VETERINÆRDAGENE 2025
12.-14. mars, Trondheim
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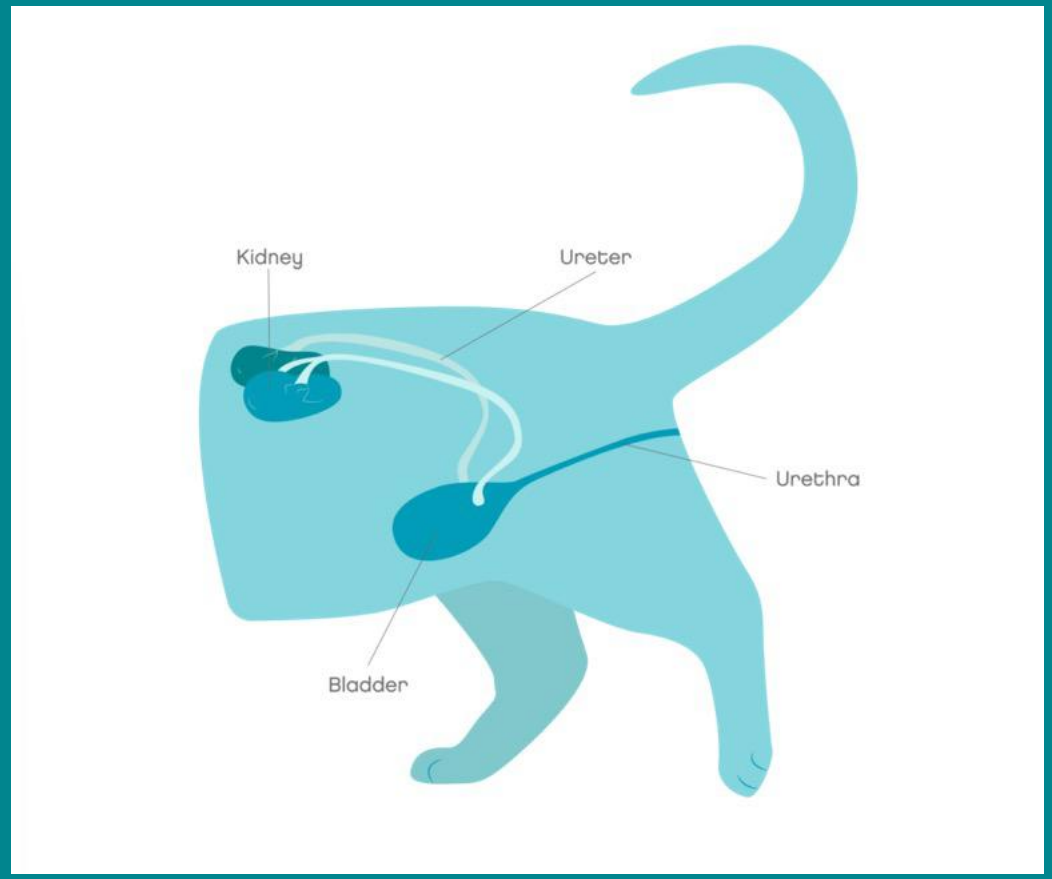

Feline Acute kidney injury: a clinical approach

Maria Lyraki DVM MSc DipECVIM-CA MRCVS

EBVS and RCVS Recognised Specialist in Small Animal Internal Medicine

DEN NORSKE VETERINÆRFORENING

Most recent guidelines

ARTICLE IN PRESS

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International Renal Interest Society best practice consensus guidelines for the diagnosis and management of acute kidney injury in cats and dogs

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[d] Department of Clinical Veterinary Medicine, Vetsuisse Faculty University of Bern, Bern, Switzerland
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ARTICLE INFO

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ABSTRACT

Acute kidney injury (AKI) is defined as an injury to the renal parenchyma, with or without a decrease in kidney function, as reflected by accumulation of uremic toxins or altered urine production (i.e., increased or decreased). AKI might result from any of several factors, including ischemia, inflammation, nephrotoxins, and infectious diseases. AKI can be community- or hospital-acquired. The latter was not previously considered a common cause for AKI in animals; however, recent evidence suggests that the prevalence of hospital-acquired AKI is increasing in veterinary medicine. This is likely due to a combination of increased recognition and awareness of AKI, as well as increased treatment intensity (e.g., ventilation and prolonged hospitalization) in some veterinary patients and increased management of geriatric veterinary patients with multiple comorbidities.

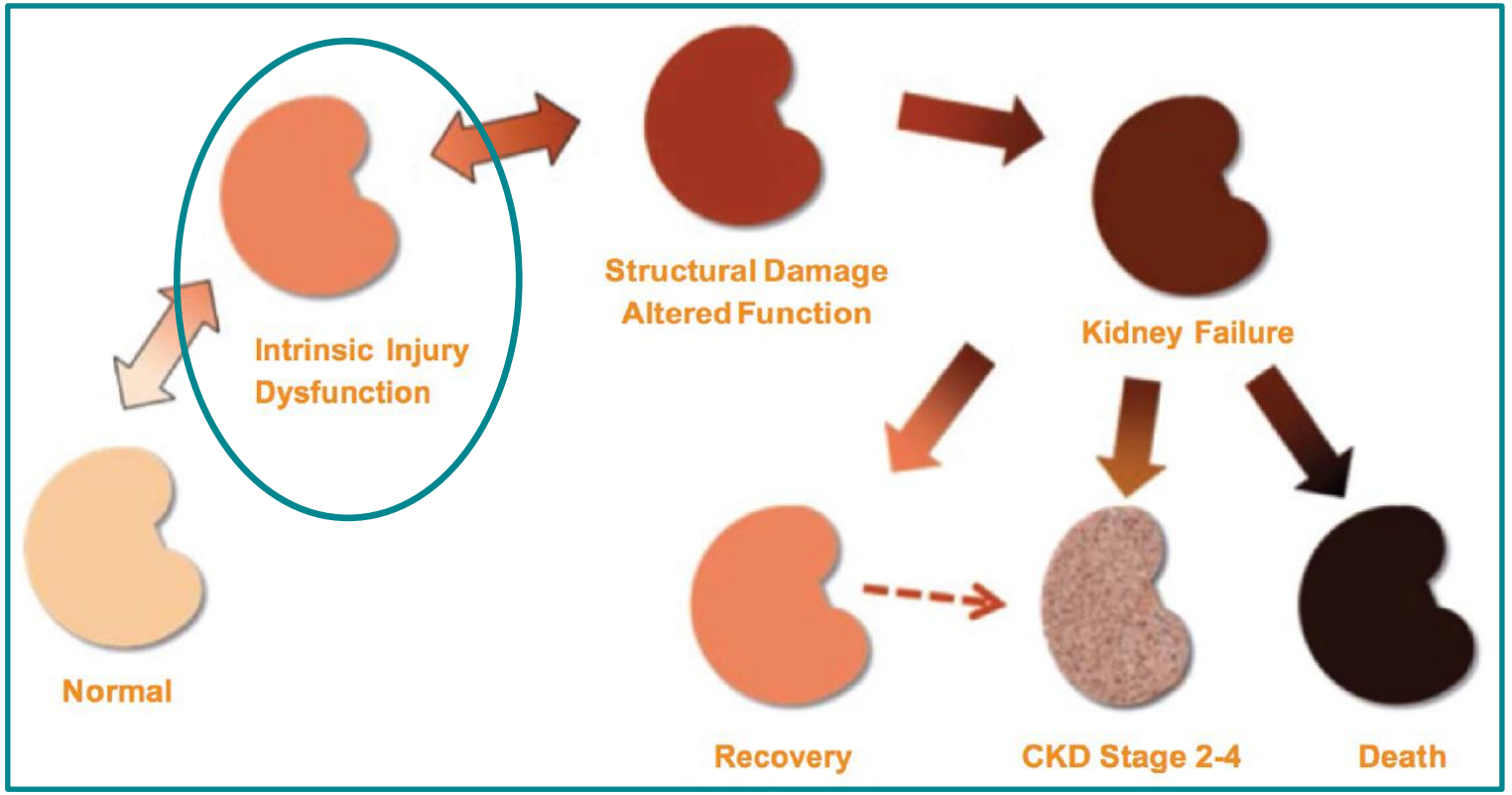
Advancements in the management of AKI, including the increased availability of renal replacement therapies, have been made; however, the overall mortality of animals with AKI remains high. Despite the high prevalence of AKI and the high mortality rate, the body of evidence regarding the diagnosis and the management of AKI in veterinary medicine is very limited. Consequently, the International Renal Interest Society (IRIS) constructed a working group to provide guidelines for animals with AKI. Recommendations are based on the available literature and the clinical experience of the members of the working group and reflect consensus of opinion. Fifty statements were generated and were voted on in all aspects of AKI and explanatory text can be found either before or after each statement

Definition

The term acute kidney failure has been replaced by the term acute kidney injury (AKI) to emphasize that if diagnosed early, the disease can be reversible and it may not lead to kidney failure.

