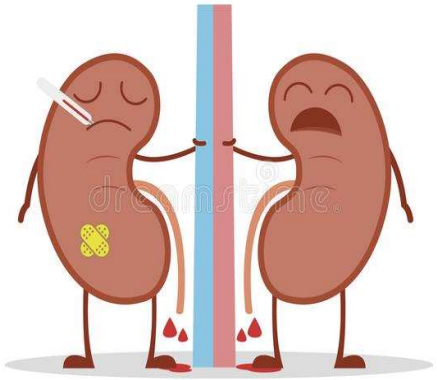
The Acute Kidney

K1



Becky Ness, PA-C, MPAS, DFAAPA, FNKF
American Academy of Nephrology PAs

K2

No disclosures

Slide 1

K1 Kidneys, not kidney's They are not possessed...they are plural
Kim, 4/10/2019

K2 PAs, not PA's...same argument, not possessed, just plural
Kim, 4/10/2019

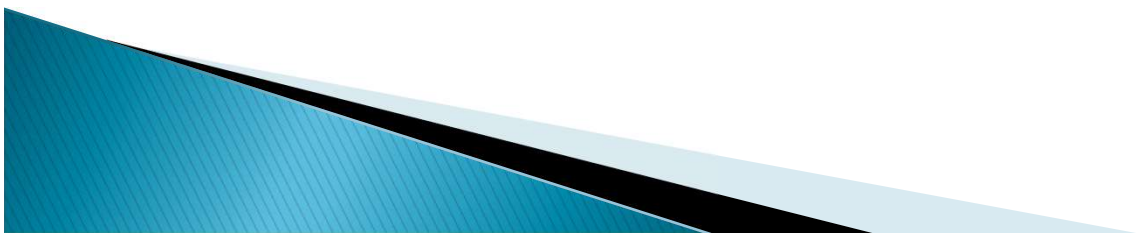
Objectives

- 1. Define Acute Kidney Injury (AKI)
- 2. Identify causes of AKI
  - Community acquired (CA) vs Hospital acquired (HA)
  - Common vs. not so common
- 3. Ascertain testing utilized to identify an AKI as well as utilization of biomarkers in predicting risk of injury/probability of recovery
- 4. Discuss treatment of AKI with objective use of which treatments and when they are indicated

Pre-Test Question #1

Which of the following is the best term to define a patient with acute kidney function changes?

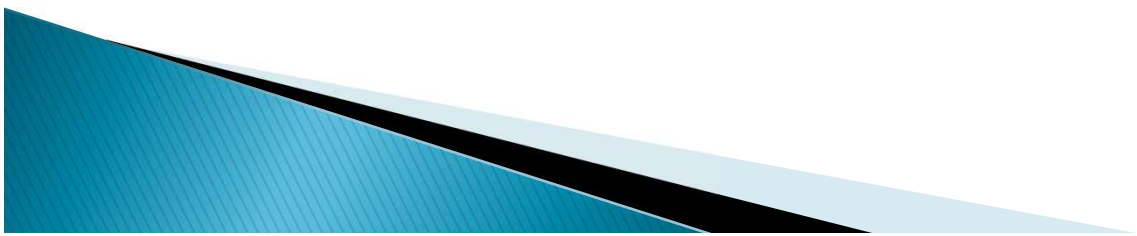
A. Acute Renal Failure
B. Acute Renal Injury
C. Acute Kidney Injury
D. Acute Kidney Dysfunction



Pre-Test Question #2

Which of the following cause AKI?

A. Dehydration
B. Medications
C. Obstructive uropathy
D. All of the above



Pre-Test #3

Which of the following would **NOT** be an indication to initiate renal replacement therapy?

A. Blood pH < 7.1, refractory to bicarbonate therapy

B. Serum potassium > 6.5mEq/L with peaked t-waves on EKG, refractory to medical therapy

C. Fluid overload w/ oliguria in a cardiac surgery patient not responding to diuretic therapy

D. Blood Urea Nitrogen level > 100mg/dL, despite volume expansion with NS

AKI vs. ARF

At the *turn of the century (almost 2 decades ago now…)*, AKIN (Acute Kidney Injury Network), recommended that the term AKI replace ARF

Why?

- Standardization- Allows definitions to be international for research and outcomes purposes
- Just the injury, without dialysis, increases long term kidney risk
- Everyone has a different point when they start dialysis

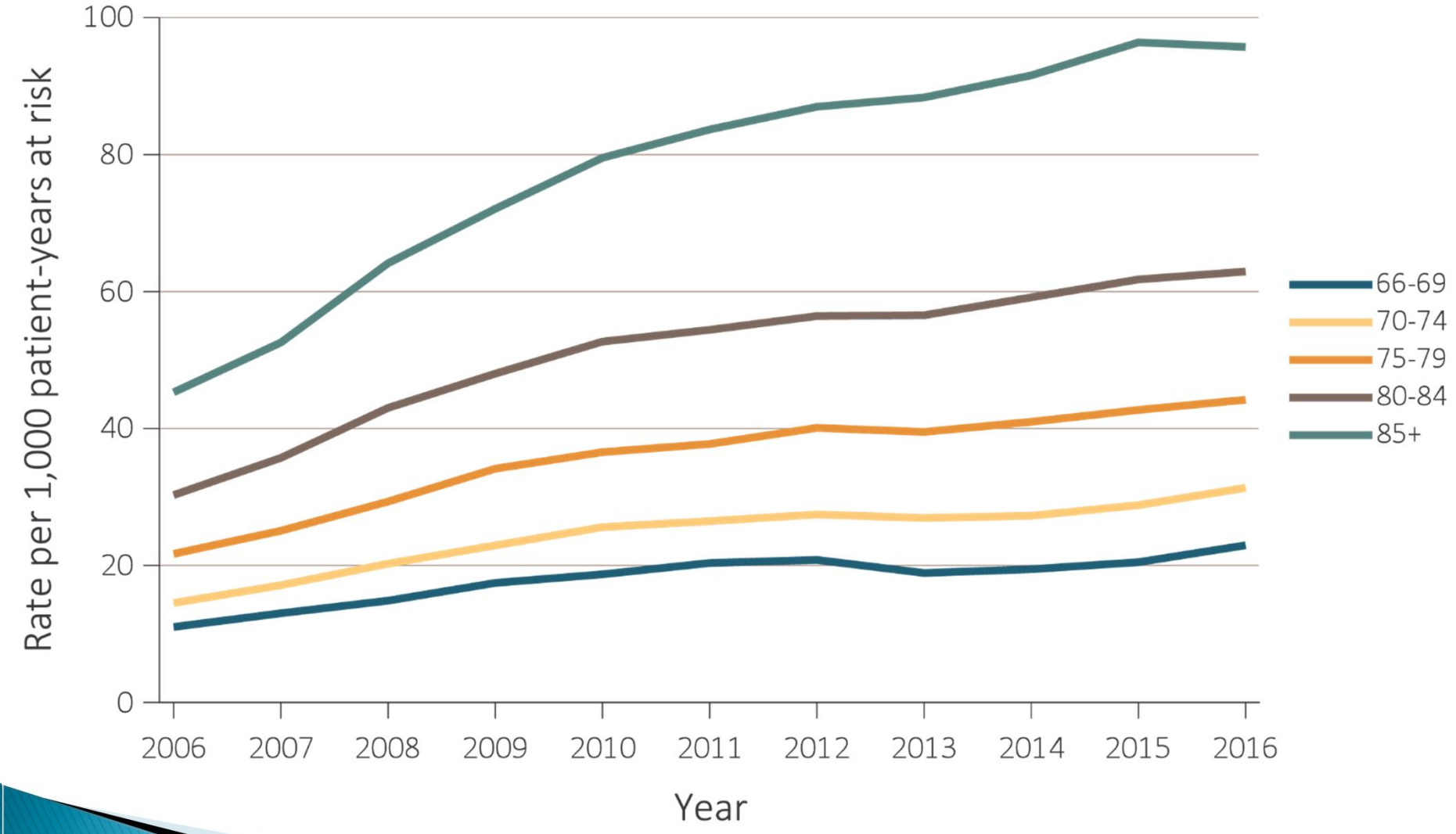
Definitions of AKI

Stage	Urine Output	RIFLE	AKIN	KDIGO
1	<0.5 ml/kg/hr for 6 hr	**Risk**: Increase in SCr of 1.5x or decrease in GFR > 25%	Increase in SCr 1.5 x baseline or **≥ 3.0 mg/dl**	Increase in SCr of 1.5-1.9 x baseline or ≥ 0.3 mg/dl increase in SCr
2	<0.5 ml/kg/h for 12 hr	**Injury**: Increase in SCr 2x or decrease in GFR > 50%	Increase in SCr 2x baseline	Increase in SCr of 2-2.9 x baseline
3	<0.3 ml/kg/hr for 24 hr or anuria for 12 hr	**Failure**: Increase in SCr 3x or decrease in GFR > 75%	Increase in SCr 3x baseline **or ≥ 4 mg/dl** (with acute rise of > 0.5mg/dl)	Increase in SCr of > 3x baseline or increase in SCr ≥ 4.0 mg/dl or initiation of RRT

Loss & ESRD of the RIFLE criteria are not included in this staging chart as they are considered outcome variables.

