Diagnosis of acute kidney injury in cats

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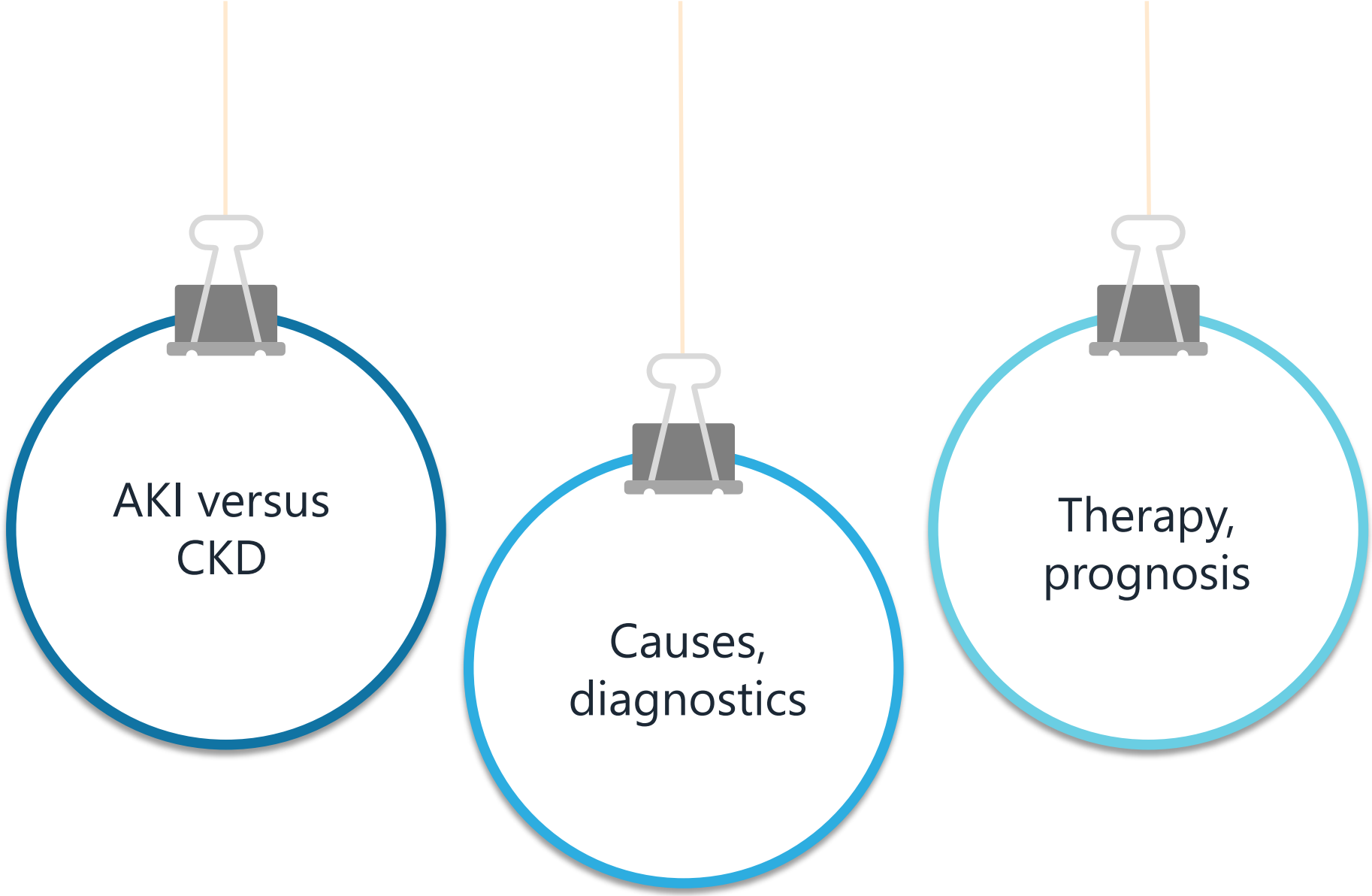

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Contrasting AKI and CKD

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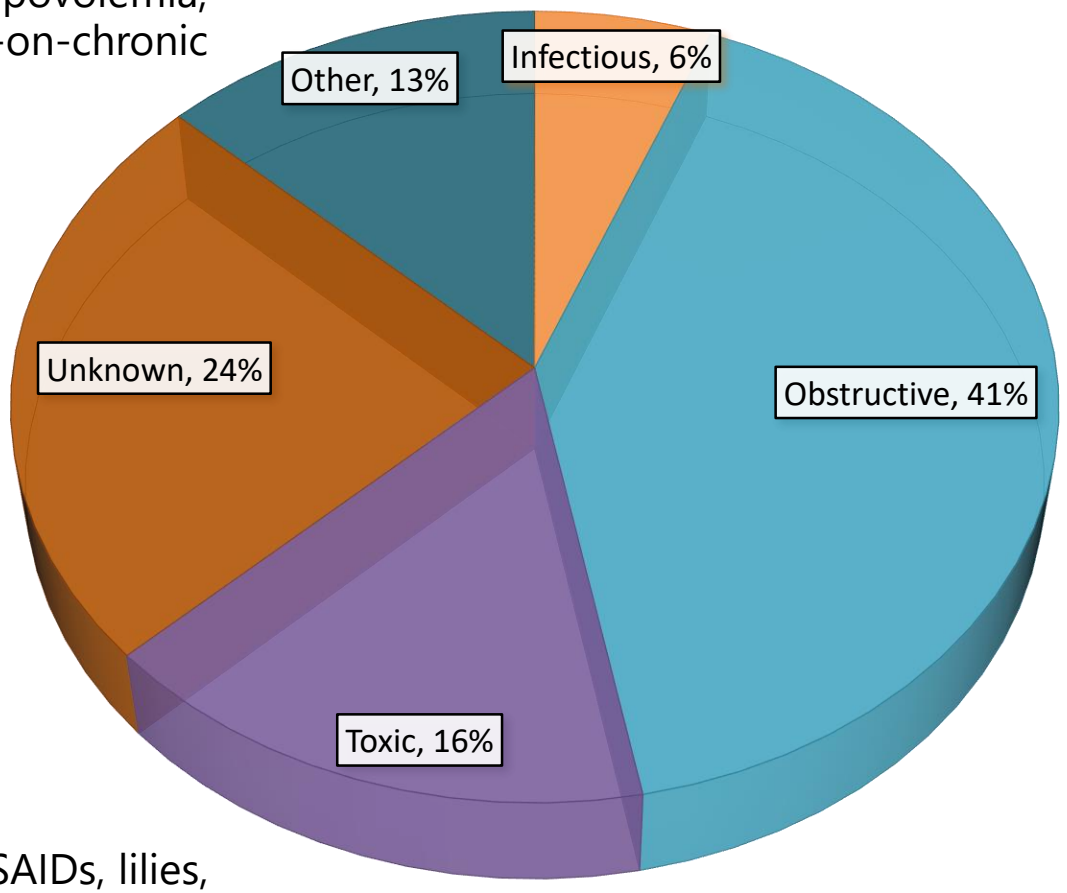
Causes of AKI in cats



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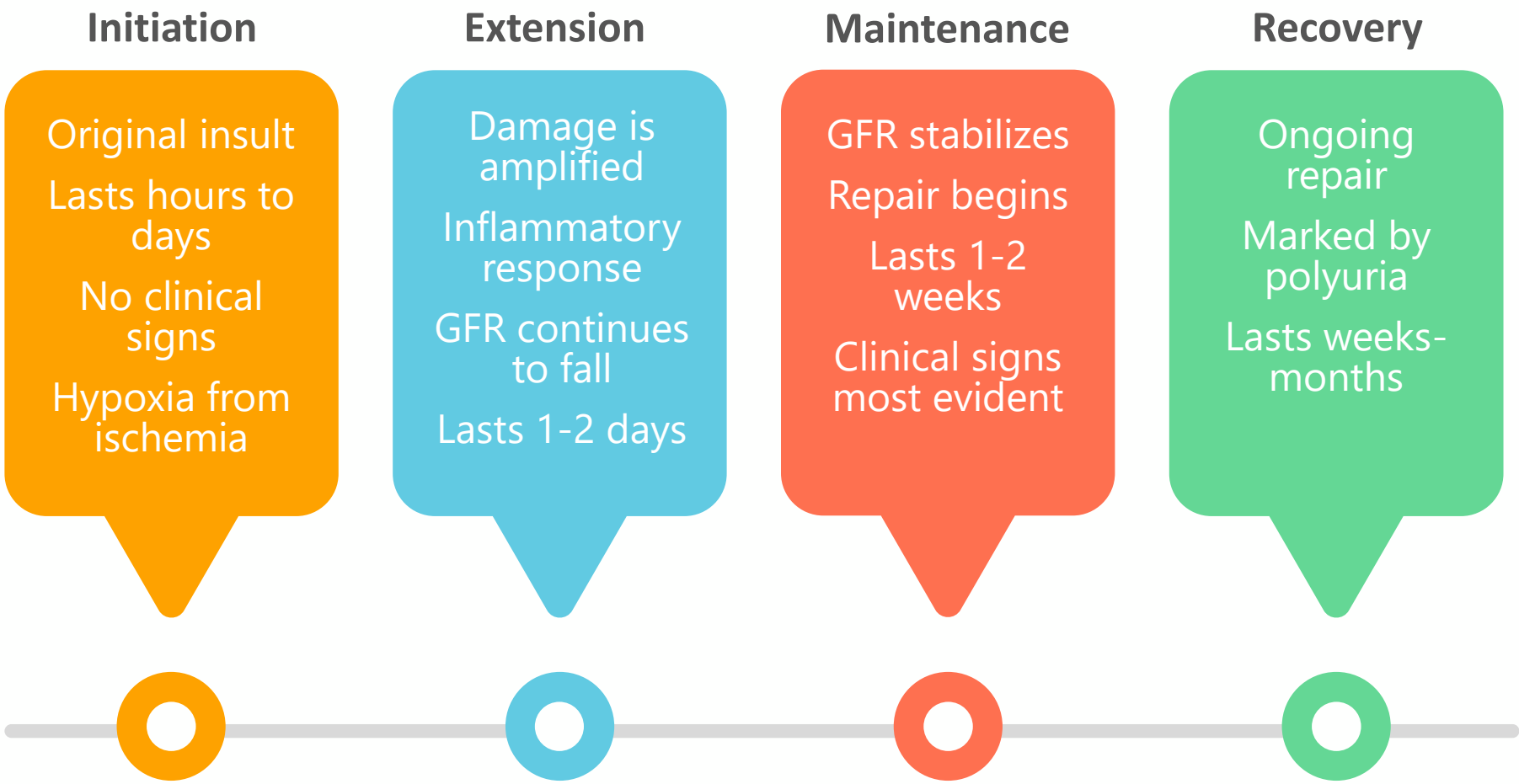
Pathophysiology



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Making the diagnosis

HISTORY

Don't forget drug/toxin exposures, environment

LAB DATA

CBC, chemistries, urinalysis, electrolytes, blood gases (if available)

EXAM, CLINICAL SIGNS

Differentiate AKI from CKD
Check blood pressure

IMAGING

Radiographs, ultrasound

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Clinical signs

- Sudden onset lethargy, collapse, vomiting
- Neurological signs
- Bradycardia, ECG abnormalities
- +/- Reduced urine production

Acute kidney injury		Chronic kidney disease
No previous history of kidney disease* Toxin/drug exposure Oliguric or anuric	**HISTORY**	Polyuria/polydipsia Hyporexia
No weight loss Kidneys normal to enlarged May be painful	**PHYSICAL EXAM**	Weight loss Dehydration Kidneys small, firm, nonpainful

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Chemistries

- May be initially normal or azotemia
- Rising creatinine over time
- Variable hyperphosphatemia
- Hyperkalemia (oligu/anuria, obstruction, etc.)