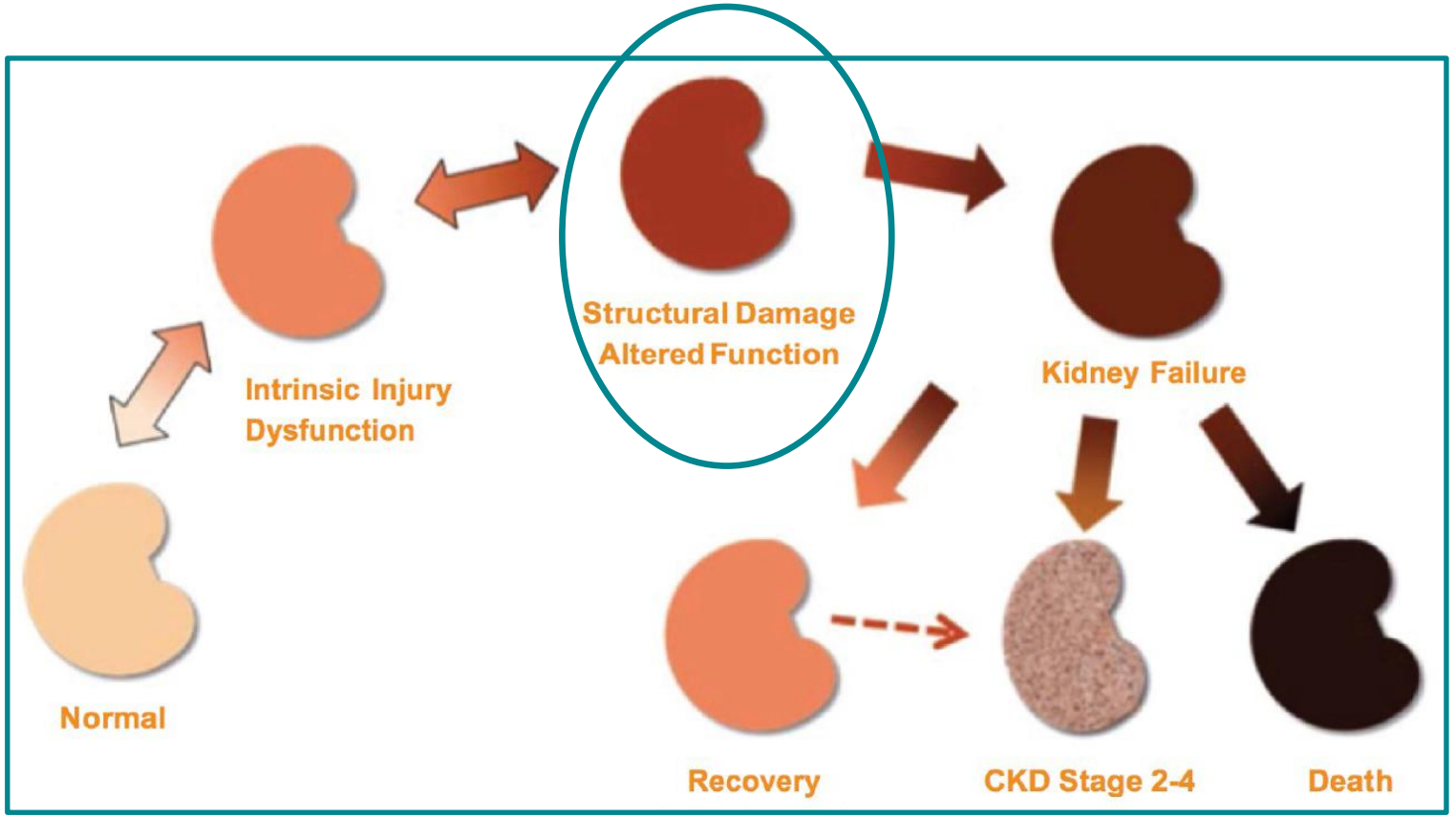
The 4 stages of AKI

- Induction



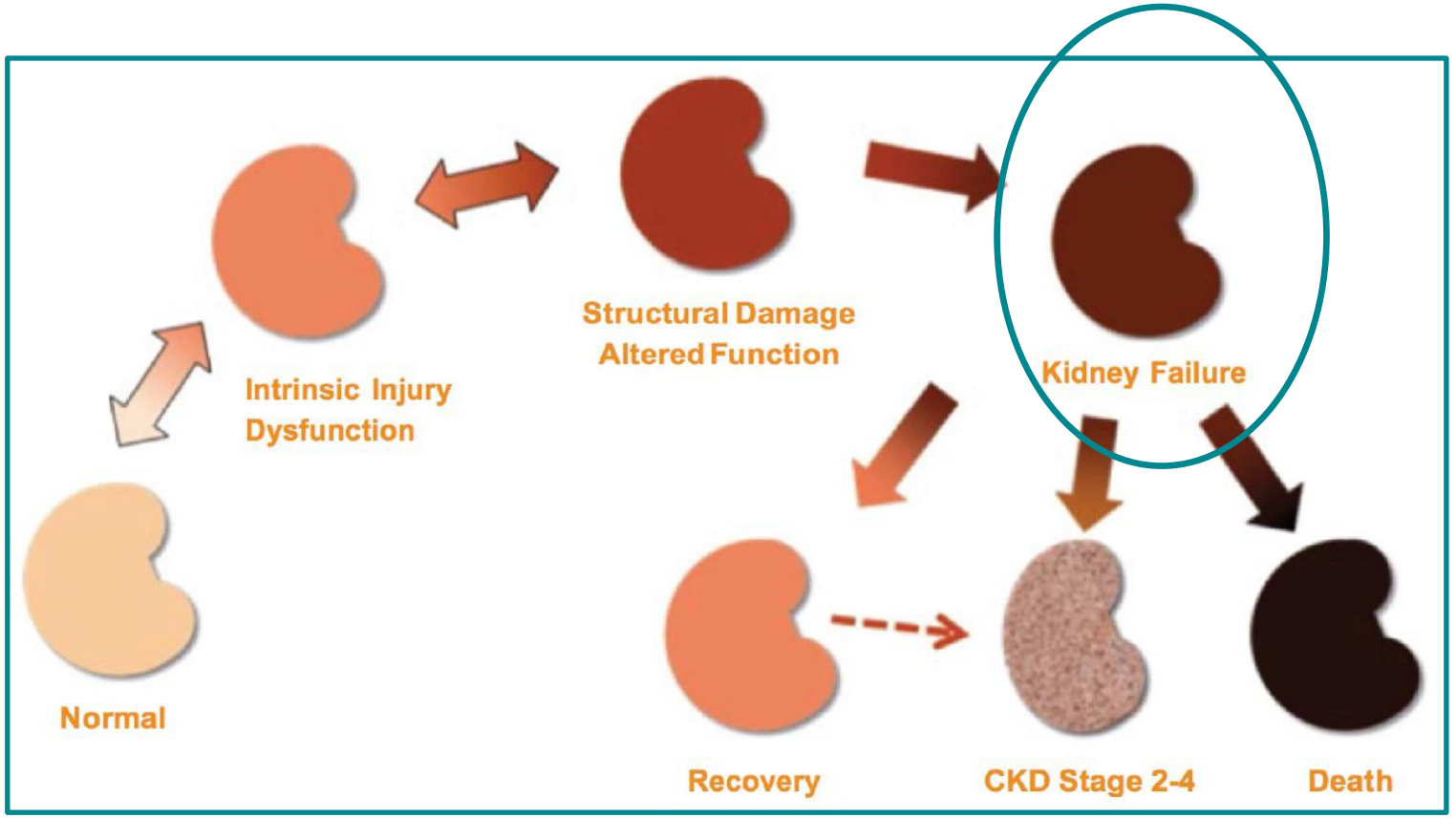
The 4 stages of AKI

- Induction
- Extension



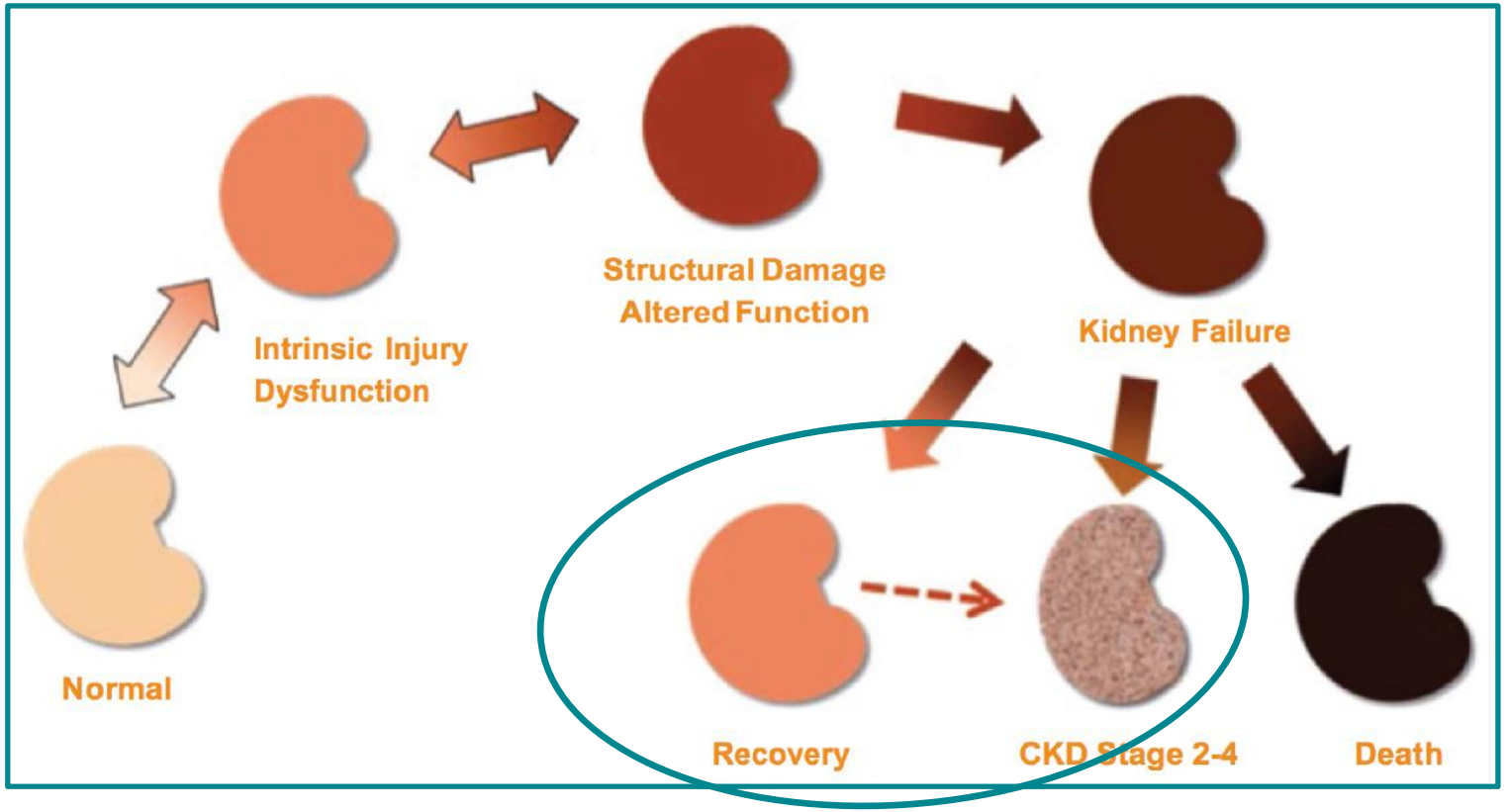
The 4 stages of AKI

- Induction
- Extension
- Maintenance



The 4 stages of AKI

- Induction
- Extension
- Maintenance
- Recovery



AKI types

- **Community-acquired**

The renal insult occurs outside of the hospital

- **Hospital-acquired**

The renal insult occurs during / after hospitalisation

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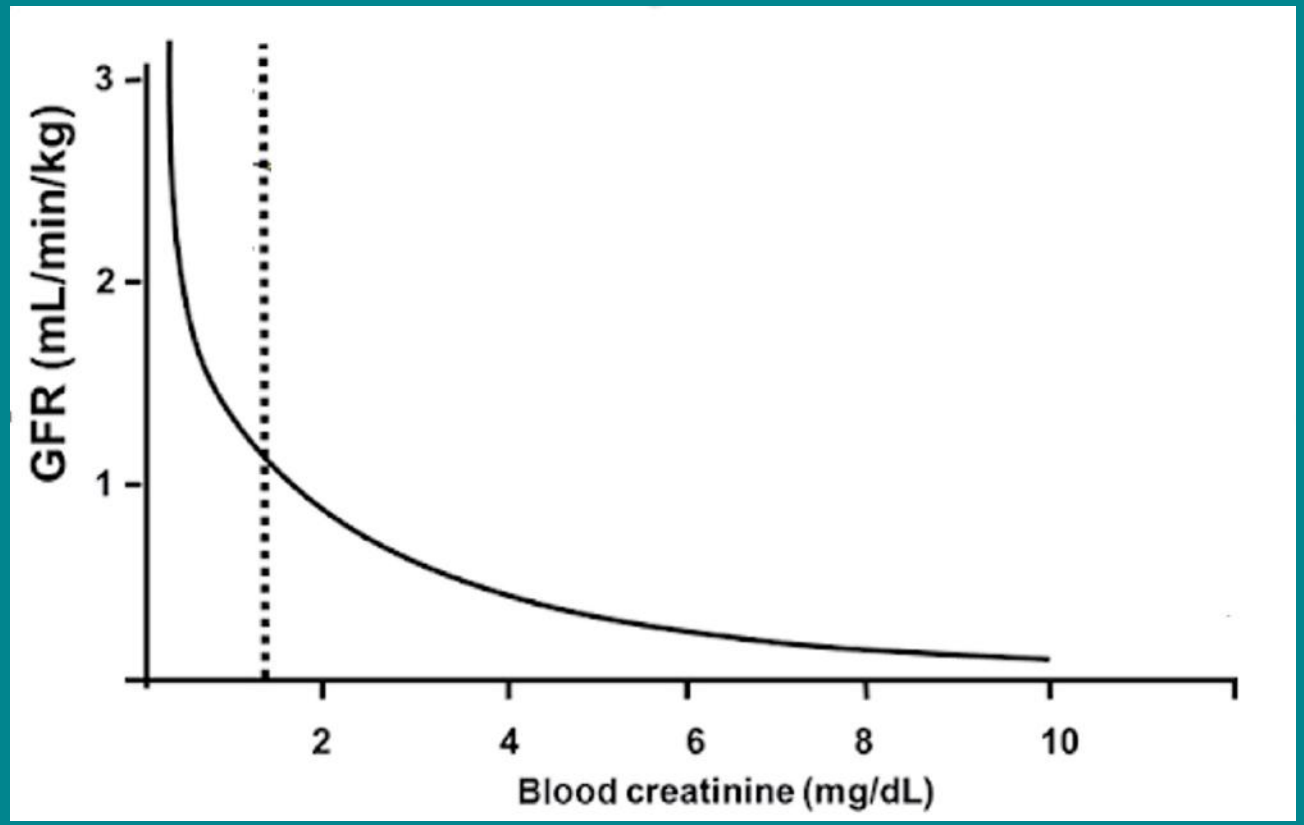
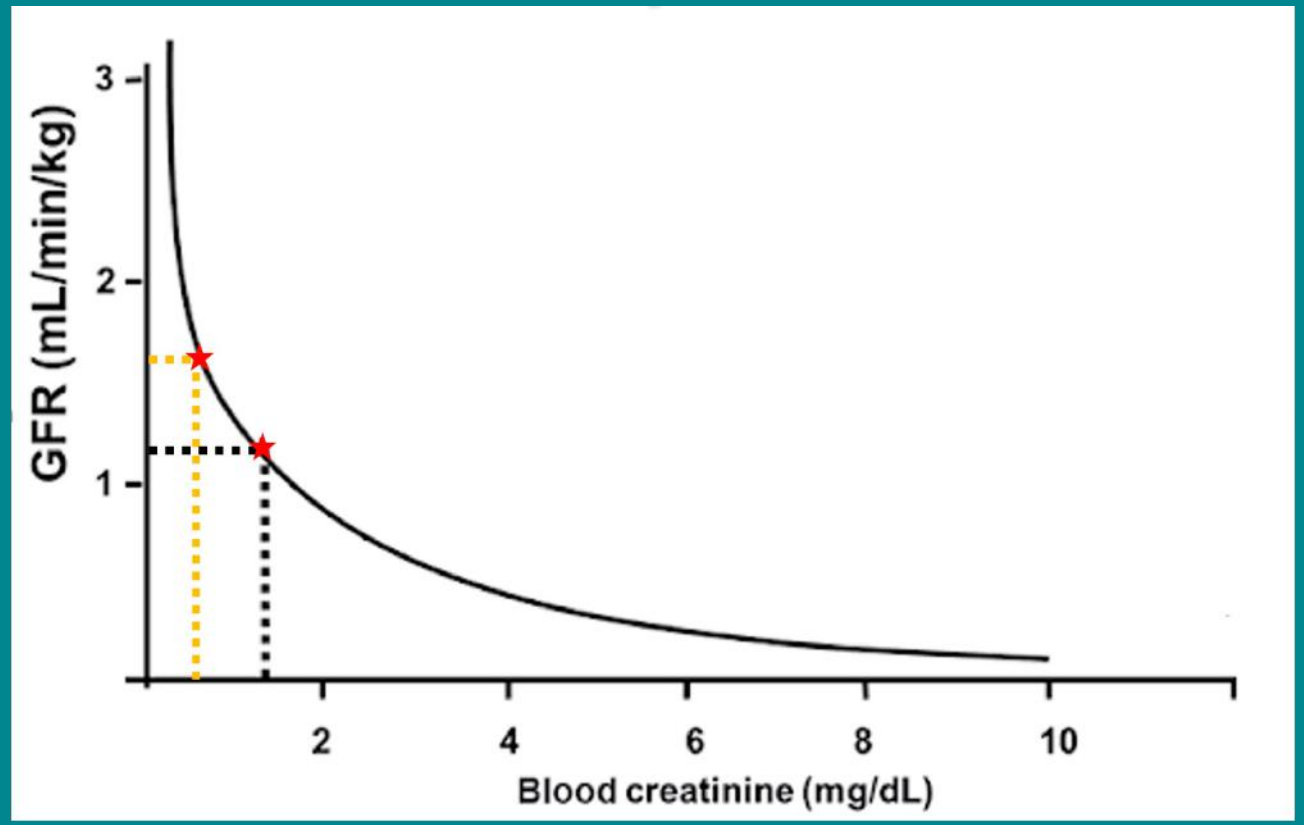
Early diagnosis!

The relationship between sCr and GFR

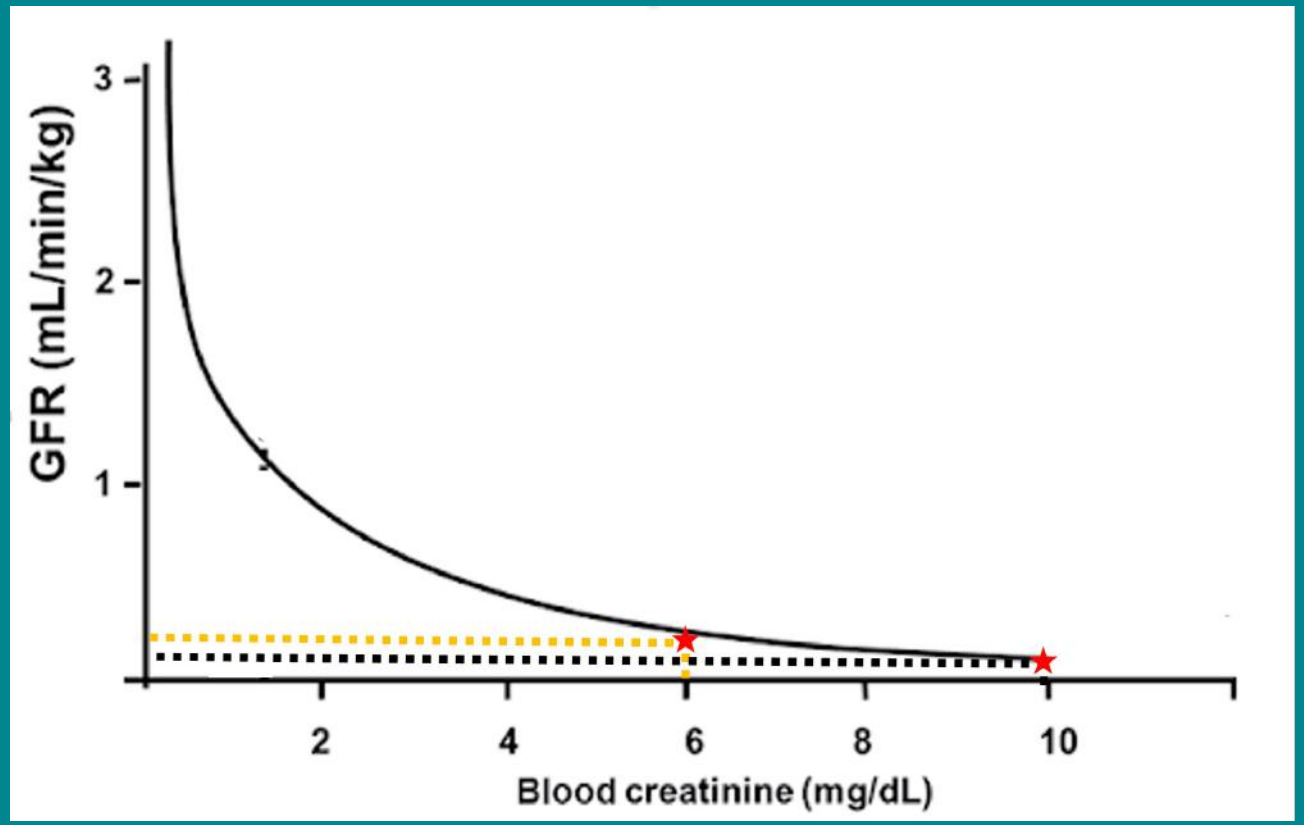


The relationship between sCr and GFR



At early stages, a small change in creatinine can lead to a big drop in GFR

The relationship between sCr and GFR



At later stages, a big change in creatinine can lead to small or no change in GFR

AKI - definitions

- **A sCr increase in comparison to admission**
  - > 0.3mg/dL (24.6 µmol/l) increase in 48 hours even if the sCr is within normal limits
  - > 1.6mg/dL (140µmol/l)

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- **Oliguria/anuria**

< 1 ml/kg/hr in 6 hours despite the patient's rehydration

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- **AKI biomarkers**
  - Glucosuria
  - Cylindruria
  - Crystalluria

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We need to know the baseline creatinine in all patients that are hospitalised. SCr, urine output and clinical picture need to be monitored every 24-48 hours

Staging

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AKI Grade	Blood Creatinine	Clinical Description