Used with permission, Erica Davis, PAC, Acute Kidney Injury: The Ugly Truth, Elsevier

AKI Epidemic – USRDS Data Survey



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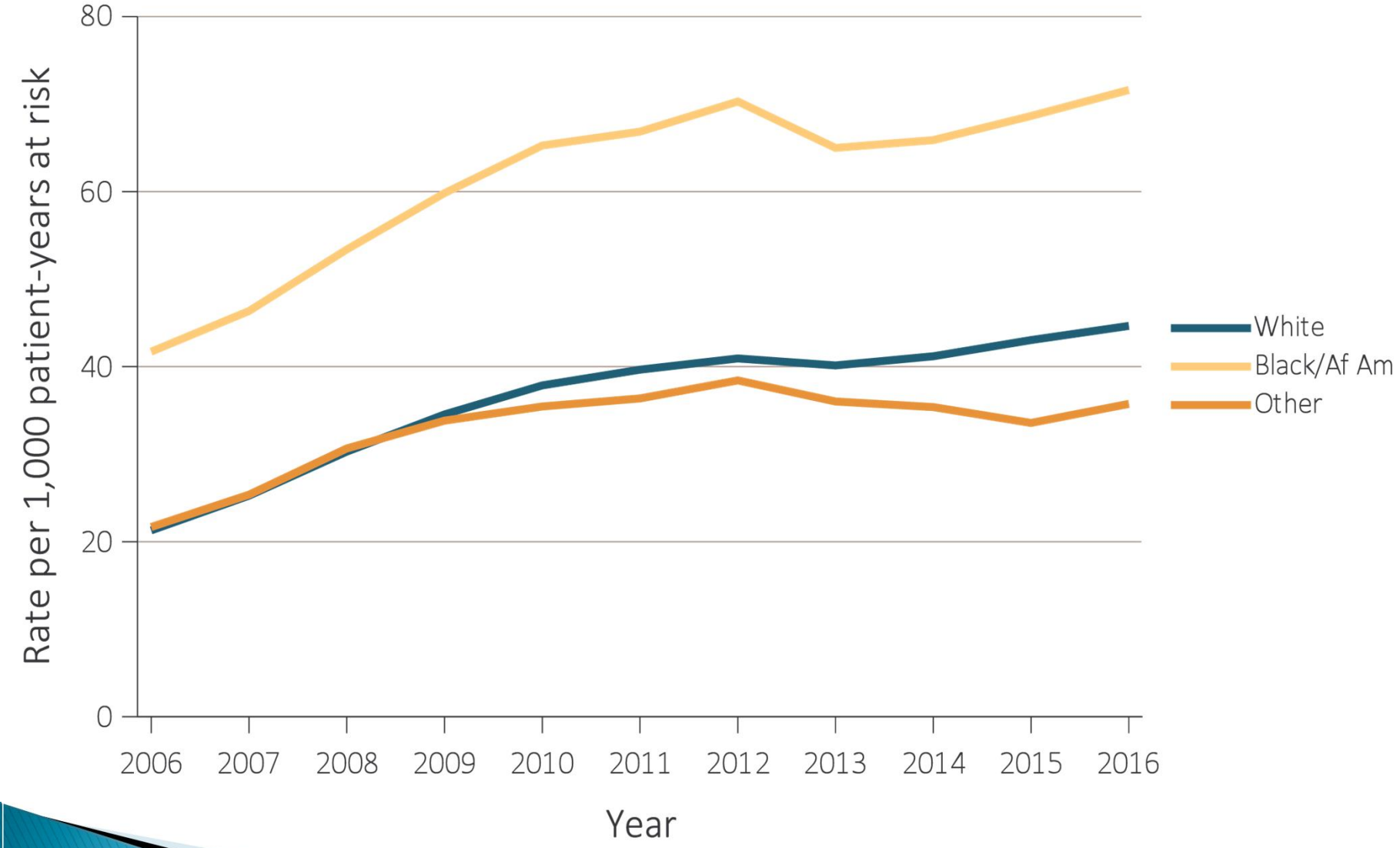
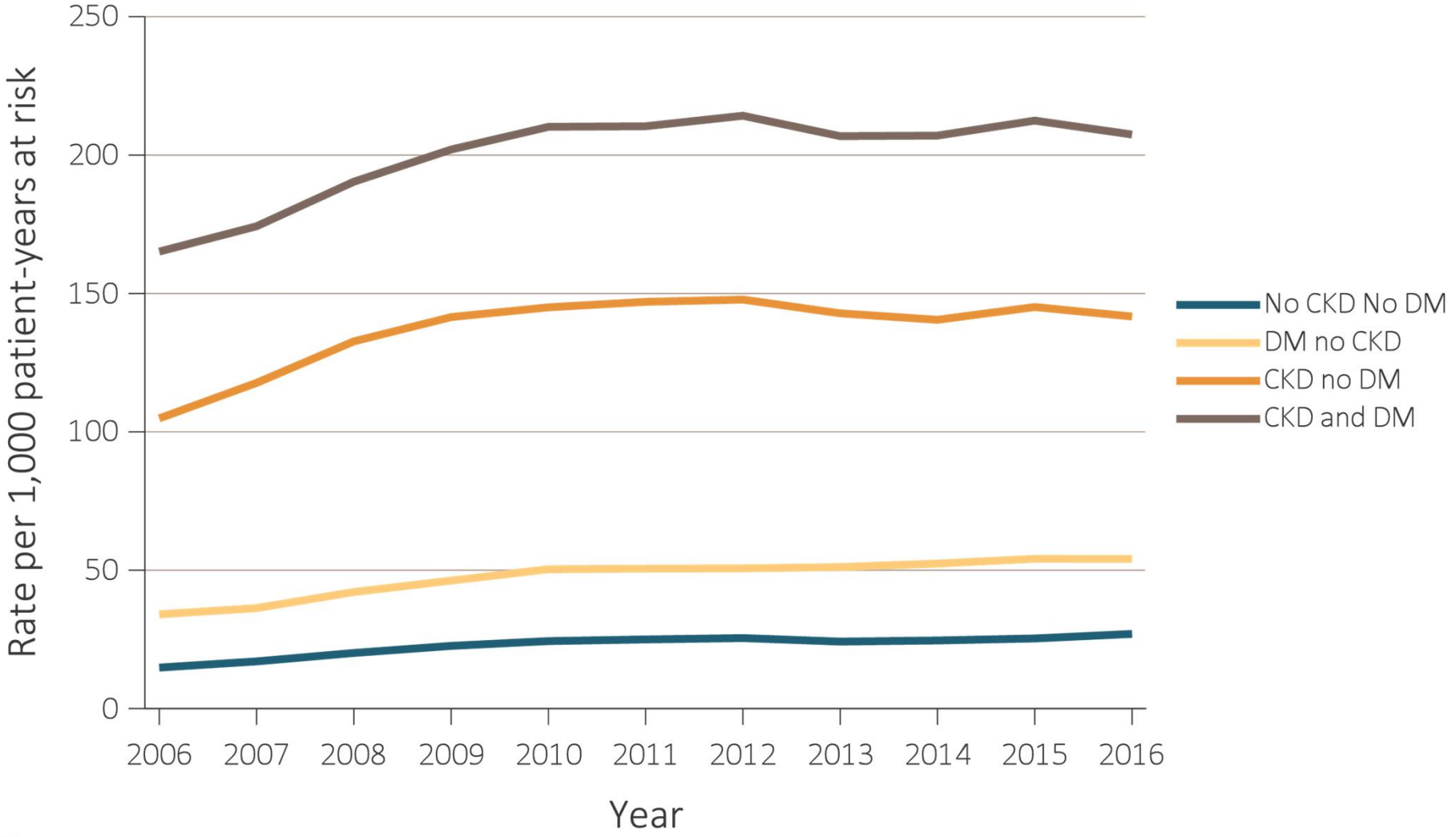
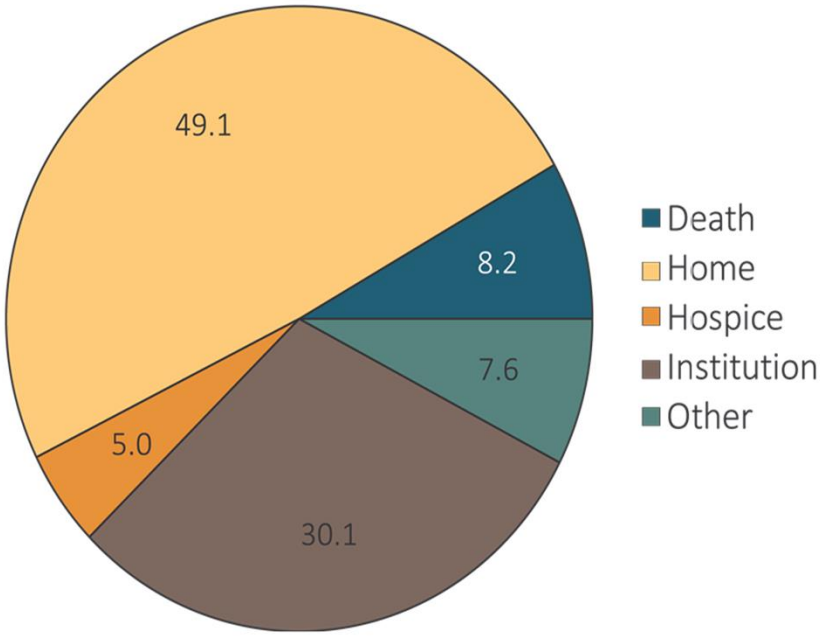


AKI Epidemic – USRDS Data Survey

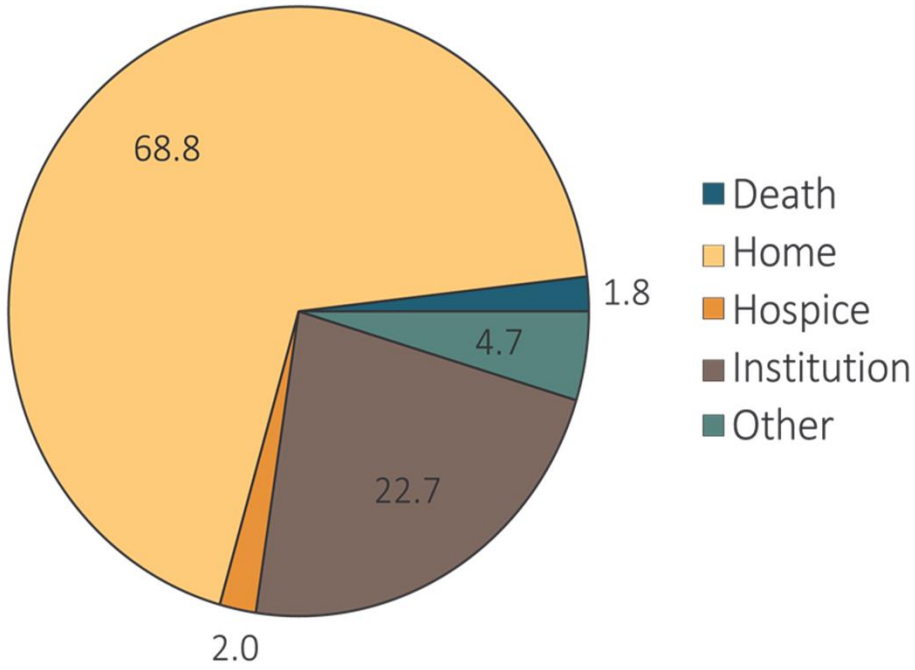


Hospital discharge status for Medicare patients

With AKI Dx



Without AKI Dx



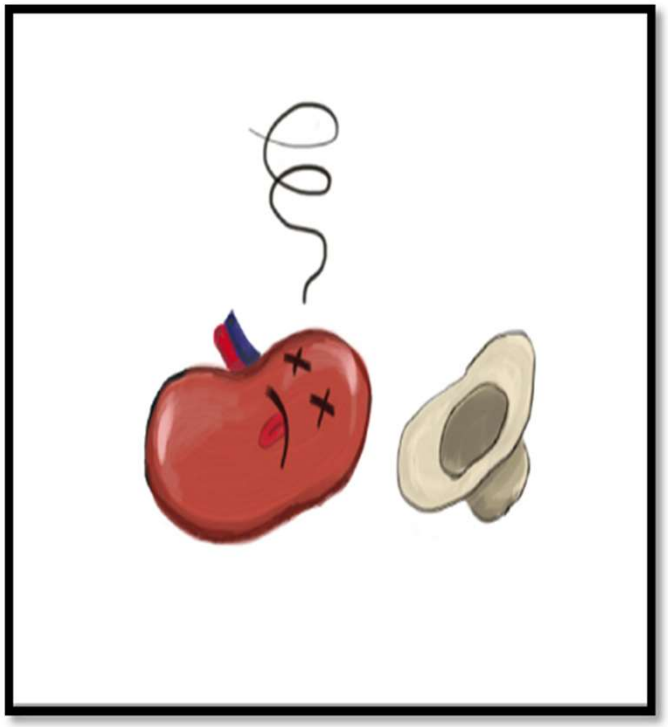
Frequency of AKI

- 7-18% of **ALL** inpatients
  - 30-70% critically ill
    - 5% require renal replacement therapy (RRT)
- 20-30% of those who undergo elective cardiac surgery

