Treatment and management of acute kidney injury

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Introduction

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CE Certificates

- Emailed to you 48 hours after the webinar
- Active participation = no quiz
- Watching video later, must complete quiz
- Call in from Smart Phone!
- Email / contact with ANY questions
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Urogenital emergencies causing AKI

- Rapid recognition
- Aggressive intervention
- Prognosis = good!

- Goals:
  - Classification
  - Etiologies
  - Treatment
  - Cases

Definition of AKI

- An "abrupt, sustained decrease in renal function and loss of kidneys' ability to:"
  - Regulate acid-base and electrolyte balance
  - Concentrate urine
  - Excrete wastes
- Previously called acute renal failure (ARF) → due to histopathologic changes that occur with an acute insult

Balakrishnan A, Drobatz KJ. Management of Urinary Tract Emergencies in Small Animals. VCNA 2014

Decreased effective renal perfusion
↓
ATP depletion
↓
Cytoskeletal changes in renal tubular epithelial cells
↓
↓ GFR
↓
Persistent injury, release of inflammatory mediators and interactions between epithelial and endothelial cells
↓
Apoptosis and acute tubular necrosis

Pathophysiology

- Usually due to acute tubular damage → necrosis and sloughing of tubular cells → obstruct tubules → loss of tight junctions between tubular cells
- Causes tubular fluid leaking out of tubule into renal interstitial space

Pathophysiology

- Renal arterial vasoconstriction → ↓ GFR → abnormal tubular function
- But… don't give up hope! Potential for compensation and repair

IRIS: Internal Renal Interest Society

How frequent is AKI?

- Humans:
  - 2-7% in all-hospital patients
  - 15% in critically ill patients → mortality 50%
- Veterinary:
  - 23.8-78.3% mortality in dogs
  - 47% in cats

Anatomically: What's up?

- Lower urinary tract: urinary bladder, urethra
- Upper urinary tract: kidneys, renal pelvis, ureters

What causes AKI?

- Pre-renal
  - Due to ↓renal perfusion, GFR, renal blood flow
  - Severe dehydration or hypovolemia → ↓effective circulating volume
    - Hypoadrenocorticism

Renal causes

- Congenital
  - Polycystic kidneys
  - Renal dysplasia
- Infectious
  - Leptospirosis
  - Pyelonephritis
  - Glomerulonephritis
- Other:
  - Ischemia
  - MODS, sepsis
  - Pigment nephropathy, etc.
- Toxicants
  - Ethylene glycol (EG)
  - Grapes/raisins
  - Lilies (cats)
  - "Calc"i-products
    - Cholecalciferol
    - Calcipotriene
    - Vitamin D
  - NSAIDs
  - Amikacin

Post-renal causes

- Due to obstruction to tubular outflow or urine leakage
  - Trauma (e.g., uroabdomen)
  - Feline urethral obstruction
  - Cystic calculi
  - Urethral calculi
  - Ureteral calculi

CLASSIFICATION… AND WHY WE CARE

RIFLE (Risk, Injury, Failure, Loss, End-Stage) renal disease

- Focuses on creatinine and UOP

	Urine output (UOP)	Serum creatinine
Risk	↓ GFR ≥ 25% < 0.5 ml/kg/hr for ≥ 6 hours	≥ 1.5 fold ↑ from baseline creatinine
Injury	↓ GFR ≥ 50% <0.5 ml/kg/hr for ≥ 12 hours	≥ 2 fold ↑ from baseline serum creatinine
Failure	↓ GFR ≥ 75% <0.3 ml/kg/hr for ≥ 24 hours or anuria ≥ 12 hours	≥ 3 fold ↑ from baseline creatinine; or an absolute creatinine ≥ 4 mg/dL with an acute ↑ > 0.5 mg/dL
Loss	Persistent ARF: complete loss of kidney function > 4 weeks	
End stage	Complete loss of kidney function > 3 months	

AKIN: Acute Kidney Injury Network

AKIN staging system	UOP	Serum Creatinine
Stage 1	<0.5 ml/kg/hr for ≥ 6 h	≥ 0.3 mg/dL or ≥150-200% ↑ from baseline creatinine
Stage 2	<0.5 ml/kg/hr for ≥ 12 h	>200-299% ↑ from baseline creatinine
Stage 3	<0.3 ml/kg/hr for ≥24 h or anuria ≥ 12 h	≥ 300% ↑ from baseline creatinine or absolute creatinine ≥ 4 mg/dL with an acute ↑ of ≥ 0.5 mg/dL

VAKI: Veterinary Acute Kidney Injury

- Uses increases in creatinine to stage patients

VAKI Staging System	
Stage 0	Creatinine ↑ < 150% from baseline
Stage 1	Creatinine ↑ of 150-199% from baseline OR Creatinine ↑ of 0.3 mg/dL from baseline
Stage 2	Creatinine ↑ of 200-299% from baseline
Stage 3	Creatinine ↑ of ≥ 300% from baseline OR Absolute creatinine > 4 mg/dL

IRIS: AKI (2013)