Grade I	<1.6 mg/dl (<140 µmol/l)	Nonazotemic AKI: a. Documented AKI: (historical, clinical, laboratory, or imaging evidence of AKI, clinical oliguria/anuria, volume responsiveness‡) and/or b. Progressive nonazotemic increase in blood creatinine: ≥ 0.3 mg/dl (≥ 26.4 µmol/l) within 48 h c. Measured oliguria (<1 ml/kg/h)# or anuria over 6 h
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 h),or volume responsiveness‡ c. Measured oliguria (<1 ml/kg/h)# or anuria over 6 h
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)	

IRIS International Renal Interest Society

Staging

In contrast to CKD, AKI's staging is not stable and we expect it to keep changing depending on clinical response to treatment.

Frequent re-evaluation during hospitalisation is required

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AKI causes

- ❑ Pre-renal
- ❑ Renal
- ❑ Post renal

Pre-renal AKI

- **Hypovolaemia**
  - Bleeding
  - Gastrointestinal ‘/ Renal / Skin burns/other losses
  - Addison’s
  - Diuretics, ACE inhibitors
- **Reduced cardiac output**
  - Heart failure
  - Restrictive pericarditis
- **Systemic vasodilation**
  - Sepsis
  - Allergic shock
  - Drugs
- **Vasoconstriction of the renal vessels**
  - Nephrotoxic drug administration (e.g. NSAIDs)
  - Hypercalcaemia
  - Septicaemia

Renal AKI

- ❑ Drugs/toxins
- ❑ Infectious (bacterial pyelonephritis, FIP, other)
- ❑ Immune-mediated (e.g. immune-mediated glomerulonephritis)
- ❑ Neoplasia
- ❑ Idiopathic

Post renal AKI

- ❑ Ureteric or urethral obstruction
- ❑ Uroperitoneum

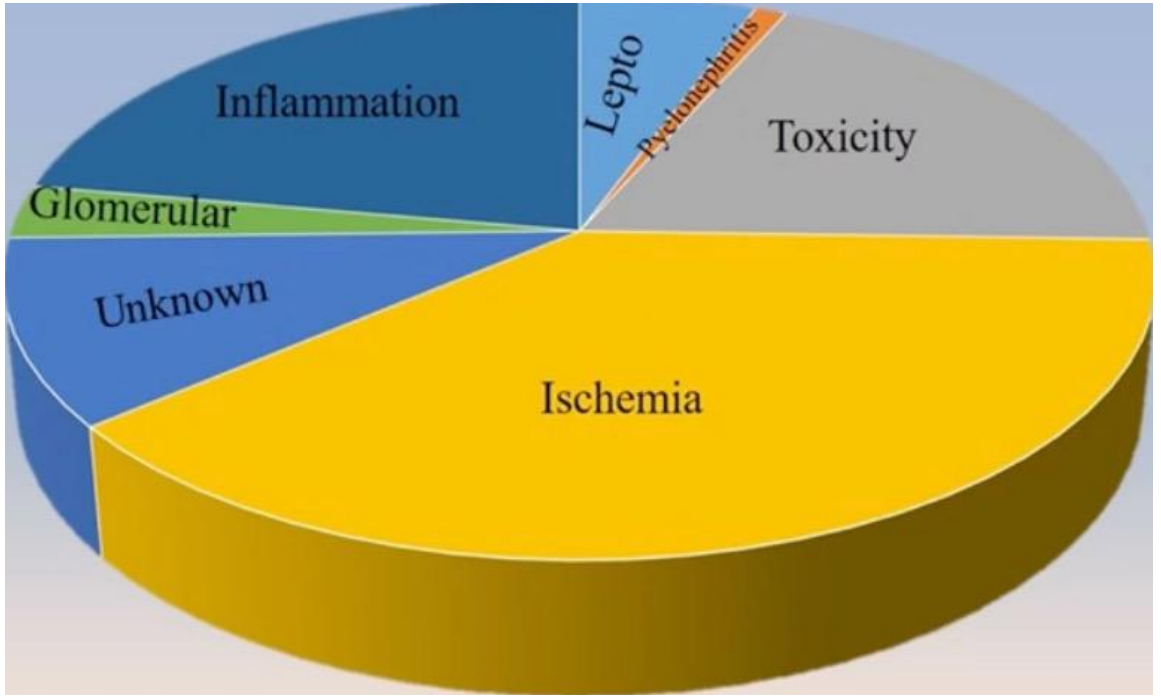
Causes of AKI

Acute kidney injury in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers, and outcome

Dar Rimer | Hilla Chen | Mali Bar-Nathan | Gilad Segev

Journal of Veterinary Internal Medicine
J Vet Intern Med. 2022;36:609–618.