1. Lewington. Raising Awareness of AKI: Global Perspective of a Silent Killer. *Kidney international Sept 2013*
2. Van Duijl, TT et al. Kidney Injury Biomarkers in an Academic Hospital Setting. *Clin Biochem Rev 40 (2) 2019*

Who will presents with AKI?

- Older
- Diabetic
- CKD
- Black
- Hispanic
- Hospitalized
- Previous AKI DX
- Anyone can present with AKI but play the odds….

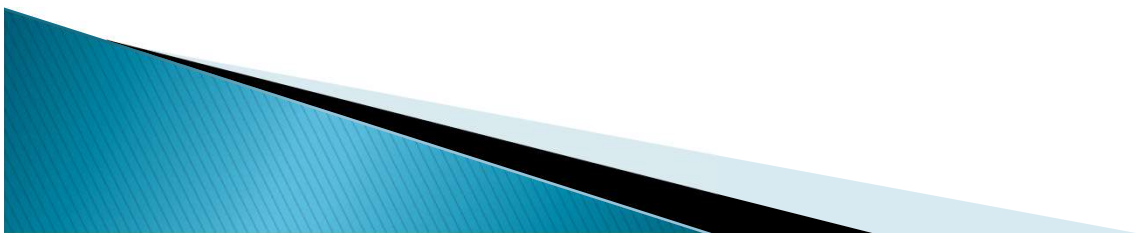


Recognizing AKI

- Not actually a “disease”, but rather a clinical syndrome
  - Heterogeneous disorder
  - Multiple etiologies
- Goal is to promptly identify and treat the underlying cause

Diagnostic approach to AKI

- Evaluation
  - Careful history
    - Drug history
    - Radiocontrast exposure
    - Recent hypotension
    - Urinary symptoms
  - Physical exam
    - Evaluation of fluid status
    - Signs of acute or chronic heart failure
    - Signs of infection/sepsis
    - Signs of systemic illnesses



Diagnostic approach to AKI

- Evaluation
  - Chemistry
    - Elevated creatinine and likely BUN
    - Possibly electrolyte abnormalities
    - Acid base disorder
  - Urinalysis and urine indices
    - RBCs or RBC casts
    - WBCs or WBC casts
    - Proteinuria
    - Hyaline or granular casts
    - Urine electrolytes (especially urine sodium, FENa)

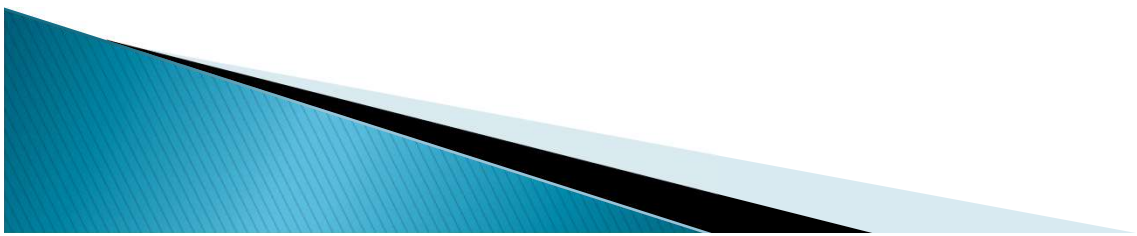
Diagnostic approach to AKI

- Renal/Bladder ultrasound
  - Size and echogenicity
  - Mass/tumor/cyst
  - Hydronephrosis
- Kidney biopsy
  - Histologic findings to confirm/support clinical diagnosis

Diagnostic approach to AKI

- What else can be done?

- Ongoing research into biomarkers
  - Traditional/Classic
  - Functional
  - Damage
    - Cell injury
    - Stress-associated
    - Inflammatory
  - Pre-injury



Diagnostic approach to AKI

- Universal attributes of an ideal biomarker
  - Easily measured – rapid test: readily available sample (blood/urine)
  - Cost effective biologic/physiologic assay with high sensitivity/specificity
  - Rapid and dynamic changes in levels that correlate with progression and/or improvement
  - Has prognostic value

Functional Biomarkers

- Creatinine
  - Limitations: poor correlation with GFR in a dynamic state
  - Affected by muscle mass, diet, medications and volume status
- Cystatin C
  - Alternative to Cr
    - Less affected by gender/diet/muscle mass
- Urinary Output

Inflammation Biomarkers

- KIM-1
  - up-regulated in renal cells after injury
  - urine levels ↑ in patients w/ ischemia induced ATN
  - predictive for AKI development 12-24 hr post CABG
- NGAL
  - ↑ in renal tubular cells during inflammatory/ischemic injury
  - ↑urinary levels 2 hr after CABG were predictive for AKI
- IL-18
  - Associated with ATN and not with eGFR defined CKD
  - ↑urinary levels associated with occurrence of AKI

Cell Cycle Arrest Biomarkers

- IGFBP7
- TIMP-2

- First FDA approved biomarker : NephroCheck
  - Product of the two
  - Urinary value predictive of development of moderate-severe AKI in post operative patients

Predictive Biomarkers for RRT in AKI

- NGAL
- IL-18
- Cystatin C
- IGFBP-7*TIMP-2
  - Insulin like growth factor binding protein-7/Tissue inhibitor of mettaloproteinase -2

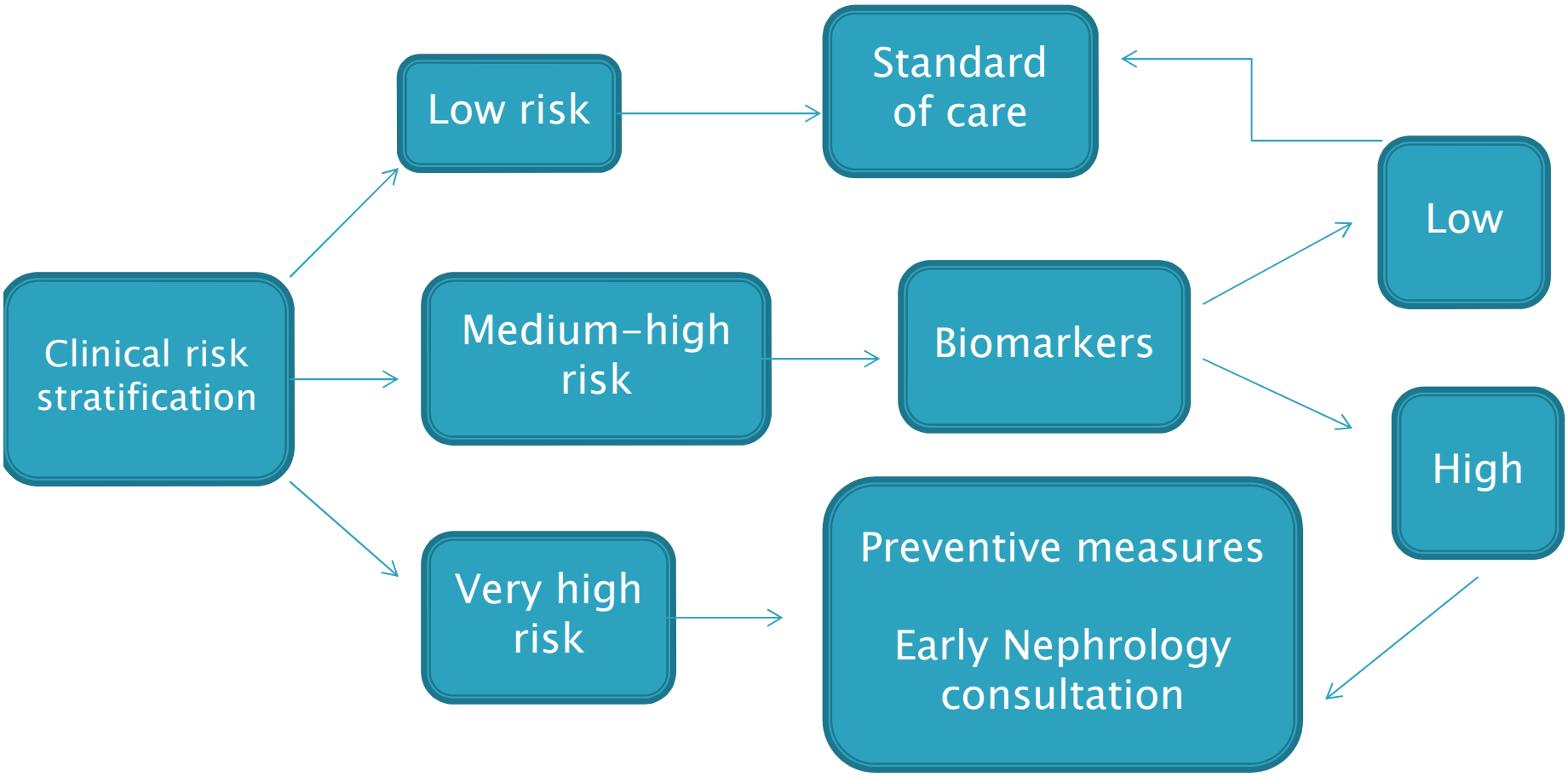
N. Shah and E. Lerma, Novel Biomarkers of Renal Function Introduction and Overview: *Medscape*: 9/20/2017