Urinalysis

- Isosthenuria
- Other findings may help determine etiology: proteinuria, pyuria, casts, etc.

Complete blood count

- Nonspecific
- Stress or inflammatory changes
- Nonregenerative anemia is uncommon (acute gastric ulceration)

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Definition of acute kidney injury

01

↑ serum creatinine by 0.3 mg/dL (26.5 µmol/L) within 48 hours

02

↑ serum creatinine to 1.5 times baseline within 7 days

03

Urine output <0.5mL/kg/hour for 6 hours (least reliable)

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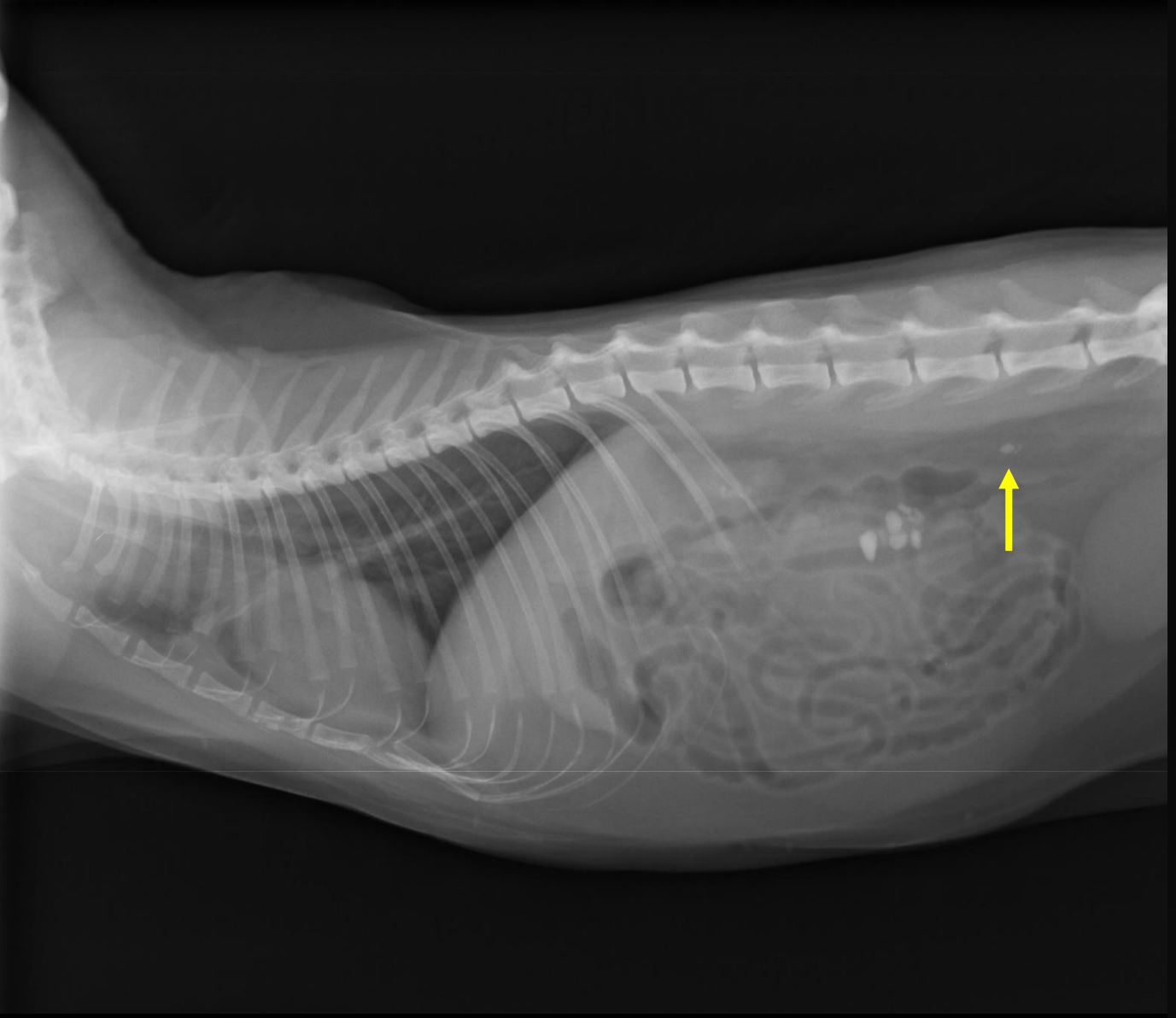

