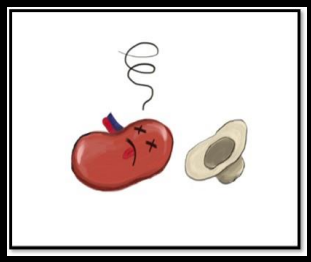
ACUTE KIDNEY INJURY – WHEN THE KIDNEYS HEAD SOUTH

Becky Ness, PA-C, MPAS, DFAAPA, FNKF

American Academy of Nephrology PAs



DISCLOSURES

- *Non-Declaration Statement: I have no relevant relationships with ineligible companies to disclose within the past 24 months. (Note: Ineligible companies are defined as those whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients.)*

OBJECTIVES

Define

- Define Acute Kidney Injury (AKI)

Identify

- Identify causes of AKI
  - Community acquired (CA) vs Hospital acquired (HA)
  - Common vs. not so common

Discuss

- 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 (over 2 decades ago)

AKIN

Recommended **AKI** replace **ARF**

Why?

- Standardization: International definitions for research/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 (2012)
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.

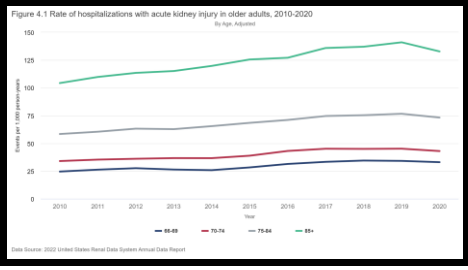
Used with permission, Erica Davis, PAC, Acute Kidney Injury: The Ugly Truth, Elsevier

ALTERNATE CRITERIA

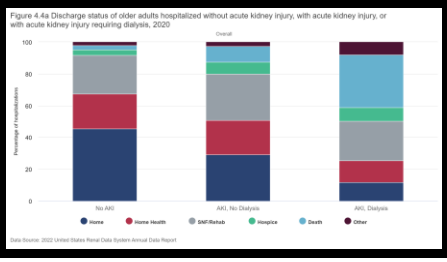
	NKD	AKI	AKD	CKD
Duration	N/A	</=7 days	<3 months	>3 months
Functional criteria	eGFR > 60, stable eGFR, stable Cr, no oliguria for > 6hrs	↑sCr by >/= 50% w/in 7 d OR >/= 0.3 mg/dL w/in 2 d OR oliguria >/= 6 hrs	AKI or eGFR < 60 ↓ eGFR >/= 35% over baseline ↑sCr > 50% over baseline	eGFR < 60
	AND	OR	OR	OR
Structural criteria	No biomarkers of kidney damage	Not yet defined	Elevated marker of kidney damage • Albuminuria • Hematuria	Elevated marker of kidney damage • Albuminuria • Hematuria

NKD no kidney damage; AKI acute kidney injury; AKD acute kidney disease; CKD chronic kidney disease

AKI Epidemic – USRDS Data Survey



HOSPITAL DISCHARGE STATUS AFTER AKI



FREQUENCY OF AKI

10-15% 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 Sept2013*
2. Ronco C; Bellomo, R; Kellum J. Acute Kidney Injury. *Lancet* 2019

WHO WILL PRESENT WITH AKI?

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

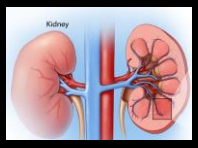
RECOGNIZING AKI

Not actually a "disease", but rather a clinical syndrome

- Heterogeneous disorder
- Multiple etiologies

Goal is to promptly identify and treat the underlying cause

Exposures
- Sepsis
- Critically ill
- Circulatory shock
- Cardiac surgery
- Nephrotoxic medication
- Burns
- Trauma



Risk factors
- Age
- Diabetes/HTN
- CKD
- Female
- Volume depletion
- Cancer
- Anemia
- Chronic disease

Pathogenesis
- ↓renal profusion
- Hypoxic injury
- Oxidative stress
- Endothelial dysfunction
- Inflammatory infiltration
- Direct tubular injury
- Obstruction
- Cytokine-induced injury

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

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?