Predictive biomarkers for AKI

- KIM-1
  - Best characteristics for both sensitivity and specificity
- NGAL
- IL_18
- L-FABP
- TIMP-2 * IGFBP7

Promising biomarkers

- microRNAs
  - Potential for early detection or prognosis
- Development-related molecules
  - Wnt/β-catenin : DKK (Dickkopf)
    - Potential in type of injury and potential outcome
- Hemojuvelin (HJV)
  - Potential as early AKI biomarker in response to Fe homeostasis in AKI
- Osteopontin (OPN)
  - Potential role in sepsis related AKI


Clinical risk stratification
Low risk
Medium-high risk
Very high risk
Standard of care
Biomarkers
Preventive measures
Early Nephrology consultation
Low
High

Types of AKI

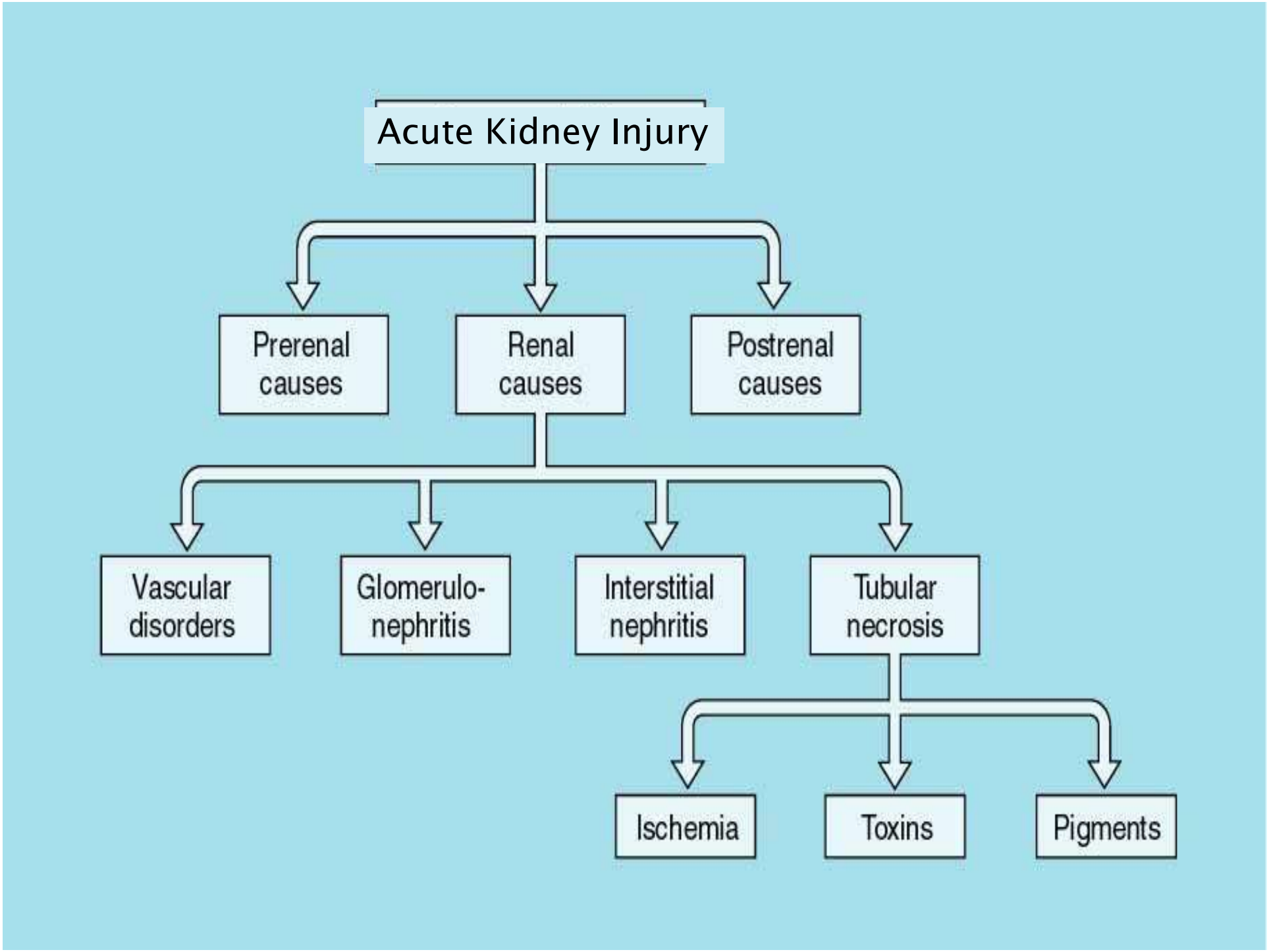
CA-AKI (Community Acquired)

- Most common
- May be as high as 65%
- Increased incidence in summer
- Increased incidence with multiple medications
- Increased incidence with multiple co-morbidities
- We have very little data

NOT CODED

HA-AKI (Hospital Acquired)

- May be missed but EHR coding is helping
- More common if nephrology is consulted
- More studied
- Increases risk for recurrence
- Increases risk for CKD (bidirectional)


Acute Kidney Injury
Prerenal causes
Renal causes
Postrenal causes
Vascular disorders
Glomerulo-nephritis
Interstitial nephritis
Tubular necrosis
Ischemia
Toxins
Pigments

Syndromes of AKI

Prerenal AKI	Intrarenal AKI	Postrenal AKI
↓ Intravascular Volume Dehydration / Hemorrhage GI, Cutaneous or Renal losses Third Spacing ↓ Effective Blood Volume CHF Cirrhosis Nephrotic Syndrome Sepsis Anesthesia Altered Renal Hemodynamics Preglomerular Constriction Postglomerular Vasodilation Medications: ACEI, NSAIDS, CSA Hepatorenal syndrome, Surgery Renal Vascular Obstruction Abdom. Compartment Synd.	Acute Tubular Necrosis Ischemic: Sepsis Hypotension Nephrotoxic: Drugs Pigments Acute Interstitial Nephritis Drug-induced Infection-related Systemic Diseases Malignancy Acute Glomerulonephritis Acute Vascular Syndrome Renal artery dissection Renal artery Throm-Emb Renal vein thrombosis Atheroembolic disease	Upper Tract Obstruction Intrinsic Stone Papillary Necrosis Blood Clot TCC Extrinsic Retroperit. Fibrosis Malignancy Ligation Pelvic Mass Lower Tract Obstruction Urethral Stricture BPH Prostate Cancer TCC of the bladder Stones: bladder Neurogenic bladder Malpositioned Foley Cath

Syndromes of AKI

Prerenal AKI

↓ **Intravascular Volume**
- Dehydration, Hemorrhage
- GI, Cutaneous or Renal losses
- Third Spacing

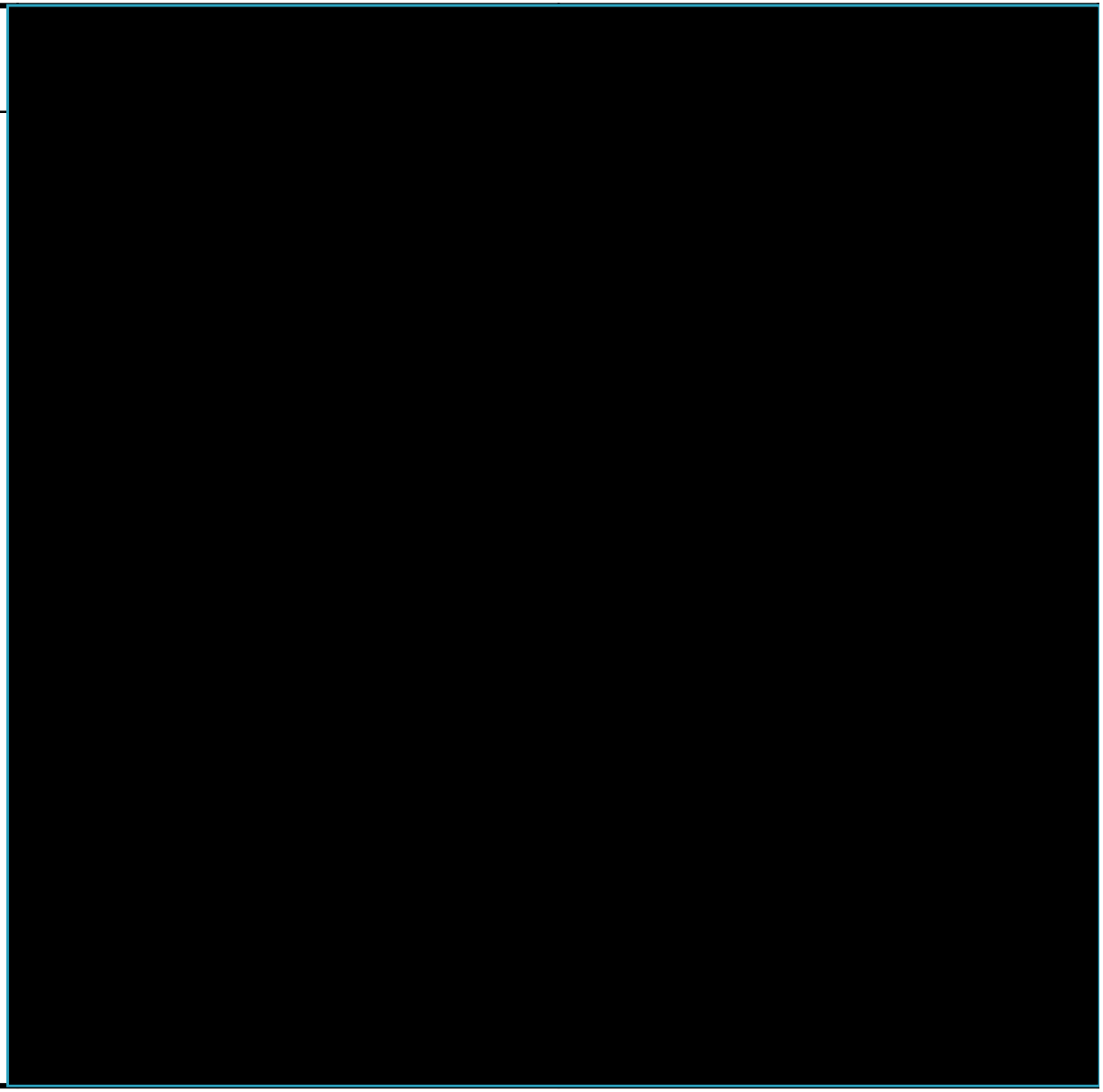
↓ **Effective Blood Volume**
- CHF
- Cirrhosis
- Nephrotic Syndrome
- Sepsis
- Anesthesia

Altered Renal Hemodynamics
- Pre-glomerular Constriction
- Post-glomerular Vasodilation

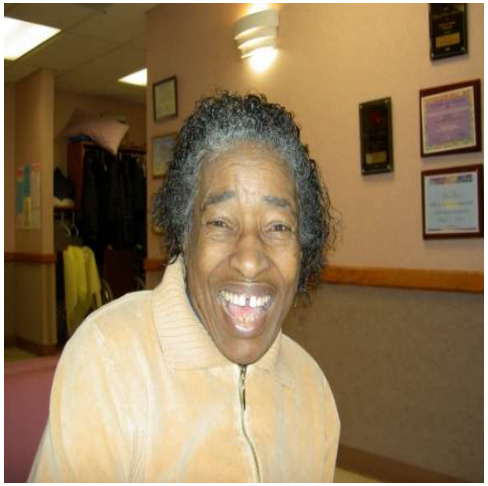
Medications: ACEI, NSAIDS, CSA
Hepatorenal syndrome, Surgery

Renal Vascular Obstruction

Abd Compartment Synd.



