Seed
GLOBAL HEALTH

Project
ECHO

AKI &CKD RELATED EMERGENCIES

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SCOPE OF DISCUSSION

- ✓ Common etiologies of AKI & CKD
- ✓ Common emergency presentation
- ✓ Patient evaluation & investigation

Acute Kidney Injury(AKI)

- Rapid decline in kidney function
- subsequent dysregulation of electrolytes, volume & nitrogenouswastes
- Prerenal, renal, post renal
- Oliguric or non oliguric AKI

- KIDGO Definition

Urea:>=0.3mg/dl in 48 hrs

Creatinine:>=1.5 X baseline(prior 7days)

Urine output:<0.5ml/kg/hr for 6hrs

AKI: RIFLE Classification

Stage	GFR criteria	UO+ Probability
Risk	SCreat 1.5 X increased OR GFR .25% decreased	UO<0.5ml/kg/hr-6hrs High sensitivity (risk>injury>failure)
Injury	Screat X2 or GFR decreased>50%	UO<0.5ml/kg/hr-12hrs
Failure	Screat X3 OR GFR reduced by>75%	UO<0.3ml/kg/hr-24hrs(oliguria) OR AnuriaX12hrs
Loss	Persistent renal failure: complete loss of kidney function >4wks	High specificity
ESKD	Complete loss of kidney function >3months	

Chronic Kidney Disease(CKD)

- Kidney damage or decreased GFR less than 60mL/min/1.73m^2 for at least 3 months
- loss of nephrones & functional renal mass
- remaining nephrones undegoing irreversible sclerosis
- progressive decline in GFR

CKD staging

Stage	GFR	
1	>90 mL/min/1.73m^2	
2	60-89 “	mild reduction
3a	45-59	moderate reduction
3b	30-44	moderate reduction
4	15-29	severe reduction
5	<15	failure

Using Creatinine based formulas for GFR(and cystatin C if available, esp in popns with lean body mass: children, malnourished, amputated) in addition to Albuminuria

Pre renal AKI causes

VOL DEPLETION

- Renal losses
- GI losses
- cutaneous losses
- Blood loss
- Pancreatitis

DECREASED CARDIAC OUTPUT

- Heart failure
- Cardiac tamponade
- P.E
- Acute MI
- Severe vulvular disease
- Abdominal compartment syndrome, tense ascites

Pre renal AKI causes

- SYSTEMIC VASODILATION
  - Sepsis
  - Anaphylaxis
  - Anaesthetics
  - Drug overdose
  - Cancer specific etiologies

DRUGS

Affarent arteriolar vasoconstrictors:

- NSAIDS
- Amphotericin B
- Norepinephrine
- Radiocontrast agents

R E N A L / I n t r i n s i c c a u s e s

VASCULAR

- Renal vesselobstruction
- Microangiopathy
- Malignant hypertension
- Scleroderma renal crisis
- Transplant rejection

GLOMERULAR

- Anti-GBM disease
- Good pasture syndrome
- PAuci immune glomerulonephritis(ANCA)
- Immune cplx
- IgA nephropathy
- Heme pigment
- Crystals
- Drugs

R E N A L / I n t r i n s i c c a u s e s

- INTERSTITIAL CAUSES
  - Drugs
  - infection
  - systemic diseasess
  - Anticoagulant related nephropathy

P O S T R E N A L C A U S E S

- Ureteric Obstruction
- Bladder neck obstruction
- Urethral obstruction
- Intraabdominal hypertension
- Renal vein thrombosis
- Tubular obstruction from crystals
- Retroperitoneal fibrosis
- Urolithiasis
- Obstructed Foley catheter
- Neurogenic bladder

Etiology in Newborn and Infants

Prerenal

- Perinatal hemorrhage
- Neonatal hemorrhage
- Adrenal hemorrhage
- Perinatal asphyxia

Renal

- ATN-neonatal asphyxia, aminoglycosides
- ACEIs given perinatally
- Acute glomerulonehritis-maternal-fetal transfer of Abs

Post renal

- Congenital malformations

E t i o l o g y i n c h i l d r e n

Prerenal:Hypovolemia

Intrinsic:

- Acute poststreptococcal glomerulonephritris
- HUS(most common cause of AKI in children ass with E.coli)
- Rhabdomyolysis