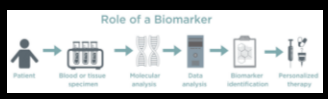
Ongoing research into biomarkers

- 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 (classic/traditional)

- 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 (classic/traditional)

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

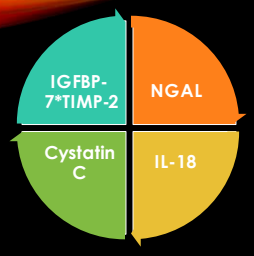
CELL CYCLE ARREST BIOMARKERS

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



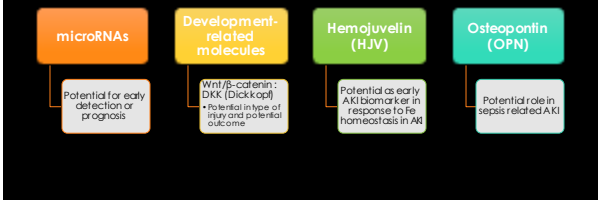
Predictive biomarkers – need for KRT in AKI

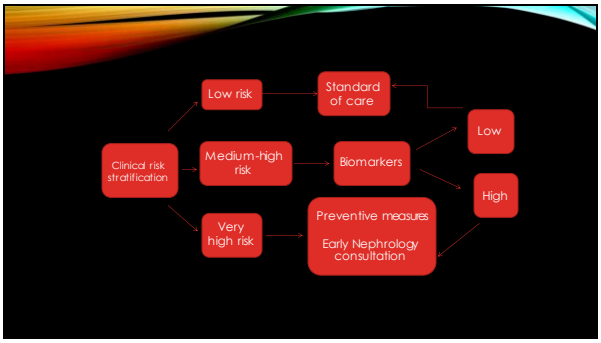
N. Shah and E. Iacma, Novel Biomarkers of Renal Function Introduction and Overview: Medscape: 9/20/2017

PREDICTIVE BIOMARKERS FOR AKI

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

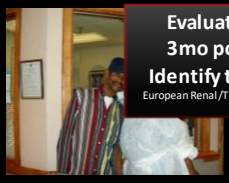
PROMISING BIOMARKERS





WHO WILL PROGRESS?

- At 3 years, 24.7% progressed



Evaluate kidney function
3mo post hospitalization
Identify the high-risk patient

European Renal/Transplant 53rd Congress, Vienna, Austria, May 2016

TYPES OF AKI

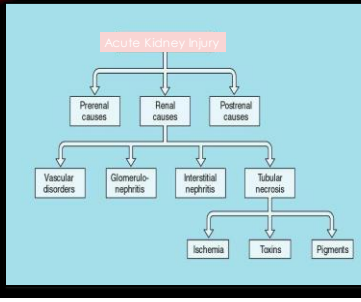
CA-AKI (Community Acquired)

- 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)

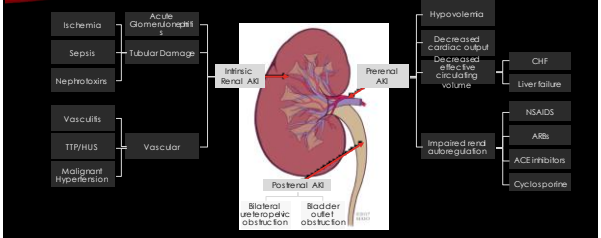
- May be missed but EHR coding is helping
- More common if nephrology is consulted
- More studied
- Increases risk for recurrence
- Increases risk for CKD (bidirectional)



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

ACUTE KIDNEY INJURY SYNDROME



Prerenal 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 Preglomerular Constriction Postglomerular Vasodilation Medications: ACEI, NSAIDS, CSA Hepatorenal syndrome, Surgery Renal Vascular Obstruction Abdom. 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

DELIA

- **What type of AKI?**
  - CA-AKI
  - HA-AKI
  - Not Coded as AKI

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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

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

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

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

SYNDROME 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: PMHx CHF
- 15 # weight gain x 1 week
- DOE, PND, unable to lie flat
- PE: 3+ BLL edema to knees, wheezes and crackles
- Admitted for IV diuresis
- Daily labs demonstrate ↑sCR

What type of AKI?

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

ALVIN

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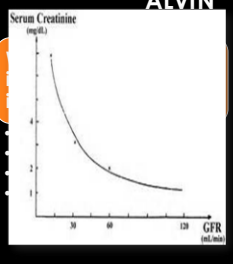
ALVIN

Which of the following is the most worrisome in Alvin?

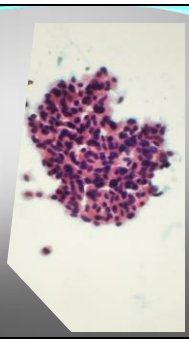
- SCr 0.7mg/dL to 0.9mg/dL
- SCr 0.8mg/dL to 1.4mg/dL
- SCr 4mg/dL to 7mg/dL
- SCr 3.3mg/dL to 3.8mg/dL

ALVIN



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 • Antibiotics – Penicillin's; Quinolones • PPIs
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 with PCP

(KDIGO guidelines state SCr to be drawn 48-72H post exposure)

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

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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

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 in PMD
- *(KDIGO guidelines state SCr to be drawn 48-72H post exposure)*
  - SCr: 2.7mg/dL (baseline 1.9mg/dL)

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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

ACUTE GLOMERULAR DISEASE

Systemic	Renal
Diffuse proliferative lupus nephritis	Anti-GBM disease (Goodpasture's)
ANCA-associated vasculitis	Post-infectious GN
Thrombotic microangiopathies (HUS/TTP)	Membranoproliferative GN
Polyarteritis nodosa	IgA nephropathy
Cryoglobulinemia	

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
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What is the cause of his AKI?