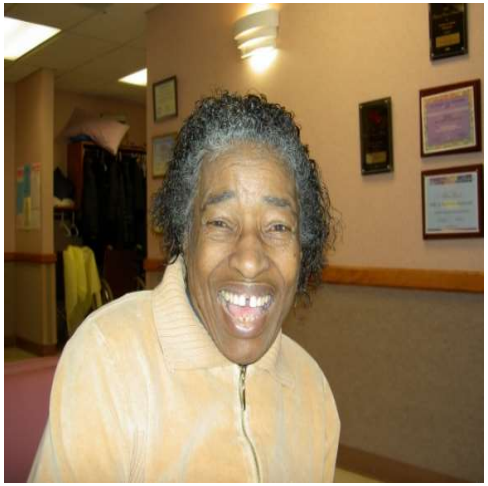
Delia



78 y/o presents ED c/o N&V for the last 48hrs
Unable to keep down intake x 48hrs
No available PMH
PE: sitting: 110/60, HR 80; standing: 80/55, HR 100
Labs: BUN 45mg/dL, SCr 1.5mg/dL, FeNa 0.3%
UA: neg heme/protein, 0-1 RBC, neg WBC,
25-100 hyaline casts

What type of AKI?

A. CA-AKI
B. HA-AKI
C. Not Coded as AKI



Delia

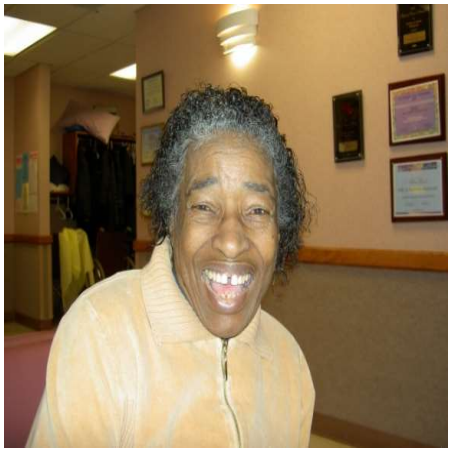
78 y/o presents ED N&V X 48H
No food held down x 48H
No available PMH
PE: 110/60 HR 80 sitting, 80/55, HR 100 standing
Labs: BUN 45mg/dL, SCr 1.5mg/dL, FeNa 0.3%
UA: neg heme/protein, 0-1 RBC, neg WBC, 25-100 hyaline casts

What type of AKI?

A. **CA-AKI**
B. HA-AKI
C. Not Coded as AKI

Pre-renal AKI

- **SYMPTOMS**
  - History of fluid losses
  - Use of NSAIDS or ACEI
  - Thirst
- **SIGNS**
  - Fluid deficit by I/O balance
  - Weight loss
  - Oliguria
  - Orthostatic hypotension
  - Tachycardia
  - Flat neck veins in the supine position
  - Lack of sweat
  - Dry skin and mucosae with loss of skin turgor



FeNa (Fractional Excretion of Na)

Remember FeNa is a urine test

	Pre-Renal	Intra-Renal	Post-Renal
FENa	<1%	>1%	>4%

Used to help differentiate between intra-renal process or extra-renal

FeNa < 1% ~ prerenal cause, volume depletion

Kidney corrects for low fluid state by reabsorbing Na, therefore functional kidney

FeNa > 1% ~ ATN

Failing kidney…cannot compensate and leaking sodium thus indicates kidney diagnosis

Syndromes of AKI

Intra-Renal AKI

Acute Tubular Necrosis

- **Ischemic:**
  - **Sepsis**
  - **Hypotension**
- **Nephrotoxic:**
  - **Drugs**
  - **Pigments**

Acute Interstitial Nephritis

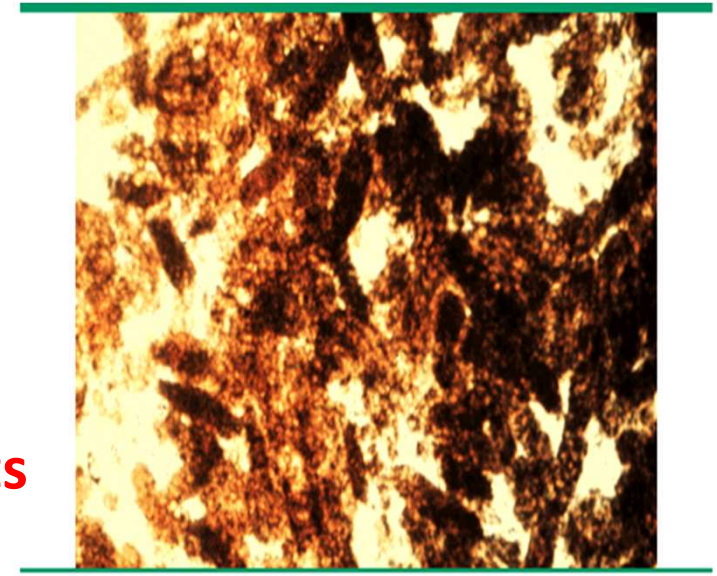
- **Drug-induced**
- **Infection-related**
- **Systemic Diseases**
- **Malignancy**

Acute Glomerulonephritis

Acute Vascular Syndrome

- **Renal artery dissection**
- **Renal artery Thrombo-Emboli**
- **Renal vein thrombosis**
- **Atheroembolic disease**

Acute Tubular Necrosis (ATN)



- Ischemic vs. Toxin vs. Sepsis
  - FeNa typically > 1%
- Characteristic casts in sediment (U/A)
  - Granular and renal tubule
  - **Pathognomonic…muddy brown casts**
  - Urine specific gravity < 1.010
- Marked by back leak and intra-tubular obstruction
- Usually Recovers
  - 3 phases: initiation, maintenance, and recovery
  - First, blame the drug….

Alvin

58 y/o male w/PMH CHF
15# weight gain over 1 week
DOE, PND, unable to lie flat
PE: 3+ BLL edema to knees, wheezes, crackles
Admit to hospital for IV furosemide
Daily labs done, as usual SCr increases

What type of AKI?

A) CA-AKI
B) HA-AKI
C) Not coded as AKI

Alvin

58 y/o male w/PMH CHF
15# weight gain over 1 w
DOE, PND, unable to lie flat
PE: 3+ BLL edema to knees, wheezes, crackles
Admit to hospital for IV lasix
Daily labs done, as usual SCr increases

What type of AKI?

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Which of the following is the most worrisome in Alvin?

A. SCr 0.7mg/dL to 0.9mg/dL
B. SCr 0.8mg/dL to 1.4mg/dL
C. SCr 4mg/dL to 7mg/dL
D. SCr 3.3mg/dL to 3.8mg/dL

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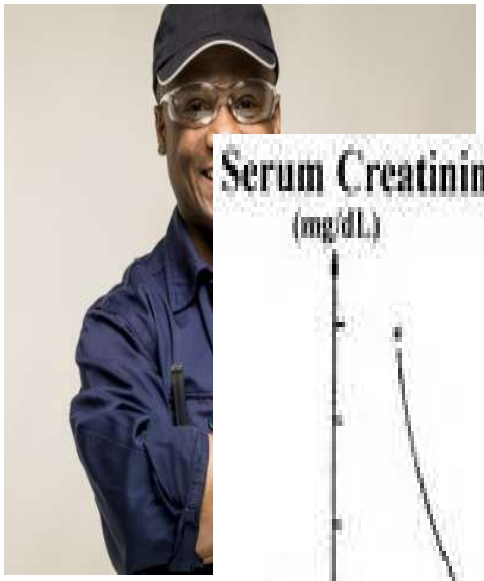
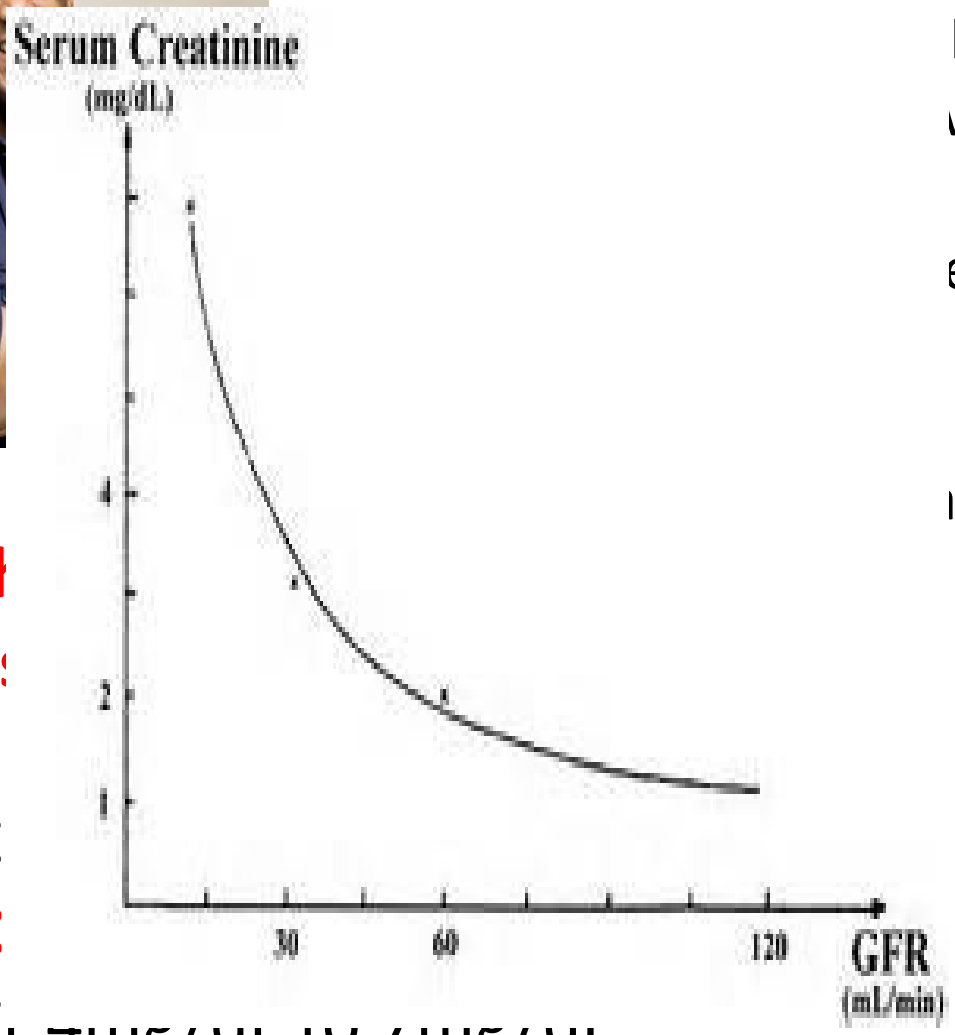
Alvin