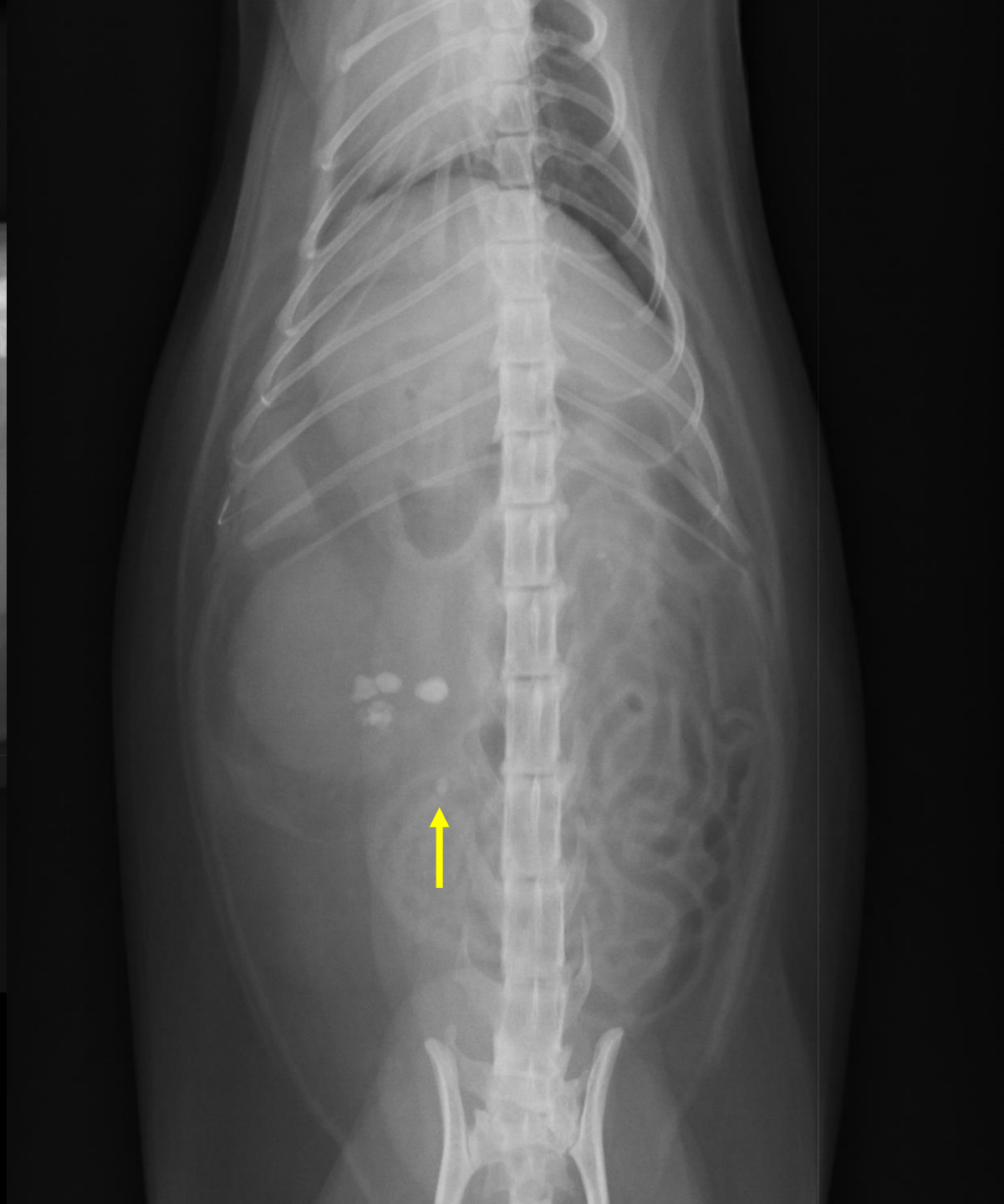

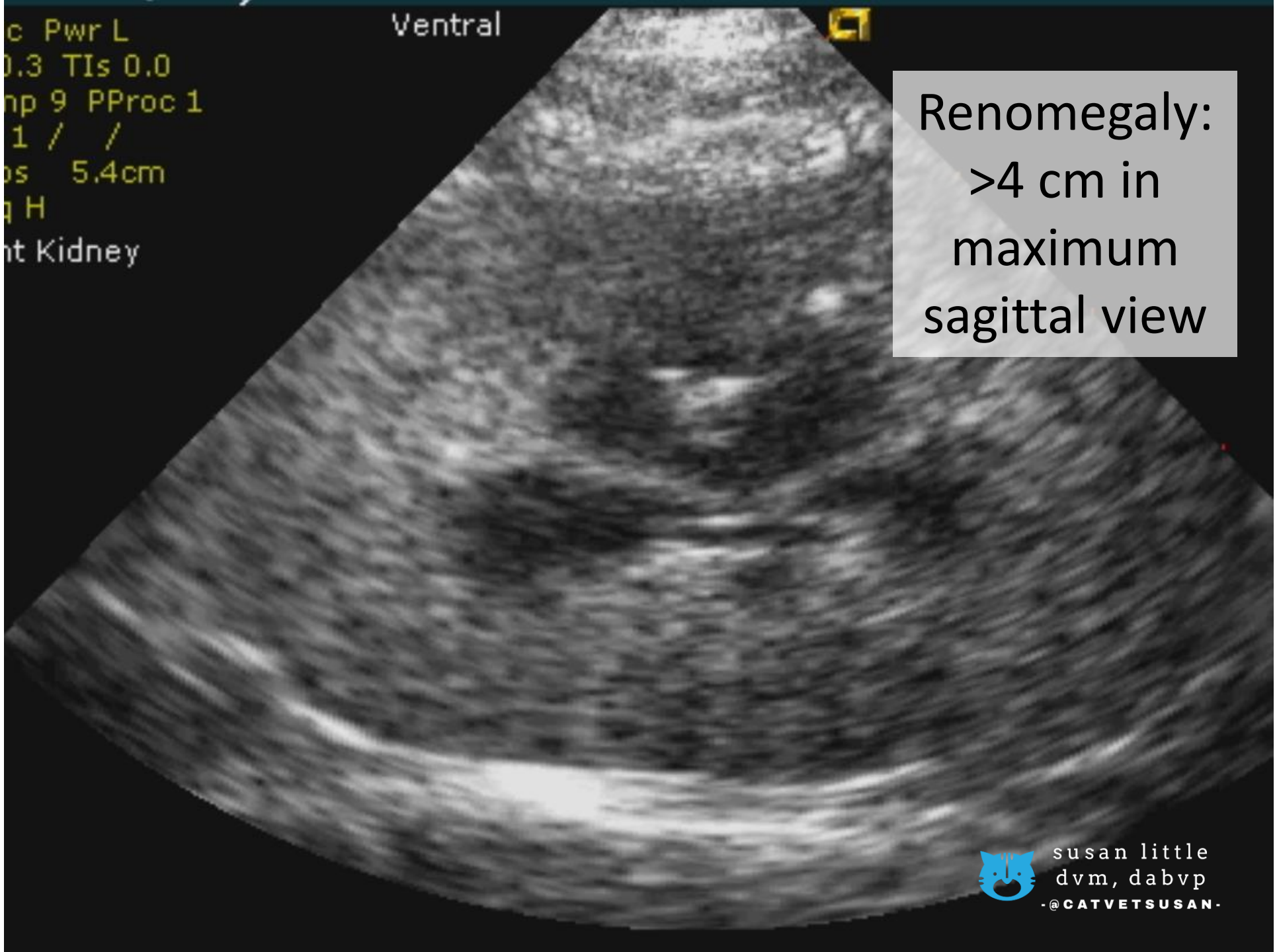
c Pwr L
0.3 TIs 0.0
np 9 PProc 1
1 / /
os 5.4cm
q H
nt Kidney
Ventral
Renomegaly: >4 cm in maximum sagittal view
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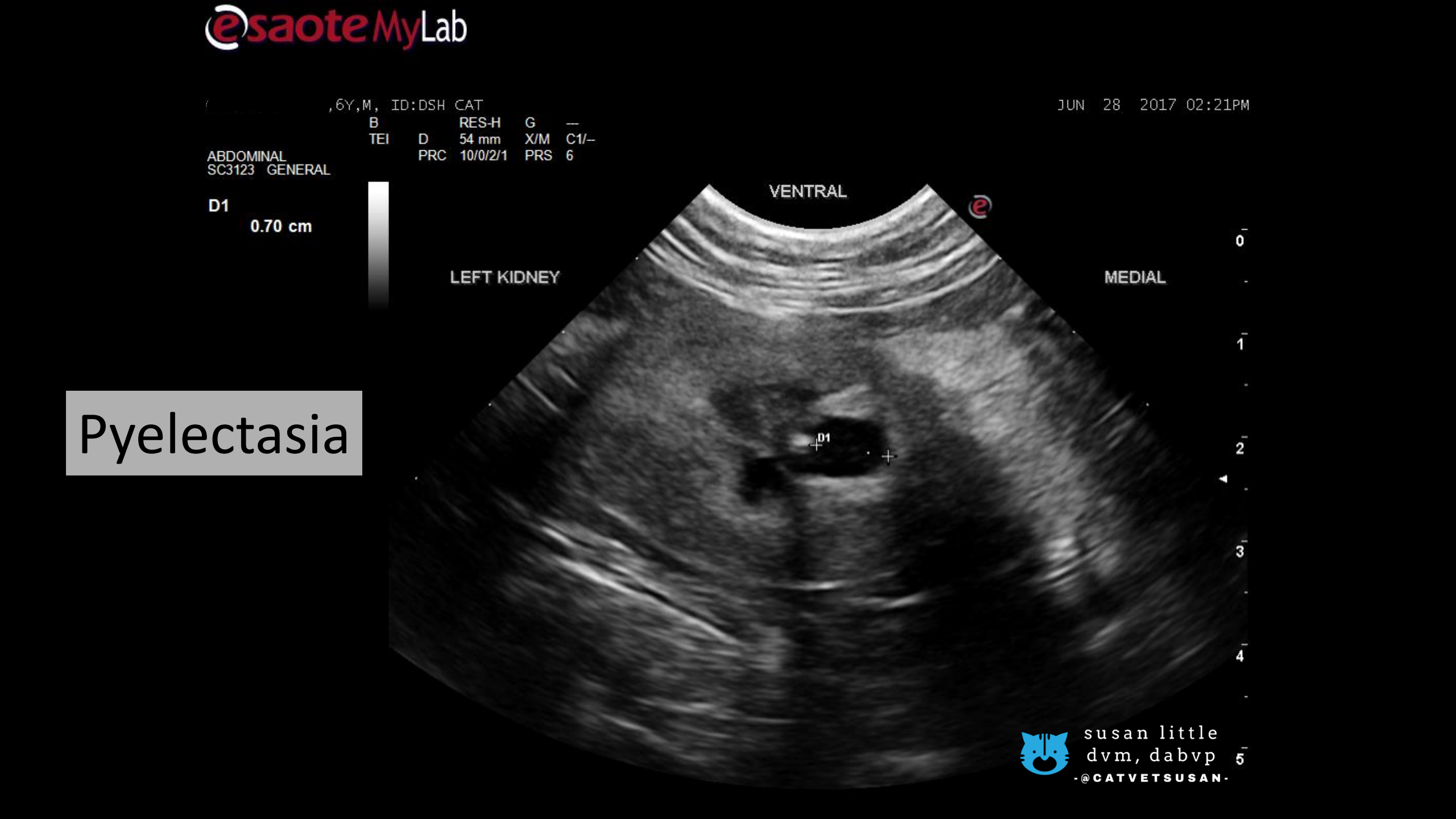
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B RES-H G —
TEI D 54 mm X/M C1/–
PRC 10/0/2/1 PRS 6
ABDOMINAL
SC3123 GENERAL
D1
0.70 cm
VENTRAL
LEFT KIDNEY
MEDIAL
D1
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2
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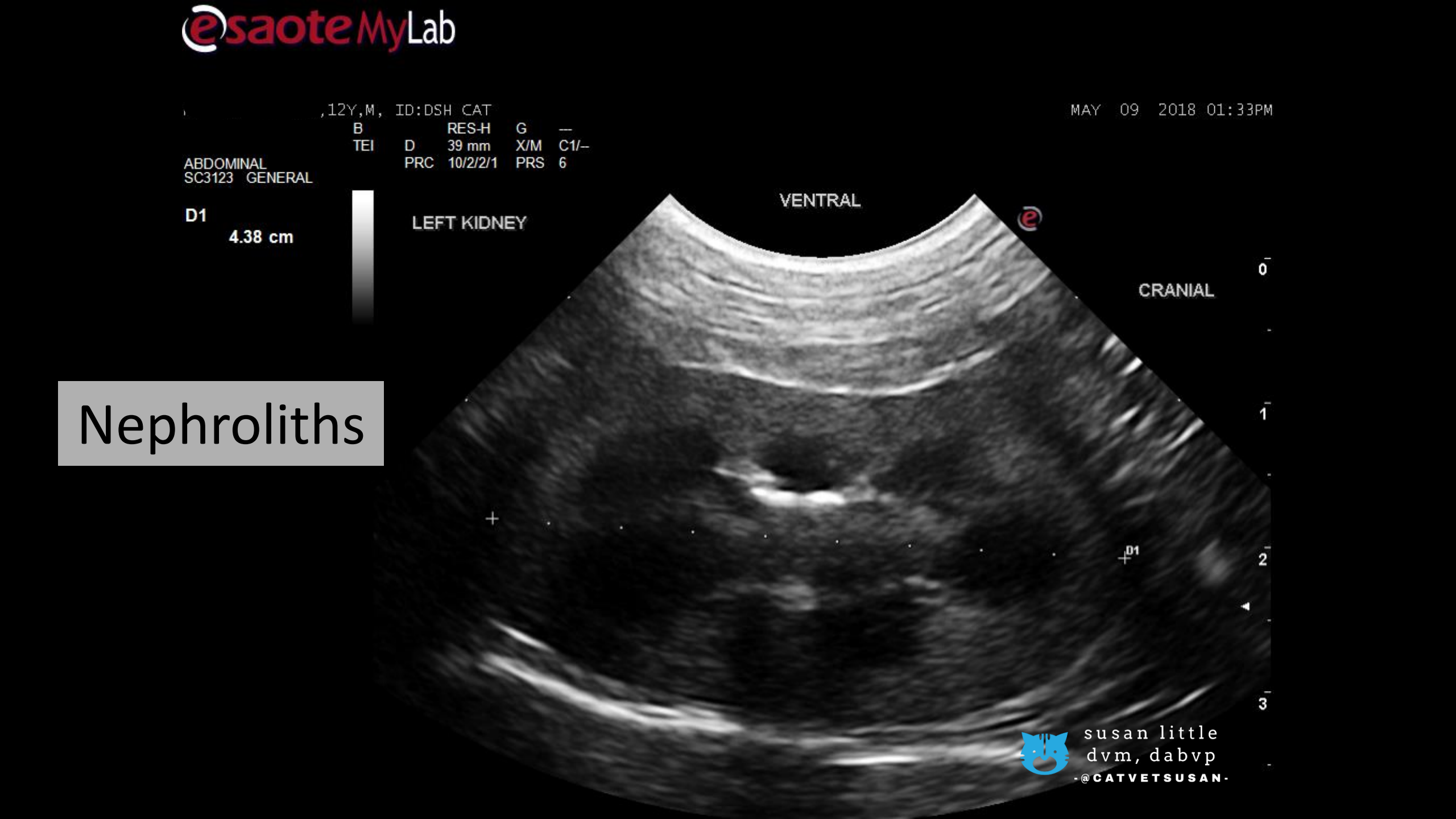
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SC3123 GENERAL
D1
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Nephroliths
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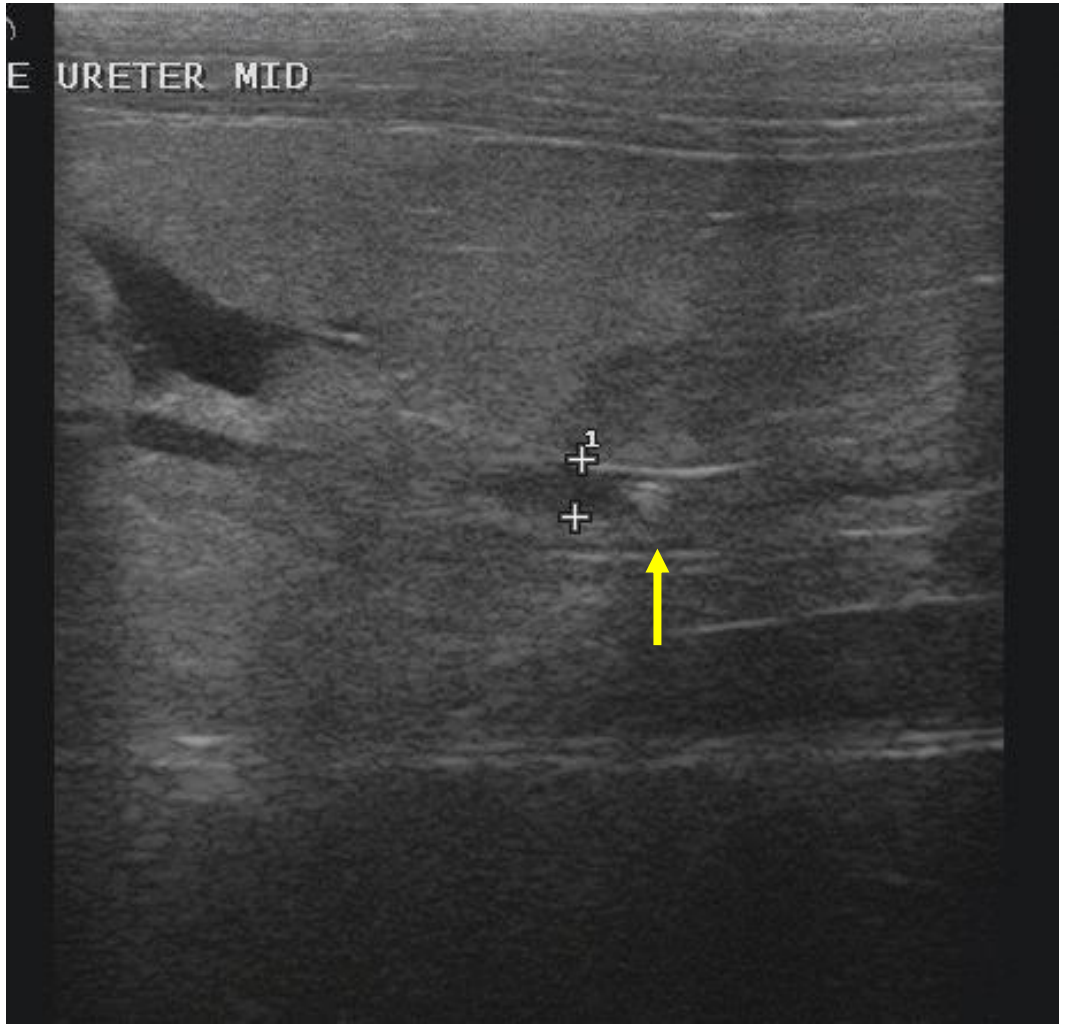