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B) GN
C) AIN
D) I have absolutely NO idea.....

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SYNDROME 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

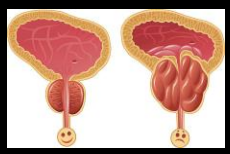
History of previous urinary tract obstruction or infection

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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B) Urine dip and cell morphology
C) Renal ultrasound
D) None of the above

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B) Foley
C) Bolus dose furosemide
D) Hold all medications

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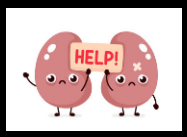
TREATMENT OF AKI

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



OUTPATIENT TREATMENT OF AKI

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

INPATIENT TREATMENT OF AKI

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

INPATIENT TREATMENT OF AKI

Volume status

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

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

HEMODYNAMIC SUPPORT

ICU TREATMENT OF AKI

Vasopressors

- Help in maintaining renal perfusion
  - Norepinephrine
  - Angiotensin II
- 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?

- NONE
- Intermittent hemodialysis (IHD)
- Continuous renal replacement therapy (CRRT)
- 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

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)

Kidney recovery in survivors

- Varies depending upon stage of AKI
  - Persistent RRT required in around 20% of Stage 3 AKI

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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?

- NONE
- Intermittent hemodialysis (IHD)
- **Continuous renal replacement therapy (CRRT)**
- **Peritoneal dialysis (PD)**
- I don't know; its why we consulted Neph!

COVID AND AKI

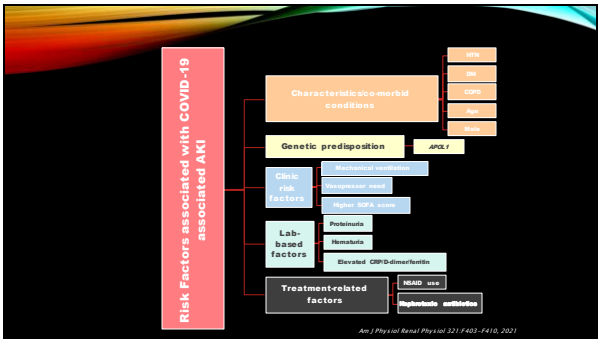
Incidence

- Inpatient: 20-50%
- ARDS due to COVID-19: 50%
  - 15-20% of those required KRT

Risk Factors

- Older age
- Male
- Underlying comorbid conditions

Am J Physiol Renal Physiol. [redacted]



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COVID AND AKI - ETIOLOGY

Direct	Indirect
Direct invasion of virus	ATN: sepsis (fevers/hypotension) and kidney ischemia
Collapsing glomerulopathy	Pre-kidney azotemia: hypovolemia
Immune dysregulation – complement activation	Rhabdomyolysis
	Oxalate nephropathy
	Interstitial nephritis (medications)
	Drug toxicity

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COVID AND AKI

Biomarkers

- Systemic inflammation – associated with more severe infection and AKI
  - IL-6
  - CRP
  - D-dimer
  - ferritin

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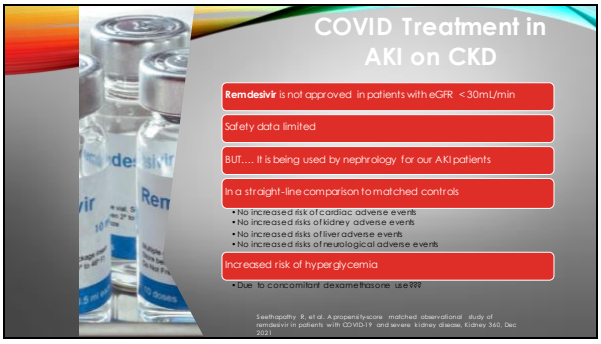
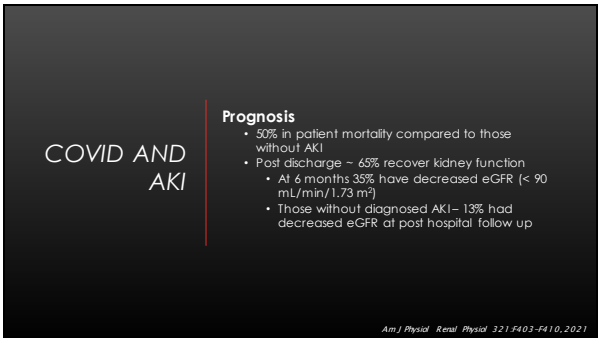
COVID and AKI