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Which
worri

A. SC

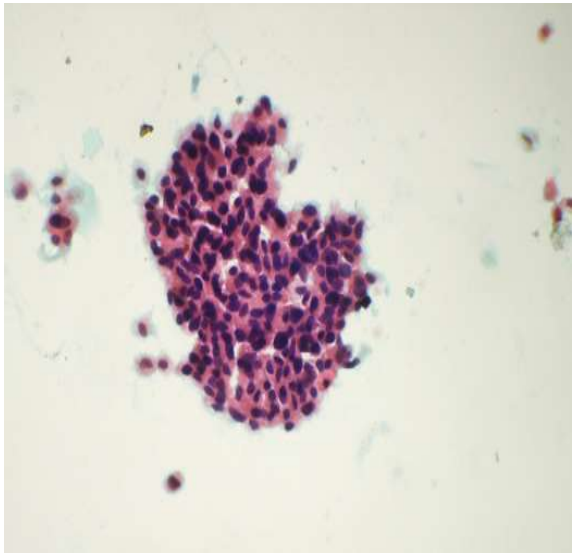
B. SC

C. SC 4mg/dL to 7mg/dL

D. SCr 3.3mg/dL to 3.8mg/dL

Acute Interstitial Nephritis (AIN)

- Classic
  - Fever, Rash, eosinophilia, and eosinophiluria
- Pyuria present
  - WBC casts common
- Rare Infectious etiology
  - Viral infections, legionella
  - leptospirosis, sarcoidosis
- Usual Suspects
  - Drugs- NSAIDs
  - Particularly Antibiotics
    - - Penicillin's; Quinolones
- U/A
  - **Hematuria**
  - Pyuria (eosinophils)
  - WBC casts



Lucy

78 y/o in ED w/CP while shoveling snow
PMH: DM, HTN, GFR 3a, all well-controlled
EKG shows ST elevation, taken to cath lab → stented

F/U labs 3 days later
(KDIGO guidelines state SCr to be drawn 48–72H post exposure)
SCr: 2.7mg/dL (baseline 1.9mg/dL)

What type of AKI?

A) CA-AKI
B) HA-AKI
C) Not coded as AKI

Lucy

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SCr: 2.7mg/dL (baseline 1.9mg/dL)

What is the most likely cause of Lucy's AKI?

A) Cholesterol embolization
B) Post renal AKI
C) Contrast nephropathy
D) Beta blocker induced hypotension

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PMH: DM, HTN, GFR 3a, all well-controlled
EKG shows ST elevation, taken to cath lab → stented

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What is the most likely cause of Lucy's AKI?

A) Cholesterol embolization
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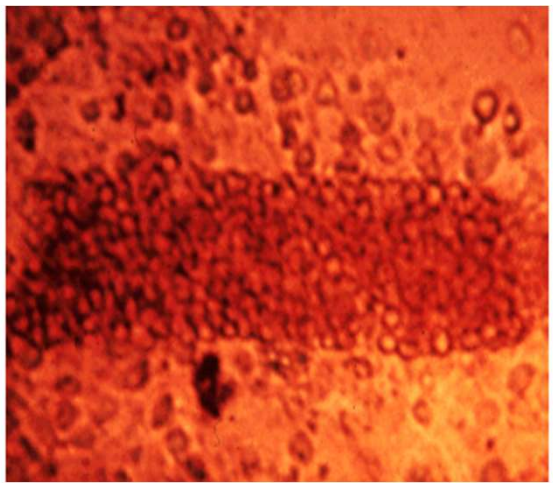
C) **Contrast nephropathy**
D) Beta blocker induced hypotension

Glomerulonephritis (GN)

- Hallmark
  - HTN, Proteinuria, and Hematuria
- Red Cell Casts and Dysmorphic Red Cells in urine sediment
- Usually associated with peripheral edema and low FeNa
- These GN diagnoses are usually nephrotic at presentation:
  - Focal Segmental Glomerulosclerosis (FSGS)
  - Membranous Nephropathy
  - Minimal Change Disease



Brendan

13 y/o male
PMH: sore throat week previously w/N&V
Dark 'coke' colored urine, brought to ED16
Labs: Na 132mEq/L, K 5mEq/L, BUN 80mg/dL, SCr 2.6mg/dL, bicarb 16mEq/L
UA: Dip 2+ blood, 1+ protein, RBC casts on micro

What type of AKI does he have?

A) CA-AKI
B) HA-AKI
C) Not coded as AKI

Brendan

13 y/o male
PMH: sore throat week previously w/N&V
Dark 'coke' colored urine, brought to ED16
Labs: Na 132mEq/L, K 5mEq/L, BUN 80mg/dL, SCr 2.6mg/dL, bicarb 16mEq/L
UA: Dip 2+ blood, 1+ protein, RBC casts on micro

What type of AKI does he have?

A) CA-AKI

B) HA-AKI

C) Not coded as AKI

Brendan

13 y/o male
PMH: sore throat week previously w/N&V
Dark ‘coke’ colored urine, brought to ED16
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B) GN
C) AIN
D) I have absolutely no idea….

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Syndromes of AKI

Postrenal AKI

Upper Tract Obstruction

- **Intrinsic**
  - **Stone**
  - **Papillary Necrosis**
  - **Blood Clot**
  - **TCC (transitional cell carcinoma)**
- **Extrinsic**
  - **Retroperitoneal Fibrosis**
  - **Malignancy**
  - **Ligation**
  - **Pelvic Mass**

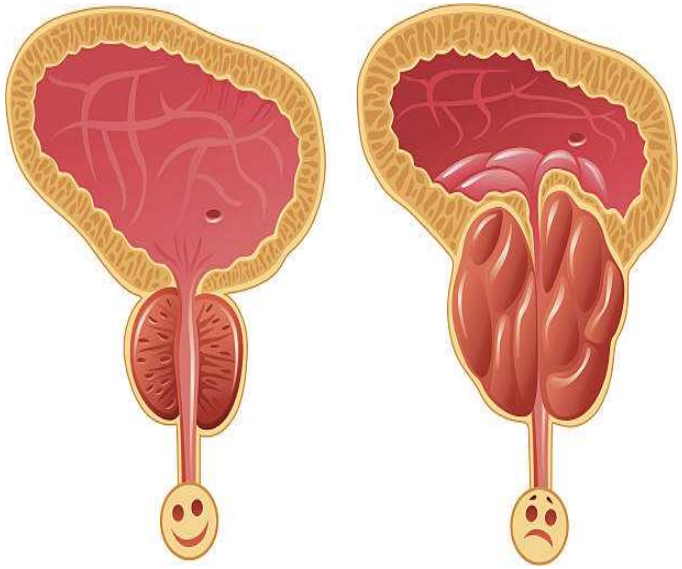
Lower Tract Obstruction

- **Urethral Stricture**
- **BPH**
- **Prostate Cancer**
- **TCC of the bladder**
- **Stones: bladder**
- **Neurogenic bladder**
- **Malpositioned Foley Catheter**

Post-Renal AKI

- **History** of previous urinary tract obstruction or infection

- Look for *bladder outflow obstruction signs*
  - Dysuria, nocturia
  - Frequency, hesitation
  - Weakening of stream, enlarged prostate
  - Distended bladder, flank mass or tenderness



- Pelvic or retroperitoneal disease or surgery

- Complete anuria or wide variations in urine output

- Normal urinalysis in the setting of progressive renal failure

Mary

82 y/o fell at home, R IT FX
ORIF done, morphine PCA, POD#1 foley removed
Admit labs : SCr 1.2mg/dL,
POD#5 SCr 6.2mg/dL
Med review – no NSAIDS, +diphenhydramine

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A) CMP to include serum BUN
B) Urine dip and cell morphology
C) Renal ultrasound
D) None of the above
E) All of the above

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A) 0.5% NS IV fluid
B) Foley
C) Bolus dose furosemide
D) Hold all medications
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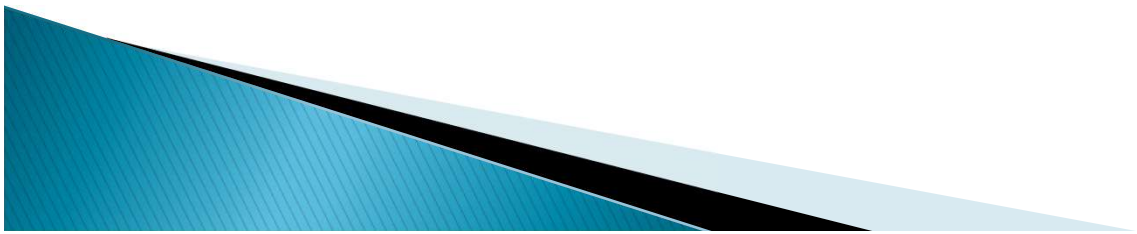
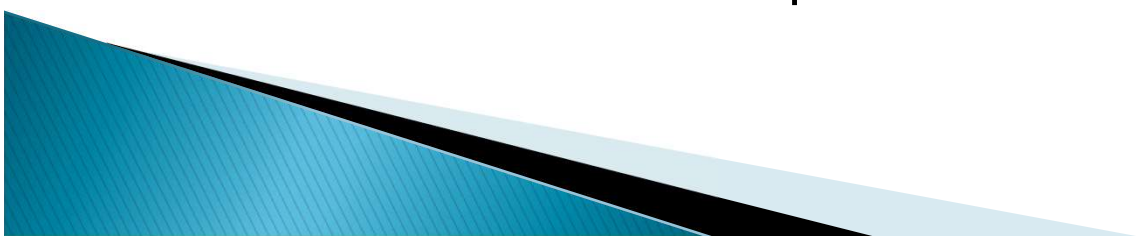
Treatment of AKI