Table 1: IRIS AKI Grading Criteria

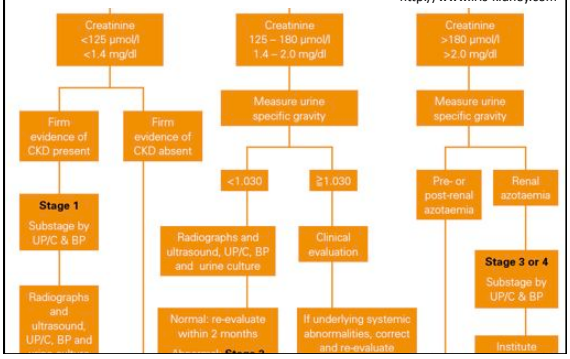
AKI Grade	Blood creatinine	Clinical Description
Grade I	<1.6 (mg/dl) (<140 µmol/l)	Non Azotemic AKI: a. Documented AKI: (Historical, clinical, laboratory, or imaging evidence of acute kidney injury, clinical oliguria/anuria, volume responsiveness‡)… and/or b. Progressive non azotemic increase in blood creatinine; ≥0.3 mg/dl (≥26.4 µmol/l) within 48 hours c. Measured oliguria (<1 ml/kg/hr) or anuria over 6 hrs
Grade II	1.7 – 2.5 mg/dl (141 – 220 µmol/l)	Mild AKI: a. Documented AKI and static or progressive azotemia b. Progressive azotemic increase in blood creatinine; ≥0.3 mg/dl ≥26.4 µmol/l) within 48 hours‡, or volume responsiveness‡ c. Measured oliguria (<1 ml/kg/hr) or anuria over 6 hrs
Grade III	2.6 – 5.0 mg/dl (221 – 439µmol/l)	
Grade IV	5.1 – 10.0 mg/dl (440 – 880 µmol/l)	Moderate to Severe AKI: a. Documented AKI and increasing severities of azotemia and functional renal failure
Grade V	>10.0 mg/dl (>880 µmol/l)	

(‡Volume responsive is an increase in urine production to >1 ml/kg/hr over 6 hours; and/or decrease in serum creatinine to baseline over 48 hours)

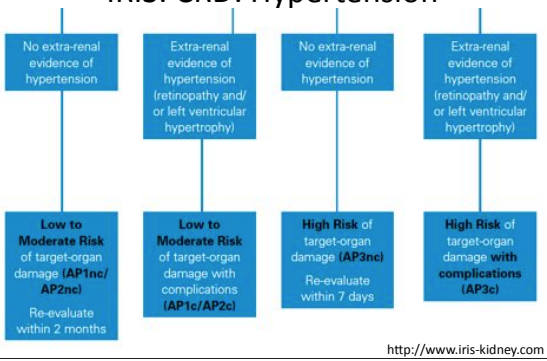
http://www.iris-kidney.com

IRIS scoring: CKD (creatinine, proteinuria, hypertension)

http://www.iris-kidney.com



IRIS: CRD: Hypertension



http://www.iris-kidney.com

In this webinar: multiple etiologies

- Grapes/raisins
- Ethylene glycol (EG)
- Lilies (cats)
- Feline urethral obstruction
- Uroabdomen

REVIEW OF GENERAL TREATMENT FOR THE AKI PATIENT

Treatment

- If toxicant: decontamination, if appropriate
  - Asymptomatic patient
  - Recent ingestion
  - Anti-emetic therapy
  - Activated charcoal? Only if it doesn't end with "ol!"

Mainstay therapy

- Correct fluid deficits with crystalloids
  - Restore renal perfusion
  - Limit further renal tubular injury
  - Restore hemodynamic state
- Avoid overhydration!

Fluid therapy

- SAFE: Saline versus Albumin Fluid Evaluation study
  - Albumin not necessary and not any more effective than saline in preventing death or dialysis needs
- Colloids? No difference between crystalloids and colloids in treating AKI
  - FDA concerns in *human* medicine with colloids

Treatment of AKI

- Gastrointestinal support:
  - Anti-emetics
    - Cerenia 1 mg/kg IV q 24
    - Ondansetron 0.5 mg/kg IV q 8-12
    - Dolasetron 0.6 mg/kg IV q 24
  - Antiulcer medication for uremic gastritis
    - Famotidine 1 mg/kg IV q 12
    - Sucralfate PO q 8
  - Phosphate binders
    - Ca X P > 70 = mineralization
    - Even in anorexic?
    - Renal secondary hyperparathyroidism

Monitoring of AKI

- Central line
- Weight q. 4-6 hours
- TPR q. 6-8 hours
- Blood pressure monitoring
- Daily PCV/TS/BG/venous blood gas/elytes
- Monitor UOP q 4
- Evaluation for proteinuria

Appropriate monitoring of hydration

- PCV/TS 35/5
- Sp. gravity 1.015-1.018
- Urine volume
- Drinking water in the cage

Appropriate urine output (UOP)

- Normal: 1-2 ml/kg/hour
- Oliguric: 0.5 ml/kg/hour
- Anuric: < 0.5 ml/kg/hour
  - Blood-tinged urine

Calculating ins and outs

- Simple!
- If FUO urinates 160 mls over 4 hours…UOP ➔ 160/4 = 40 ml/hour
- If you gave 80 mls of IV fluids over 4 hours… 80/4 = 20 ml/hour
- In vs. out?

