Acute renal failure

Prolonged,reversible abrupt loss of renal function

Dr. Jumana Albaramki

Types of ARF

- RIFLE classification based on Cr,urine output.(risk,injury,renal failure,renal loss,ESRD)
- prerenal,renal,postrenal
- Oliguric (<1ml/kg/h) ,nonoliguric

Prerenal causes

- 1.intravascular volume depletion:bleeding,GIT losses,third spacing
- 2.low cardiac output:CHF
- 3.Sepsis

- Treatment: fluid management and restoration of effective circulatory volume

Renal causes

- 1.ATN,acute cortical necrosis from drugs,ischemic vasoconstrictive injury
- 2.Glomerulonephritis:PSGN,SLE,HSP
- 3.HUS:Most common cause
- 4.Acute interstital nephritis
- 5.drug induced ARF

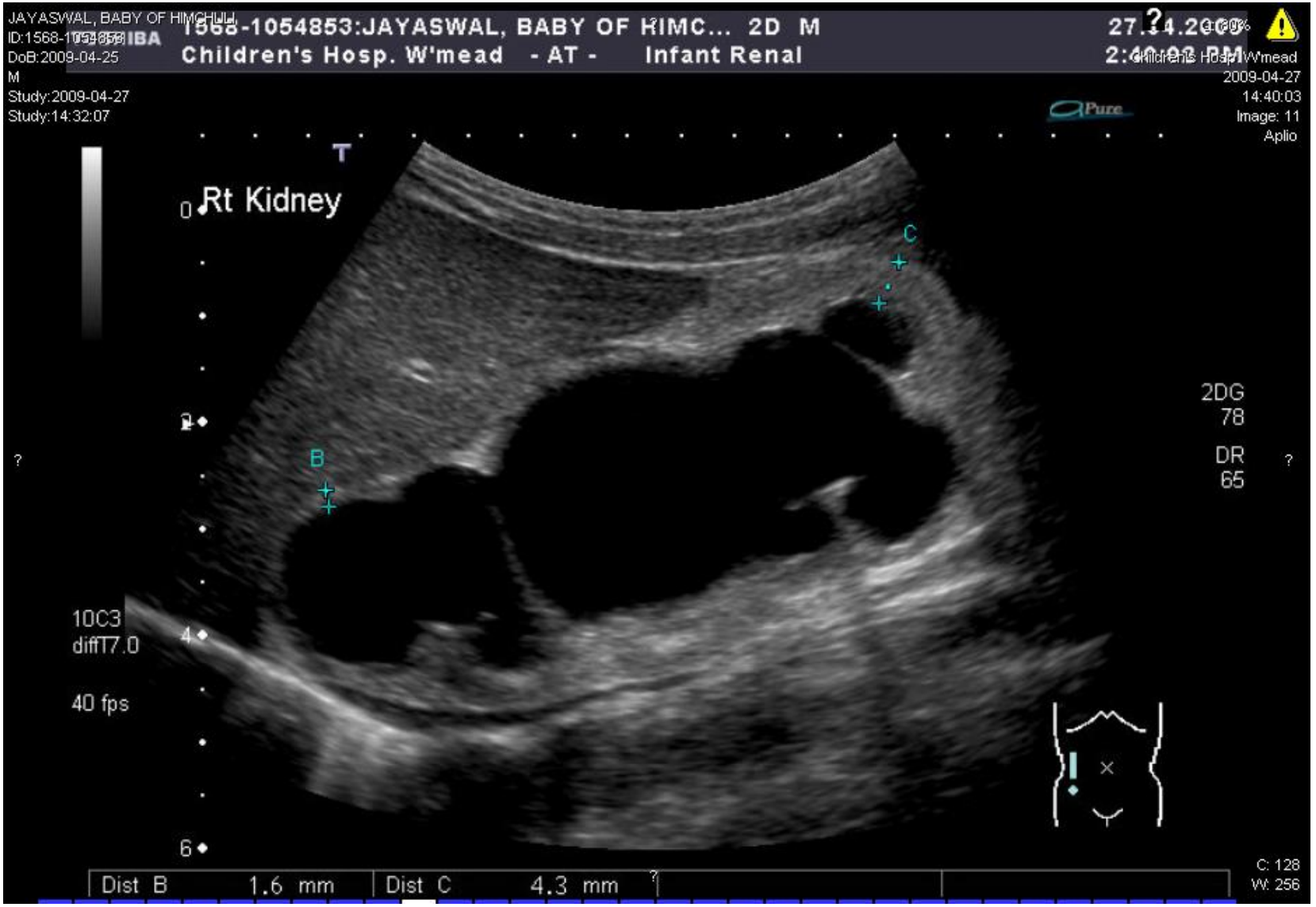
- 6.pigment nephropathy:rhabdomyolysis myoglobinuria,result trauma,status epilepticus,hereditary
- tx with fluids,alkali,diuretics

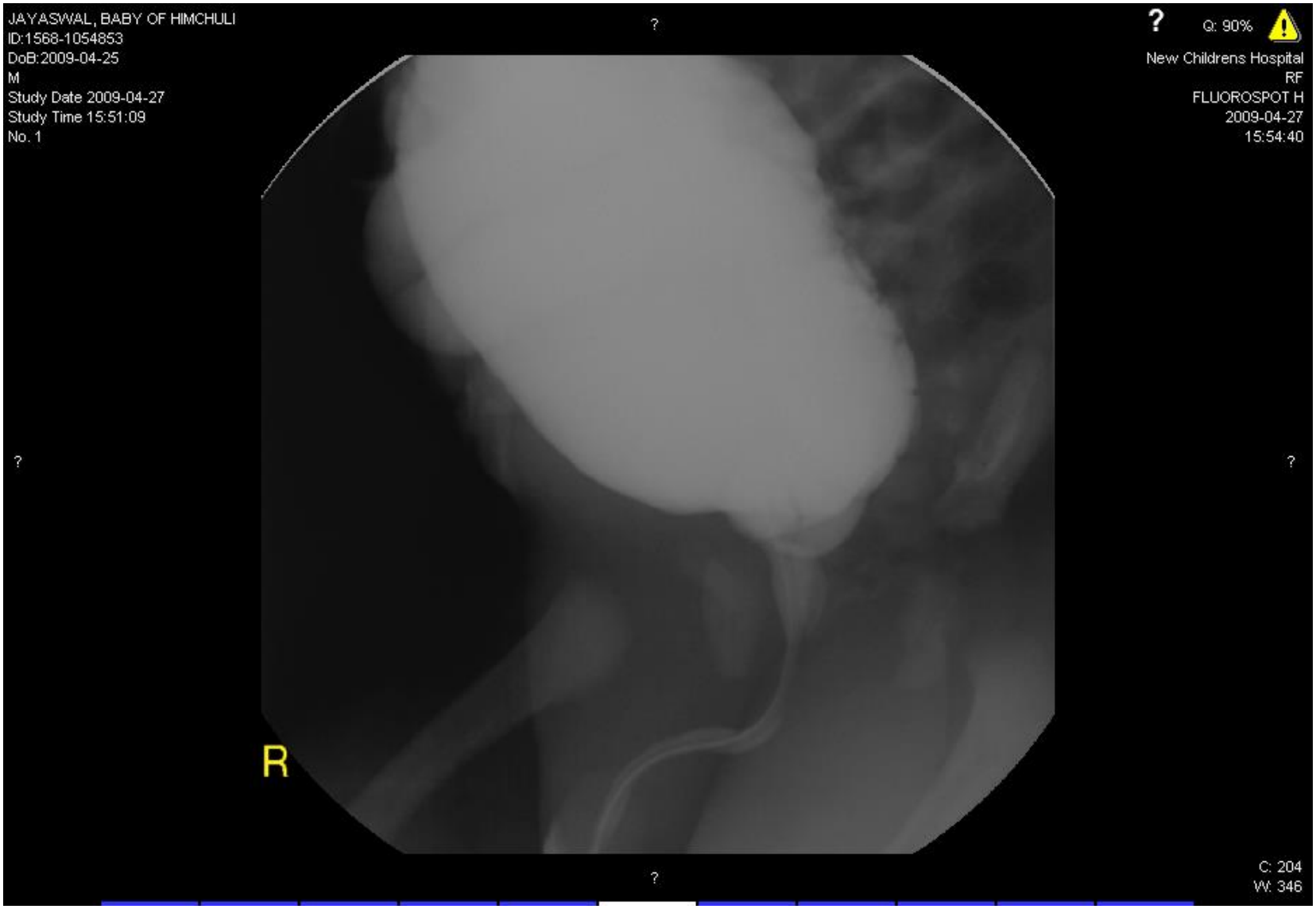
- 7.Tumor lysis syndrome:high uric acid,phosphate,low calcium.Tx allopurinol,alkalinazation of urine ph 7