- General paradigm
  - Discontinue all nephrotoxic agents
  - Ensure volume status and perfusion pressure
  - Consider functional hemodynamic monitoring
  - Monitor serum creatinine and urine output
  - Avoid hyperglycemia
  - Consider alternatives to radiocontrast procedures
  - Check for changes in drug dosing
  - Consider renal replacement therapy



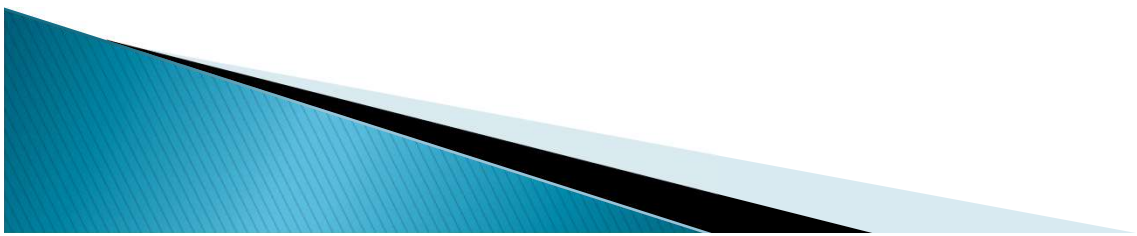
Treatment of AKI

- Outpatient
  - Stable vs unstable
    - Stable patients
      - Identify cause
        - Remove offending medication
        - Encourage fluids
        - Close interval follow to include lab monitoring
    - Unstable patients
      - ED evaluation
      - Admission to hospital



Treatment of AKI

- Inpatient
  - Medical Floor vs ICU
    - Initial treatment usually the SAME
      - STOP offending medication(s)
      - Treat obstruction if present
        - Urinary catheter
      - Measure I/O's
      - Closely monitor vitals, labs

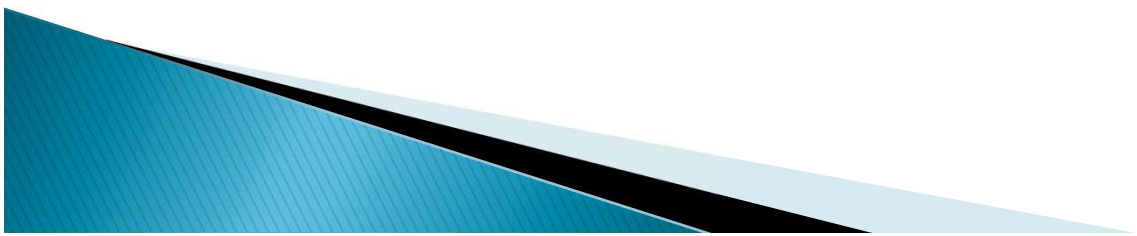


Treatment of AKI

- Volume status
  - Most important aspect of HA-AKI
  - Volume responsiveness vs. Volume unresponsiveness
  - Often existing as a continuum

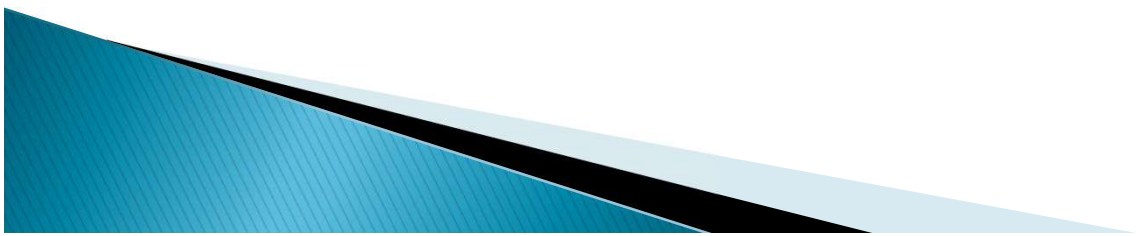
Hemodynamic support

- Fluid management
  - Initial management of most AKI
  - Choice of solution
    - Crystalloid vs. colloid
      - No significant difference in renal outcomes or mortality
      - Colloid considerably more expensive
    - *In most case crystalloid is first choice*
      - Exceptions:
        - Hemorrhagic shock
        - Hepatorenal Syndrome
        - Burn patients
        - Massive fluid resuscitation



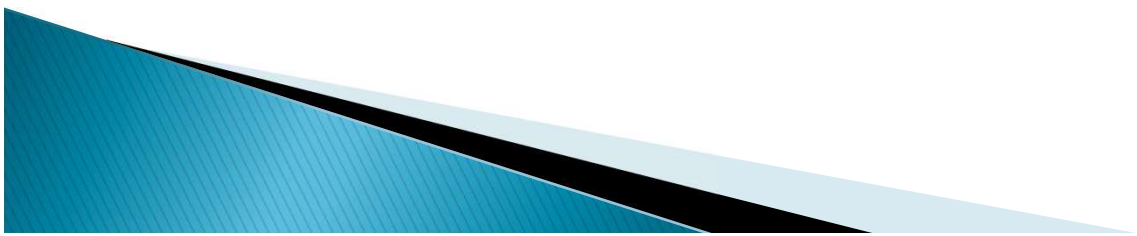
ICU Treatment of AKI

- Vasopressors
  - Help in maintaining renal perfusion
  - Use AFTER intravascular volume replete
  - Vasomotor shock – particularly helpful
    - Sepsis
    - Pancreatitis
    - Anaphylaxis
    - Burns
    - Liver failure



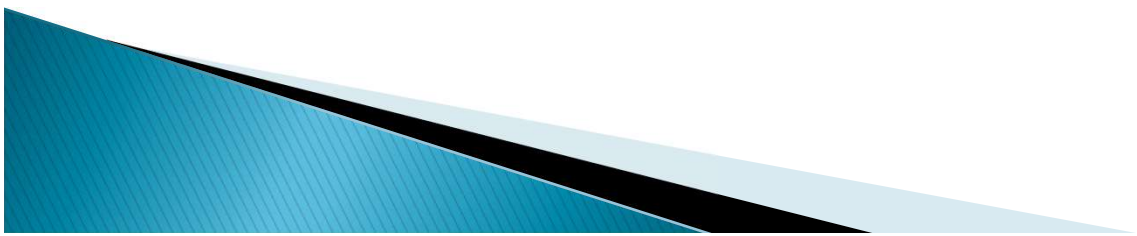
ICU Treatment of AKI

- Glycemic control
  - Stress induced hyperglycemia is common in ICU patients
  - Prior controversy in literature regarding high “tight” glycemic control is needed
  - KDIGO guidelines currently recommend target goal of 110-149 mg/dL



ICU Treatment of AKI

- Diuretics
  - Loop diuretics
    - Rationale – decrease active Na transport therefore O2 demand
    - May help to " wash out" debris from tubules
    - Renal vasodilation, increased renal blood flow
    - Current recommendations
      - **NOT** for routine prevention/treatment of AKI
      - May be useful in managing fluid overload or electrolyte disturbances (potassium)



Alvin

Cr rose 0.8 – 1.4, Tx with IV furosemide with improvement in fluid status however Cr continued to rise 2.0 > 3.6> 8.2
UOP diminished and now < 100 mL/day
K+ is now 5.9 despite IV furosemide
BPs unable to rise > 80 systolic despite 2 pressors

What is the best form of dialysis for Alvin?

A. NONE
B. Intermittent hemodialysis (IHD)
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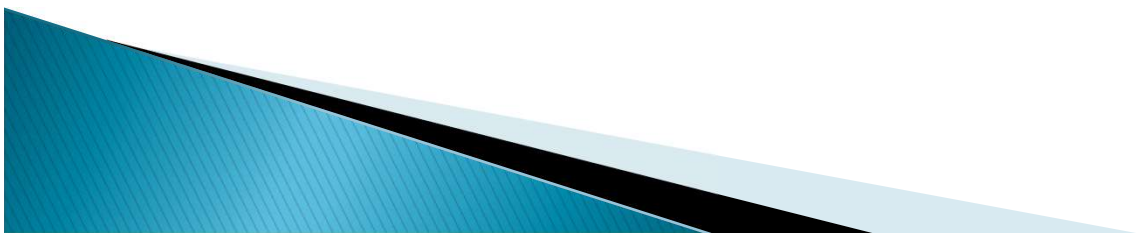
D. I don't know; its why we consulted Neph!