Additional care

- Nutritional support
- ± antibiotic therapy
  - Did you get your urine culture first?
  - Leptospirosis status? Zoonosis!
- Antihypertensive therapy?

ADDITIONAL THERAPY FOR AKI

GOAL: ⇧UOP WITHIN 30-60 MINUTES

Additional therapy

- Vasopressors
  - ⇧renal vasodilation; induces natriuresis and diuresis
  - Dopamine 2.5-5 mcg/kg/min CRI
  - CONS: can vasoconstrict and impair GFR, dogs only
- Osmotic diuresis
  - ⇧tubular flow, renal blood flow and intravascular volume
  - Free radical scavenger, reduces post-ischemic swelling
  - ⇧urine flow through the nephron
  - Mannitol 0.5-1 g/kg IV bolus followed by 60-120 mg/kg/hour X 1-2 days
  - CONS: dehydration/volume depletion, hypernatremia

Additional therapy

- Diuretics
  - ⇧urine flow to flush out intratubular casts, reduces tubular obstruction, inhibits Na^+ transport, reducing energy requirements of cells, prevents ↓ in GFR
  - Furosemide: 0.25-4 mg/kg IV followed by 0.1-2 mg/kg/hr CRI
  - CONS: dehydration/volume contraction, pre-renal azotemia, electrolyte abnormalities, hypochloremic metabolic alkalosis, ototoxicity

Miscellaneous additional therapy

- Diltiazem (Calcium channel blocker)
  - Reverses vasoconstriction by preglomerular dilation, cytoprotective, reversal of thromboxane A2-induced vasoconstriction
  - DOSE: 0.1-0.5 mg/kg slow IV, followed by 1-5 mcg/kg/min CRI
  - CONS: Hypotension
- Fenoldopam
  - Selective DA-1 agonist
  - Produces systemic and renal vasodilation, ⇧renal blood flow
  - DOSE: 0.8 mcg/kg/min CRI

ETIOLOGIES FOR AKI

Grapes and Raisins

Grapes and raisins

- Eubig et al, 2001
  - Red, green, seedless, seeded
  - Grapes: 0.7 oz/kg
  - Raisins: 0.11 oz/kg
- VIN:
  - Grape seed extract OK?
  - Raisin juice? Grape juice?
  - Baking process?
- More than a handful…

Grapes and Raisins

- Clinical signs:
  - Anorexia
  - Vomiting
  - Diarrhea
  - Kidney pain
  - Azotemia
  - Oliguria (0.5-1 ml/kg/hr)
  - Anurua (< 0.5 ml/kg hr)
- Azotemic? R/O nephrotoxicants:
  - Grapes/raisins/currants
  - NSAIDs
  - Ethylene glycol

Harper, 3 yo black lab, 30 kgs

- PC: ingested 12 oz. raisins 3 days prior – o unaware of toxicity
  - Vomiting, loose stool, anorexia, depression X 24 hours
- PMHX: healthy
- Went to RDVM:
  - CBC/CHEM:
    - BUN: 180 mg/dL
    - Creatinine 5.4 mg/dL → referred
- Presentation:
  - T: 37.9°C/100.2°F, HR 80, RR 16
  - 5% dry
  - Mild abdominal and renal pain, no palpable bladder

Harper

- Diagnostics:
  - I-stat
  - Catheterize, empty bladder, quantitate urine, pull UA, CULTURE
    - FAST U/S – no bladder seen
    - 10 mls
- Treatment
  - Bolused 1 liter LRS IV once
    - Post-bolus, small bladder seen on FAST U/S
  - Blood pressure 170/100 mmHg (MAP 150 mmHg)
  - Aluminum hydroxide 10 mls PO q. 8
  - Sucralfate 1 gram PO q. 8
  - Famotidine 0.5 mg/g IV q. 24
  - Ampicillin 22 mg/kg IV q. 8

Harper

- Treatment:
  - Antihypertensives:
    - Enalapril 0.5 mg/kg PO SID
    - Hydralazine 0.5 mg/kg PO BID
  - Anti-emetics:
    - Maropitant 1 mg/kg SC SID
  - Blood pressure monitoring
  - UOP monitoring (UCS)

	BUN mg/dL	Creat mg/dL	UOP	Fluid rate	PCV	TS	kg	
Day 2	229	11	Oliguric	200-300 ml/hr		35	5	33
Day 3	300	14.7	Oliguric	100 ml/hr		30	4	33
Day 4	>300	18	Oliguric	50 ml/hr		30	4	35
Day 5	>300	22	Anuric	20 ml/hr; bolused 1 L		34	6	31
Day 8	32	0.8	13 ml/kg/hr	400 ml/hr		27	4	31