- 8. vascular :renal artery,vein thrombosis

Post renal ARF

- Obstruction at level of ureter till urethra
- Elevated tubular pressure decrease GFR
- Duration of obstruction affects recovery
- Congenital (PUJ,PUV),acquired (stones)
- Post obstructive diuresis:dilute urine with large Na losses,reduced excretion of H,K


JAYASWAL, BABY OF HIMCHUL
ID:1568-1054853
DoB:2009-04-25
M
Study:2009-04-27
Study:14:32:07
1568-1054853:JAYASWAL, BABY OF HIMC... 2D M
Children's Hosp. W'mead - AT - Infant Renal
Children's Hosp. W'mead
2009-04-27
14:40:03
Image: 11
Aplio
Rt Kidney
B
C
2DG
78
DR
65
10C3
diff7.0
40 fps
Dist B 1.6 mm
Dist C 4.3 mm
C: 128
W: 256


JAYASWAL, BABY OF HIMCHULI
ID:1568-1054853
DoB:2009-04-25
M
Study Date 2009-04-27
Study Time 15:51:09
No. 1
?
?
Q: 90%
New Childrens Hospital
RF
FLUOROSPOT H
2009-04-27
15:54:40
?
?
R
?
C: 204
W: 346

- Important points in history :
- History of losses,gastroenteritis prerenal
- Previous GE,previous throat infection,red urine think PSGN
- Bloody diarrhea HUS
- Fever,rash,joint pain HSP.SLE
- Drug history
- O/E: state of hydration,hemodynamic status
- Edema,HTN,palpable bladder

Investigations

- Elevated urea,creatinine,spot Na
- FeNa=(Una X Pcr)/(Pna XUcr) X 100%
- FeNa < 1% in prerenal,2-3% renal
- FeNa unreliable in diuretics,neonate
- High in bartter,CRD.
- FBC: anemia usualy chronic

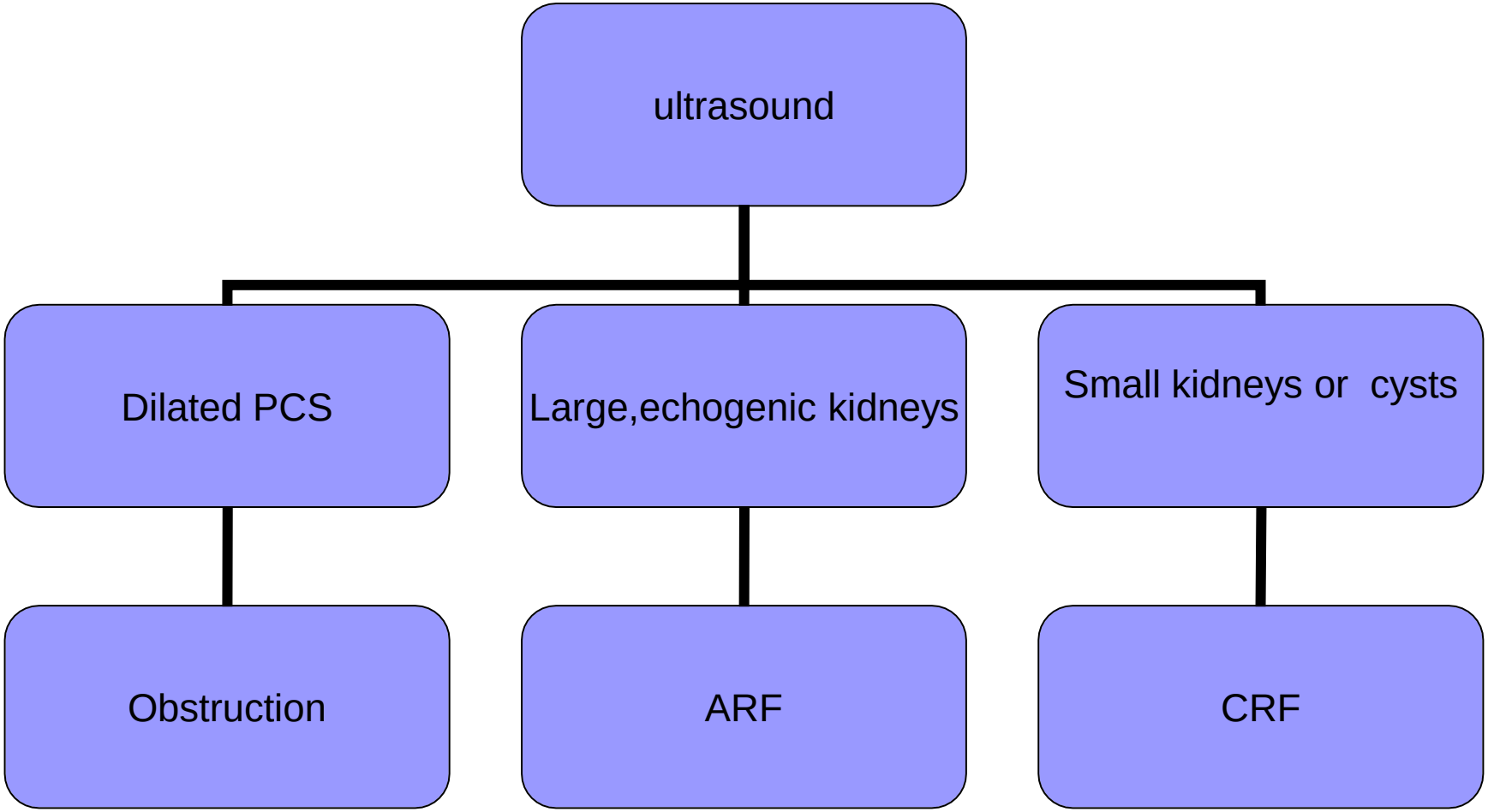
Laboratory studies used to differentiate between prerenal acute renal failure (ARF) and acute tubular necrosis (ATN)

	Prerenal ARF	ATN
Urine sodium (meq/L)	<20	>30
Fractional excretion of sodium	<1 percent	>2 percent
Urine osmolality (mOsm/L)	>500	<350
Serum BUN/Cr ratio*	>20:1	<20:1

* Used only in adolescents and older children.

- Blood smear:schistocytes of HUS
- Electrolytes:high K,acidosis,phosphate,dilutional hyponatremia,low calcium
- Complements,ANA,antiDNAs,ANCA,
- Imaging:U/S ,Doppler (kidney size,echogenecity
- Renal biobsy

Role of U/S in renal failure



- Urine analysis for proteinuria (glomerular,tubular)
- Urine sediment:RBC,WBC casts,crystals,myoglobin,red brown granular,tubular epithelial casts in ischemic,nephrotoxic ATN,eosinophiluria
- New kidney markers of injury:KIM 1,cystatin C

Management

- Monitoring:weight,input/output chart,B.P

- Fluids:bolus of crystalloid,furosemide as indicated by hydration
- Fluid restriction to U.O.P and insensible losses at 400/m2
- ?? Role of renal dose dopamine in reversing oliguria
- ?? Role of lasix in reversing oliguria
- Conservative: Recovery phase:polyuria

- Hyponatremia:dilutional,fluid restriction
- Na < 120, Tx with hypertonic saline= (125-measures) X .6 X wt over 2-4 h.
- High PO4,low Ca:diet restriction,phosphate binders
- Acidosis:correct Ca before,give I.V sodium biocarbonate