- 249 dogs
- 58% ischaemia or non-specific inflammation
- 14% toxicosis
- 8% infectious
- 5 % miscellaneous



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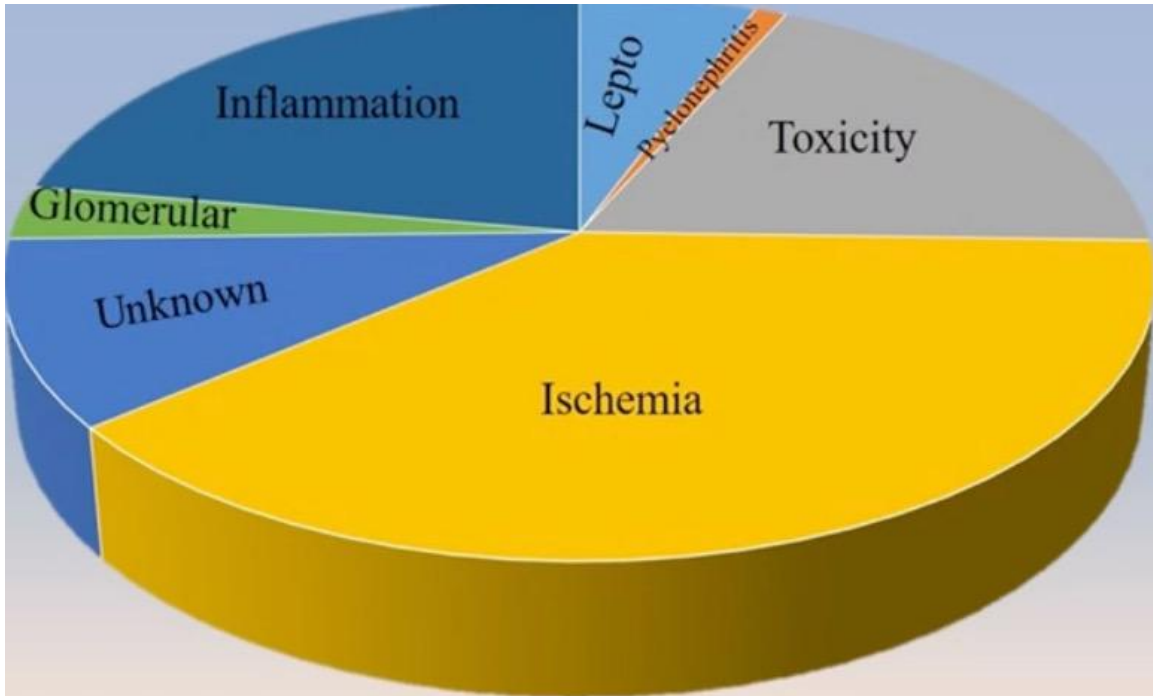

Cats – 15 % obstructive disease e.g. ureteric obstruction!

History –taking in AKI

- Detailed history is required
  - Toxin / drug exposure
  - Lifestyle
  - Recent general anaesthetic history
  - Recent neutering surgery (female)
  - Nephrotoxic drug administration OR transfusion
  - Causes of dehydration / hypovolaemia
  - Infection

Acute-on-chronic AKI

Etiology	All dogs (n = 100) n (%)	Survivors (n = 65) n (%)	Nonsurvivors (n = 35) n (%)
Unknown	45 (45)	25 (39)	20 (57)
Inflammatory	30 (30)	22 (34)	8 (23)
Ischemic	7 (7)	5 (8)	2 (6)
Pyelonephritis	15 (15)	10 (15)	5 (14)
Other[a]	3 (3)	3 (5)	0 (0)

Acute on chronic kidney disease in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers, and survival

Asia Dunaevich | Hilla Chen | Danielle Musseri | Sharon Kuzi
Michal Mazaki-Tovi | Itamar Aroch | Gilad Segev

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Etiology	All cats (n = 100) n (%)	Survivors (n = 58) n (%)	Nonsurvivors (n = 42) n (%)
Unknown	66 (66)	34 (51.5)	32 (48.5)
Ureteral obstruction	11 (11)	9 (81.8)	2 (18.2)
Ischemia	9 (9)	8 (88.9)	1 (11.1)
Pyelonephritis	8 (8)	4 (50.0)	4 (50.0)
Other[a]	6 (6)	3 (50.0)	3 (50.0)

Clinical signs

- ❑ Depression, lethargy – 90%
- ❑ Anorexia – 83%
- ❑ Vomiting - 68%
- ❑ Diarrhoea – 41%
- ❑ Polyuria/Polydipsia
- ❑ Oliguria / Anuria
- ❑ Hypersalivation
- ❑ Abdominal pain
- ❑ Bleeding diathesis

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DOI: 10.1111/jvim.16375
STANDARD ARTICLE
Journal of Veterinary Internal Medicine Open Access ACVIM American College of Veterinary Internal Medicine

Acute kidney injury in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers, and outcome

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Acute vs Chronic kidney injury

- ❑ Chronic vs acute history of clinical signs
- ❑ Good body condition score
- ❑ Small versus enlarged or normal size kidneys
- ❑ Painful palpation of the kidneys
- ❑ Blood work

Diagnosis

- ❑ Physical examination
- ❑ Arterial blood pressure – 2-4 times a day
- ❑ Haematology, serum biochemistry, electrolytes
- ❑ Urinalysis, sediment, UPC, culture
- ❑ Abdominal ultrasound

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- ❑ Urinalysis, sediment, UPC, culture
- ❑ Abdominal ultrasound

Minimum tests in all cases

Arterial blood pressure measurement

- Oscillometric
- Doppler
  - More reliable in cats
  - Systolic blood pressure

Blood pressure

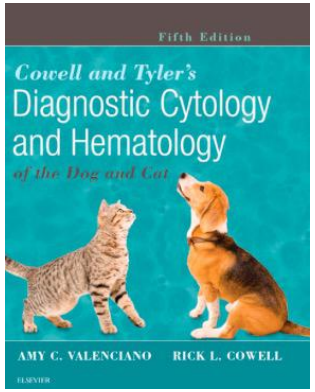
SBP (mm Hg)	Substage of SBP	Risk of future TOD
<140	Normotensive	Minimal
140 - 159	Prehypertensive	Low
160 - 179	Hypertensive	Moderate
≥180	Severely hypertensive	High

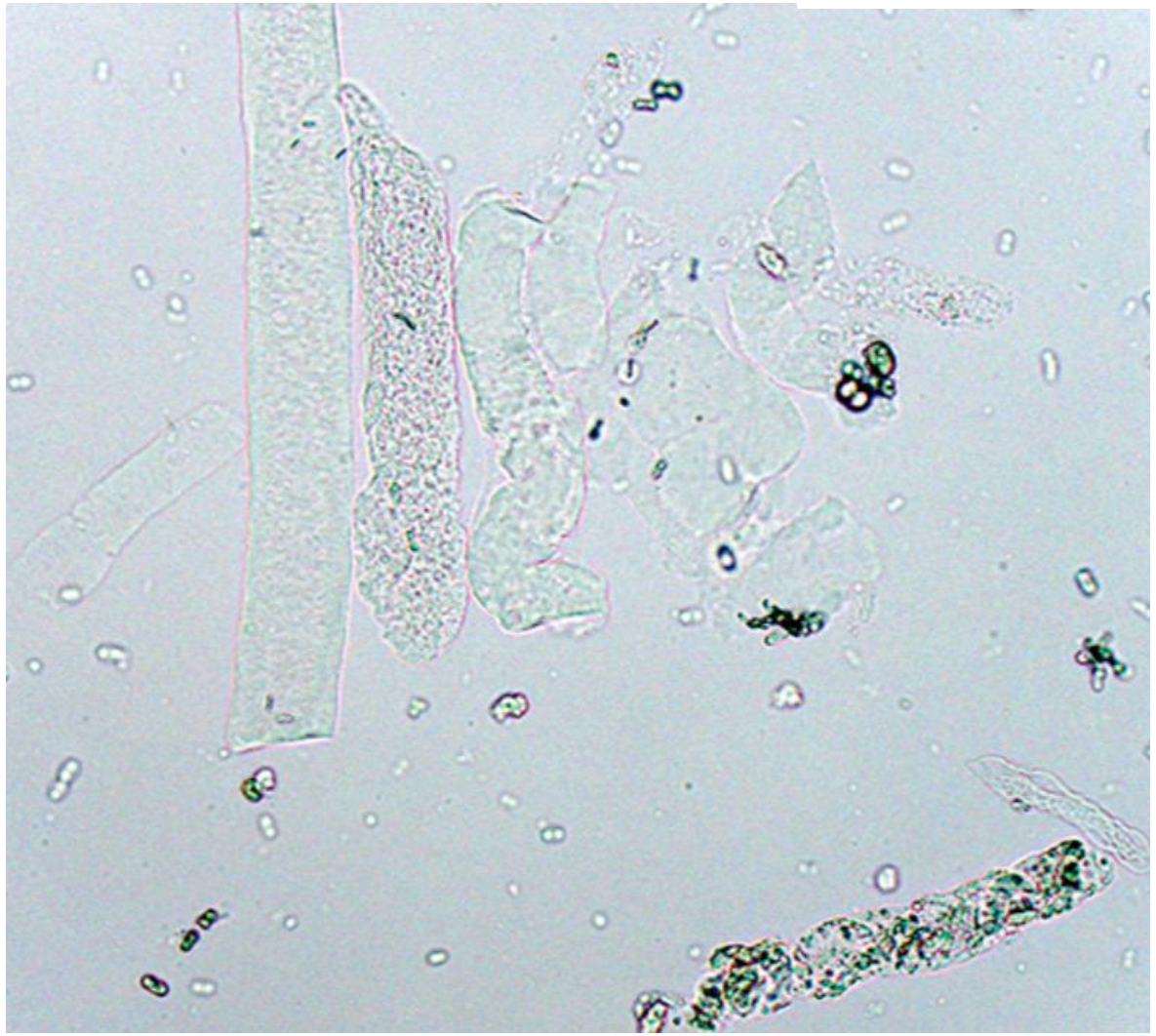
Urinalysis

- Urine specific gravity
- Dipstick
- UPC measurement
- Sediment analysis
  - Crystals
  - Cylinders
  - Glucosuria

Urinalysis

Urinalysis


Fifth Edition
Cowell and Tyler's
Diagnostic Cytology
and Hematology
of the Dog and Cat
AMY C. VALENCIANO
RICK L. COWELL