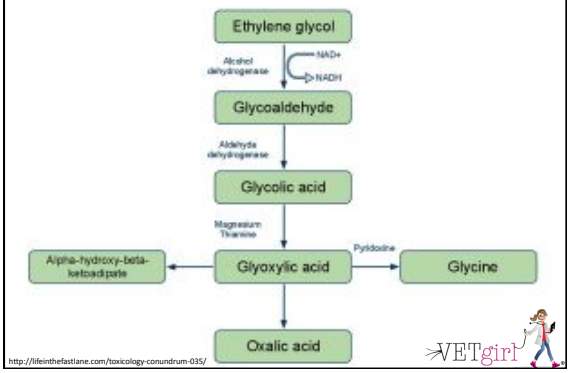
Lilium or *Hemerocallis* spp.

- True lilies
  - Easter lily, Tiger lily, Day lily, Stargazer lily, all Asiatic lilies
  - Common sound-alikes *not* peace, lily-of-the-valley, Peruvian!
- Cats only?
- Toxic portion
  - All of the plant, even pollen!
  - Water-soluble toxicant
  - Still unknown
  - 2-3 leaves = poisonous

What's the prognosis?

- Treat aggressively for 48 hours
- 100% survival! (Bennett AJ, JAVMA 2013)
- 27% of pet owners knew lilies poisonous (Slater MR, JAAHA 2011)
- Aggressive therapy!
  - Decontamination
  - Monitoring renal function
  - No SQ fluids!
  - 48 hour care

Ethylene Glycol (EG)



Ethylene glycol (EG)

- EG isn't toxic…
- Metabolites are *deadly* → lead to calcium oxalate → ARF
- Goal of tx: inhibit metabolism → just drunk!
- Antidotes:
  - 4-MP (fomepizole)
  - Ethanol

Ethylene glycol (EG)

- Generally only toxic if high concentration! (>90%)
  - Rapid absorption
  - Peak blood levels: 3 hours
- Toxic dose:
  - CATS: 1.4 mL/kg
  - Dogs: 4.4-6.6 mL/kg
- Aggressive treatment with the antidote or fatal!
  - Reported mortalities 96-100% (cats) and 59-70% (dogs)
  - CATS: 3 hours
  - DOGS: 8-12 hours
  - GRAVE once azotemic

Stage	Onset/duration	Clinical signs (CS)
Stage 1	30 minutes-12 hours	Drooling Drunk Ataxia pu/pd Metabolic acidosis Crystalluria + EG
Stage 2	12-24 hours	Dogs: Resolved clinical signs Cats: Continued lethargy Subtle signs: Dehydration Tachycardia Tachypnea
Stage 3	Cats: 12-24 hours Dogs: 36-72 hours	Oliguric renal failure Kidney pain Severe lethargy/coma Dehdyration Vomiting/uremic gastritis Seizures/coma/death

Ethylene glycol (EG)

- Decontamination if early/witnessed → no ACC!
- Diagnosis?
  - Human quantiative test
  - Veterinary EG tests (Kacey, PRN, Catachem)
  - Lots of false +
  - CaOx crystalluria
  - Metabolic acidosis
  - Anion gap
  - AUS "bright" kidneys

Treatment for EG

- ANTIDOTE: Goal to inhibit EG conversion by alcohol dehydrogenase (ADH)
- Fomepizole (Antizol®)
  - Ideal
  - No longer made
  - $$$$$
  - Varying dog vs. cat dose
  - Removed by dialysis
- Ethanol (IV grain alcohol or vodka)

4-MP (Fomepizole)

- DOGS:
  - Two protocols:
  - OPTION 1:
    - Initial load of 20 mg/kg IV
    - 12 hours post: 15 mg/kg IV
    - 24 hours post: 15 mg/kg IV
    - 36 hours post: 5 mg/kg
  - OPTION 2:
    - Time 0: 20 mg/kg IV
    - 17 hours: 15 mg/kg IV
    - 25 hours: 5 mg/kg
    - 36 hours: 5 mg/kg
- CATS:
  - Much higher dose! (> 6X!)
  - 125 mg/kg slow IV
  - 12, 24, 36 hours post: 31.25 mg/kg IV

Ethanol

- DOGS:
  - Multiple modalities:
  - CRI:
    - Make a 7% solution (70 mg/ml) and…
    - Load with 8.6 ml/kg (600 mg/kg) slow IV, followed by 1.43 ml/kg/hour (100 mg/kg/hour) IV for 24-36 hours CRI
  - Intermittent:
    - Make a 20% solution (200 mg/ml) and…
    - 5.5 ml/kg IV q 4 hours X 5 doses, followed by 5.5 ml/kg q 6 hours X 4 doses

Seriously, the hardest part of EG?