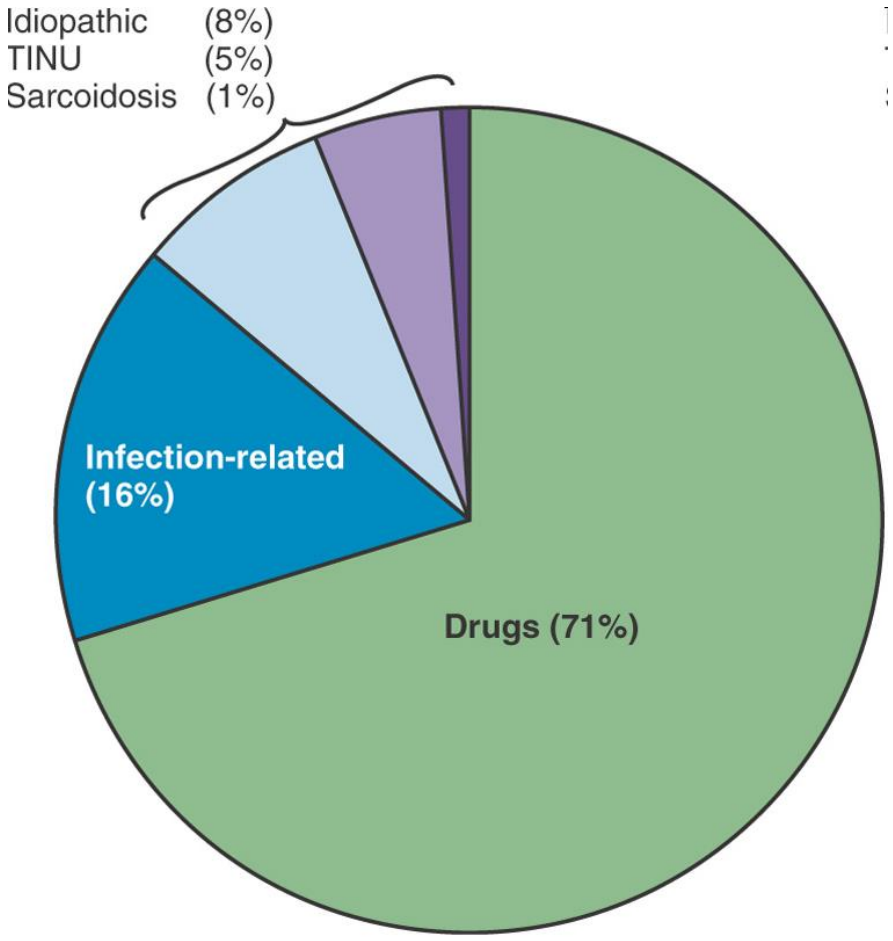
- HTN: fluid overload or vasoconstriction due hypovolemia
- Tx:diuretics,dialysis,nifedipine
- Hyperkalemia:low potassium diet,Ca gluconate,insulin and glucose,sodium biocarbonate,ventolin,oral resins
- Order ECG

- Nutrition:fluid restriction,catabolism result in high urea,K,need sufficient calories

Indications for renal replacement therapy

- Persistent hyperkalemia
- Diuretic resistant volume overload and associated hypertension and heart failure
- Refractory acidosis
- Severe uremia with risk of encephalopathy and /or pericarditis

Interstitial Nephritis



(From Baker RJ, Pusey CD: The changing profile of acute tubulointerstitial nephritis, *Nephrol Dial Transplant* 19:8-11, 2004.)

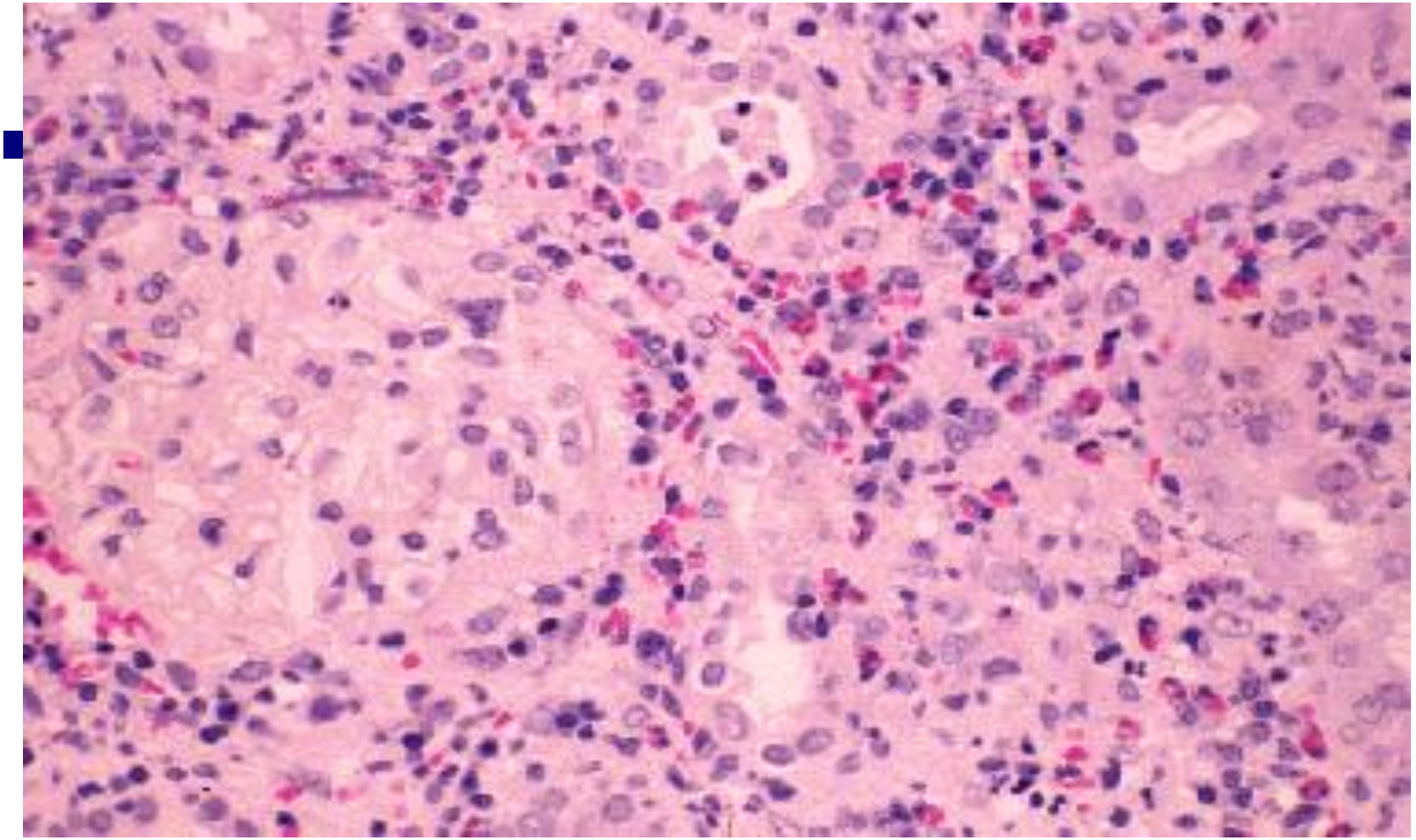
TIN

- The absence of HTN,significant proteinuria,RBC casts,exposure to drugs,sterile pyuria,good urine output,evidence of tubular dysfunction favor TIN

- Present nonspecific with fever,flank pain,skin rash,arthralgia

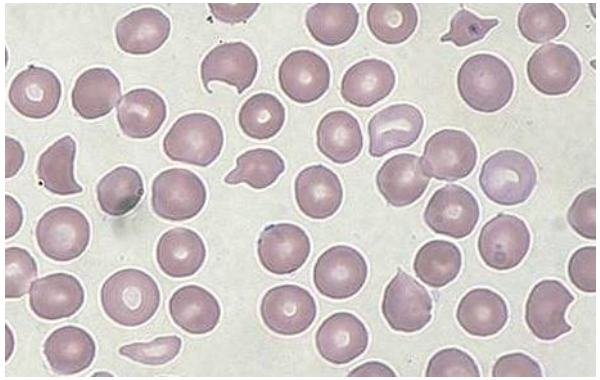
- Leucocytosis,eosinophilia,high ESR,ARF
- Hematuria,non-nephrotic proteinuria