Post renal:

- Posterior urethral valve, stones, tumor, neurogenic bladder

C K D e t i o l o g y

Adults

Diabetes mellitus

Hypertension

Glomerulonephritis

Polycystic kidney disease

Chronic pyelonephritis

Medications

Children

Congenital anomalies of kidney/UT

Hereditary diseases like Alport syndrome,

polycystic KD

Glomerulonephritis

Nphrotic syndrome

Reflux nephropathy

Diabetes M

Medications

AKI-CKD relationship & TB

- AKI predisposes to CKD
- CKD likely to promote AKI(acute on chronic)

- TB & antiTBs cause AKI
- Study by Grace Kansiime, Kalyesubula et al
- TB itself causes AKI on addition to anti-TBs
- PLHIV had earlier diagnosis of TB than those without HIV
- Mortality from AKI was lower in PT?PLHIV than TB alone

Common AKI-CKD Emergencies

- Hyperkalemia
- Fluid overload
- Hypertensive crises
- Severe anaemia
- Metabolic acidosis
- Infections
- Other Electrolyte imbakance Ca, PO4, Mg
- Uremic encephalopathy
- Bleeding

Pt Approach & Evaluation

- ✓ Primary Survey
- ✓ Attach monitors
- ✓ SAMPLE hitory & Detailed history
- ✓ Secondary survey
- ✓ Adjuncts
- ✓ Investigation
- ✓ Consultations
- ✓ Disposition

Initial Assessment & Stabilization

Parameter	Assess & Act
Airway	Ensure Patency
Breathing	Adequate breathing
Circulation	Adequate perfusion
Disability	AVPU,GCS, RBS
Exposure	Temp, wounds, cutaneous complications

Adjuncts

- Bedside POCUS(pericardial effusion, effectiveness of myocardial contraction, Pleural effusion, P. edema, echogenic kidneys, hydronephrosis, size of kidneys, C-M diff)
- Attach monitors(spo2, BP, EKG)

History

- SAMPLE, detailed

Parameter	Focus
Signs& symptoms	
Allergies	
Medications	
Past medical/surgical	
Last meal	
Events(recent)	

Secondary survey

- Head to toe
- General condition
- Face
- ABCD
- Respiratory
- CVS
- CNS
- GIT
- GUS
- MSK
- Other

Investigations

BLOOD

- CBC, RBS+/_ HbA1c, RFTS/Extended electrolytes
- ABGs, PT/INR, HIV
- LFTS
- B/S for malaria esp in ped