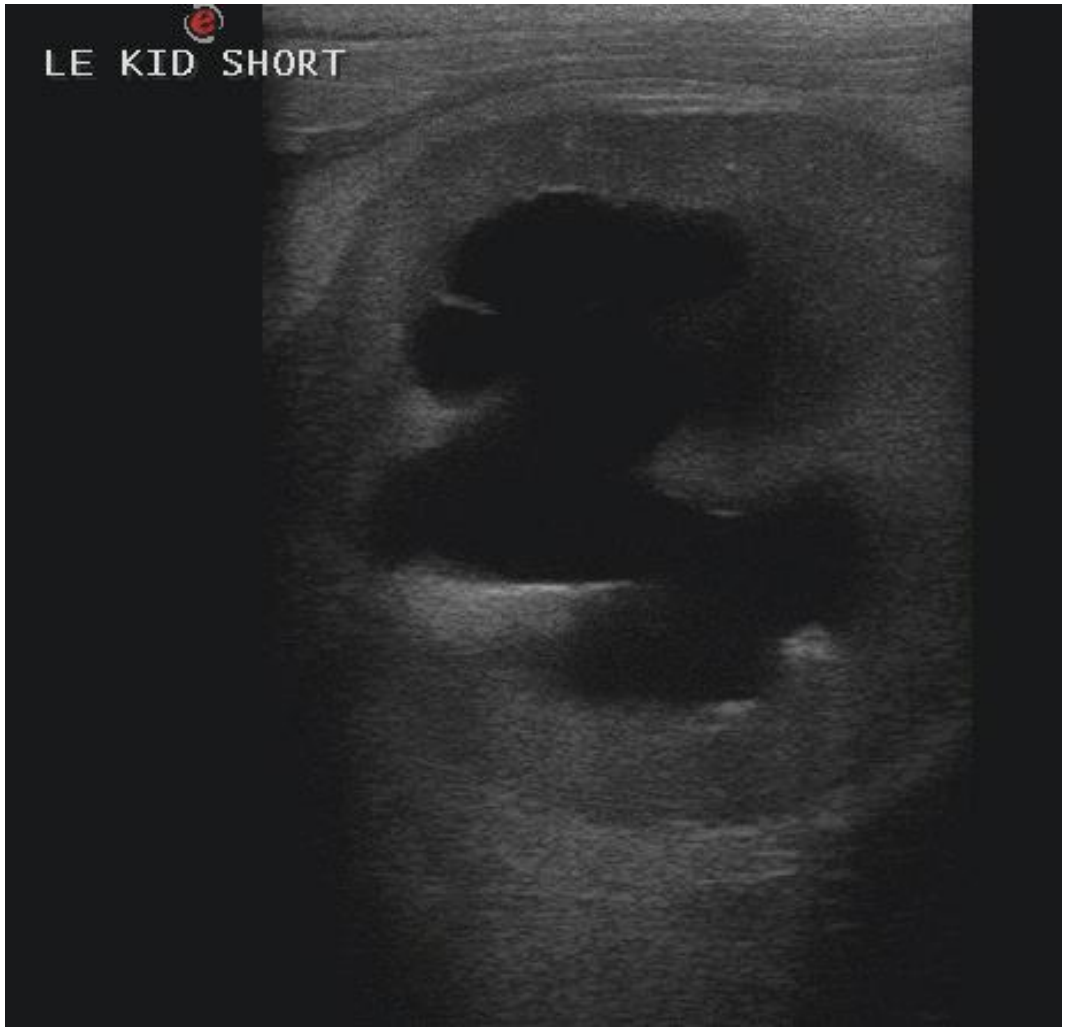

Monaghan K et al, J Feline Med Surg, 2012

Other ultrasound changes in AKI

01 Retroperitoneal fluid

02 Increased cortical +/- medullary echogenicity

03 [Corticomedullary junction changes]

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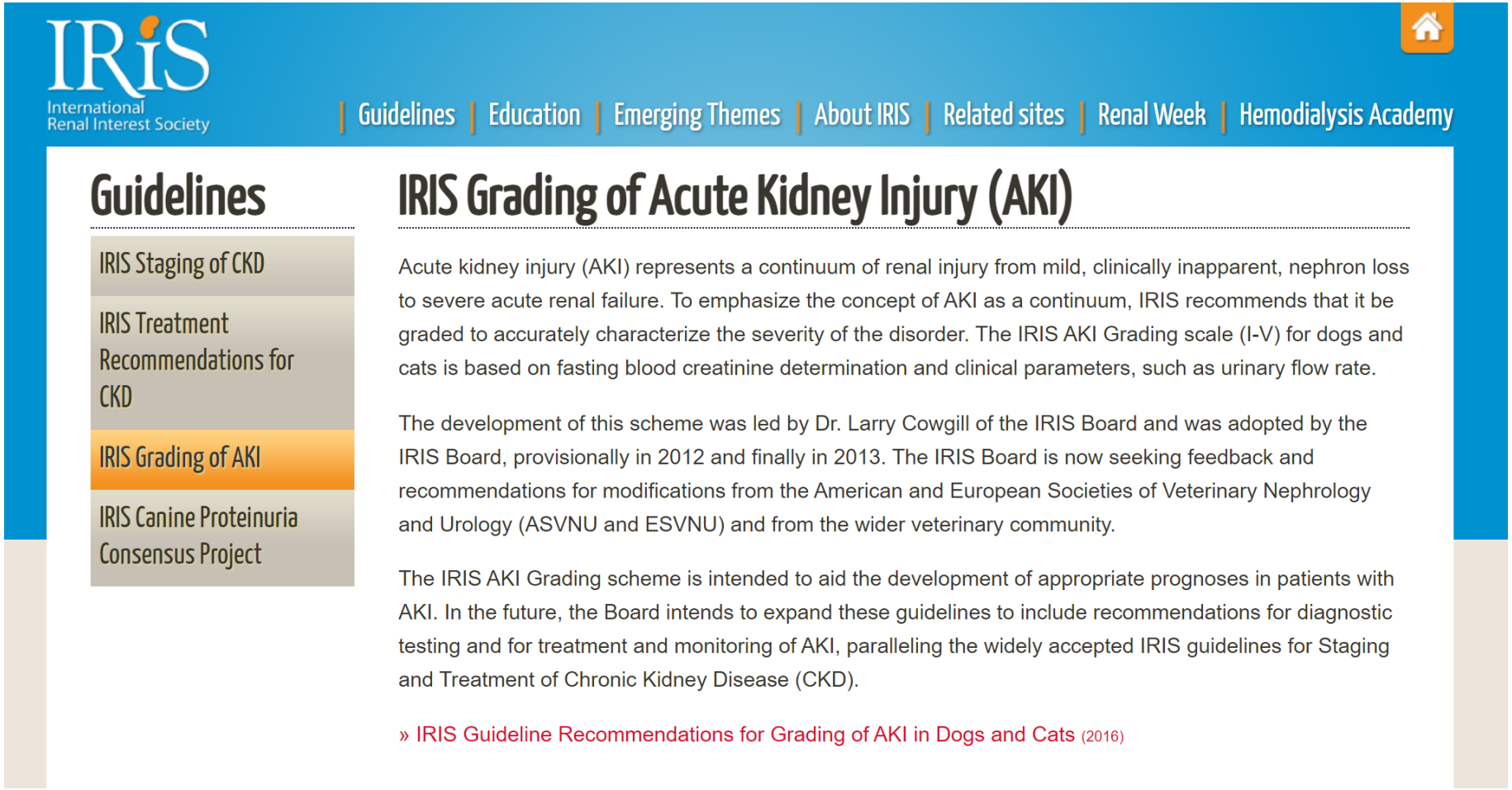
www.iris-kidney.com



Why should we grade AKI?