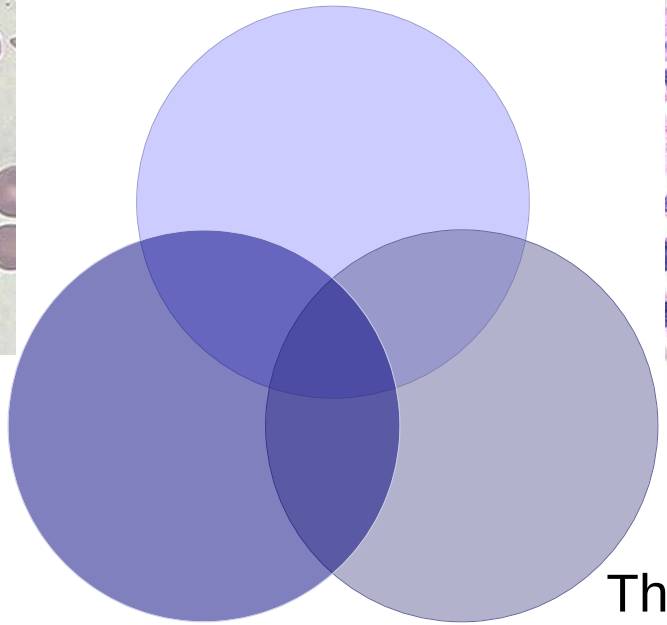
Eosinophils in acute interstitial nephritis

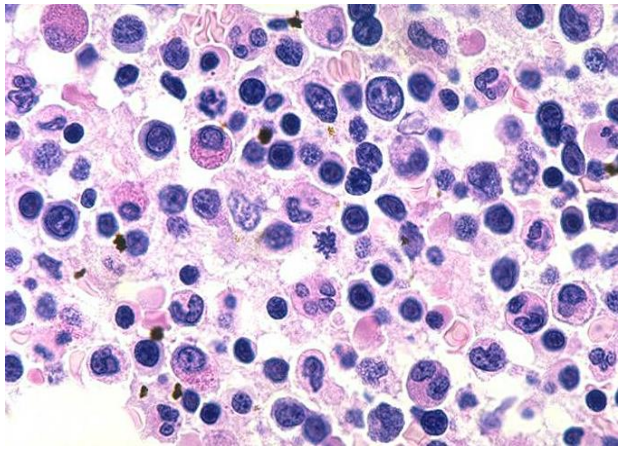


HUS

Acute haemolytic anaemia







Reduced GFR

Thrombocytopaenia

Classification of HUS

- Infectious (Stx)
  - E coli 0157:H7
  - Shigella dysenteriae type I (D+ HUS)
- Hereditary
  - Factor H deficiency, VWF proteinase def,ADAMTS-13
- Secondary
  - Pregnancy
  - Malignancy
- Medication
  - CNIs

Diarrhae + HUS

- D+HUS:follows STEC, shigella
- Transmitted undercooked hamburgers,milk,person to person
- O157:H7 E. coli most common serotype
- 5-15% of kids infected STEC develop HUS

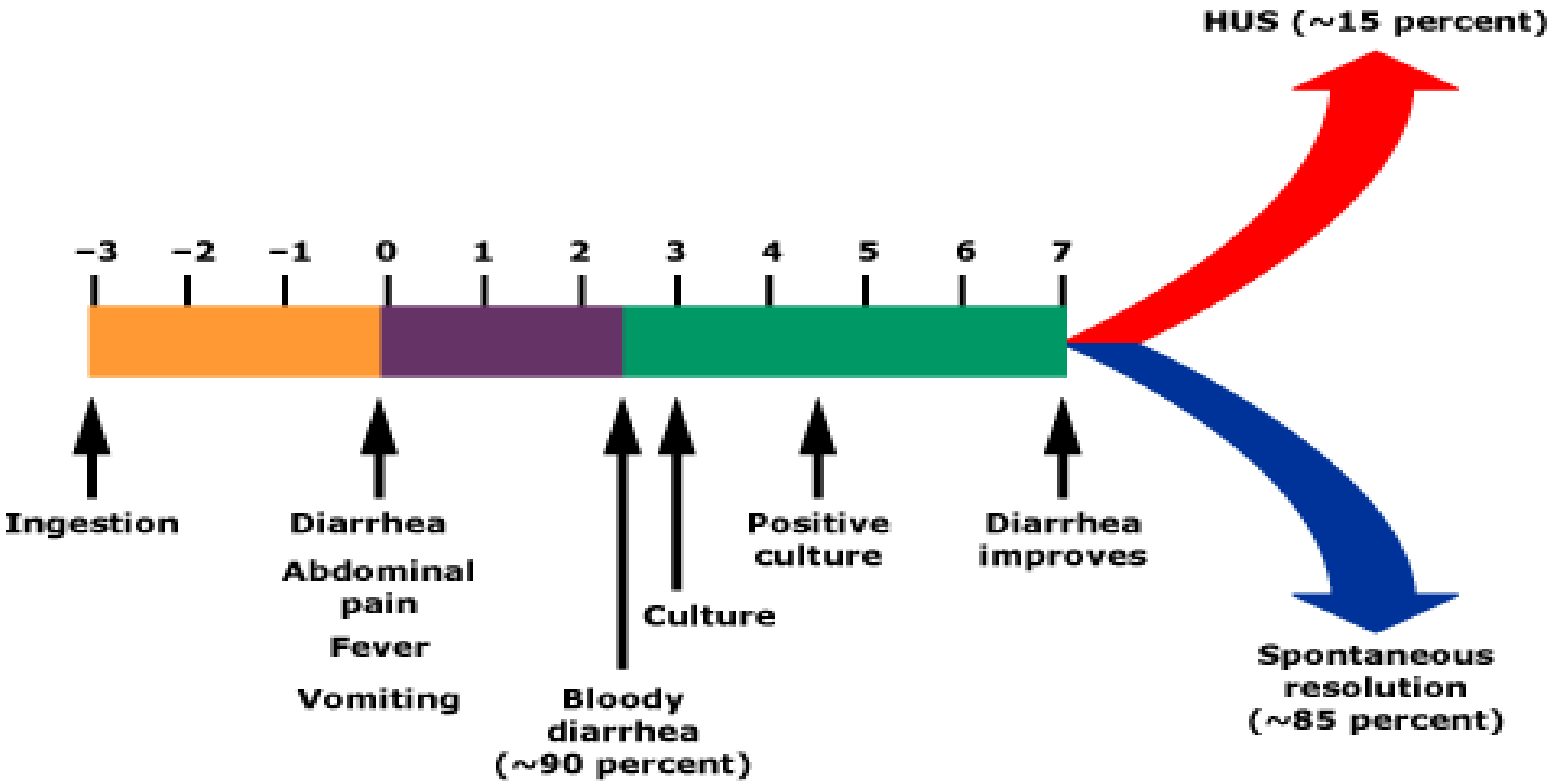
- Risk of HUS increase with age <5y, WBC >13,000/mm^3,antimotiliy drugs (retention of toxin
- Antibiotic can increase risk?? Release toxin

Clinical Manifestations

- Diarrhea 3-7 d after exposure to STEC,mostly bloody
- Pallor,oliguria 4-7 d post diarrhoea
- GIT:severe colitis,transmural
- necrosis,perforation,stricture,rectal prolapse
- Hepatitis,jaundice

- Pancreatitis
- Glucose intolerance,IDDM
- CNS: seizures,irritability,confusion
- Mycardium ischemia rare,rhabdomyolysis
- HTN,renal cortical necrosis, 50% are anuric,75% needs dialysis