Ureteric obstruction

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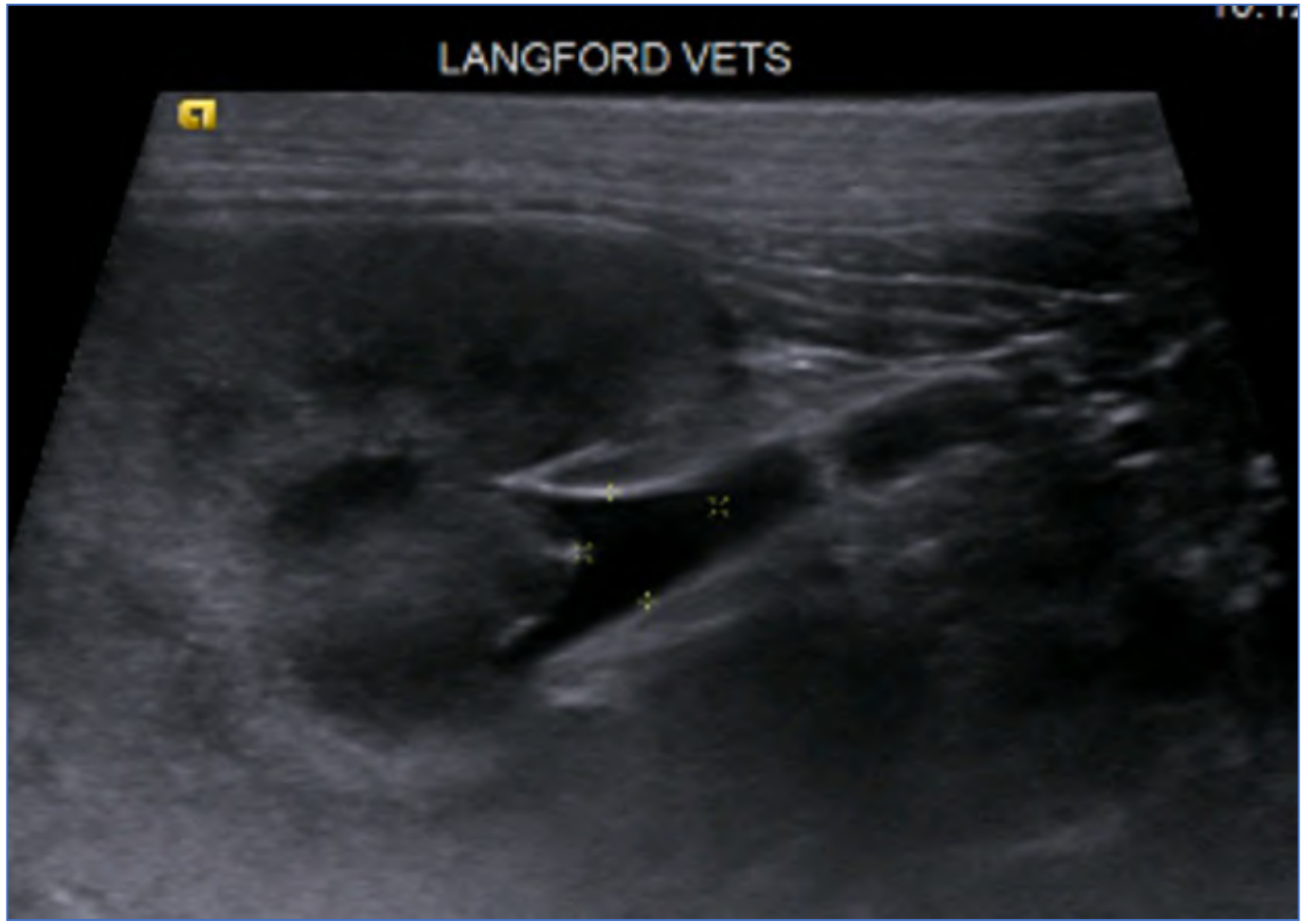

Diagnosis - SDMA

- SDMA (symmetric dimethylarginine) is the amino acid, arginine, that contains two methyl groups (dimethyl) in a symmetrical orientation
- Earlier marker of the GFR (detects 24-40% GFR reduction vs 75% of sCr)
- Increases in AKI
- The magnitude of increase may be indicative of the renal function
- Useful when sCr is still normal and baseline sCr is not known
- Cannot differentiate acute from chronic disease

Received: 19 January 2022 | Accepted: 7 July 2022
DOI: 10.1111/jvim.16497

Journal of Veterinary Internal Medicine Open Access ACVIM American College of Veterinary Internal Medicine

STANDARD ARTICLE

Evaluation of symmetric dimethylarginine in cats with acute kidney injury and chronic kidney disease

Samantha C. Loane | James M. Thomson | Timothy L. Williams | Katie E. McCallum

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- Earlier marker of the GFR (detects 24-40% GFR reduction vs 75% of sCr)
- Increases in AKI
- The magnitude of increase may be indicative of the renal function
- Useful whe
- Cannot diff

New studies report that the range 14-18 ug/mL is a grey area where cats may not necessarily have kidney disease

Evaluation of symmetric dimethylarginine in cats with acute kidney injury and chronic kidney disease

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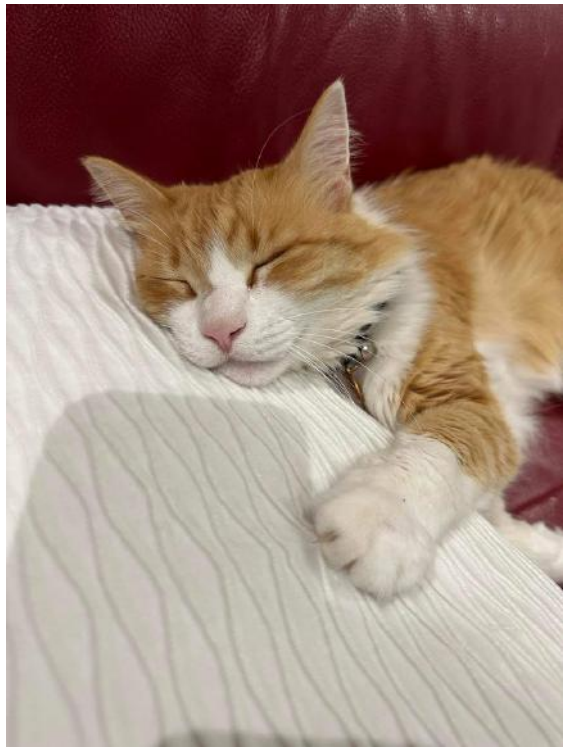
Specific tests for causes of AKI

- ❑ Urinary tract infection
- ❑ Feline Leukaemia Virus (FeLV)
- ❑ Feline Immunodeficiency Virus (FIV)
- ❑ Feline Infectious Peritonitis
  - ❑ Serology is not helpful
  - ❑ PCR FCoV in the free fluid or FNA kidneys / lymph nodes
- ❑ *Toxoplasma gondii*
- ❑ *Bartonella spp*
- ❑ Panleukopenia virus
- ❑ *Dirofilaria spp*
- ❑ *Tick-borne*
- ❑ *Leishmania spp*
- ❑ *Leptospira spp*



Specific tests for causes of AKI

- Abdominal radiographs – useful to detect radiopaque uroliths

Specific tests for causes of AKI

- Abdominal radiographs – useful to detect radiopaque uroliths
- CT scan – contrast can be nephrotoxic, we usually avoid it
- Cytology
  - Not needed routinely
  - Diagnostic in FIP and lymphoma
- Renal biopsies
  - Rarely indicated
  - Glomerulonephritis
  - Neoplasia

Treatment

- ❑ Cause - specific
- ❑ Symptomatic, non-specific care

Cause specific

- ❑ Nephrotoxic drug discontinuation
- ❑ Toxicosis - specific treatments
- ❑ Infection – antimicrobial treatment as indicated
- ❑ Obstructive disease – relieve the obstruction or by-pass the obstruction

Symptomatic non-specific care

- **Renal perfusion**
  - **Hypovolaemia – correction within 1-2 hours**
    - CRT <1, tachycardia, tachypnoea, hypothermia, depression, cold extremities, pale mucous membranes
    - Hartmann's fluid bolus 5-10 ml/kg
    - 1-3 boluses

Close monitoring for fluid overload!

Symptomatic non-specific care

- **Renal perfusion**
  - **Dehydration**
    - Dry mucous membranes, weight loss, enophthalmos, depression, skin tent
    - 3, 5, 7, or 10% dehydration
    - % dehydration x BW (kg) = deficit in litres
    - Slow correction within 12-48 hours
    - Once hydrated, I reduce fluids to minimum (matching ins and outs)

Close monitoring for fluid overload!

Colloids and AKI

DEN NORSKE VETERINÆRFORENING

Assessment of Hydroxyethyl Starch (6% HES 130/0.4) Kidney Storage in Critically Ill Dogs: A Post-mortem Prospective Study

frontiers in Veterinary Science

Katja-Nicole Adamik[1*], Michael H. Stoffel[2], Simone Tangermann[3], Bettina de Breuyn Dietler[4] and Nadine Stokar-Regenscheit[2]

RETROSPECTIVE STUDY

Retrospective evaluation of paired plasma creatinine and chloride concentrations following hetastarch administration in anesthetized dogs (2002–2015): 244 cases

Kristin M. Zersen DVM | Khursheed Mama DVM, DACVAA | Justin C. Mathis DVM, MS, DACVECC

Original Study

Journal of Veterinary Emergency and Critical Care 26(1) 2016, pp 35–40
doi: 10.1111/vec.12412

Retrospective cohort study on the incidence of acute kidney injury and death following hydroxyethyl starch (HES 10% 250/0.5/5:1) administration in dogs (2007–2010)

Galina Hayes, PhD, DVM, DACVECC, DACVS; Leontine Benedicenti, DVM and Karol Mathews, DACVECC, DVSc

Effects of 6% Tetrastarch and Lactated Ringer's Solution on Extravascular Lung Water and Markers of Acute Renal Injury in Hemorrhaged, Isoflurane-Anesthetized Healthy Dogs

M.S. Diniz, F.J. Teixeira-Neto, N. Celeita-Rodríguez, C.H. Girotto, M.W. Fonseca, A.C. Oliveira-Garcia, and B. López-Castañeda

Evaluation of biomarkers of kidney injury following 4% succinylated gelatin and 6% hydroxyethyl starch 130/0.4 administration in a canine hemorrhagic shock model

Corrin J. Boyd BSc, BVMS(Hons), GradDipEd, MVetClinStud, MANZCVS, DACVECC[1] | Melissa A. Claus DVM, DACVECC[1] | Anthea L. Raisis BVSc, MVetClinStud, PhD, MANZCVS, DVA[1] | Rachel Cianciolo VMD, PhD, DACVP[2] | Erika Bosio BSc(Hons), PhD[3,4] | Giselle Hosgood BVSc(Hons), MS, PhD, FANZCVS, DACVS[1] | Mary Nabity DVM, PhD, DACVP[5] | Trevor Mori BSc(Hons), PhD[6] | Anne Barden BSc, PhD[6] | Claire R. Sharp BSc, BVMS, MS, DACVECC[1] | Lisa Smart BVSc(Hons), DACVECC[1,3,4]

Veterinary Anaesthesia and Analgesia
Formerly the Journal of Veterinary Anaesthesia
Veterinary Anaesthesia and Analgesia, 2016, 43, 262–270 doi:10.1111/vaa.12311

RESEARCH PAPER

Urinary neutrophil gelatinase-associated lipocalin concentration changes after acute haemorrhage and colloid-mediated reperfusion in anaesthetized dogs

Jennifer Davis*, Anthea L Raisis*, Rachel E Cianciolo†, David W Miller*, Robert E Shiel*, Mary B Nabity‡ & Giselle L Hosgood*

Symptomatic non-specific care

- Renal perfusion
  - Avoid colloids
    - Possibly nephrotoxic
    - No evidence that they are more effective than crystalloids

Close monitoring for fluid overload! Tachypnoea, dyspnoea, serous nasal discharge, pleural effusion or ascites

Symptomatic non-specific care

- Vasopressors (noradrenaline, dopamine)
  - only if there if fluid-resistant hypotension
  - care with the vasoconstriction that will reduce renal perfusion

Patients with AKI are usually hypertensive – if there is refractory hypotension then consider septic shock, hypovolaemia

