It also highlights the use of Oxyglobin® and blood transfusion for the treatment of patients with haemoabdomen.

Relevant papers

Beatty JA et al (2002).Spontaneous hepatic rupture in six cats with systemic amyloidosis. J Small Anim Pract **43**:355-63.

Spontaneous hepatic rupture, secondary to the accumulation of hepatic amyloid, was diagnosed in six cats over a two-year period. Previous reports of feline hepatic amyloidosis have documented clusters of cases from breeding catteries. Most affected cats have been Siamese or a related breed and the disease is generally regarded as familial. In contrast, the cases presented here were sporadic, with relatives and other cats in the household not clinically affected. They included a Devon rex, a breed not previously reported with this condition, and a domestic shorthair. Clinical signs in three of these cases had, prior to referral, been misinterpreted as resulting from blunt trauma, immune-mediated haemolysis or a coagulopathy. Antemortem diagnostic features, including new data on the value of hepatic ultrasonography and fine-needle aspirate

cytology, are reported. These cases illustrate how the course of this disease can vary between individuals and that, despite the dramatic underlying pathology, hepatic amyloidosis can present a diagnostic challenge and should be suspected in any young adult cat with consistent clinical signs, irrespective of breed or environment.

Case Challenge

Ollie

Signalment:
An 8 year old male castrated Border Collie

History:
Ollie presented for evaluation of sudden onset hindlimb paresis and pain. He had been out for a walk that evening and had suddenly yelped in pain and started "trying to walk backwards". He was able to walk home although his owner felt he was not using his left hindlimb normally. He had lain down at home and seemed very subdued with occasional vocalisation as if in pain so his owners had sought veterinary attention. He had been totally normal earlier that day and the owners could not associate anything with the initial episode of vocalisation and pain on the walk – he had been walking a few metres ahead of them and they had not noticed anything unusual.

Ollie's vaccinations were up to date and he had been castrated at a young age. He had a history of intermittent colitis and was fed principally on an exclusion diet of fish and tapioca. He had eaten his meal this evening normally at 6pm. His owner reported that over the last week he had maybe urinated a little more frequently on his walks but his water intake had been normal and there had been no straining on urination. He last urinated on his walk this evening normally.

Physical examination – major body systems

Cardiovascular system: HR 100 beats per minute with regular strong femoral pulses. Mucous membranes pink with a CRT of 1.5s

Respiratory system: Unremarkable

Neurological system: Quiet. Cranial nerve evaluation was within normal limits. Appeared unwilling to walk with a hunched stance. Both hindlimbs appeared to have increased tone and there appeared to be decreased proprioception in his right hindlimb. Forelimbs appeared normal. Back pain does not appear to be localiaable

Abdo palpation: Mildly uncomfortable but difficult to palpate due to stance.

Temperature: 37.8ºC

WHAT FURTHER QUESTIONS WOULD YOU LIKE TO ASK REGARDING HISTORY AND PHYSICAL EXAMINATION?

Problem list	Differential diagnosis

It is now 10pm at night – what is your plan for overnight?

Diagnostics?

Treatment?

Please do not turn over yet!!

Initial database

PCV	51%
TS	84 g/L

Blood smear evaluation;

Red blood cell morphology appeared normal. Platelet count adequate. White cell count subjectively within normal limits with normal morphology.

In house coagulation:

PT 15s (reference 12-17s)
PTT 79s (reference 70-102s)

Biochemistry (vetscan):

Albumin	Did not read	
Globulin	Did not read	
ALP	84 U/L	20-150 U/L
ALT	52 U/L	10-118 U/L
Amylase	333 U/L	200-1200 U/L
Tbilirubin	5 μmol/L	2-10 μmol/L
Total calcium	2.07 mmol/L	2.15-2.95 mmol/L
Phosphorus	0.81 mmol/L	0.93-2.13 mmol/L
Creatinine	99 μmol/L	27-124 μmol/L
BUN	6.0 mmol/L	2.5-8.9 mol/L
Glucose	6.4 mmol/L	3.3-6.1 mmol/L
Na	148 mmol/L	138-160 mmol/L
K	3.8 mmol/L	3.7-5.8 mmol/L

Interpretation?

Any change to your overnight plan?

Please do not turn over yet!!

Ollie was treated with Hartmanns at 4ml/kg/hr overnight with morphine 0.3mg/kg im as necessary.

At 5am he was taken for a short walk outside. He postured to urinate and passed a few small drops of bloody urine. He continued to strain for a period of time but did not pass anything else.

At 8.30am, he vomited.

Evaluation 9am following day

Cardiovascular: HR 150bpm with moderate-poor quality pulses – certainly not as good as the previous evening. Mucous membranes pink with 1s CRT

Respiratory: Unremarkable

Neurological: Seems more depressed. Hindlimbs still appear to have increased tone. Proprioception in right hindlimb seems to have improved

Abdominal palpation: More painful, especially in caudal abdomen

Temperature: 39.2ºC

How would you describe/interpret Ollie's clinical progression?

Revised plan?

Please do not turn over yet!!