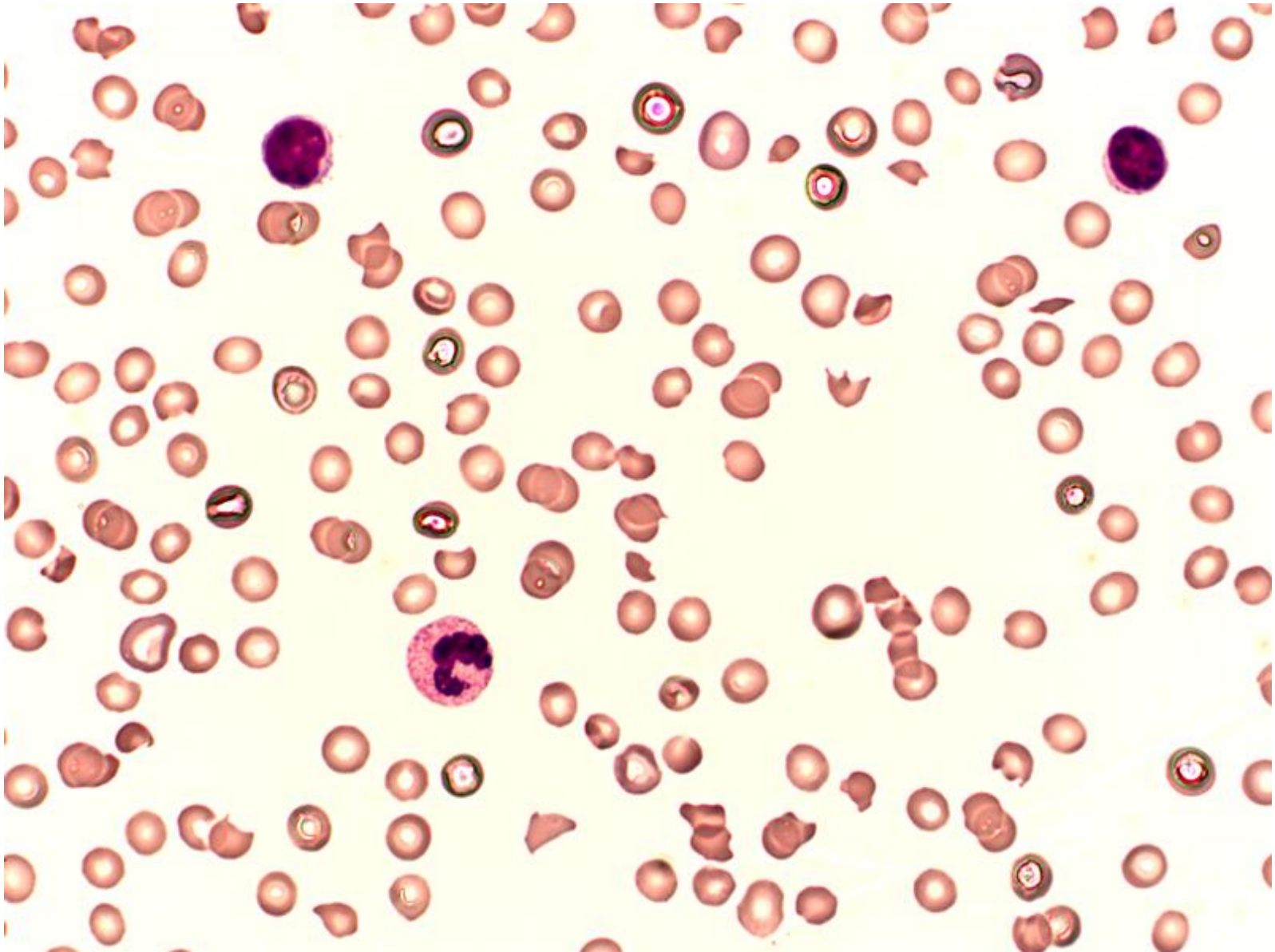
Progression of E coli O157:H7 infections in children



Investigations

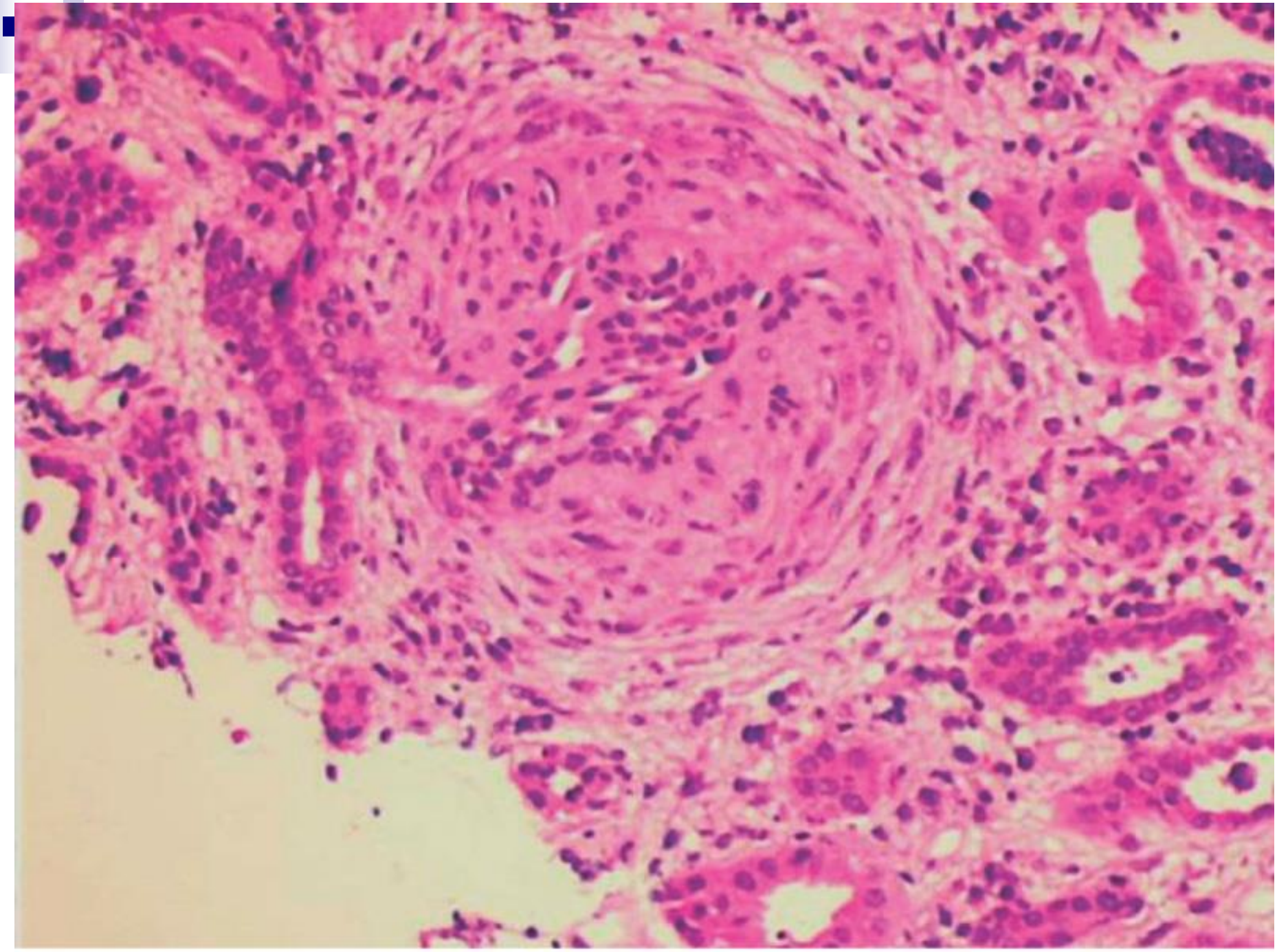
- FBC,showes anemia and low platlets.
- LDH high,blood film shows schizocytes,fragmented RBC
- High urea and creatinine
- Elevated liver enzymes hematuria,proteinuria

Microangiopathic hemolytic



RPGN

- 1.PSGN
- 2.MPGN
- 3.Lupus nephritis
- 4.Wegner,good pasture
- Treatment with high dose pulse steriods



PSGN

Epidemiology of PSGN

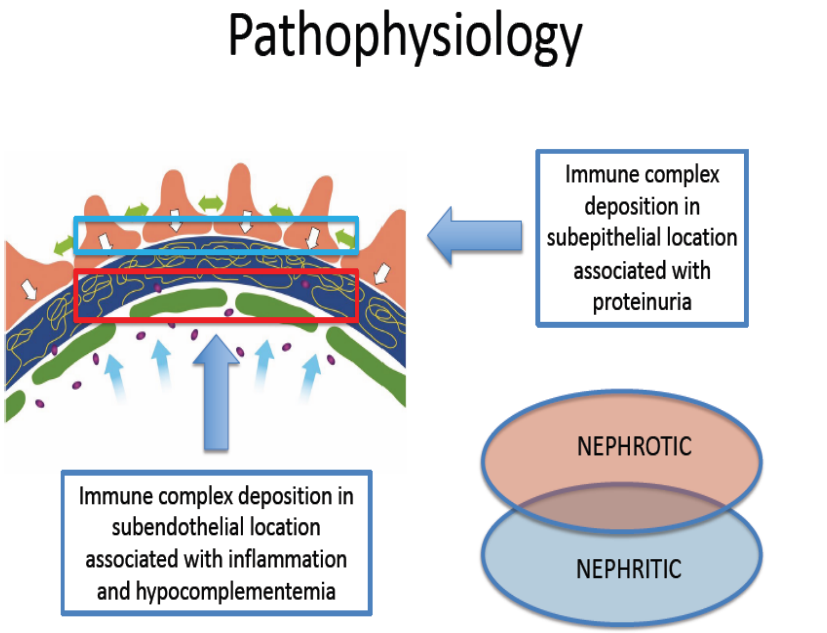
Follows GABHS pharyngitis in winter,pyodrema in summer