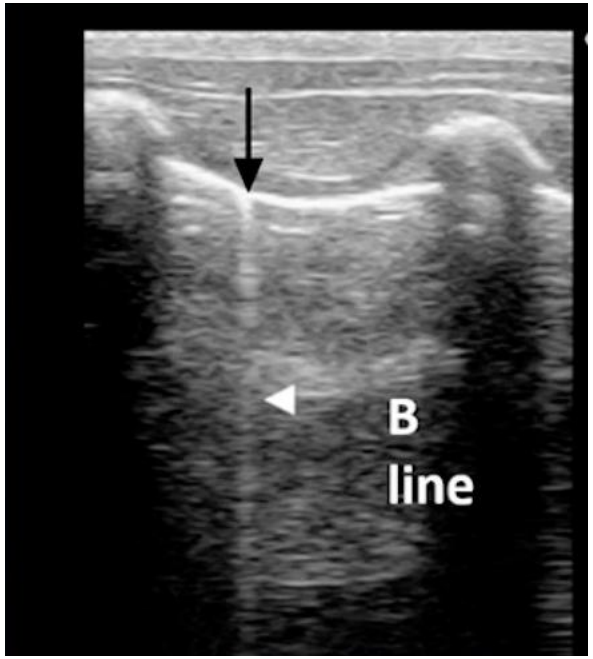
Fluid overload

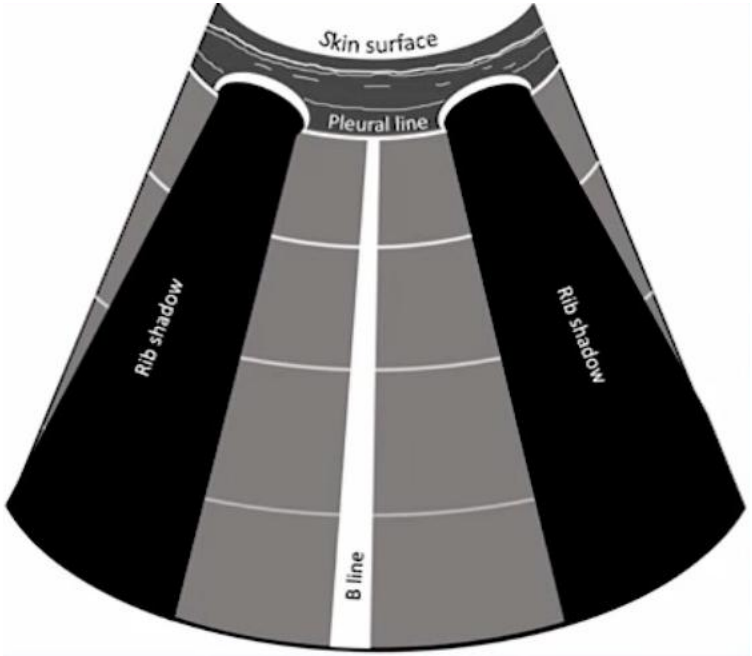
- Most common mortality cause in AKI
- Physical examination findings
  - Tachypnoea
  - Dyspnoea
  - Serous nasal discharge
  - Pleural effusion or ascites
  - Weight increase
  - Crackles in thoracic auscultation
  - Hypertension
  - Chemosis
  - Jugular vein pulse

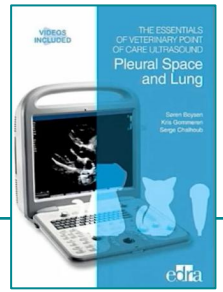
Fluid overload

- Most common mortality cause in AKI
- Imaging findings
  - Pleural effusion
  - Pulmonary oedema
  - Left atrial enlargement