$C_1 \times V_1 = C_2 \times V_2$

- Make a 7% ethanol solution with Everclear (*check proof* - 190 proof, 95% alcohol):
- (95%)(X ml) = (7%)(1000 ml)
- (X ml) = 74 ml
- Remove 74 mls from a liter bag of fluids and replace it with 74 ml of 190 proof vodka

MOLSON, 1 YO, MC LAB

Molson, 1 yo, MC Labrador, 30 kgs

- HX: Jumped into the truck 3 days ago, and yelped out when landed on the tail gate
- PC:
  - Vomiting and anorexic last 24 hours
  - Lethargic for 48 hours

Molson, 1 yo, MC Labrador, 30 kgs

- Physical exam:
  - Depressed
  - Non-ambulatory but weak with support
  - T 99.5 F (37.5 C), HR 60 bpm, RR 40, pink tacky mm, CRT < 2 seconds
  - Attempts to bite you when you palpate abdomen

Molson, 1 yo, MC Labrador, 30 kgs

- Shave abdomen for FAST US
- Notice peri-umbilical bruising
- What is the significance of this bruising?

Photo courtesy of Dr. Soren Boysen, DACVECC

Molson, 1 yo, MC Labrador, 30 kgs

- Hct: 57%
- TS: 5.2 g/dl (52 mmol/L)
- Glucose: 79.2 mg/dl (4.4 mmol/L)
- BUN 50-80 mg/dl
- Lactate 6.7 mmol/L

Emergency electrolytes

pH	7.200	
PCO_2	27.8	mmHg
PO_2	40.5	mmHg
Hct	47.	%
Hb	15.0	g/dL
Na^+	140.7	mmol/L
K^+	10.2	mmol/L
Cl^-	115.	mmol/L
Ca^{++}	1.15	mmol/L
Mg^{++}	0.17	mmol/L
Glu	4.4	mmol/L
Lac	6.7	mmol/L
Urea	U.C	mmol/L
Calculated Results		
BE-ECF	-17.2	mmol/L
BE-B	-14.8	mmol/L
SBC	12.4	mmol/L
HCO_3	10.7	mmol/L

Photo courtesy of Dr. Soren Boysen, DACVECC

Molson, 1 yo, MC Labrador, 30 kgs

- Doppler blood pressure: 90 mm Hg
- ECG - interpretation?

Molson, 1 yo, MC Labrador, 30 kgs

- Fluid therapy:
  - Volume resuscitate, despite bradycardia
  - IV crystalloids
  - IV colloids
- Molson:
  - Hypotensive at 90 mm Hg
  - Bolus 1 L LRS IV over 20 minutes
  - Re-assess
  - BP 110 mm Hg, HR 70

Molson, 1 yo, MC Labrador, 30 kgs

- Started at 250 mls/hour LRS
- Started at 30 ml/hour of Hetastarch
- Correct elytes before surgery
- Does he need treatment for hyperkalemia?
  - Sodium bicarbonate 1 mEq/kg slow IV
  - 10% calcium gluconate 1 ml/kg slow IV
  - Dextrose/insulin

Venous blood gas analysis

- Severe metabolic acidosis!
- Respiratory alkalosis
- Severe hyperkalemia -> bradycardia in the face of shock
- Lactic acidosis

Blood gas courtesy of Dr. Soren Boysen, DACVECC

Molson, 1 yo, MC Labrador, 30 kgs

Molson, 1 yo, MC Labrador, 30 kgs

Photo courtesy of Dr. Soren Boysen

Molson, 1 yo, MC Labrador, 30 kgs

Photos courtesy of Dr. Soren Boysen, DACVECC

Abdominal fluid PCV

Peripheral (57%)

Abdominal (13%)

Molson, 1 yo, MC Labrador, 30 kgs

- Abdominal to peripheral blood creatinine 2.6:1

Fluid analysis - suspected diagnosis?

- Peripheral blood
  - PCV 57%
  - TS 5.2 g/dl (52 mmol/L)
  - Glucose 102 mg/dl (5.7 mmol/L)
  - Lactate 3.6 mmol/L
  - Urea (azostix) 50-80
  - Sodium 152 meq/l
  - Potassium 5.1 meq/l
  - Creatinine 4.2
- Abdominal fluid
  - PCV 13%
  - TS 2.0 g/dl (20 mmol/L)
  - Glucose 64 mg/dl (3.6 mmol/L)
  - Lactate 6.8 mmol/L
  - Urea (azostix) 50-80
  - Sodium 146 meq/l
  - Potassium 10.6 meq/l
  - Creatinine 8.6

Contrast study

- Urethrocystogram - leakage from the bladder

Molson, 1 yo, MC Labrador, 30 kgs

- Urinary catheter placement to prevent more leakage
- Abdominocentesis to remove as much effusion as possible
- 8 hours of stabilization followed by surgery at 12 hours

Molson, 1 yo, MC Labrador, 30 kgs

- Fluid therapy:
  - 200 mls of LRS/hour
  - 30 mls HES/hour
- Fentanyl 5 mcg/kg/hour CRI
- Maropitant 1 mg/kg SQ q 24
- Monitoring UOP – in place 24 hours

Molson, 1 yo, MC Labrador, 30 kgs

- TPR/blood pressure monitoring q. 4
- Venous blood gas + elytes + creatinine q. 12
- Post-op, small amounts water
- Nutritional support 12 hours later
- Did well → discharged 36 hours later
  - Tramadol, Clavamox, carprofen

So, how do we treat the uroabdomen patient?