01 Understand that AKI is a continuum of renal injury

02 Take advantage of lessons from human nephrology

03 Discriminate pathophysiologic & therapeutic spectrum

04 Earlier recognition of AKI

05 Better prediction of outcomes

06 Better data collection to assess treatment outcomes

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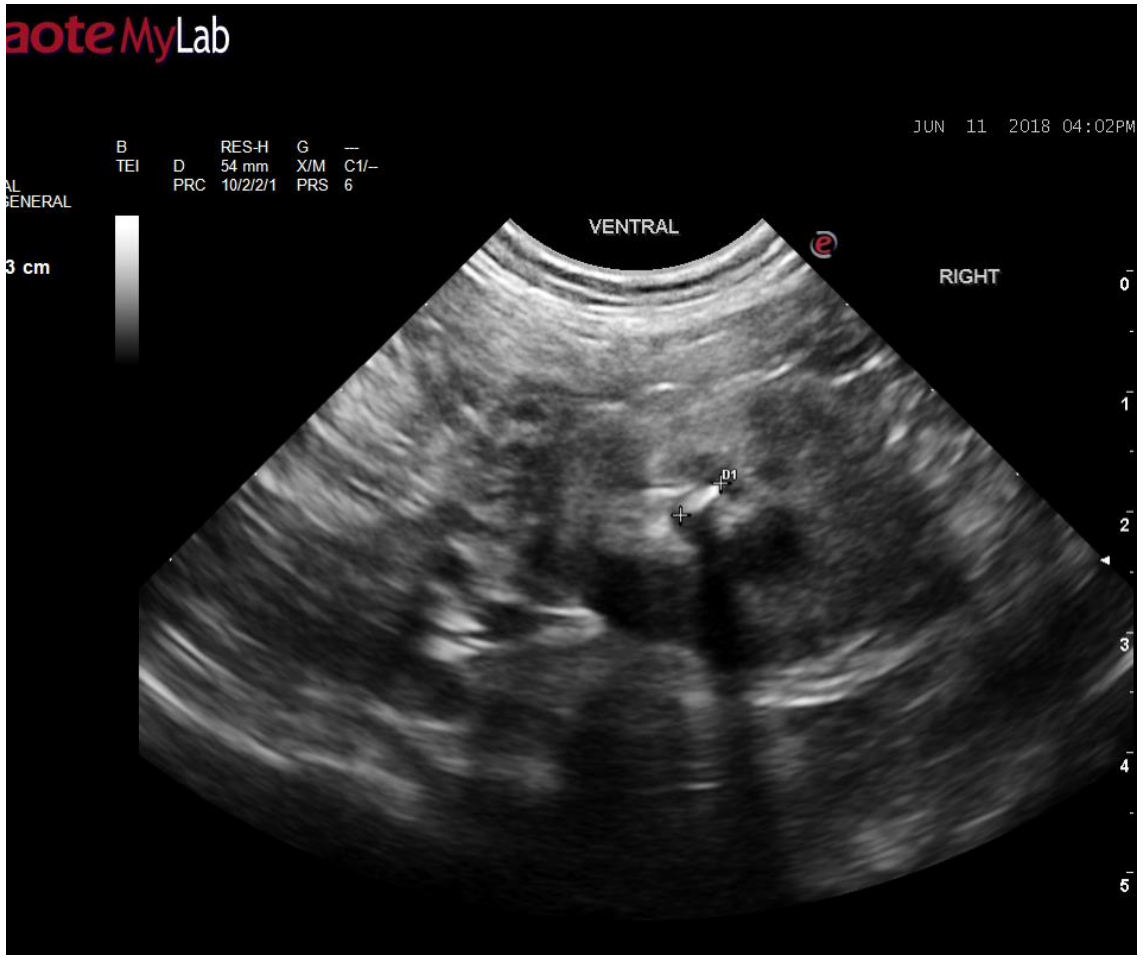
AKI Grade	Creatinine mg/dL [µmol/L]	Description
Grade 1	<1.6 [<140]	**Non-azotemic AKI** a) Documented AKI and/or b) Progressive non-azotemic ↑ in creatinine by ≥0.3 mg/dL [≥26.5 µmol/L] within 48 hours c) Oliguria (<1 mL/kg/hour) or anuria over 6 hours
Grade 2	1.7-2.5 [141-220]	**Mild AKI** a) Documented AKI + static or progressive azotemia b) Progressive azotemia: ↑ creatinine by ≥0.3 mg/dL [≥26.5 µmol/L] within 48 hours or volume unresponsiveness c) Oliguria (<1 mL/kg/hour) or anuria over 6 hours

AKI Grade	Creatinine mg/dL [µmol/L]	Description
Grade 3	2.6-5.0 [221-439]	**Moderate to severe AKI:** Documented AKI and increasing severities of azotemia & functional renal failure.
Grade 4	5.1-10.0 [440-880]	
Grade 5	>10.0 [>880]	
Each grade is further sub-graded as: • NO (non-oliguric) or O (oligo-anuric) • RRT (requiring renal replacement therapy		

Ginger



- 8 year old spayed female
- Lethargy, poor appetite
- History of renal & ureteral stones
- Previously diagnosed with IRIS CKD stage 2

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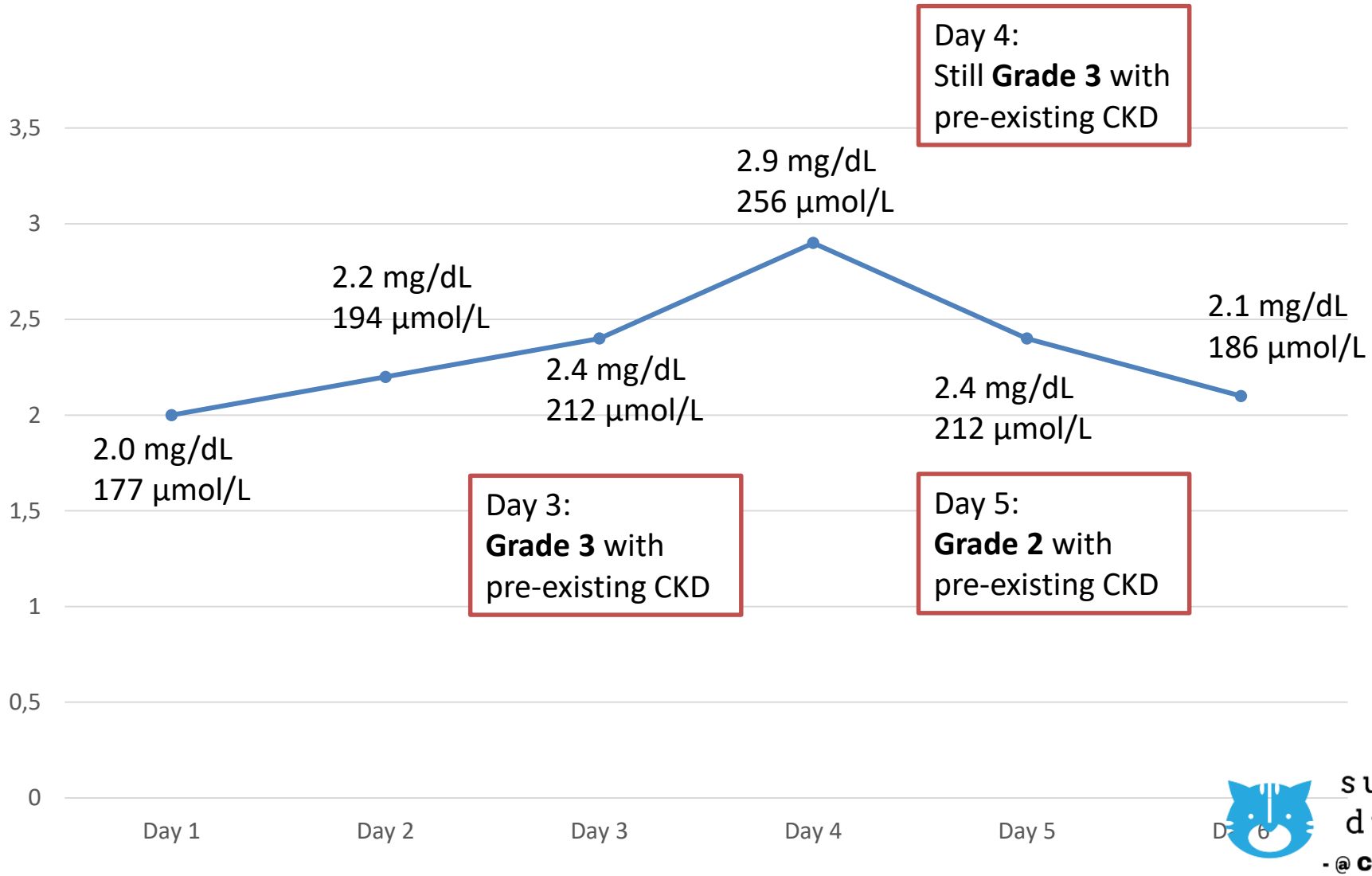
Ginger's serum creatinine



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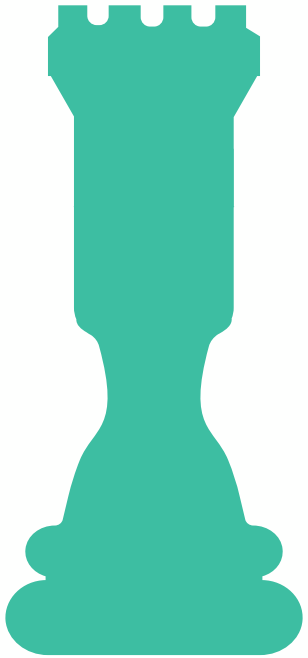
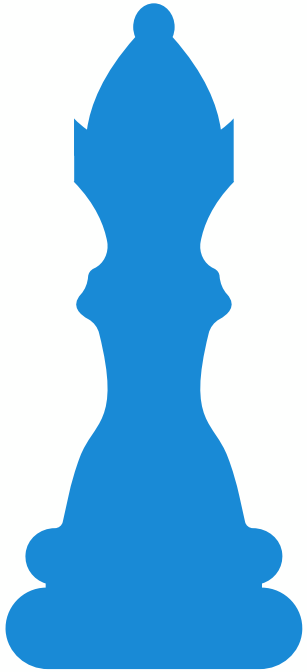
Intensive care is needed for the best outcome



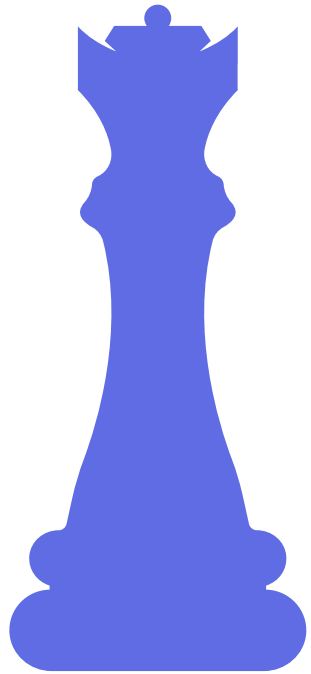
Fluid therapy

Urine output



Frequent monitoring

Constant rate infusions



Dialysis

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Keys to managing AKI

01

Specific therapy when possible

02

Correct hypovolemia, fluid deficits

03

Restore urine output

04

Correct acid-base & electrolyte disturbances

05

Treat hypertension if present

06

Supportive care

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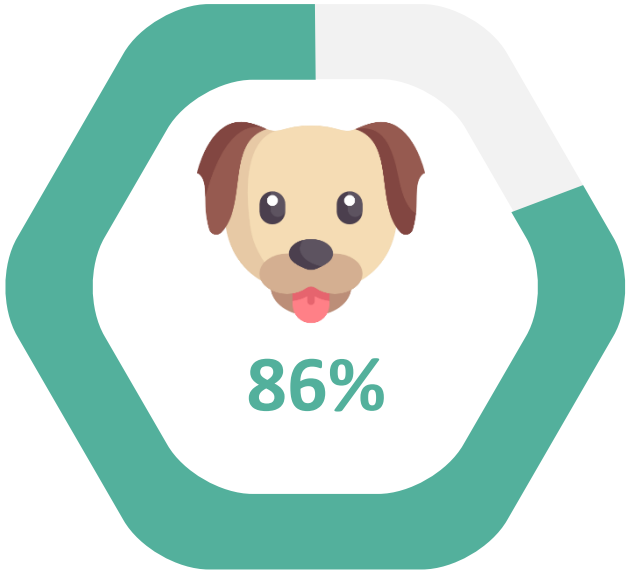
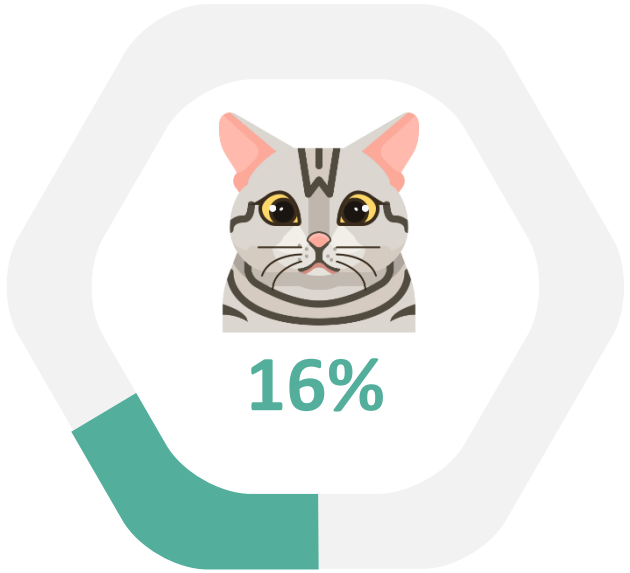
Companion animal exposures to potentially poisonous substances reported to a national poison control center in the United States in 2005 through 2014



Chocolate!

versus



Lilies

Swirski AL et al. J Am Vet Med Assoc 2020

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Lily toxicity

- Almost all lily species are toxic to cats
- Exposure to any part of the plant is toxic
- Must treat as quickly as possible!