Certain nephritogenic M types,age 5-15 y,M:F 2:1

Risk of PSGN following GABHS is 15%

Antibiotic treatment doesn't prevent PSGN

Clinical features:latent period 10-14 days after pharyngitis, 3-6 wk pyoderma



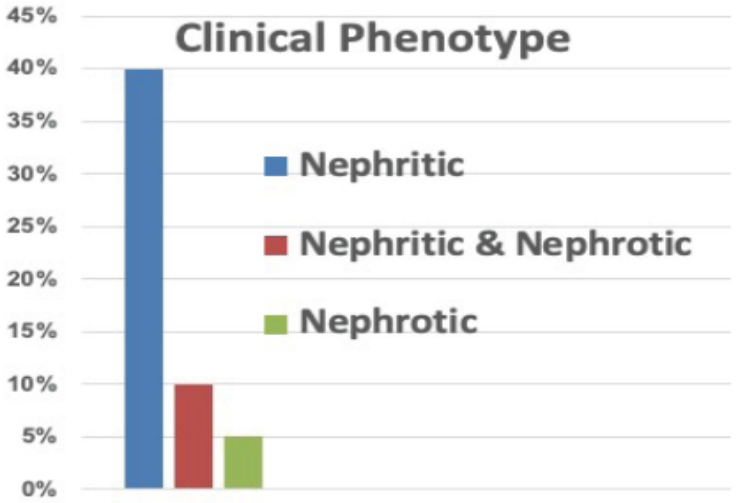
Clinical Characteristics at Presentation

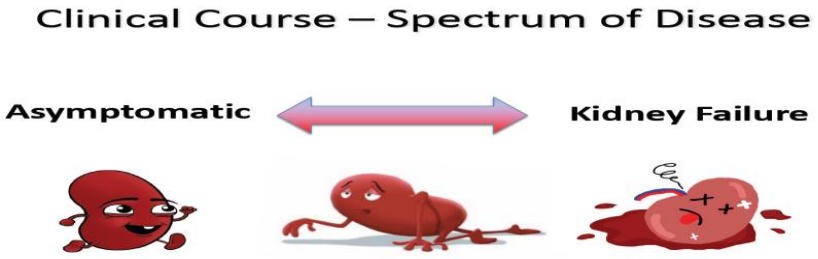
Hematuria
- Microscopic or gross
- Discolored urine reported in up to 80%

Hypertension
- Reported in 60-75%

Azotemia/Increased Cr
- Reported in 30-40%

Oliguria
- Reported in 25-35%





Laboratory investigations

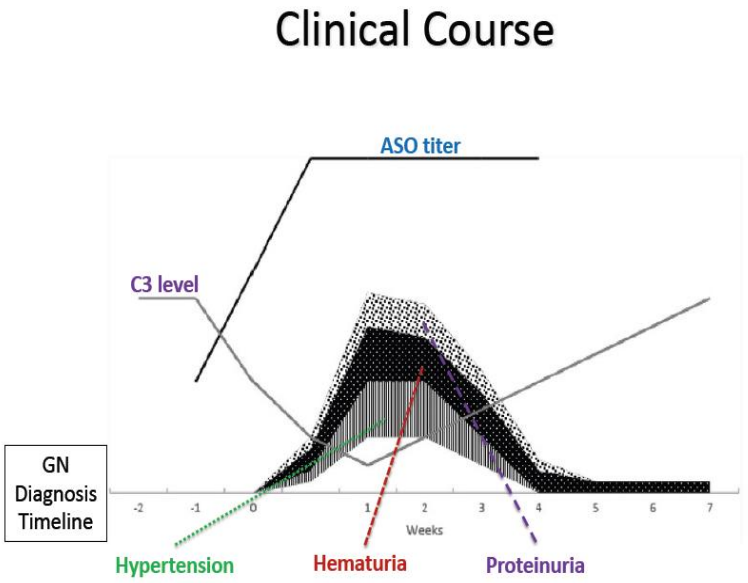
Urine : RBC casts

Low C3

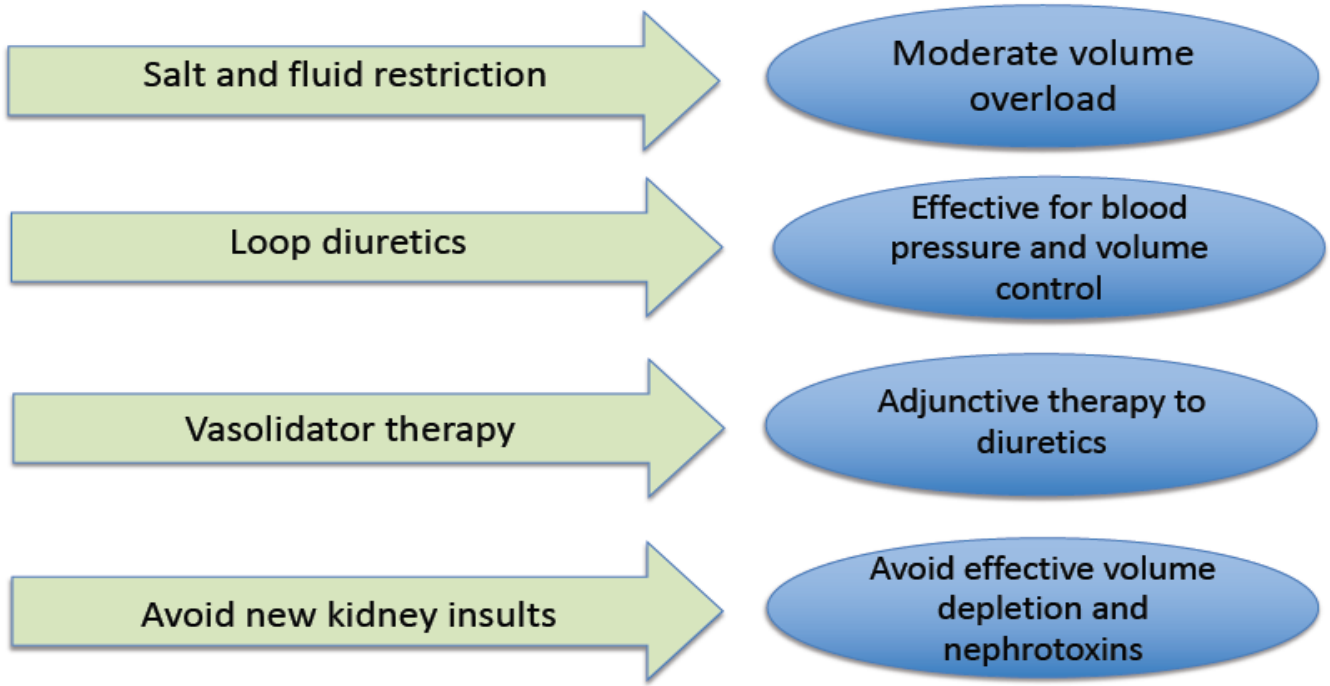
Positive ASOT

Renal azotemia

Hematuria and proteinurea stays for months



Management:
General Medical Care



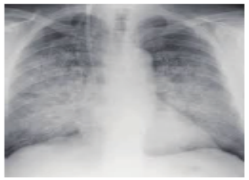
Clinical Course: Serious Sequelae

Encephalopathy/Seizures

- Around 5% of most large cohorts
- Generally related to hypertension

Symptomatic Pulmonary Edema/CHF

- 5-15% of most large cohorts
- Chest radiograph changes in up to 50%



Dialysis

- 1-2% of most large cohorts
- Most often related to RPGN

General Clinical Expectations

Most clinical signs and symptoms resolve spontaneously and within weeks

Hypocomplementemia >3 months should raise concern for a chronic hypocomplementemic GN