- Stabilize before surgery!
- Therapeutic abdominocentesis if no immediate surgery?
- Antibiotic therapy
- Pain control – excruciatingly painful!

2nd Hour – Lower Urinary Tract Disease

Goals

1. Review FLUTD in cats
2. Examine feline urethral obstruction
3. Discuss medical / dietary goals

2nd Hour Goals – "Lower" Urinary

- Review FLUTD in cats
  - UTI
  - Idiopathic Cystitis / Crystalluria
  - Stones
- Closely examine feline urethral obstruction
  - Clinical signs
  - Diagnostics
  - Treatments
  - Pathophysiology
- Discuss medical and dietary management of FLUTD

FLUTD - Etiology

- Young to middle-aged cats
- 55-69% are idiopathic
- Rarely seen in older cats
  - Bacterial cystitis
  - Urolithiasis (bladder stones)
  - Neoplasia.

FLUTD – Clinical Signs

- Dysuria (difficult urination)
- Pollakiuria (increased frequency of urination)
- Hematuria
- Agitation
- Vocalization (crying or howling)
- Owners perception?
  - Loss of house-training
  - Aggression
  - Constipation
  - Over-grooming
  - Abdominal pain

Urethral Plugs

- Colloid matrix leaks from the bladder wall as a result of inflammation.
- Combinations of a protein-colloid matrix
  - Mucoproteins
  - Albumin
  - Globulin
  - Cells
  - Blood clots
  - Crystalline material (struvite)

Infectious Causes?

- Rare cause of FLUTD
  - Older
  - Following urethral catheterization
  - Chronic renal failure (66%)
  - Reported to be easier to grow bacteria in dilute cat urine [specific gravity <1.025]
- Secondary to:
  - Urolithiasis
  - Neoplasia (TCC, ACA, Leiomyoma)
  - Anatomical defect of the urinary tract

How I Diagnose and Treat a Blocked Cat

- The severity and duration of the obstruction will determine the severity of the clinical signs.
- History
- Clinical Signs
- And examination….

Size does not matter…

- Physical examination

How do I handle the case?

- Exam
  - HR, RR, RE, MM, CRT
  - Temperature
- IV catheter
- Pull bloods
- Start fluids
- Doppler (?)
- Conversation with owners

Hyperkalemia – ECG Changes

Hyperkalemia – ECG Changes

- ECG Changes:
  - Peaked/narrow T-waves.
  - Widened QRS
  - Decreased or absent P-waves
  - Sine wave.
- Correlation between ECG changes and K+ are inconsistent:
  - Ca2+, Acid/base, Na+

Hyperkalemia

- Potassium plays a major role in cell function and neuromuscular transmission.
- Severity of the signs does not correlate with the magnitude of the change in the plasma potassium concentration
- Treatment should be guided by its functional consequences by monitoring the electrocardiogram.

Historical and physical parameters as predictors of severe hyperkalemia in male cats with urethral obstruction

Justine A. Lee, DVM, DACVECC and Kenneth J. Drobatz, DVM, MSCE, DACVECC, DACVIM

Journal of Veterinary Emergency and Critical Care **16**(2) 2006, pp 104–111
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Abstract

Objective: To evaluate selected historical and physical parameters as predictors of hyperkalemia in male cats with urethral obstruction.
Design: Retrospective study.
Setting: Veterinary teaching hospital.
Animals: Two hundred and twenty-three male cats.
Interventions: None.
Measurements and main results: The metabolic derangements of 223 male cats that presented with urethral obstruction from 1997 through 1999 were reported in a companion article. Approximately 12% of the cats had multiple, life-threatening metabolic derangements. In the present study, historical and physical parameters were evaluated as predictors of hyperkalemia ($K^+ \geq 8.0$ mmol/L) in cats with urethral obstruction. The 4 historical parameters significantly associated with hyperkalemia were: first time obstruction, outdoor status, anorexia, or vomiting. The 5 physical parameters significantly associated with hyperkalemia were: rectal temperature, heart rate, respiratory rate, pulse quality, and the presence of arrhythmia. Of the physical parameters, a rectal temperature below 95–96.6 °F (35–35.9 °C) or a heart rate below 120 b.p.m. were the most accurate predictors. When used in combination (i.e., evidence of bradycardia and hypothermia), the specificity for hyperkalemia was 98–100%.
Conclusions: Rectal temperature and heart rate were the best parameters for predicting hyperkalemia in this population.