Treatment

Incorporate same KDIGO AKI guidelines mentioned throughout presentation

- STOP nephrotoxic medications
- Hemodynamic monitoring
- Fluid management
  - Including STRICT I/O
- Lung-protective ventilation
  - To reduce cytokine and hemodynamic instability secondary to volutrauma and barotrauma
- Initiate KRT


COVID Treatment in AKI on CKD
Remdesivir is not approved in patients with eGFR <30mL/min
Safety data limited
BUT.... It is being used by nephrology for our AKI patients
in a straight-line comparison to matched controls
• No increased risk of cardiac adverse events
• No increased risks of kidney adverse events
• No increased risks of liver adverse events
• No increased risks of neurological adverse events
Increased risk of hyperglycemia
• Due to concomitant dexamethasone use???
Seethapathy R, et al. A propensity-score matched observational study of remdesivir in patients with COVID-19 and severe kidney disease, Kidney 360, Dec 2021


COVID AND AKI
Prognosis
• 50% in patient mortality compared to those without AKI
• Post discharge ~ 65% recover kidney function
• At 6 months 35% have decreased eGFR (< 90 mL/min/1.73 m²)
• Those without diagnosed AKI – 13% had decreased eGFR at post hospital follow up
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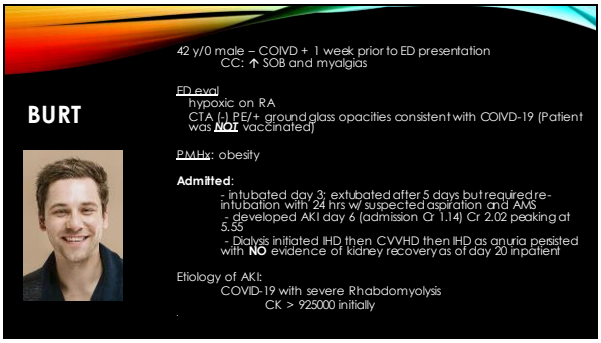
BURT
42 y/0 male – COIVD + 1 week prior to ED presentation
CC: ↑ SOB and myalgias
ED eval
hypoxic on RA
CTA (-) PE/+ ground glass opacities consistent with COIVD-19 (Patient was NOT vaccinated)
PMHx: obesity
Admitted:
- intubated day 3; extubated after 5 days but required re-intubation with 24 hrs w/ suspected aspiration and AMS
- developed AKI day 6 (admission Cr 1.14) Cr 2.02 peaking at 5.55
- Dialysis initiated IHD then CVVHD then IHD as anuria persisted with NO evidence of kidney recovery as of day 20 inpatient
Etiology of AKI:
COVID-19 with severe Rhabdomyolysis
CK > 925000 initially

CAROL

72 y/0 female – COIVD +5 days prior with initial Sx of nausea, fatigue, loose stools, poor intake and weakness

CC: ↑ symptoms with O2 in the low 90's on RA at local UC

ED eval:

hypoxic on RA

CTA (-) PE/+ ground glass opacities consistent with COIVD-19 (Patient was **NOT** vaccinated)

PMHx: Obesity and HTN

Admitted:

Maintained O2 on O2 via NC

DAY 6 ↑ abdominal pain → CT abd/pelvis showed a lg rectus sheath hematoma extending into the pelvis (active bleed noted)

Embolization performed by IR to multiple areas

Severe mass affect to bladder and right ureter (mod-severe) right hydronephrosis

CAROL

Labs

Cr on admission was 0.74

- AKI developed day 7 with Cr 1.56
- Peak Cr 4.41 day 8

Procedures

- Attempted aspiration of hematoma day 7: unable as hematoma was solid
- Right nephroureteral stent placed day 7 due to oliguria (hydronephrosis resolved 2 days later)
- Tunneled hemodialysis catheter placed day 9
- IHD initiated to which she responded well after 2 session
- Ex-lap day 12 due to perforated diverticulum with hematoma and abscess

Discussion

- placed on prophylactic anticoagulation at time of COVID -19 diagnosis per local protocol due to increased VTE risk with resultant hematoma and complications there of

AKI PEARLS

ISN 0BY25 AKI INITIATIVE

THINK KIDNEYS

- 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

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

PRE-TEST QUESTION #2

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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

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THANK YOU!

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