Repeat Biochemistry (vetscan) at 9am:

Albumin	34 g/L	25-44 g/L
Globulin	27 g/L	23-52 g/L
ALP	86 U/L	20-150 U/L
ALT	89 U/L	10-118 U/L
Amylase	500 U/L	200-1200 U/L
Tbilirubin	6 μmol/L	2-10 μmol/L
Total calcium	2.56 mmol/L	2.15-2.95 mmol/L
Phosphorus	1.41 mmol/L	0.93-2.13 mmol/L
Creatinine	326 μmol/L	27-124 μmol/L
BUN	11.4 mmol/L	2.5-8.9 mol/L
Glucose	6.6 mmol/L	3.3-6.1 mmol/L
Na	149 mmol/L	138-160 mmol/L
K	4.9 mmol/L	3.7-5.8 mmol/L

Revised problem list	Differential diagnosis

Ongoing treatment?

Please do not turn over yet!!

Imaging (emergency clinician)

Abdominal ultrasound reveals a moderate amount of free peritoneal fluid. Bladder appears small but intact although the cranial pole looks abnormal.

Abdominocentesis

Reveals a haemorrhagic fluid.

Meanwhile Ollie is receiving a 20ml/kg bolus of Hartmanns fluid

What would you like to do with abdominal fluid sample? How will you interpret any results?

Please do not turn over yet!!!

Analysis of the abdominal fluid reveals

	Peripheral	Abdominal fluid
PCV (%)	57	12
TS (g/L)	88	30
Urea (mmol/L)	12.1	28.4
Creatinine (μmol/L)	323	1656
Potassium (mmol/L)	4.6	>8.5

Cytology on the abdominal fluid reveals crenated red cells, non degenerate neutrophils and some macrophages.

Following the fluid bolus, Ollie's HR is 140bpm and his pulses are slightly improved.

What is your assessment of these results?

What is the next step you would recommend to the owners?

Please do not turn over yet

The diagnosis of uroabdomen is made. Following a second fluid bolus, Ollie is anaesthetised for a retrograde cystourethrogram.

Radiographic Interpretation

Extravasation of contrast into the peritoneal cavity with irregular margin of bladder

Exploratory laparotomy

A large grapefruit sized brown-green mass was identified ventral to the bladder – on closer inspection this appeared to be a large blood clot. It was bluntly dissected away to reveal a walnut sized mass protruding form the bladder mucosa which was visible due to the bladder being ruptured.

A partial cystectomy was performed and the bladder closed with a simple continuous layer in the mucosa and a simple interrupted layer in the muscularis and serosa. All other organs were considered to be within normal limits. The abdomen was lavaged with 3L of saline and the abdomen closed.

What would you like to monitor post-operatively?

How should the patient be instrumented post-operatively?

Progression

Six hours post-operatively, Ollie developed tachycardia with intermittent runs of ventricular tachycardia at >200bpm. These were treated with an intravenous lignocaine infusion. Urine output and central venous pressure were monitored.

Ollie improved slowly with resolution of the azotaemia by 48 hours. Oral ulceration did develop 1 day post-operatively but was mild and resolved within 3 days.

The urinary catheter was removed on day 3 by which time Ollie had started to eat. A free catch urine sample obtained 12 hours after the urinary catheter was removed yielded a growth of E. coli and Proteus both sensitive to enrofloxacin.

Histopathology

Was received on day 5 and showed bladder leiomyoma with focal haematoma formation.

Ollie was discharged on day 6.

Follow-up at one month

Ollie was reported to be well at home with normal urination – ultrasound of the bladder was unremarkable as was his bloodwork and US. He remains well one year later.

Case summary

This cases highlights:

- Difficulty of distinguishing neurological abnormalities from the "acute abdomen" especially in the early stages
- If patients do not progress systemically as well as you expect, problem and differential diagnosis lists should be revised
- The diagnostic criteria for uroabdomen
- The time frame over which the clinical signs of uroabdomen develop

Relevant papers

Burrows and Bovee. (1974) Metabolic changes due to experimentally induced rupture of the canine urinary bladder. Am J Vet Res. 1974 Aug;35(8):1083-8.

78% dogs died 47 – 90 hours later
Clinical signs within 12-24 hours and included CNS depression, anorexia, increasing reluctance to walk, abdominal discomfort, vomiting, progressive and severe dehydration, haematuria (54 %) and stranguria (82 %).
Haematology and Biochemistry from 21 hrs:

- Marked leukocytosis: mean = 31 x 10^9/l, predominantly neutrophils
- Haemoconcentration: mean PCV = 50%
- Azotaemia: BUN doubled within 5 hours and creatinine was clearly increased by 21 hours
- ↑ phosphorus: noted by 21 hours
- ↑ potassium: but mean not >5.5mmol/l until 53 hrs, other studies have found no increase.
- ↓ sodium
- ↓ chloride

Schmeidt et al (2001) Evaluation of abdominal fluid: peripheral blood creatinine and potassium ratios for diagnosis of uroperitoneum in dogs. Journal of Veterinary Emergency and Critical Care **11** 275-280.

Abdominal fluid potassium and creatinine were recorded as were concurrent peripheral blood values. An abdominal fluid creatinine to peripheral blood creatinine ratio of >2:1 was predictive of uroabdomen (specificity 100%, sensitivity 86%). An abdominal fluid potassium to peripheral blood potassium ratio of >1.4:1 is also predictive (specificity 100%, sensitivity 100%). Abdominal fluid to peripheral blood potassium and creatinine ratios provide a means to diagnose uroperitoneum in dogs without elevated peripheral blood creatinine.