Clinical signs of lily toxicity

- Salivation
- Vomiting
- Anorexia
- Depression
- Polyuria > oliguria > anuria
- Death in 3-6 days

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Treating lily toxicity

INDUCE VOMITING

Xylazine 0.4-0.5 mg/kg, IV/IM
Dexmedetomidine 7 µg/kg IM

DECONTAMINATE

Activated charcoal once rehydrated, 1-2 g/kg every 4-6 hours, up to 4 doses

IV FLUIDS

Replacement solution; monitor for overhydration

REASSESS & ADJUST

Renal values, electrolytes, urine output

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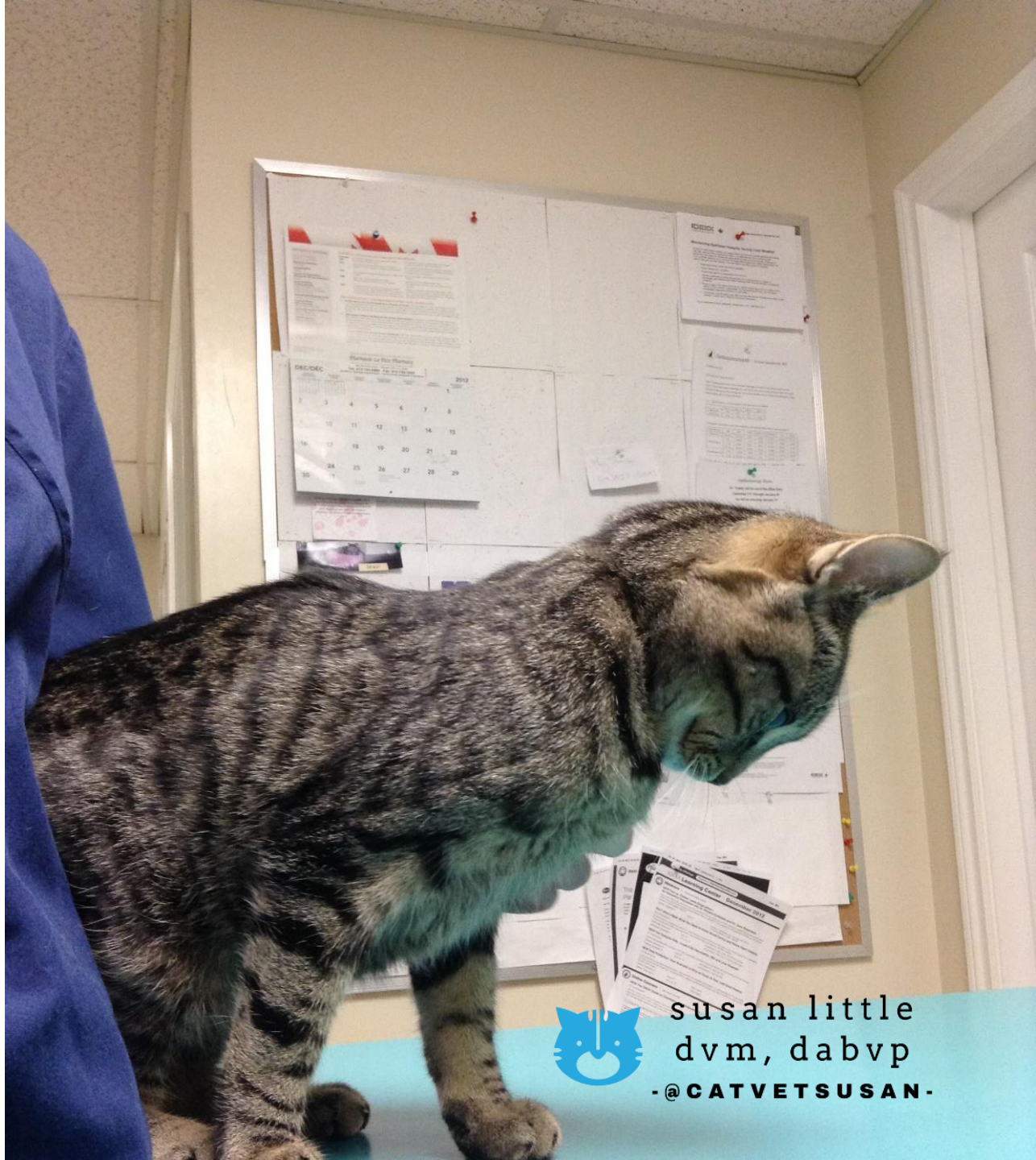
Scout

- Spayed female, **8 months old**
- Poor appetite, polyuric/polydipsic for a few days
- Weak, cervical ventroflexion



	Scout's result	Reference range
ALP	33	14-111 U/L
ALT	64	12-130 U/L
Urea	**23.2**	5.7-12.9 mmol/L
Creatinine	**509**	71-212 µmol/L
Glucose	5.35	4.11-8.83 mmol/L
Potassium	**2.9**	3.7-5.2 mmol/L
Total Calcium	**2.87**	1.95-2.83 mmol/L

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Fluid therapy + careful monitoring

- Use replacement solution
- % dehydration x body weight (kg) = deficit (L)
- Replace deficit over 4-12 hours
- Duration? Start weaning when hydration is normal & renal values have plateaued for 24 hours

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Avoid overhydration!

01 Easy to overhydrate cats

02 Increases morbidity & mortality

03 Congestive heart failure
Pulmonary edema
Hypertension

04 Renal edema & secondary anuria*

05 Reassess often & adjust as needed

06 Forcing diuresis is controversial!

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Overhydration

Early signs

Trembling
Restlessness
Excitation
Vocalization
Panting
Tachypnea
Vomiting
Nausea
Polyuria

Late signs

Tachy- or bradycardia
Nasal discharge
Chemosis
Coughing, dyspnea
Subcutaneous edema
Diarrhea
Exophthalmia
Depression
Pulmonary edema
Pleural effusion

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Monitoring fluid therapy

Urine output

Urinary catheter or material to soak urine (weigh before & after urination)

Body weight

Weight at least 3 times/day
1 kg weight gain = 1 L fluids

Central venous pressure

Trends over time are most helpful

PCV & total solids

Crude measurement
Influenced by other things

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The terms **'oliguria'** and **'anuria'** are only applicable to a well-hydrated patient that is appropriately volume resuscitated.

Monaghan K, et al. Feline acute kidney injury: 2. Approach to diagnosis, treatment and prognosis. J Feline Med Surg. 2012 Nov;14(11):785-93.

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Restore urine output if oliguric or anuric

Only after hypovolemia & dehydration are corrected!

01

DRUGS

Furosemide
1-2 mg/kg, IV bolus
0.25-1 mg/kg/hour CRI

Mannitol (20%)
1-2 mg/kg/min CRI

*Fenoldopam
0.1-1 µg/kg/min CRI

02

DIALYSIS

Best hope for anuric cats

03

CRITICAL CARE

Intensive monitoring is needed

Urine output goal

- >1-2 mL/kg/hour
- 2-5 mL/kg/hour is ideal

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Renal replacement therapy

Indications

- Oliguric/anuric despite therapy
- Refractory to fluid therapy
- Severely uremic
- Life-threatening electrolyte abnormalities
- Volume overload
- Certain toxins

Options

- Peritoneal dialysis
- Intermittent hemodialysis
- Continuous renal replacement therapy

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Supportive care

01

Antiemetics

Maropitant: 1 mg/kg every 24 hours (PO, IV, SC)
Ondansetron: 1 mg/kg every 6-8 hours (PO, IV, SC)

02

Gastroprotectants

Omeprazole: 1 mg/kg every 12 hours (PO)
Pantoprazole: 1 mg/kg, slow IV, every 24 hours

03

Nutrition

Feeding tube may be needed

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Prognosticating

Overall mortality is 47-64%

Bad things

- Oliguria/anuria
- Increased potassium
- Decreased albumin

Good things

- Increase in urine production to >1 mL/kg/hour over 6 hours
- Decreasing BUN & creatinine within 3 days

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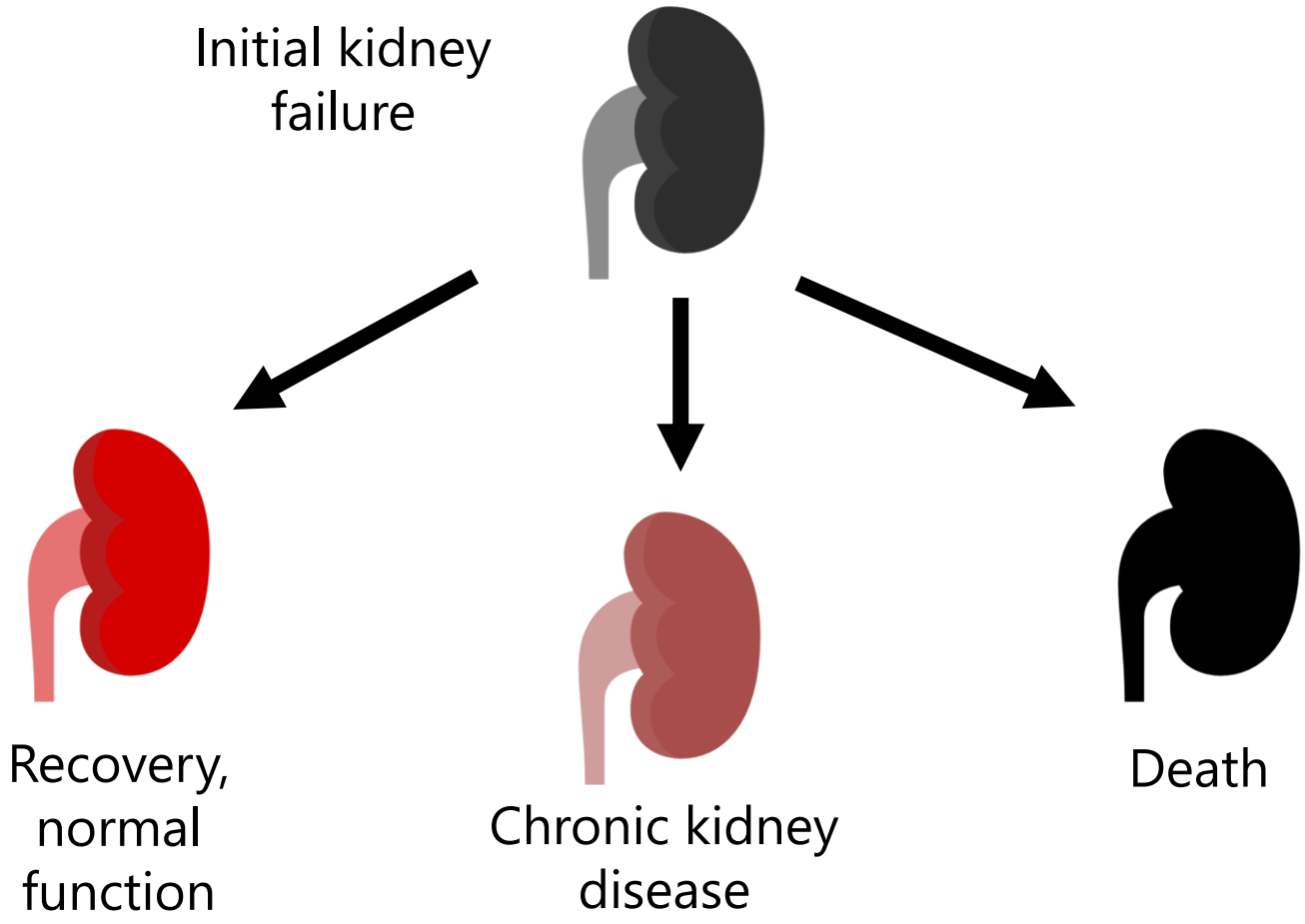
Acute kidney injury - potential outcomes