Original Study

Journal of Veterinary Emergency and Critical Care **18**(1) 2008, pp 61–67
doi: 10.1111/j.1476-4431.2007.00268.x

Electrocardiographic assessment of hyperkalemia in dogs and cats

Tanya L. Tag, DVM and Thomas K. Day, DVM, MS, DACVA, DACVECC

Objective: To determine if electrocardiogram (ECG) changes induced by hyperkalemia in clinical patients correspond with previously reported changes in experimental animals.
Design: Prospective clinical study.
Setting: Two private practice 24-hour emergency and critical care facilities.
Animals: Fifteen dogs and 22 cats with serum potassium levels >5.5 mEq/L.
Interventions: None.
Measurements: The following data were collected when hyperkalemia was documented: ECG ($n = 37$), sodium and chloride (mEq/L) ($n = 35$), total magnesium (mg/dL) ($n = 18$), total calcium (mg/dL) ($n = 30$), and venous pH ($n = 18$). Animals were divided into five groups based on severity of hyperkalemia and ECG interpretation included rate, rhythm and P-QRS-T evaluation.
Main Results: Twenty-two of 37 (59%) of the ECGs were normal or revealed abnormalities that have not been previously described in conjunction with hyperkalemia. In dogs, there was no correlation ($r = 0$) between potassium blood levels and heart rate ($n = 15$). There was weak correlation ($r = 0.40$; $P = 0.06$) between potassium blood levels and heart rate in cats ($n = 22$). The correlation was stronger ($r = 0.64$; $P < 0.05$) when data were compared in cats with serum potassium level >8.5 mEq/L (Groups 4 and Group 5; $n = 11$).
Conclusions: ECGs obtained from ill dogs and cats with hyperkalemia are inconsistent with ECGs from experimentally induced hyperkalemia. It is difficult to determine the clinical relevance of heart rate differences between cats with serum potassium levels >8.5 mEq/L and animals with experimentally induced hyperkalemia; this may be due to the presence of other biochemical abnormalities in diseased animals.

Hyperkalemia - Treatment

- Fluid Therapy
  - Crystalloid
  - Correct dehydration
  - Correct hypovolemia
    - 0.9% NaCl, Norm-R, P-Lyte, LRS, etc.

Potassium free???

Original Study

Journal of Veterinary Emergency and Critical Care **18**(4) 2008, pp 355–361
doi: 10.1111/j.1476-4431.2008.00328.x

The influence of crystalloid type on acid–base and electrolyte status of cats with urethral obstruction

Kenneth J. Drobatz, DVM, MSCE, DACVIM (Medicine), DACVECC and Steven G. Cole, DVM, DACVECC, DACVIM (Cardiology)

Conclusions: While both crystalloid solutions appear safe and effective for fluid therapy in cats with urethral obstruction, the use of a balanced electrolyte solution may allow more rapid correction of blood acid–base status within the first 12 hours of fluid therapy. The use of a potassium-containing balanced electrolyte solution does not appear to affect the rate of normalization of blood potassium in treated cats with urethral obstruction.

Hyperkalemia – Calcium Gluconate

- Calcium gluconate 10% (diluted 1:1 with normal saline)
- Slow intravenous infusion over 5-10 minutes
- 3 cc/ cat
- Immediate (short-term) myocardial protection (no effect on K)
- Lasts 20-30 minutes.
- Can repeat dose if effects wear off.

Hyperkalemia – Insulin / Dextrose

- Insulin/Dextrose
- Regular insulin at 0.1 U/kg to 0.25 U/kg IV
- Followed by a glucose bolus of 0.5g/kg
- Begins lowering plasma potassium concentration within several minutes to one hour.
- Stimulates Na/K+ ATPase
- K+ moves intracellularly.
- Monitor BG

Hyperkalemia – Sodium Bicarbonate

- Sodium Bicarbonate
  - Raises pH
  - Drives potassium into the cells
  - Effects begin in 30 to 60 minutes and last for several hours

- Ketamine 5mg/kg
- Diazepam / Midazolam 0.2-0.3mg/kg
- Butorphanol 0.4mg/kg

Setup

Extruding the Penis

- DORSAL OR LATERAL recumbency
- Extend penis and displace dorsally …
- Urethra should be parallel to vertebral column

Tomcat Catheter

Advance catheter until resistance is met …

Flush and advance the catheter with gentle pressure.

Tomcat Catheter

Urinary Catheter Suture

Urinary Catheter Management

Tape catheter to tail and set up closed urine collection system …

Urinary Catheter Management

J Am Anim Hosp Assoc. 2008 Jan-Feb;44(1):2-4.

Aerobic bacterial culture of used intravenous fluid bags intended for use as urine collection reservoirs.

Barrett M[1], Campbell VL.

Author information

Abstract

Numerous studies have shown a relationship between indwelling urinary catheters and bacterial urinary tract infection. Some veterinary hospitals utilize stored, used intravenous (IV) fluid bags as part of the urine collection system. The authors cultured 95 such bags to see if they were potential sources of bacterial contamination. Forty-two unused IV bags were emptied of their contents for use as controls. Results indicated no aerobic bacterial growth in either group. The authors conclude that properly stored, used IV bags are unlikely sources of aerobic bacterial contamination when used in a urine collection system.