URINE Urinalysis+ urine protein+ urine electrolytes Na, Urea

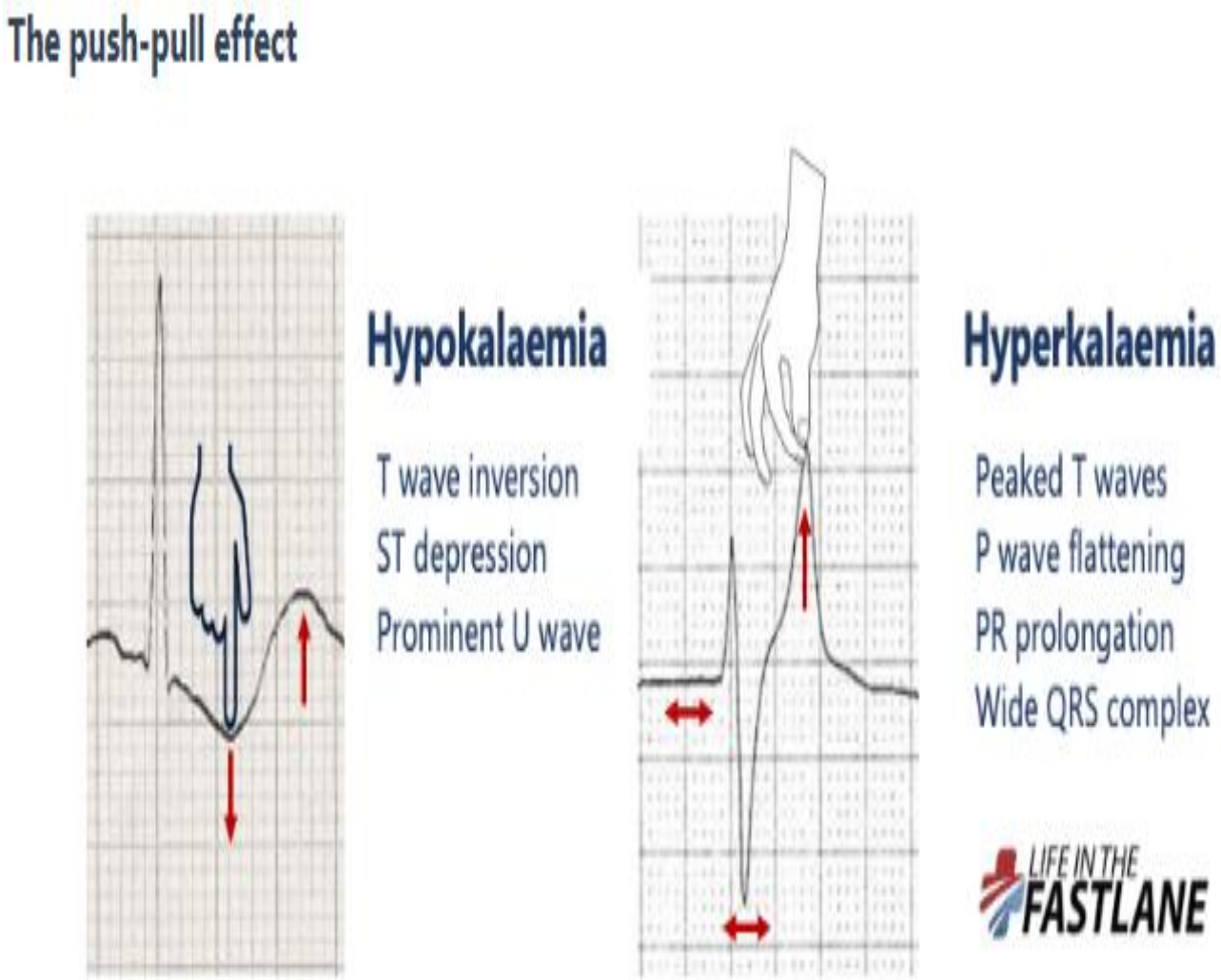
IMAGING

- EKG+/-Echo
- CXR
- Abdominal US scan
- Brain CT in Altered LOC

Fluid overload

- Edema, with pulmonary congestion
- Elevate head of bed at 30^{o}
- Positive pressure ventilation, CPAP, BIPAP
- Fluid restriction
- ? Diuretics
- Folley catheter-UO

Hyperkalemia



Potassium level (mmol/L)	Mechanism	ECG changes
5.5 – 6.5	Repolarisation abnormalities	Peaked T waves
6.5 – 7.0	Progressive atrial paralysis	P wave widening/flattening PR prolongation P waves eventually disappear
7.0 – 9.0	Conduction abnormalities	**Bradyarrhythmias:** Sinus bradycardia; high-grade AV block with slow junctional and ventricular escape rhythms; slow AF Conduction blocks (bundle branch block, fascicular blocks) Prolonged QRS interval with bizarre QRS morphology
> 9.0	All of above	Development of sine wave appearance (pre-terminal rhythm) Asystole Ventricular fibrillation PEA with bizarre, wide complex rhythm

HyperKalemia Approach

- Membrane stabilization: Ca gluconate
- Insulin +Glucose infusion
- Salbutamol
- K restriction
- Dialysis if severe

Bleeding/ severe anaemia

- Pressure dressing if external
- Transfuse with Platelets/ fresh WB
- Erythropoietin/thrombopoietin
- Do coagulation studies
- Consult Haematology

Other Emergencies

Uremia

- Encephalopathy, pericarditis, Gastritis
- R/o other causes SOL, investigate
- Dialysis

Hypertensive crisis

- Lower BPs gradually
- Investigate

Other Emergencies

Metabolic Acidosis

- Sodium bicarbonate if severe
- Investigate the cause
- Manage the underlying cause

Infections

- CBC, Blood culture, urine culture
- Broad spectrum safe antibiotic

Team work/Disposition

- Consultations
- Nephrologist
- ICU
- Dialysis
- Urology
- Cardiology
- Nursing team
- Others

I THANK YOU ALL