- Depends on cause, time to presentation, response to treatment
- Degree of azotemia is not prognostic

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Journal of Feline Medicine and Surgery (2012) **14**, 775–784

CLINICAL REVIEW

FELINE ACUTE KIDNEY INJURY
1. Pathophysiology, etiology and etiology-specific management considerations

Kelly Monaghan, Benjamin Nolan and Mary Labato

New terminology – and a new emphasis

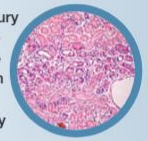
Acute kidney injury (AKI) is a relatively new term in the nephrology literature that largely replaces the use of 'acute renal failure' (ARF). Originally introduced in the human literature, AKI allows for greater stratification of cases with regard to severity and prognosis. By suggesting that a patient has injury, rather than failure, one can recognize the potential for earlier treatment and recovery. In human medicine, the older term 'ARF' refers only to patients requiring renal replacement therapy (RRT). Additionally, the more common term 'kidney', rather than 'renal', facilitates communication with and understanding by clients of AKI.

The incidence of AKI in cats is not known, but it is not uncommon and can be caused by a variety of different insults. A uniform definition for AKI does not exist in the veterinary literature and varies among publications. Generally accepted criteria include an abrupt reduction in kidney function resulting in alterations in glomerular filtration, urine production and tubular function. These alterations result in an inability to maintain fluid, electrolyte and acid–base balance, and may lead to azotemia.

Acute kidney injury is a relatively new term that largely replaces the use of 'acute renal failure' and recognizes the potential for earlier treatment and recovery.

Pathophysiology

The pathophysiology of AKI is complex, but can be described by four stages: initiation, extension, maintenance and recovery.



Practical relevance: Acute kidney injury (AKI) is a frequently recognized disease process in cats that requires immediate and aggressive intervention. A thorough understanding of the pathophysiologic processes underlying AKI and familiarity with the most common etiologies are essential for providing the most effective and timely therapy. Possessing this knowledge will also allow a more accurate prognosis to be given, and afford the best chance of a favorable outcome.

Clinical challenges: Feline patients often present with vague signs of AKI, which may delay treatment and adversely affect the prognosis. Their response to injury and treatment is often different to that of other species.

Audience: This two-part review article is directed at small animal practitioners as well as specialists. Part 1 reviews mechanisms underlying AKI in the cat, as well as etiologies and treatments related to some specific causes of AKI.

Evidence base: The veterinary literature is limited with regards to the pathophysiology of AKI unique to the cat. However, there are numerous feline studies evaluating causes of AKI.

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PART 2
Part 2 of this review article, discussing more general aspects of diagnosis, and setting out an approach to treatment, appears on pages 785–793 of this issue of *J Feline Med Surg* and at:
DOI: 10.1177/1098612X12464460

Journal of Feline Medicine and Surgery (2012) **14**, 785–793

CLINICAL REVIEW

FELINE ACUTE KIDNEY INJURY
2. Approach to diagnosis, treatment and prognosis

Kelly Monaghan, Benjamin Nolan and Mary Labato

Prompt recognition – a priority

Acute kidney injury (AKI) is a term used to describe the condition in which there is an abrupt reduction in renal function. This can occur due to several causes, as discussed in Part 1 of this article. In all cases prompt recognition of AKI is important to maximize the chance of a favorable outcome.

Diagnosis

Diagnosis of AKI should focus on attempts to identify an underlying cause and establish the severity of disease.

History and physical examination

A thorough history should be obtained from the owner regarding time course, previous therapies, medication history and potential exposure to toxins. Physical examination may reveal various degrees of lethargy and depression depending on the severity of systemic illness. With severe disease, patients may have oral ulceration and a 'uremic breath' odor (ammonia-like smell). Melena may be noted in patients with secondary gastrointestinal ulceration and bleeding. Attention should be paid to the size of the urinary bladder to evaluate for obstruction as well as hint towards urine production. Kidneys are often palpably normal or enlarged, and may be painful. A patient with a renal tumor or a ureteral obstruction may have asymmetry in renal size and shape. However, it should not be forgotten that patients with AKI may have pre-existing chronic kidney disease (CKD) as well and this must be considered when evaluating physical examination findings, as well as laboratory and imaging results.

Patients with AKI may have pre-existing chronic kidney disease as well.

Practical relevance: Feline acute kidney injury (AKI) is a commonly recognized problem in small animal practice that requires prompt diagnosis and directed therapy. There are many treatment methods with which practitioners should be familiar, including medical options, surgical interventions and renal replacement therapy (dialysis). It is important to know which option is most appropriate for each cause and stage of AKI to deliver the most effective therapy.

Clinical challenges: AKI can cause vague clinical signs, but a vast array of life-threatening sequelae. Rapid recognition of potential complications and knowledge of treatment options is imperative for successful management. Feline patients also require an understanding of their unique physiology as it relates to the therapeutic plan.

Audience: This two-part review article is directed at small animal practitioners as well as specialists. Part 2 discusses the diagnosis of AKI in cats using physical examination findings, clinicopathologic results and imaging modalities. The treatment of AKI and its sequelae is also reviewed, with information on recent advances in this area.

Evidence base: While there is very limited data comparing the outcomes of various treatment options, there is literature addressing the use of several medications, as well as renal replacement therapy, in cats.

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PART 1
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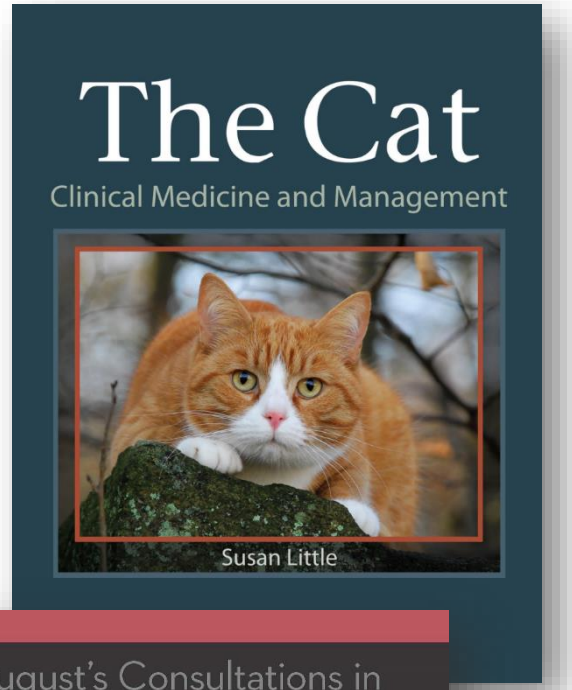
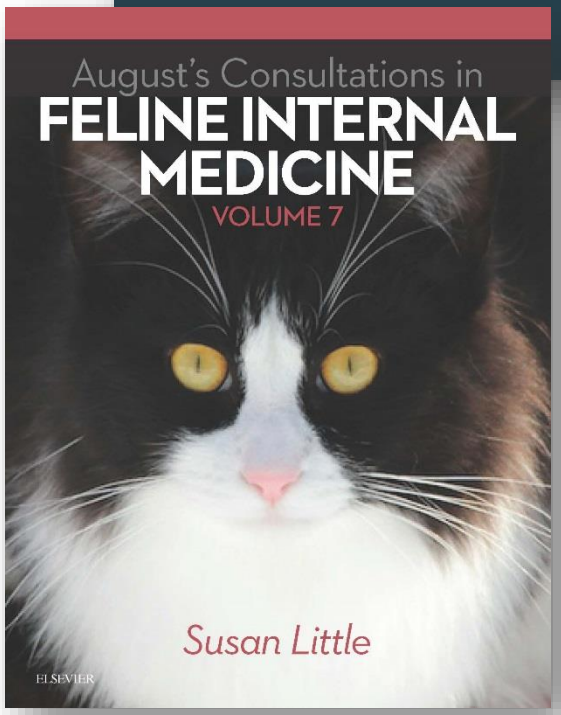
DOI: 10.1177/1098612X12464458

Reading list

- Chen H et al. Acute on chronic kidney disease in cats: Etiology, clinical and clinicopathologic findings, prognostic markers, and outcome. J Vet Intern Med, 2020.
- Cole L et al. Ultrasonographic findings in cats with acute kidney injury: a retrospective study. J Feline Med Surg, 2019.
- Bennett A et al. Outcome following gastrointestinal tract decontamination and intravenous fluid diuresis in cats with known lily ingestion: 25 cases (2001-2010). J Am Vet Med Assoc, 2013.