Radiograph

Initial treatment factors associated with feline urethral obstruction recurrence rate: 192 cases (2004–2010)

JAVMA, Vol 243, No. 4, August 15, 2013

Peter F. Hetrick, DVM, and Elizabeth B. Davidow, DVM, DACVECC

Objective—To evaluate the association of treatment factors during initial urinary catheterization (IUC) of cats with recurrence of urethral obstruction at 24 hours and 30 days after catheter removal.

Design—Retrospective case series.

Animals—192 male cats with urethral obstruction that were treated at an emergency and specialty center from 2004 through 2010.

Procedures—Data were obtained from the cats' medical records. Duration of indwelling catheterization, catheterization with a 5F versus 3.5F urinary catheter, treatment with phenoxybenzamine versus prazosin, consistent administration of pain medication, and treatment with meloxicam or antimicrobials during IUC were reviewed for association with rate of recurrent urethral obstruction (rUO).

Results—Overall rUO rates were 10.94% (21/192 cats) at 24 hours and 23.57% (37/157 cats) at 30 days after IUC. At 24 hours and 30 days after IUC, rUO developed in 10 of 140 (7.14%) and 20 of 110 (18.18%) prazosin-treated cats, respectively, compared with 10 of 46 (21.74%) and 16 of 41 (39.02%) phenoxybenzamine-treated cats, respectively. Reobstruction developed following use of a 5F or 3.5F urinary catheter in 11 of 58 (18.97%) and 7 of 105 (6.67%) cats, respectively, through 24 hours. There was no association between rUO and duration of urinary catheterization, administration of antimicrobials or meloxicam, or consistent administration of pain medication during IUC.

Conclusions and Clinical Relevance—At 24 hours and 30 days after IUC, rUO rates in prazosin-treated cats were significantly lower than rates in phenoxybenzamine-treated cats. Reobstruction rate at 24 hours was significantly lower when a 3.5F versus 5F urinary catheter was used. (*J Am Vet Med Assoc* 2013;243:512–519)

Recovery

-Recover with heat support
if T < 98 degrees
-Keep collection bag BELOW level of the patient

Urine

Sometimes the urine is normal in appearance..

Helpful Tips!

If initial attempts fail

- (1) decompression
- (2) re-attempt catheterization

PLEASE AVOID!!!

1. Grasping prepuce with hemostats
2. Suturing a "rigid" catheter in place
3. Performing the unblocking procedure without gloves / aseptic technique

Treatment Plan

- Fluid / IVC / RR/RE
- PCV/TS/BG/LYTES (+/- Creat)
- Food / H20
- E-Collar
- Buprenorphine
- Prazosin:0.25-1.0 mg/**cat** PO q8-12h
- Phenoxybenzamine: 0.5-1.0 mg/kg PO q12h--give for 5 days before evaluating efficiency.
- Cosequin?
- Ins/Outs
- Diet

Diagnostics

- The standard diagnostic work-up for a cat with lower urinary tract disease includes
  - bloodwork (CBC/CHEM)
  - Urinalysis (+/- urine culture & sensitivity)
  - Abdominal radiographs (+/- AUS)

Bacterial Infections

- Empirical vs. C&S
- Risk vs. benefit with placement of Urinary Catheter
- Concurrent disease?
  - Stones?
  - CRF?
  - Hypert4?
  - Traumatic / Contaminated catheter placement?

Long Term Management

Discharge?

- Usually these patients remain in the hospital for 36-72 hours.
- IV Fluids/diuresis until urine is clear, ins=outs, electrolyte, acid/base, fluid deficit corrected
- U-Cath is then removed and cat is monitored for a period of time (hours).
- Cat is then discharged for monitoring at home.

Post-Obstructive Diuresis

- **Post-obstructive diuresis** – usually happens in patients with a severe obstruction.
- Etiology – not completely known
  - Renal tubular damage?
  - Osmotic diuresis from osmotic particles that have been built up rather than excreted.
- Important to match ins/outs

Summary

- The majority of cats are relatively stable.
- Initial evaluation and therapy in critically ill cats may make the difference between life and death.
- Fluid deficits, electrolyte changes, and acid/base disturbances make treatment of these cats challenging and interesting.

Questions?

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Where is VetGirl going to be? Check out our upcoming 2014 lectures here:

Dr. Justine Lee

- April 1, 2014: Connecticut Veterinary Medical Association, Hartford, CT.
- April 22-24, 2014: Russian Veterinary Conference, Moscow, Russia
- May 1-4, 2014: International Veterinary Seminars, New Orleans, LA.
- May 13, 2014: New Jersey Veterinary Medical Association, NJ.
- June 19, 2014: Minnesota Veterinary Medical Association, Saint Paul, MN

Dr. Garret Pachtinger

April 29, 2014: Delaware Veterinary Medical Association, Dover Downs, DE

May 8, 2014: Pennsylvania Veterinary Medical Association, Lancaster, Pa

August 16-18, 2014: Pennsylvania Veterinary Medical Association Keystone Veterinary Conference in Hershey, PA

September 5, 2014: Leon Veterinary Conference, Guadalajara, Mexico

September 24-28, 2014: Southwest Veterinary Symposium, Ft. Worth, Texas