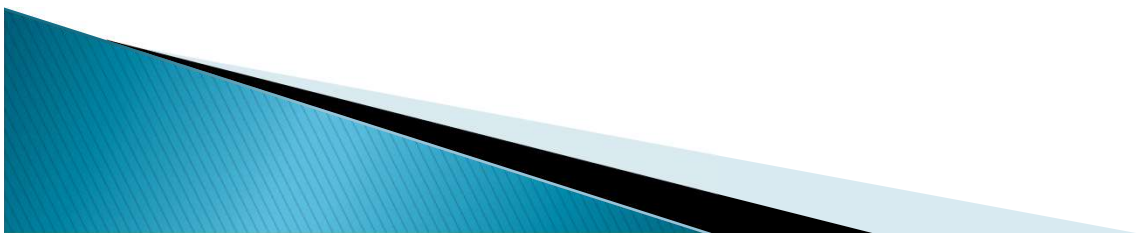
Dialysis in the treatment of AKI

- Timing of renal replacement therapy
  - Optimal timing not defined
    - More and more studies have found no significant difference in " earlier" vs. " later"
  - Potential concerns
    - Risks of RRT
      - Hypotension
      - Arrhythmia
      - Membrane bio incompatibility
      - Vascular access complications
      - Use of anticoagulation administration
  - May delay renal recovery
  - May increase progression of CKD



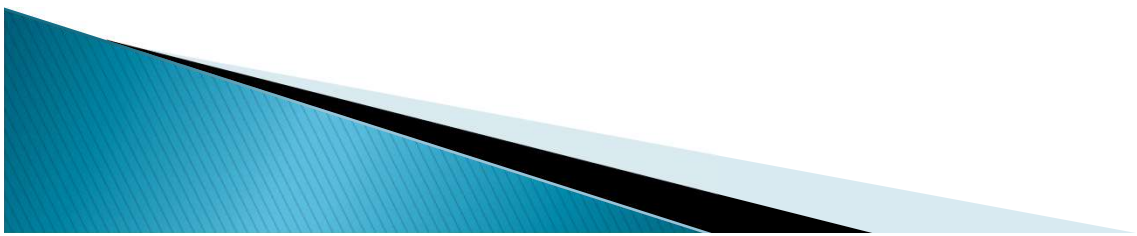
Dialysis in the treatment of AKI

- Timing
  - Absolute indications
    - Severe hyperkalemia
    - Severe acidosis
    - Volume overload
    - Uremic complications
  - Other considerations
    - Severity of underlying illness
    - Degree of dysfunction of other organs
    - Solute burden
    - Need for fluid input for nutrition or medications



Dialysis in the treatment of AKI

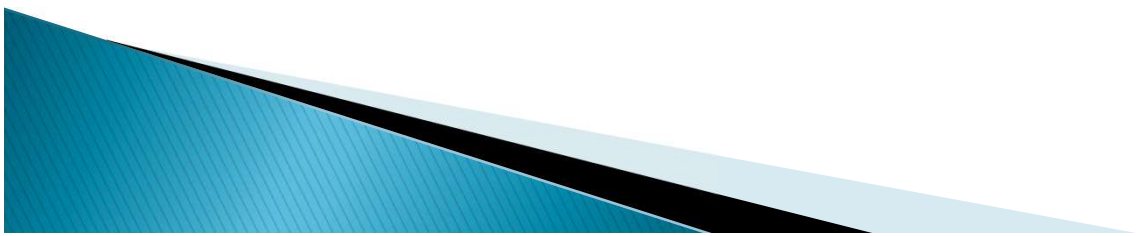
- Modality
  - IHD (intermittent hemodialysis)
  - SLED/EDD (sustained low-efficiency daily dialfiltration)/ (extended daily dialysis)
  - CRRT (continuous renal replacement therapy)
    - CVVH(F)
    - CVVHD
    - CVVHDF
    - SCUF



Dialysis in the treatment of AKI

- Hemodialysis
  - Blood runs countercurrent to dialysate
  - Solute clearance by **DIFFUSION**
  - Size-dependent process

- Hemofiltration
  - Fluid removed via pressure gradient
  - Replaced with equal amount of replacement fluid
  - Solute clearance by **CONVECTION**
  - Size-independent process



Dialysis in the treatment of AKI

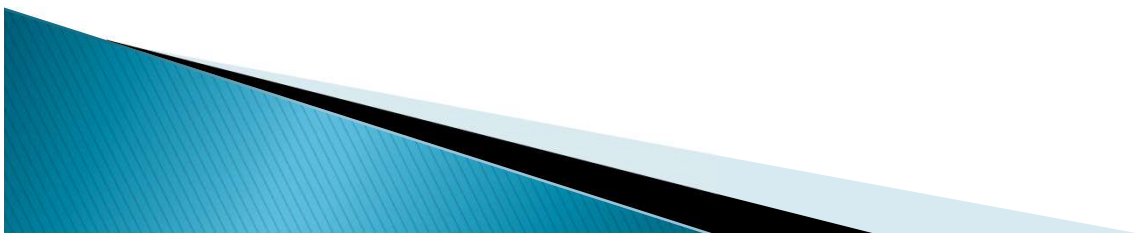
- Modality
  - How to choose?
    - Advantages of CRRT
      - Slower fluid removal
      - More hemodynamic stability
      - Better control of fluid balance
      - Slower control of solute concentration
      - Avoiding large fluctuations in fluid shifts
      - Greater flexibility
      - User-friendly machines
    - Advantages of IHD
      - Fast removal of toxins
      - Restricted treatment time frame
      - Cost

Prognosis

- Mortality remains high
  - Reported rates from 40% – 70%
  - Features associated with higher mortality
    - Age
    - Sepsis
    - Respiratory failure
    - Liver failure
    - Thrombocytopenia

Prognosis

- Mortality
  - Linear relationship between stage of AKI and mortality
    - Stage 1 AKI (RR of 2.4)
    - Stage 2 (RR of 4.15)
    - Stage 3 (RR of 6.37)
- Renal recovery in survivors
  - Varies depending upon stage of AKI
    - Persistent RRT required in around 20% of Stage 3 AKI



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AKI Pearls

- If you look for it, you will find it
- CA-AKI is more common than you think
- Inpatient management of AKI is evolving and a lecture all by itself
- 'Sick day rules' as championed by the UK will decrease CA-AKI
- Worldwide push to decrease AKI
  - 0by25
  - Think Kidneys

ISN 0BY25 AKI INITIATIVE

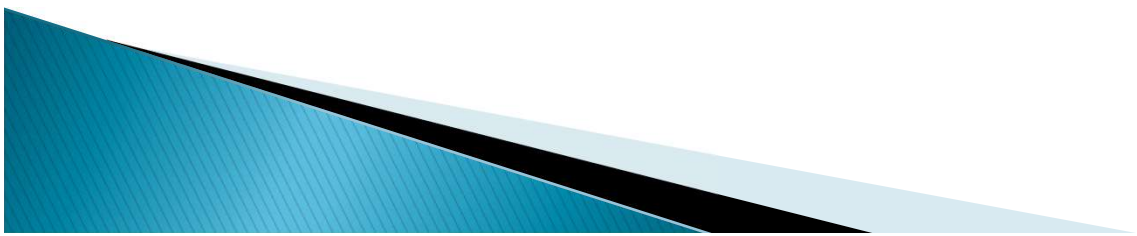
'THINK KIDNEYS'

NHS

Pre-Test Question #1

Which of the following is the best term to define a patient with acute kidney function changes?

A. Acute Renal Failure
B. Acute Renal Injury
C. Acute Kidney Injury
D. Acute Kidney Dysfunction



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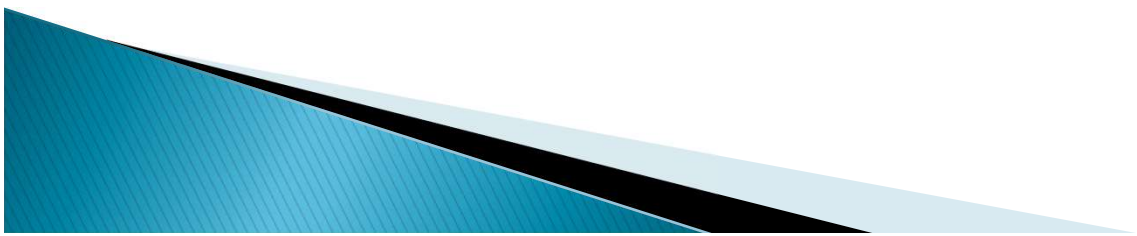
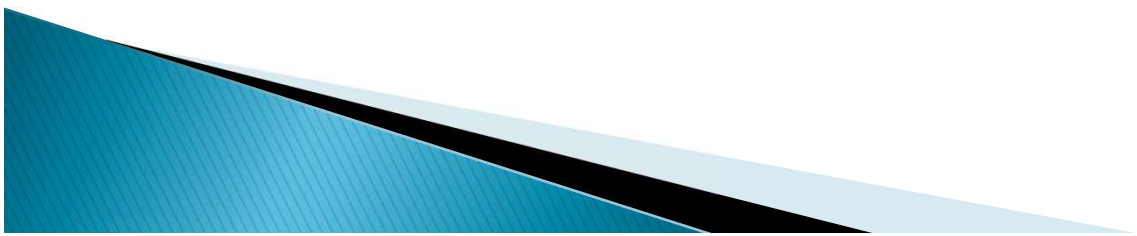


Pre-Test Question #2

Which of the following cause AKI?

A. Dehydration
B. Medications
C. Obstructive uropathy
D. All of the above



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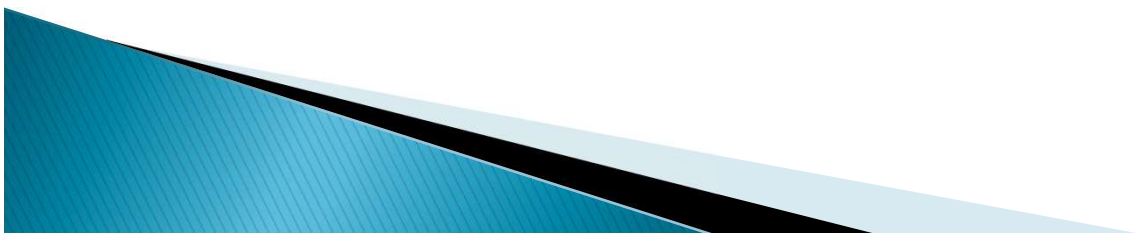
Which of the following would NOT be an indication to initiate renal replacement therapy?

A. Blood pH < 7.1, refractory to bicarbonate therapy
B. Serum potassium > 6.5mEq/L with peaked t-waves on EKG, refractory to medical therapy
C. Fluid overload w/ oliguria in a cardiac surgery patient not responding to diuretic therapy
D. Blood Urea Nitrogen level > 100mg/dL, despite volume expansion with NS



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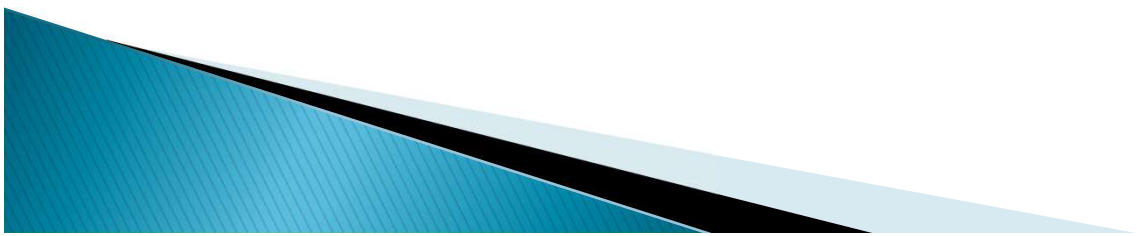

THANK YOU!

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AANPA

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