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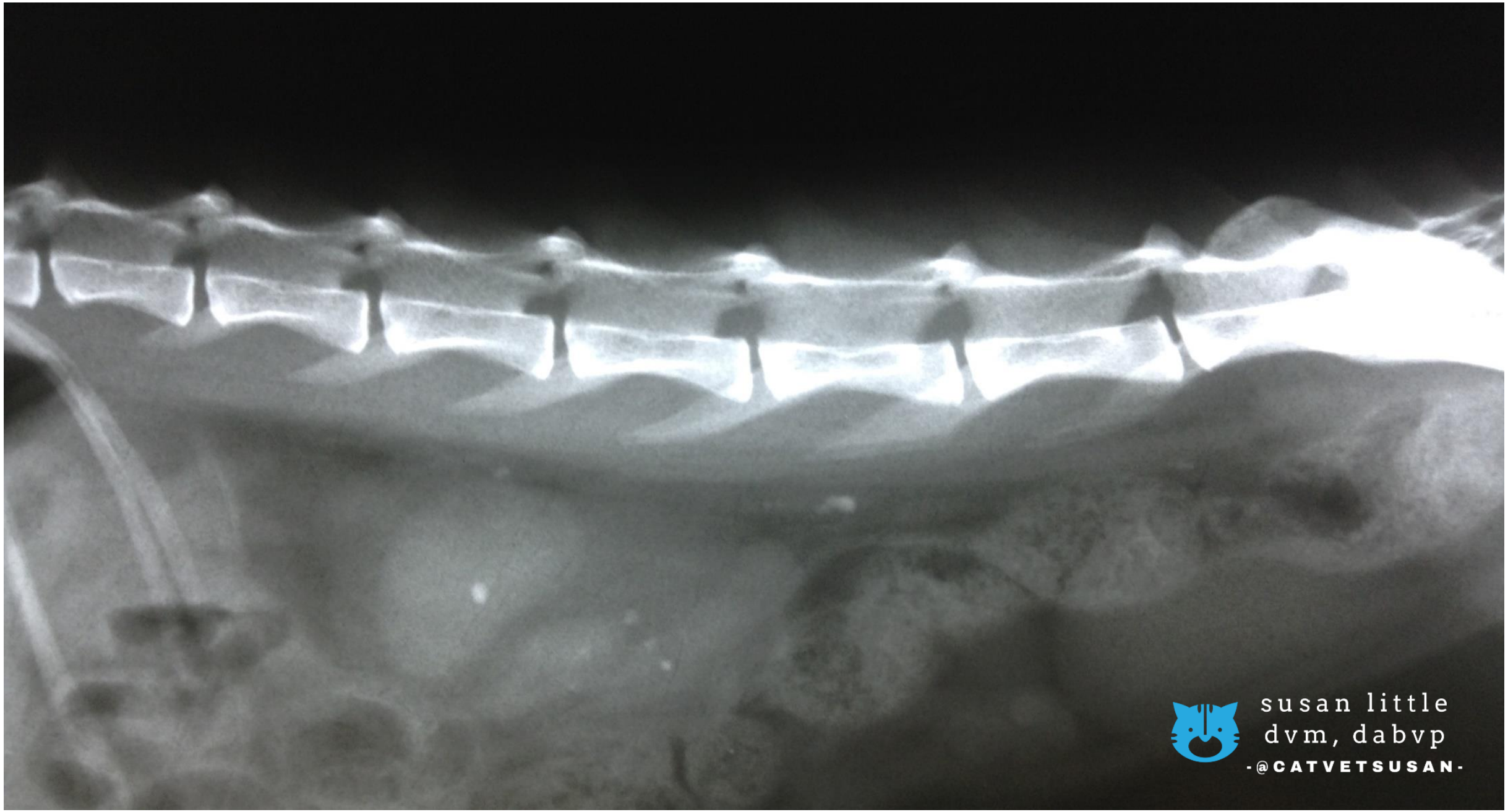
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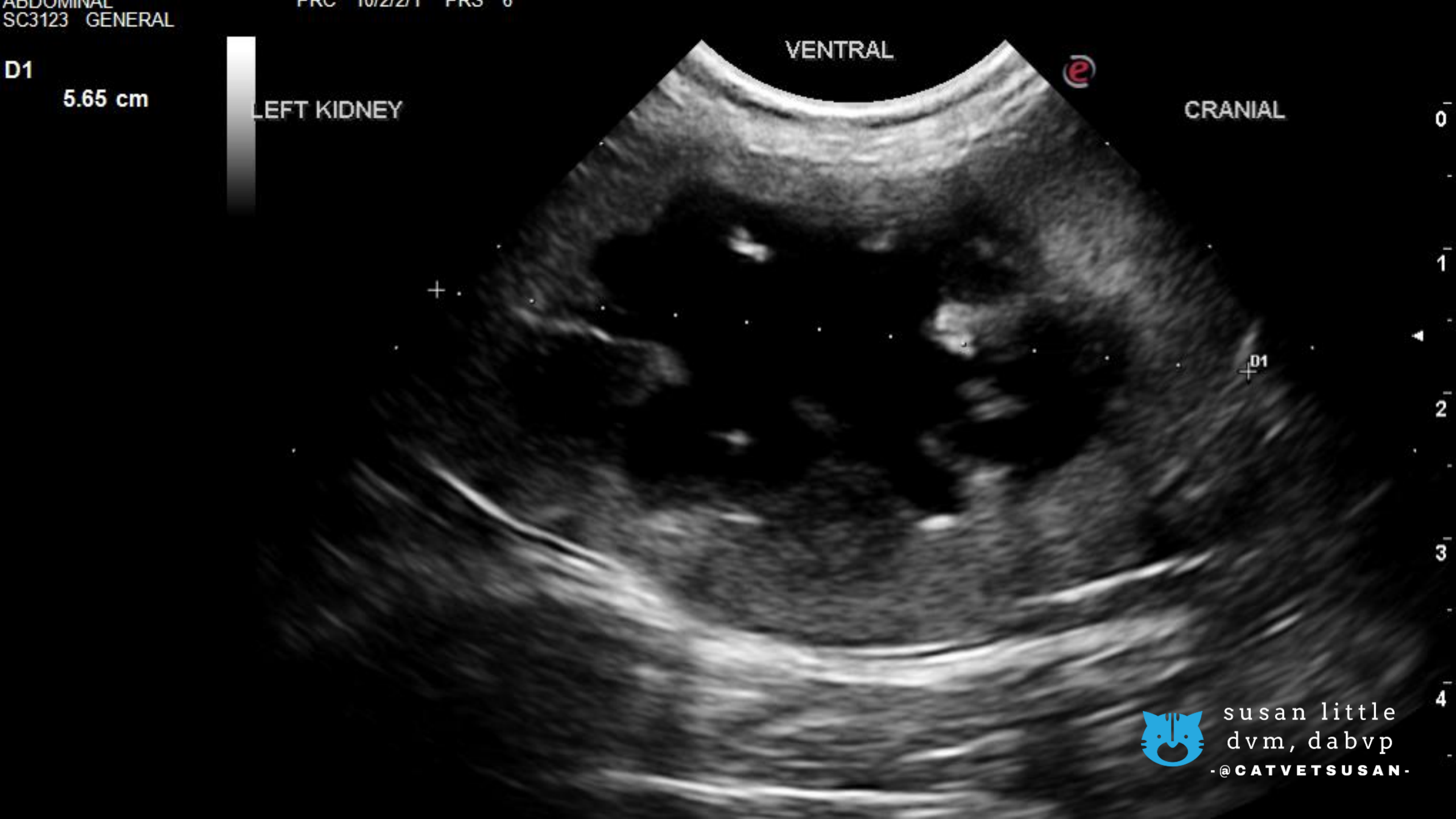
ABDOMINAL
SC3123 GENERAL
D1
5.65 cm
LEFT KIDNEY
VENTRAL
CRANIAL
D1
0
1
2
3
4
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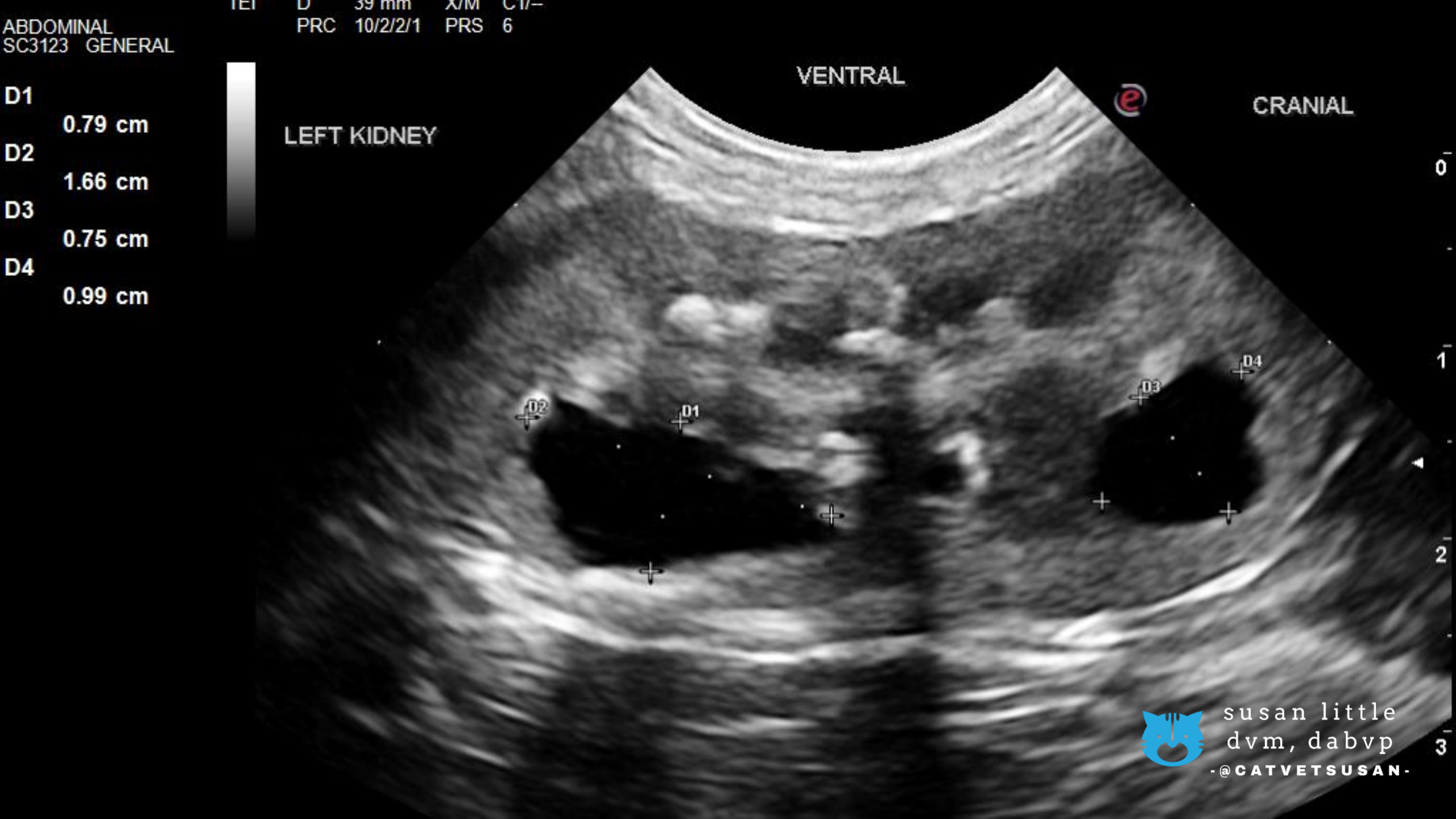
TEI D 39 mm X/M C1/–
PRC 10/2/2/1 PRS 6
ABDOMINAL
SC3123 GENERAL
D1
0.79 cm
D2
1.66 cm
D3
0.75 cm
D4
0.99 cm
LEFT KIDNEY
VENTRAL
CRANIAL
D1
D2
D3
D4
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Injury to normal kidneys

Acute on chronic disease

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Differentiation of uremia

Concentrated urine	Isosthenuric	Rule out urinary obstruction or rupture first!
Pre-renal	Renal	Post-renal

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Treatment for hyperkalemia

- 0.5-1.0 mL/kg calcium gluconate 10% IV over 5-10 min; monitor ECG
- 0.25-0.5 IU/kg regular insulin + 1 mL/kg dextrose 50%
- 1-2 mEq/kg IV sodium bicarbonate over 10-15 min, repeat if needed

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Cooper et al Peritoneal Dialysis in Cats with Acute Kidney Injury: 22 Cases (2001–2006)

- In cases of acute kidney injury refractory to fluid therapy, both hemodialysis and PD are treatment options that can be considered. Hemodialysis requires specialized training, equipment, and water purification systems and is also not readily available to most veterinarians. PD is labor intensive, but does not require specialized equipment and may be performed in any clinic with adequate technical assistance and supervision. Patient personality also must be considered when deciding to perform PD because the patient must tolerate frequent manipulations.

Treatment of hyperkalemia

Severity	Serum potassium	Treatment options
Mild	<6 mEq/L	**Fluid** therapy for dilution
Moderate	6-8 mEq/L	**Sodium bicarbonate**: 1-2 mEq/kg IV over 10-15 min, repeat if needed, maximum 4 mEq/kg **Dextrose** (50%): 1 mL/kg IV, dilute to 10-20% **Regular insulin**: 1 unit IV with dextrose
Severe	>8 mEq/L	**Calcium gluconate** (10%): 0.5 mL/kg IV, over 5-10 min, monitor ECG Followed by **regular insulin with dextrose**

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