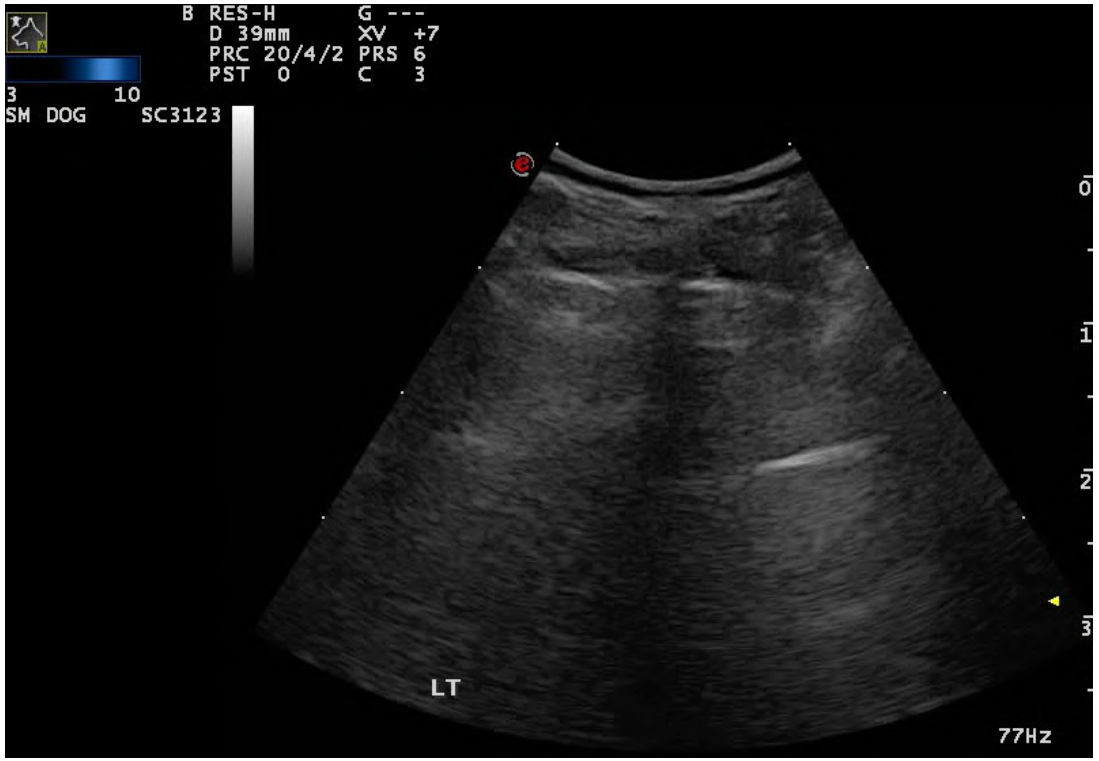
Fluid overload

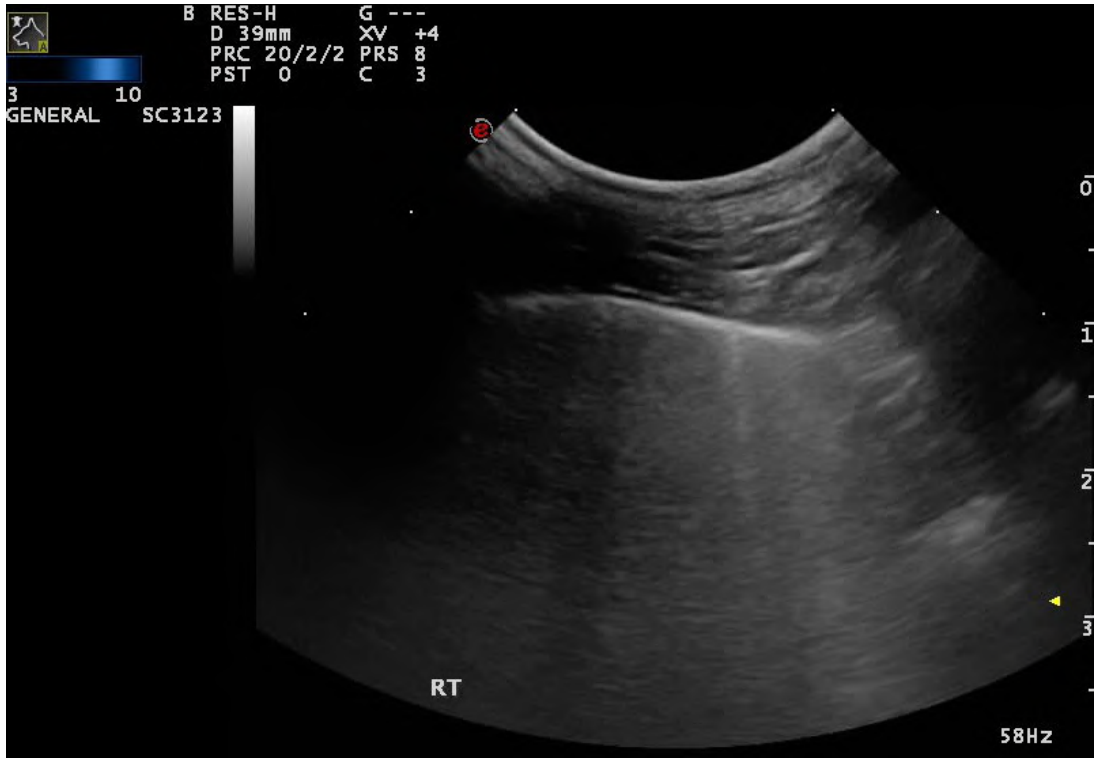




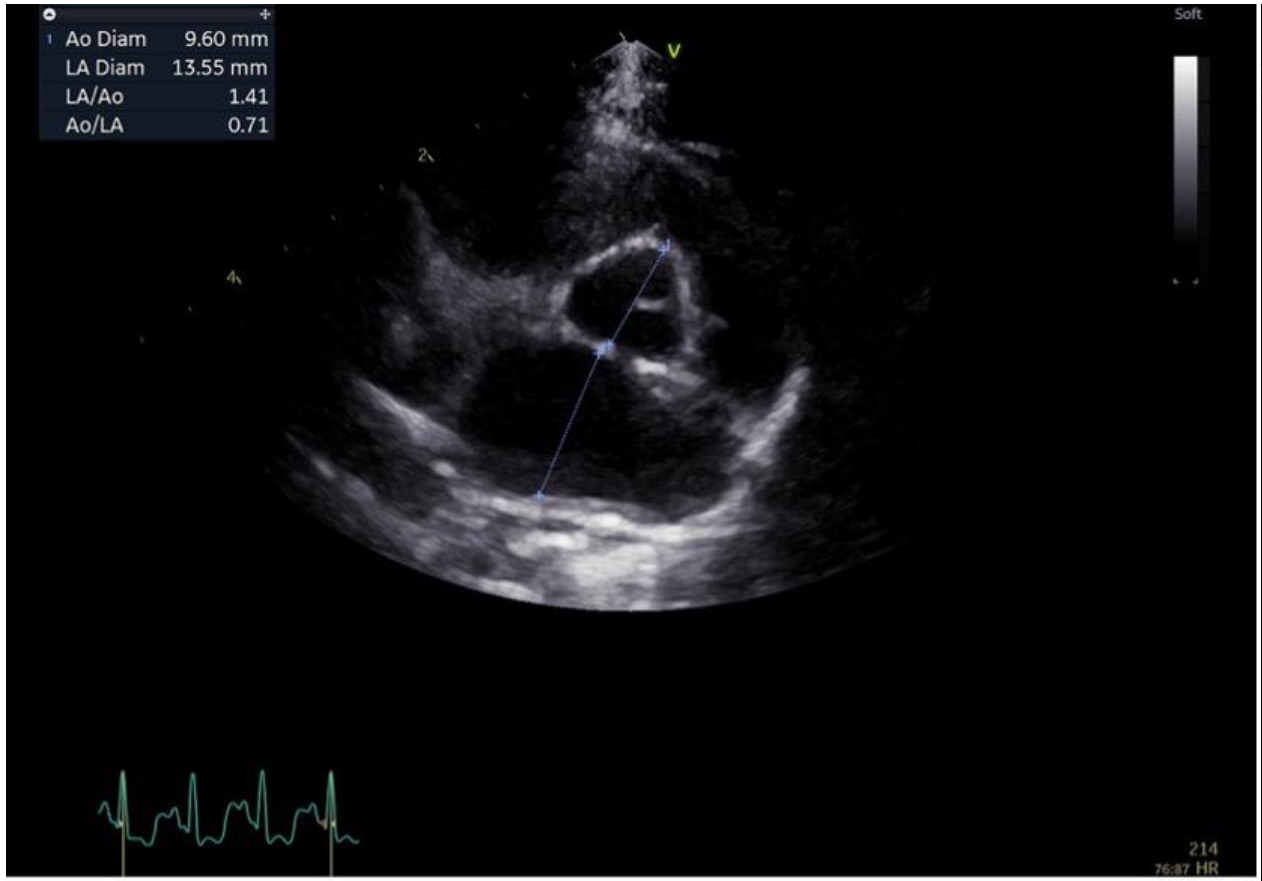


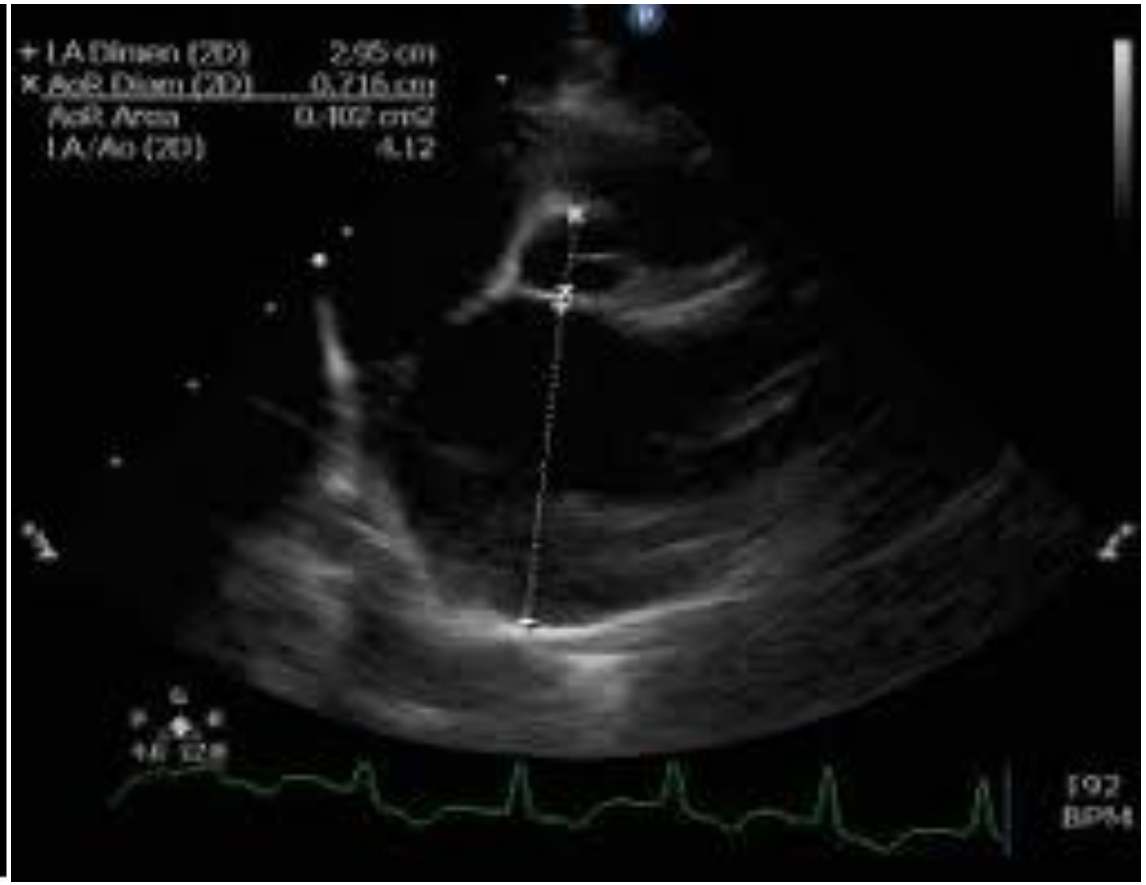
Normal lung ultrasound vs B-lines





Left atrial : aorta ratio





Treating fluid overload

- Stop fluids
- Urinary catheter placement for measuring urinary output
- Diuretics may be needed for controlling hyperkalaemia and pulmonary oedema associated with fluid overload
  - Furosemide
    - Bolus 1-2 mg/kg IV , maintenance CRI 0.7-1 mg/kg/hr IV
  - Mannitol
    - bolus slowly IV 0,25-0.5 g/kg, maintenance CRI 60-120 mg/kg/hr IV

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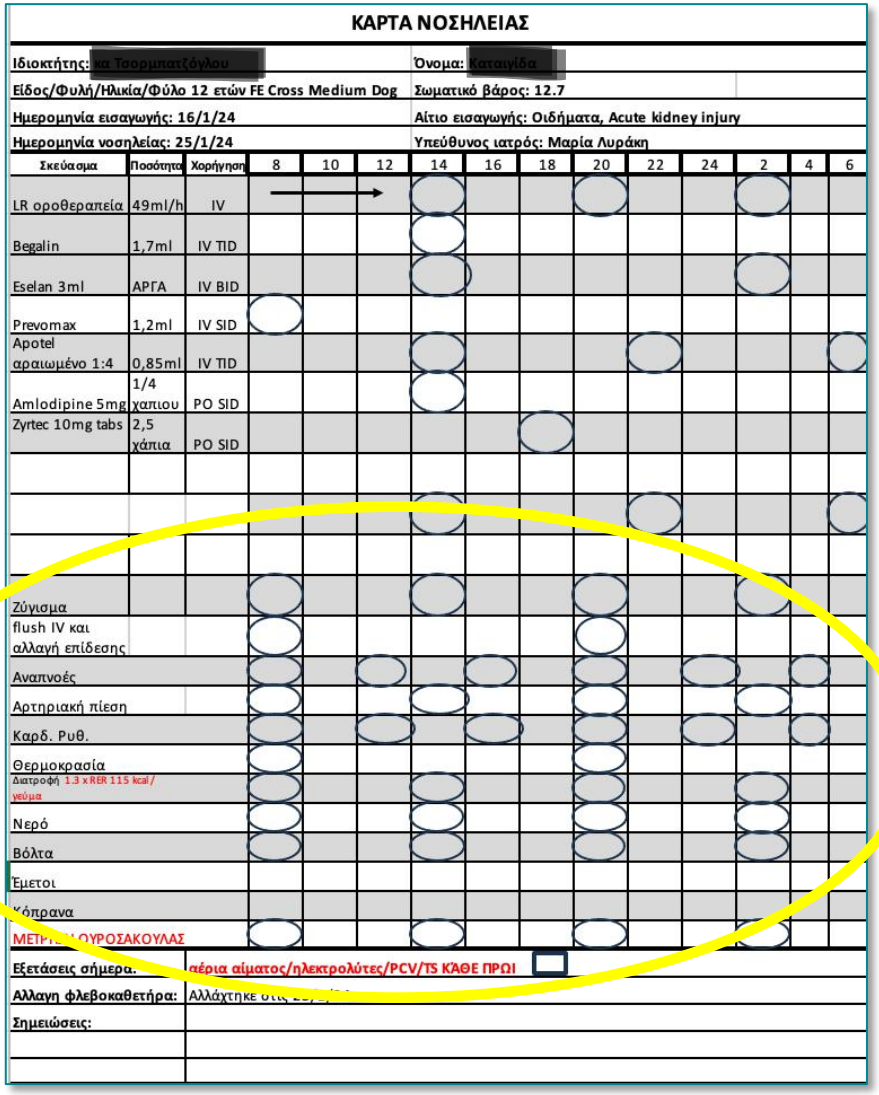
Diuretic use carries the risk of deteriorating renal function, risk/benefit should be considered

Intensive care



ΚΑΡΤΑ ΝΟΣΗΛΕΙΑΣ

Ιδιοκτήτης: [redacted]							**Όνομα:** [redacted]							
Είδος/Φυλή/Ηλικία/Φύλο 12 ετών FE Cross Medium Dog							**Σωματικό βάρος:** 12.7							
Ημερομηνία εισαγωγής: 16/1/24							**Αίτιο εισαγωγής:** Οιδήματα, Acute kidney injury							
Ημερομηνία νοσηλείας: 25/1/24							**Υπεύθυνος ιατρός:** Μαρία Λυράκη							
Σκεύασμα	**Ποσότητα**	**Χορήγηση**	8	10	12	14	16	18	20	22	24	2	4	6
LR οροθεραπεία	49ml/h	IV	→	→	→									
Begalin	1,7ml	IV TID												
Eselan 3ml	ΑΡΓΑ	IV BID												
Prevomax	1,2ml	IV SID												
Apotel αραιωμένο 1:4	0,85ml	IV TID												
Amlodipine 5mg	1/4 χαπιου	PO SID												
Zyrtec 10mg tabs	2,5 χάπια	PO SID												
Ζύγισμα														
flush IV και αλλαγή επίδεσης														
Αναπνοές														
Αρτηριακή πίεση														
Καρδ. Ρυθ.														
Θερμοκρασία														
Διατροφή 1.3 x RER 115 kcal/ γεύμα														
Νερό														
Βόλτα														
Έμετοι														
Κόπρανα														
ΜΕΤΡΗ[illegible]ΟΥΡΟΣΑΚΟΥΛΑΣ														
Εξετάσεις σήμερα:		αέρια αίματος/ηλεκτρολύτες/PCV/TS ΚΑΘΕ ΠΡΩΙ ☐												
Αλλαγη φλεβοκαθετήρα:		Αλλάχτηκε στις [illegible]												
Σημειώσεις:														

- ❑ Heart rate, pulse quality
- ❑ Respiratory rate, thoracic auscultation
- ❑ Blood pressure
- ❑ Weight
- ❑ Temperature
- ❑ Urine output measurement
- ❑ Fluid rate re-assessment based on the above
  - ❑ Fluid rate after hydration: **INS = OUTS**

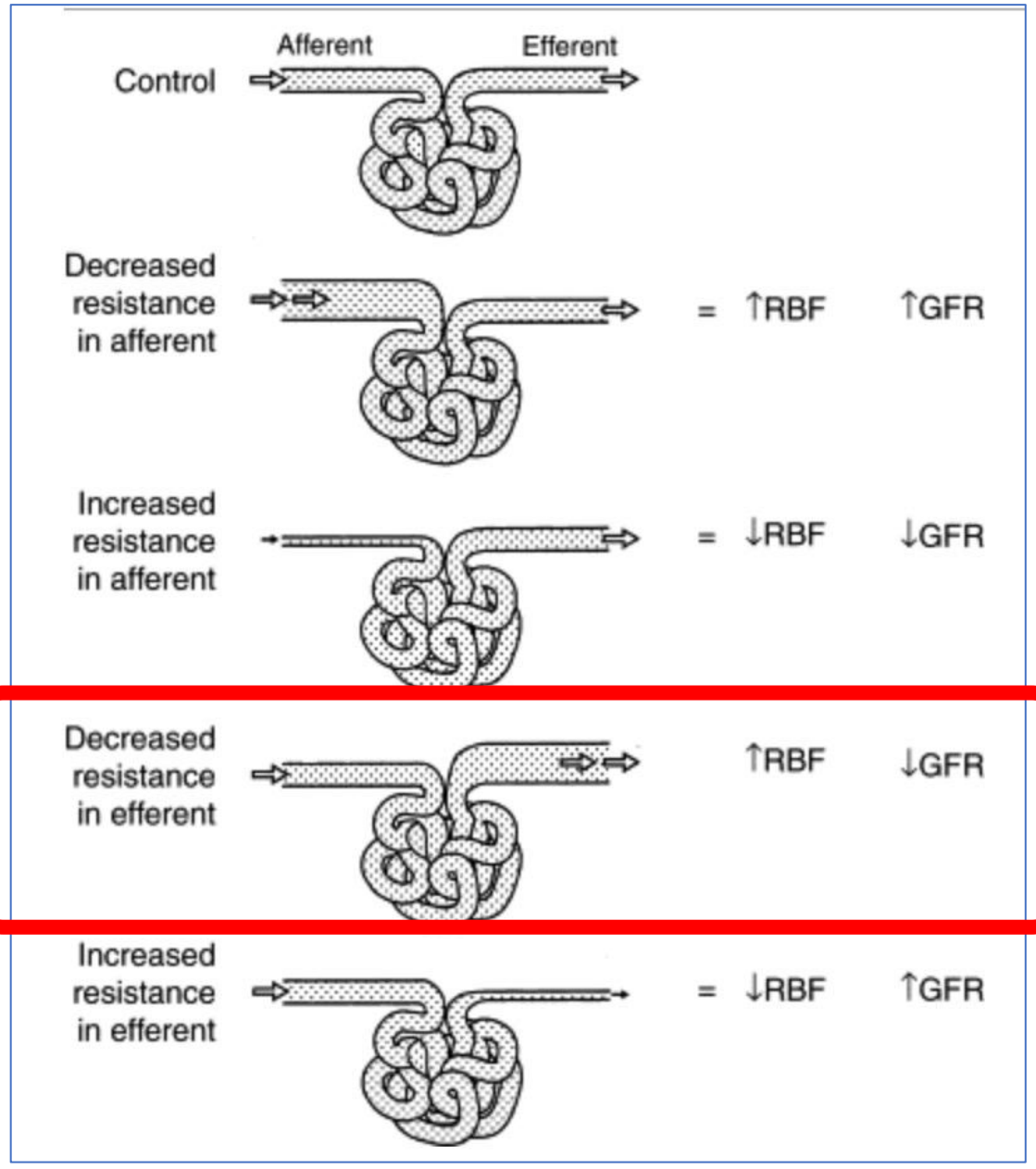
Blood work every 24-48 hours

- ❑ pH, base excess, electrolytes, Ca^{2+}, PCV, total solids, glucose
- ❑ Urea, Creatinine, Phosphate