Recurrent gross hematuria is common with new acute illness early after diagnosis

Recurrent APSGN is quite rare

ESKD from APSGN is uncommon

Clinical features differentiating acute kidney injury from chronic kidney disease in children

Finding	Acute kidney injury (AKI)	Chronic kidney disease
Serum BUN and Cr	Progressive rise in BUN and Cr	Stable elevated BUN and Cr
Historical clues	Positive history for AKI etiology (eg, recent streptococcal infection: poststreptococcal glomerulonephritis)	History of chronic hypertension
Growth	Normal growth	Impaired growth
Bone status	Normal bones	Evidence of renal osteodystrophy: History of fractures, abnormal tibial torsion
Urine sediment	No broad urinary casts	Broad waxy urinary casts
Hematocrit	Anemia usually mild	Anemia usually severe
Renal ultrasound	Normal or enlarged kidney size	Small shrunken kidneys

BUN: blood urea nitrogen, Cr: creatinine

UpToDate®

Chronic renal failure

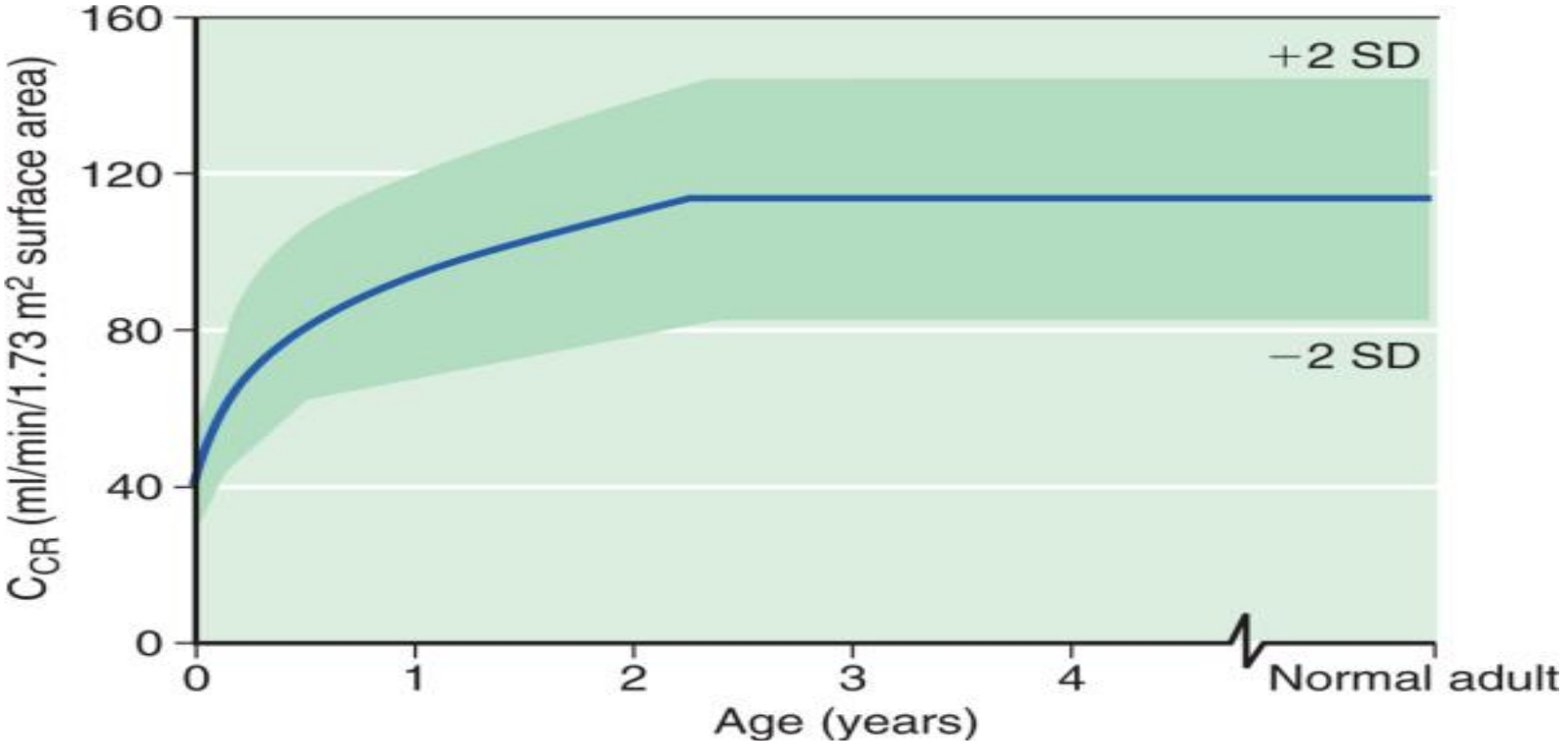
Plasma Cr does not rise until renal function has fallen to less than half normal levels

Cr affected by muscle bulk

GRF= height x k /creat in mg/dl ml/min/1.73 m2

Modified schwartz formula. k .0.413

Normal progression of GFR with age



Creatinine with age

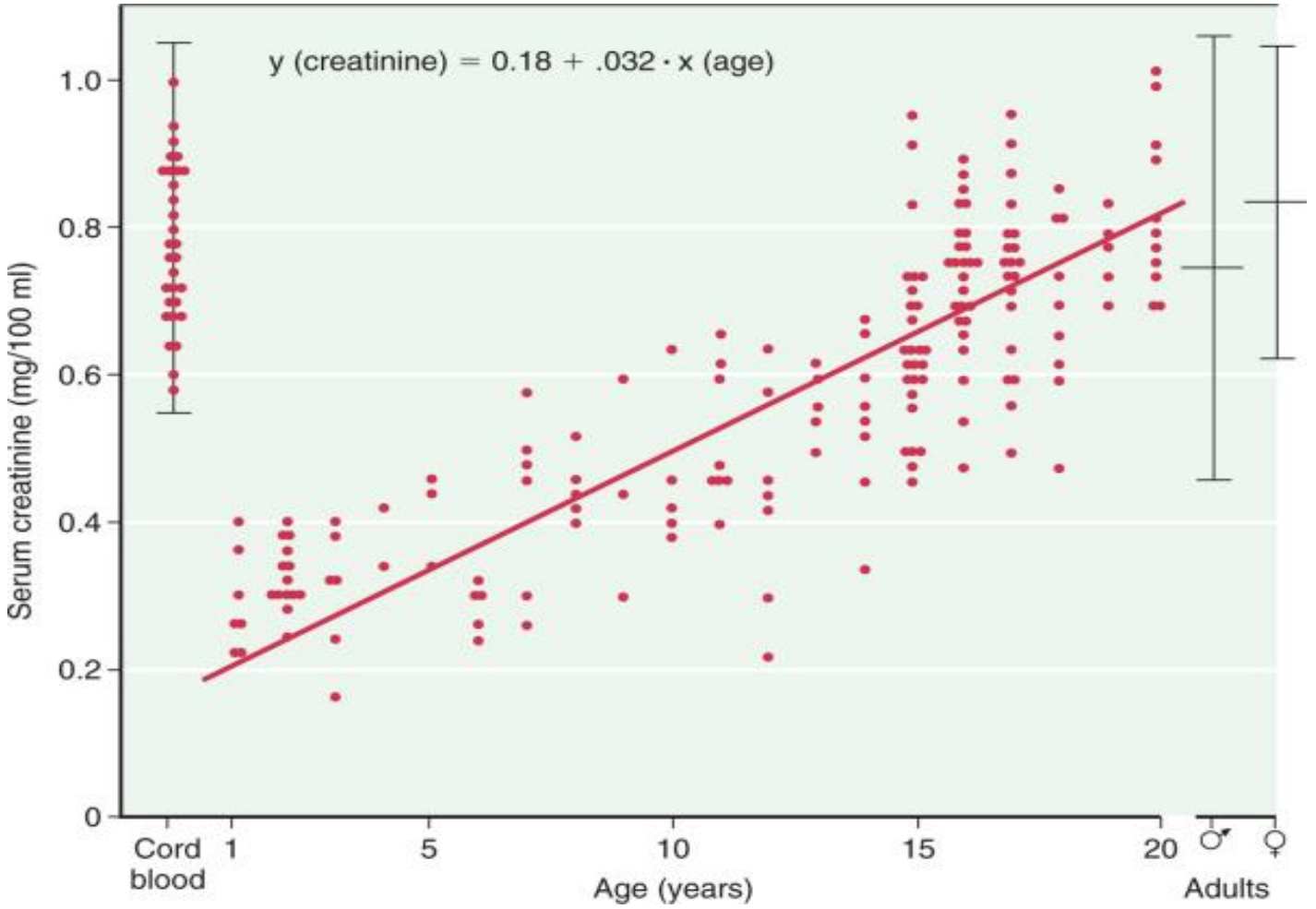


Table 10. Stages of Chronic Kidney Disease

Stage	Description	GFR (mL/min/1.73 m^2)
1	Kidney damage with normal or ↑ GFR	≥90
2	Kidney damage with mild ↓ GFR	60–89
3	Moderate ↓ GFR	30–59
4	Severe ↓ GFR	15–29
5	Kidney failure	<15 (or dialysis)

Chronic kidney disease is defined as either kidney damage or GFR <60 mL/min/1.73 m^2 for ≥3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies.

presentation

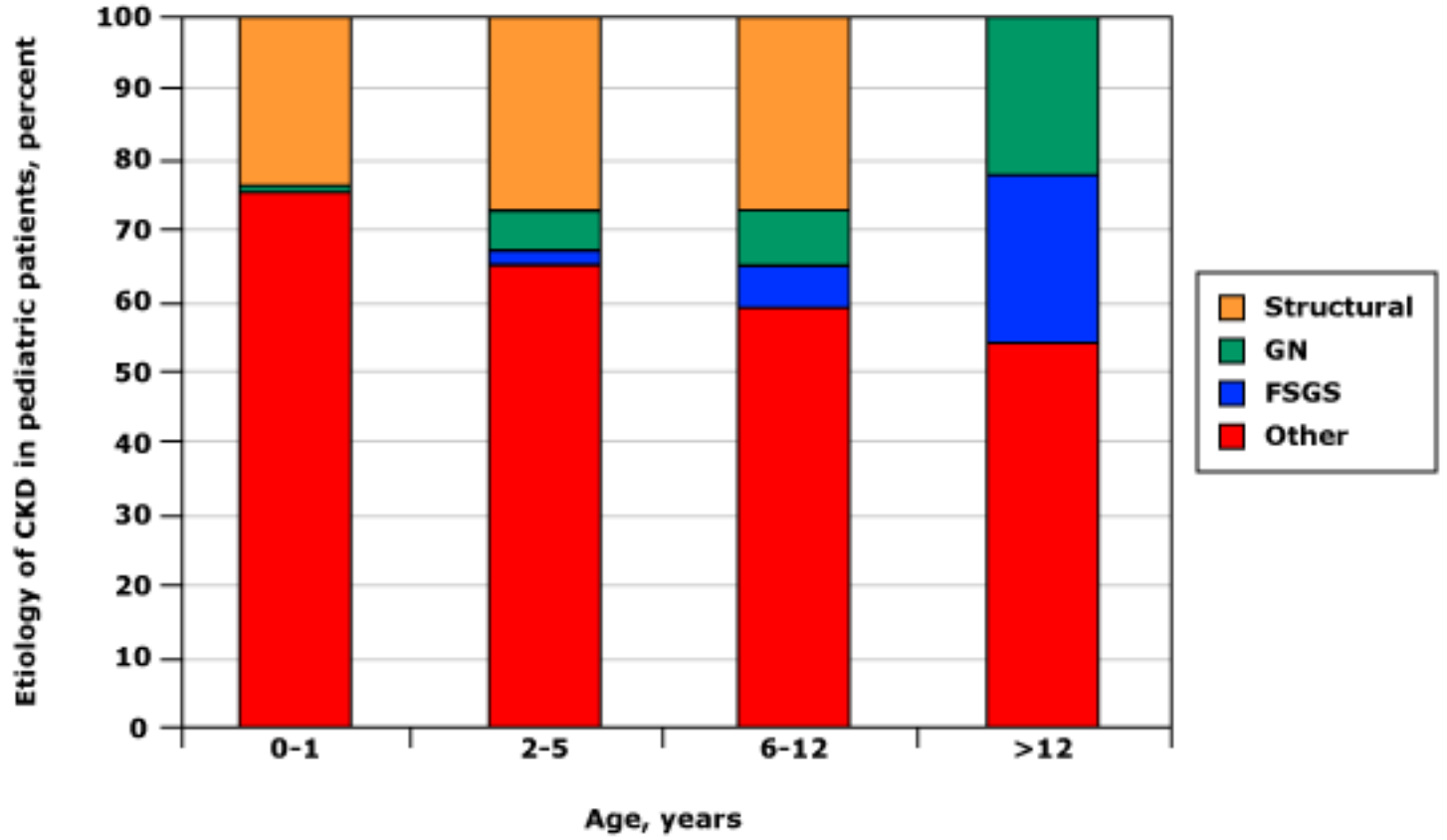
Asymptomatic
Anorexia,lethargy
Polydipsia,polyuria
Anemia
FTT,bone osteodystrophy
In GN present HTN,edema,hematuria

etiology

Causes:

1. Structural congenital malformations (CAKUT) as VUR,obstructive uropathy,dysplasia
2. Hereditary nephropathy ARPCKD,hyperoxaluria
3. Glomerulonephritis as FSGS,CNS

Distribution of the etiology of chronic kidney disease (CKD) in children based upon age



FSGS: focal segmental glomerulosclerosis; GN: glomerulonephritis; Structural: structural anomalies of the kidney and urinary tract.

Adapted from: NAPRTCS: 2007 Annual Report, Rockville, MD, EMMES, 2007. Available at https://web.emmes.com/study/ped/announce.htm. (accessed on April 9, 2008).

Management

Investigations:CBC,iron studies

Electolyte,urea ,creatinine, biocarbonte,Ca,PO4,,PTH

Urine protein,lipid profile

Prevention of progression of CRF

1. Proteinuria:due to hyperfilteration,ACEI,ARB dilate afferent art and reduce intraglomerular pressure

ACEI cause anemia,high K,Cr,

2. HTN

3. hyperphosphatemia

Renal osteodystrophy

- Reduced 1,25 OH vit D impairs intestinal Ca absorption leads to low Ca and increase PTH stimulates 1hydroxylase increase Vit D,Ca.
- High phosphate stimulate PTH

- Symptoms : bone pain ,skeletal deformities bowing
- Treatment : low phosphate diet,phosphate binders as calcium carbonate
- Active form of vitamin D

Anemia

Erythropoietin Deficiency

Blood loss (HD lines,GIT losses due to impaired platelet function)

Decreased RBC survival

Hyperparathyroidism decrease BM production.

Iron deficiency

Vitamin B12,folate deficiency

Inflammation,infection

Clinical effects of anemia

Systemic symptoms of fatigue,loss of appetite,decrease exercise tolerance

Evaluation:CBC,,ferritin,iron,TIBC

TSAT:Iron/TIBC should be >20%

Target Hb levels based on KDOQI guidelines is between 11-13

Treatment by recombinant erythropoetin

Treatment

- Nutrition
- Children should take recommended energy and protein requirements
- Treat metabolic acidosis

Recombinant growth hormone

Treat HTN with ACEI

BLE 165.5

mmon Complications and Treatments of Chronic Kidney Disea

MPLICATION	TREATMENT
. Poor growth	Increased caloric intake, treat acidosis, treat CKD-MBD, rec
. Anemia	Erythropoietin, iron supplementation
. CKD-Mineral Bone Disorder/secondary hyperparathyroidism	1,25-Dihydroxyvitamin D supplementation, calcium supplen dietary phosphorous restriction, phosphate binders
. Cardiovascular	
a. Hypertension	Antihypertensive medications
b. Left ventricular hypertrophy	Volume control
. Electrolyte abnormalities	
a. Hyperkalemia	Low K diet, furosemide, sodium polystyrene sulfonate
b. Hyponatremia	Sodium supplementation
c. Metabolic acidosis	Alkali replacement

Treatment of ESRD

Peritoneal dialysis:CAPD

CCPD:Uses an automated machine with 7 night cycles with a long day time dwell

Acute intermittent hemodialysis:needs vacular acsess as AV fistula,permcath

Requires 3 X 4 hour sessions/week

Diffusion through a semipermeable membrane,ultrafilteration of fluids

Renal transplantation


THANK YOU