Systemic hypertension

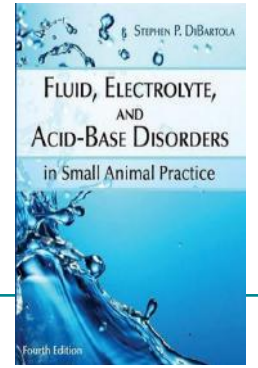
- Prolonged hypertension over multiple measurements
- Re-assessed after correction of the dehydration / improve renal perfusion
- Could be due to fluid overload – correct fluid overload first
- Important complication – increases the mortality risk
- Drugs
  - Amlodipine 0.1-0.6 mg/kg/day, starting at a low dose.
  - Hydralazine 0,5-3 mg/kg IV BID and/or CRI 1.5-5 mg/kg/minute IV

AVOID RAAS inhibitors



RAAS inhibitors

- ACE inhibitors (benazepril, enalapril)
- Angiotensin receptor blockers



Symptomatic care

- Severe uremia often causes gastrointestinal signs
- Gastric ulcers or oesophagitis may occur
- Treatment
  - Omeprazole 1mg/kg BID
  - Maropitant 1mg/kg IV SID
  - Ondansetron 0.5-1 mg/kg BID
  - Metoclopramide 2mg/kg/day CRI IV
- Analgesia
  - Paracetamol 10mg/kg IV TID
  - Buprenorphine 0,02 mg/kg IV TID

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Consider a dose reduction in azotaemia 25-50%

animals

MDPI

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Review

Drug-Dosing Adjustment in Dogs and Cats with Chronic Kidney Disease

Francesca De Santis [1], Andrea Boari [1], Francesco Dondi [2,*] and Paolo Emidio Crisi [1]

[1] Faculty of Veterinary Medicine, Veterinary Teaching Hospital, University of Teramo, Località Piano d'Accio, 64100 Teramo, Italy; fdesantis@unite.it (F.D.S.); aboari@unite.it (A.B.); pecrisi@unite.it (P.E.C.)
[2] Department of Veterinary Medical Sciences, Alma Mater Studiorum, University of Bologna, Via Tolara di Sopra 50, 40064 Bologna, Italy
* Correspondence: f.dondi@unibo.it

Simple Summary: Adjusting drug dosages in dogs and cats with chronic kidney disease (CKD) can be challenging in clinical practice due to the lack of specific indications in the current literature; moreover, the evaluation of renal function through the measurement of glomerular filtration rate (GFR), which is unanimously considered as a requisite for most adjustment strategies, is often hard to perform in clinical settings. Therefore, the present review aims to provide practical guidelines for dosage adjustment in CKD patients through an overview of the available literature.

Abstract: Chronic kidney disease is a common kidney disorder in adult and aged dogs and cats; the management of associated complications and comorbidities generally requires a life-long medical treatment to ensure a good quality of life of affected patients. However, indications and the literature on drug dosing in dogs and cats with chronic kidney disease are often lacking. The aim of this review is to revise the current literature on drug dosing in canine and feline patients with renal impairment, with a special focus on the most commonly used medications to manage chronic kidney disease and possible comorbidities.

Keywords: chronic kidney disease; small animals; dose adjustment; nephrology; pharmacology

check for updates

Citation: De Santis, F.; Boari, A.; Dondi, F.; Crisi, P.E. Drug-Dosing

Symptomatic care

- In cases of severe proteinuria and risk of thromboembolism
  - Clopidogrel 2-4mg/kg/SID
  - Rivaroxaban 1-2 mg/kg/SID

Nutrition

- AKI patients are in a catabolic state
- Protein, energy, vitamin deficiencies may delay recovery
- Energy requirement during hospitalisation = 1.5 x RER
  - RER = (30 x BW) + 70 = kcal/day
    - Alternatively, RER = 70 x $BW^{0,75}$
- Renal diet is not necessary any may be too restricted in proteins and too high in fat
- Phosphate binders can be added if required

Nutrition

- ❑ Pancreatitis often co-exists in dogs, we do not have data in cats
- ❑ Diagnosing pancreatitis can be difficult in light of azotaemia (similar clinical signs)
- ❑Avoid high – fat diets (e.g. renal)

J Vet Intern Med. 2018 Mar-Apr; 32(2): 722–726. PMCID: PMC5867007
Published online 2018 Feb 22. doi: 10.1111/jvim.15047 PMID: 29469974

Assessment of Canine Pancreas-Specific Lipase and Outcomes in Dogs with Hemodialysis-Dependent Acute Kidney Injury

K. Takada, [1] C.A. Palm, [2] S.E. Epstein, [3] and L.D. Cowgill [2]

Renal diet

- ❑ Do not use force feeding
- ❑ Appetite stimulants
  - ❑Maropitant 1mg/kg IV SID
  - ❑ Μιρταζαπίνη 0,5 mg/kg PO/ transdermal SID (2mg/cat)
- ❑ Feeding tube placement early on during hospitalisation

Haemodialysis

- ❑ Not widely available
- ❑ It is helpful in acute disease after toxin ingestion
- ❑ It may help other AKI patients at the stage of oliguria / anuria

Peritoneal dialysis

- ❑ Requires surgical placement of special catheters in the abdomen
- ❑ No special haemodialysis machine is required – less expensive
- ❑ Intensive 24-hr care is required

Peritoneal Dialysis in Veterinary Medicine

Rachel L. Cooper, DVM[a], Mary Anna Labato, DVM[b,*]

KEYWORDS

• Peritoneal dialysis • Acute kidney injury • Anuria • Urea kinetic

Peritoneal dialysis is a modality of renal replacement therapy that is commonly used in human medicine for treatment of chronic kidney disease and end-stage kidney failure. Peritoneal dialysis employs the same principle as other forms of renal replacement therapy: the removal of uremic solutes by diffusion across a semipermeable membrane. In hemodialysis and continuous renal replacement therapy, blood is passed through straw-like semipermeable membranes, which are bathed in a dialysate. By contrast, peritoneal dialysis uses the peritoneum as a membrane across which fluids and uremic solutes are exchanged. In this process, dialysate is instilled into the peritoneal cavity and, through the process of diffusion and osmosis, water, toxins, electrolytes, and other small molecules are allowed to equilibrate. The dialysate is then removed and discarded, carrying with it uremic toxins and water. This process is repeated continuously as needed to achieve control of uremia.

Although peritoneal dialysis is used primarily for the treatment of chronic kidney disease in people, reports from as early as 1923 demonstrate its role in treating acute kidney injury.[1] Its use has also been described for removal of dialyzable toxins and to treat pancreatitis, electrolyte and acid base abnormalities, refractory congestive heart failure, and inborn errors of metabolism. In veterinary medicine, the most common use of peritoneal dialysis is to treat acute kidney injury, though it can be used for any of the aforementioned indications as well.

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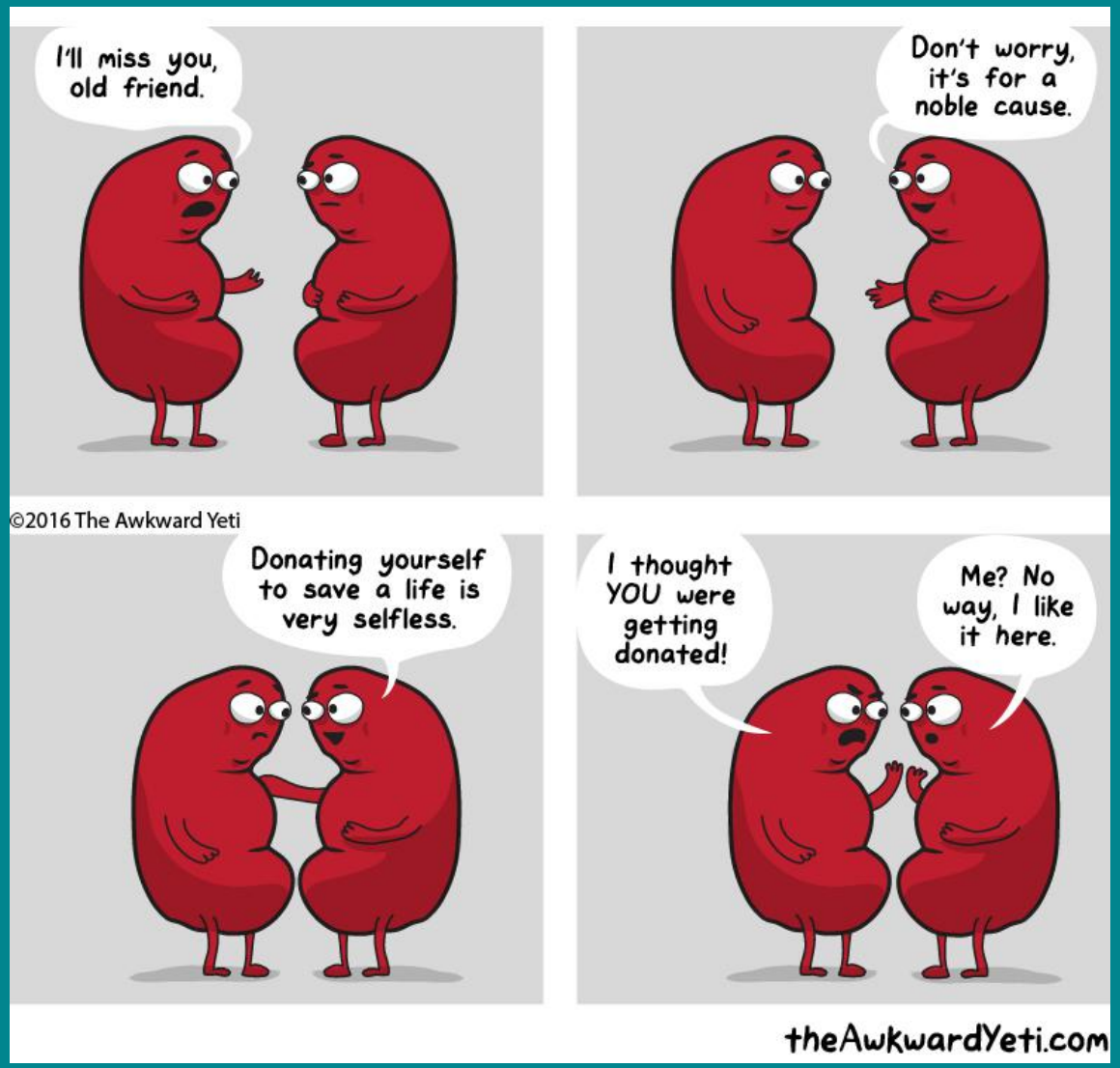
Prognosis

- Depends on the underlying cause
- Acute-on-chronic disease has a worse prognosis
- Azotaemia is often reversible regardless of the magnitude
  - Takes time
  - Intensive care for a couple of days may be needed (average duration is a week)

Summary

- ❑ Every patient in our wards is at risk for AKI and urea/creatinine/urine output should be monitored in all patients
- ❑ Early diagnosis and treatment will improve outcome
- ❑ Fluid overload is a common complication
- ❑ Under-feeding is also common in the hospitalisation ward and frequent hydration and weight checks are recommended



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Thank you for your attention!

Any questions?

marialyraki@outlook.com